QUESTION

A 26-year-old male presents to the hospital with fever and arthritis. Echocardiography confirms the presence of carditis in this patient. He gives a past history of myocardial infarction and peptic ulcer disease. Which of the following is the most appropriate anti-inflammatory agent in this patient?

A Diclofenac 50 mg TDS

B Naproxen 250 mg BD

C **Celecoxib 200 mg BD** CORRECT

D Ibuprofen 400 mg TDS

OK RIGHT ANSWER

C. Celecoxib 200 mg BD

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

Determine the total body clearance for a drug in a 70 kg male patient. The drug has an elimination half-life of 3 hours with an apparent volume of distribution of 100 ml/kg.

A 23 ml/h

B 230 ml/h

C **1617 ml/h** CORRECT

D 16 ml/h

RIGHT ANSWER

OK

C. 1617 ml/h

WHY THIS IS RIGHT

Total Vd = 100 ml/kg × 70 kg = 7000 ml.

Clearance = (0.693 × Vd) / t½ = (0.693 × 7000) / 3 ≈ 1617 ml/h.
This uses the standard clearance-half-life relationship. Hence total body clearance ≈ 1617 ml/h.

$$Cl = \frac{0.693 \times V_d}{t_{1/2}}$$

QUESTION

Which of the following drugs is not likely to cause a pleural pathology?

A **Metformin** CORRECT

B Methysergide

C Bleomycin

D Nitrofurantoin

OK RIGHT ANSWER

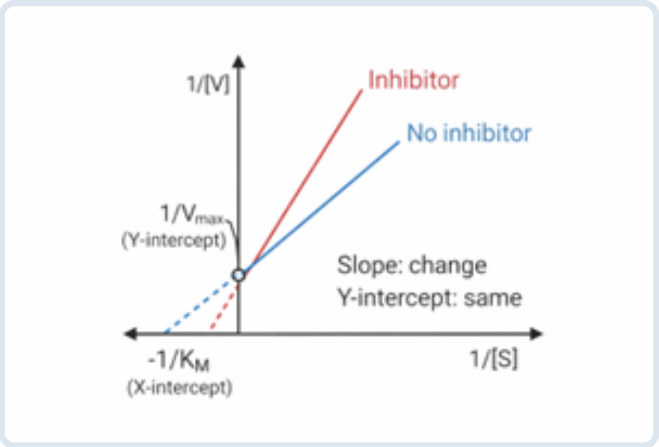
A. Metformin

WHY THIS IS RIGHT

Drug-induced pleural disease
Several drugs cause pleuropulmonary fibrosis or pleural reactions, including bleomycin, methysergide, and nitrofurantoin. These are classic causes of drug-induced pleural pathology. Metformin has no known association with pleural disease. Hence metformin is the exception.

QUESTION

Identify the type of inhibition from the given graph.



A **Competitive** CORRECT

B Non-competitive

C Un-competitive

D Allosteric

OK RIGHT ANSWER

A. Competitive

WHY THIS IS RIGHT

In competitive inhibition graphs (Michaelis-Menten or dose-response):
• Curve shifts RIGHT
• Same maximum effect (Emax)
• Increased Km (↓ affinity)

Drug (agonist) + receptor
↓
Competitive inhibitor present
↓
Competes for SAME site
↓
Need more agonist to achieve same effect
↓
Right shift in curve
The Lineweaver-Burk plot shows the same Y-intercept (1/Vmax) with a steeper slope in the presence of inhibitor. This indicates Vmax is unchanged while Km increases. Such a pattern is characteristic of competitive inhibition. Competitive inhibitors shift the X-intercept without altering Vmax.

Type	Km	Vmax	Graph Shift
Competitive	↑	Same	Right shift
Non-competitive	Same	↓	↓ height
Uncompetitive	↓	↓	Parallel shift

QUESTION

Lignocaine is used in a loading dose for the treatment of arrhythmias. The loading dose of this drug depends upon which of the following factors?

A Clearance

B **Volume of distribution** CORRECT

C Half-life

D Bioavailability

OK RIGHT ANSWER

B. Volume of distribution

WHY THIS IS RIGHT

Goal: Achieve target plasma concentration
↓
Drug distributes in body compartments
↓
Extent depends on Vd
↓
Higher Vd -> higher loading dose
Loading dose is determined by the amount of drug needed to rapidly achieve the target plasma concentration. It depends primarily on the volume of distribution, which reflects how extensively the drug distributes into tissues. Clearance and half-life influence maintenance dosing, not the loading dose. Hence Vd is the key determinant.

$$\text{Loading Dose} = \frac{C_p \times V_d}{F}$$

QUESTION

Concerning the mechanism of action of various antimicrobial drugs, which of the following statements is FALSE?

A **Linezolid: Binds to 30S ribosome and inhibits protein synthesis.** CORRECT

B Rifampicin: Inhibits DNA-dependent RNA polymerase.

C Aztreonam: Inhibits cell wall synthesis.

D Daptomycin: Causes depolarization of membranes.

OK

RIGHT ANSWER

A. Linezolid: Binds to 30S ribosome and inhibits protein synthesis.

WHY THIS IS RIGHT

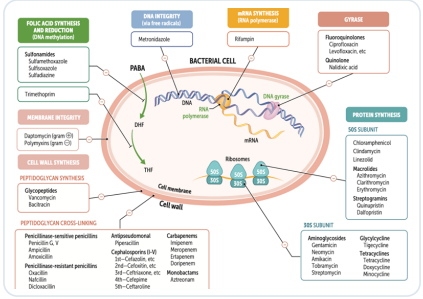
A) Linezolid: Binds to 30S

ribosome □ (FALSE)
• Linezolid is an oxazolidinone.
• It binds to the 50S ribosomal subunit, not 30S.
• Mechanism: prevents formation of initiation complex (70S).
• □□ Hence, statement is incorrect.

B) Rifampicin: Inhibits DNA-dependent RNA polymerase □
• Rifampicin blocks bacterial RNA synthesis by inhibiting DNA-dependent RNA polymerase.
• □□ Correct.

C) Aztreonam: Inhibits cell wall synthesis □
• Aztreonam is a monobactam (β-lactam).
• Acts on penicillin-binding proteins (PBPs) -> inhibits peptidoglycan cross-linking.
• □□ Correct.

D) Daptomycin: Causes membrane depolarization □
• Daptomycin inserts into bacterial membrane -> rapid depolarization -> cell death.
• □□ Correct.



QUESTION

5. Which of the following is a suicide inhibitor?
1. Nor-ethindrone
2. Ritonavir
3. Spironolactone
4. Benzbromarone

A 1, 2 and 3

B **1 and 3** CORRECT

C 2, 3 and 4

D 1, 2, 3 and 4

OK RIGHT ANSWER

B. 1 and 3

WHY THIS IS RIGHT

Enzyme + drug (looks like substrate)
↓
Drug gets activated inside enzyme
↓
Forms covalent bond
↓
Enzyme permanently inactivated
A suicide inhibitor irreversibly inactivates its target enzyme during metabolism. Norethindrone and spironolactone form reactive intermediates that permanently inhibit steroid-metabolizing CYP enzymes. Ritonavir is a potent but reversible CYP inhibitor, and benzbromarone acts mainly as a uricosuric drug. Hence only 1 and 3 are classic mechanism-based inhibitors.

QUESTION

A young male weighing 46 kg was treated with a drug. It has a Vd of 6 L/kg and clearance of 4.6 L/hour. What is the approximate half-life of this drug?

A 0.9 hours

B **40 hours** CORRECT

C 0.5 hours

D 20 hours

RIGHT ANSWER

OK **B. 40 hours**

WHY THIS IS RIGHT

Given:
• Vd = 6 L/kg
• Weight = 46 kg

Total Vd = 6 L/kg × 46 kg = 276 L.
Half-life t½ = 0.693 × Vd / Clearance = 0.693 × 276 / 4.6 ≈ 0.693 × 60 ≈ 41.6 hours.
Thus the approximate half-life is \~40 hours.
This uses the standard pharmacokinetic half-life equation.

Factor	Effect
Vd ↑	Drug stored in tissues -> longer half-life
Clearance ↓	Drug removed slowly -> longer half-life

$$t_{1/2} = \frac{0.693 \times V_d}{Cl}$$

QUESTION

All of the following statements are true regarding thiopentone except?

A It is highly lipid-soluble drug

B It is quickly concentrated in the brain after IV administration

C It shows the phenomenon of redistribution

D **It has a short duration of action due to rapid renal excretion** CORRECT

OK

RIGHT ANSWER

D. It has a short duration of action due to rapid renal excretion

WHY THIS IS RIGHT

Thiopentone (Thiopental)- An ultra-short-acting barbiturate IV anesthetic used for induction of anesthesia.

Thiopentone is highly lipid soluble and rapidly enters the brain after IV injection. Its brief anesthetic effect is due to redistribution from brain to muscle and fat, not elimination.
Metabolism occurs mainly in the liver, not rapid renal excretion. Hence the short duration is redistribution-dependent, making option D false.

QUESTION

Which of the following drugs is most teratogenic and strongly associated with neural tube defects (NTDs) when used during pregnancy?

A Lamotrigine

B Oxcarbazepine

C **Valproic acid** CORRECT

D Levetiracetam

OK RIGHT ANSWER

C. Valproic acid

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

Which pharmacokinetic parameters are used to determine the loading dose of an orally administered drug?
1. Volume of distribution (Vd)
2. Half-life
3. Plasma concentration
4. Bioavailability

A 1, 2 and 3

B Only 1

C **1, 3 and 4** CORRECT

D 1, 2, 3 and 4

OK RIGHT ANSWER

C. 1, 3 and 4

WHY THIS IS RIGHT

Loading dose = (Target plasma concentration × Volume of distribution) / Bioavailability.
Thus it depends on Vd, desired plasma concentration, and bioavailability for oral drugs.

Goal: Achieve target plasma concentration quickly
↓
Need to fill drug in body compartments
↓
Depends on:
Volume of distribution
Desired plasma concentration
Bioavailability (oral)
↓
Calculate loading dose
Half-life determines dosing interval and maintenance dose, not loading dose. Hence parameters 1, 3, and 4 are required.

$$\text{Loading Dose} = \frac{C_p \times V_d}{F}$$

QUESTION

Which of the following drug is associated with cholestasis without significant portal inflammation?

A Acetaminophen

B Amoxicillin-clavulanic acid

C Isoniazid

D **Oestrogen plus androgen** CORRECT

RIGHT ANSWER

OK

D. Oestrogen plus androgen

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

Match the following drugs with their respective Schedules:

Choose the option where the match for Morphine is correct

Drugs	Schedule Options
A. Hepatitis B vaccine	1. Schedule X
B. Veterinary drugs	2. Schedule H
C. Morphine	3. Schedule G
D. Insulin	4. Schedule Z

A. A-3, B-1, C-2, D-4

B. A-4, B-1, C-3, D-2

C. A-2, B-1, C-4, D-3

D. **A-3, B-4, C-1, D-2** CORRECT

RIGHT ANSWER

OK

D. A-3, B-4, C-1, D-2

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1c” here and does not include a written explanation for this item.

QUESTION

A 30-year-old lady who is a known case of seizures was on carbamazepine. She was prescribed erythromycin for her recent URTI. A few days later, she developed ataxia. What is the most likely cause of her symptoms?

A **Carbamazepine toxicity** CORRECT

B Erythromycin toxicity

C Rebound epileptic episode

D Increased hepatic metabolism

RIGHT ANSWER

OK

A. Carbamazepine toxicity

WHY THIS IS RIGHT

Carbamazepine -> metabolized by CYP3A4 -> normal levels
Erythromycin inhibits CYP3A4, the main enzyme responsible for carbamazepine metabolism. This raises serum carbamazepine levels, causing toxicity with neurologic signs like ataxia and diplopia. The interaction is due to reduced hepatic clearance, not increased metabolism. Drug-drug inhibition leading to accumulation explains the symptoms.
B) Erythromycin toxicity
• Causes GI upset, QT prolongation
• NOT ataxia

C) Rebound epileptic episode
• Would cause seizures, not ataxia

D) Increased hepatic metabolism
• Opposite happened!
• Metabolism is inhibited, not increased

QUESTION

A 42-year-old male is brought to the ED after ingesting multiple acetaminophen tablets along with alcohol in a suspected suicide attempt. He has no chronic alcohol use history. The physician informs the family that acute ethanol ingestion may reduce acetaminophen toxicity. Which mechanism best explains this protective effect?

A **Ethanol competitively inhibits hepatic cytochrome P450** CORRECT

B Ethanol depletes hepatic glutathione levels

C Ethanol induces hepatic cytochrome P450

D Ethanol impairs intestinal absorption of acetaminophen

OK RIGHT ANSWER

A. Ethanol competitively inhibits hepatic cytochrome P450

WHY THIS IS RIGHT

Acetaminophen is metabolized mainly by glucuronidation and sulfation, but a small fraction is converted by CYP2E1into the toxic metabolite NAPQI.
In acute ethanol ingestion --> ethanol competes for CYP2E1 --> inhibiting NAPQI formation and thereby reducing hepatotoxicity. This competitive inhibition is protective when ethanol and acetaminophen are ingested together in a patient without chronic alcohol use.

Why other options are incorrect:
• B: Glutathione depletion increases toxicity, seen in chronic alcoholism.
• C: CYP induction occurs with chronic, not acute, ethanol use.
• D: Ethanol does not significantly impair acetaminophen absorption.

QUESTION

All of the following are true for non-microsomal enzymes, except:

A Found in cytoplasm and mitochondria

B Not inducible

C **Carry out glucuronidation reactions** CORRECT

D Include esterases and amidases

RIGHT ANSWER

OK

C. Carry out glucuronidation reactions

WHY THIS IS RIGHT

Non-microsomal drug-metabolizing enzymes are located mainly in the cytoplasm and mitochondria and include enzymes such as esterases, amidases, alcohol dehydrogenase, and monoamine oxidase. These enzymes are generally not inducible and participate in reactions like hydrolysis, oxidation, and reduction. Glucuronidation, however, is a Phase II conjugation reaction catalyzed by UDP-glucuronyl transferase, which is a microsomal enzyme located in the smooth endoplasmic reticulum, not a non-microsomal system.

Why other options are incorrect:
• Found in cytoplasm and mitochondria: True for non-microsomal enzymes.
• Not inducible: Correct characteristic.
• Include esterases and amidases: Correct examples of non-microsomal enzymes.

QUESTION

All of the following are correctly matched drug-induced liver pathologies, except:

A Valproate - Microvesicular steatosis

B Halothane - Massive necrosis

C **Methotrexate - Cholestatic pattern** CORRECT

D Allopurinol - Fibrin ring granulomas

RIGHT ANSWER

OK **C. Methotrexate - Cholestatic pattern**

WHY THIS IS RIGHT

Methotrexate causes chronic hepatotoxicity characterized by hepatic fibrosis, cirrhosis, and macrovesicular steatosis, especially with long-term use. A cholestatic pattern is not its typical liver injury profile, making this pairing incorrect.

Why other options are incorrect (i.e., correctly matched):
• Valproate - Microvesicular steatosis: Valproate impairs mitochondrial β-oxidation, leading to microvesicular fatty change.
• Halothane - Massive necrosis: Halothane can cause immune-mediated fulminant hepatic necrosis (“halothane hepatitis”).
• Allopurinol - Fibrin ring granulomas: Allopurinol is classically associated with granulomatous hepatitis featuring fibrin ring granulomas.

QUESTION

All of the following drugs show increased absorption with food, except:

A Halofantrine

B Lithium

C Griseofulvin

D **Azithromycin** CORRECT

RIGHT ANSWER

OK

D. Azithromycin

WHY THIS IS RIGHT

Azithromycin shows reduced absorption when taken with food, especially oral suspension and capsules, as food delays gastric emptying and lowers peak plasma concentration. Hence, it is recommended to be taken on an empty stomach to ensure optimal bioavailability. This makes azithromycin the exception among the listed drugs.

Why other options are incorrect:
• Halofantrine: Absorption is significantly increased with fatty meals, improving bioavailability.
• Lithium: Food enhances GI tolerance and absorption, reducing gastric irritation.
• Griseofulvin: Fatty food markedly increases absorption by improving its dissolution and uptake.

QUESTION

A 78-year-old woman is brought to the hospital due to 2 days of nausea, vomiting, abdominal pain, and headache. She was recently hospitalized due to a stroke, and the hospitalization was complicated by a urinary tract infection and delirium. The patient has a history of chronic obstructive pulmonary disease, and her treatment regimen includes theophylline. Temperature is 37 C (98.6 F), blood pressure is 110/70 mm Hg, pulse is 118/min, and respirations are 22/min. On physical examination, the patient appears agitated and restless. There is a coarse tremor of the outstretched arms. Chest auscultation reveals mild expiratory wheezing. Serum theophylline levels are markedly elevated. Treatment with which of the following agents most likely precipitated this patient's current symptoms?

A Amoxicillin

B Cephalexin

C **Ciprofloxacin** CORRECT

D Haloperidol

RIGHT ANSWER

OK **C. Ciprofloxacin**

WHY THIS IS RIGHT

The patient has theophylline toxicity, suggested by nausea, vomiting, headache, agitation, tremor, tachycardia, and elevated serum theophylline levels. Ciprofloxacin is a strong CYP1A2 inhibitor, the primary enzyme responsible for theophylline metabolism. Inhibition of this pathway markedly reduces theophylline clearance, leading to drug accumulation and toxicity. Elderly patients are particularly susceptible to this interaction, especially during acute illness.

Why other options are incorrect:
• Amoxicillin: Does not affect theophylline metabolism.
• Cephalexin: No significant CYP interaction with theophylline.
• Haloperidol: May cause extrapyramidal symptoms but does not elevate theophylline levels.

QUESTION

Which of the following drugs need labelling as “Dangerous to use except under medical supervision?

A **Schedule G drugs** CORRECT

B Schedule H drugs

C Schedule J drugs

D Schedule X drugs

RIGHT ANSWER

OK **A. Schedule G drugs**

WHY THIS IS RIGHT

Schedule G drugs are required to carry the warning: “Caution: It is dangerous to take this preparation except under medical supervision.” These drugs can cause significant harm if misused and therefore mandate strict medical oversight, although they may not be as tightly controlled as Schedule H or X drugs. The labeling is a legal requirement to alert patients about potential risks and the need for professional supervision.

Why other options are incorrect:
• Schedule H: Prescription-only drugs; require Rx but no specific “dangerous” warning label.
• Schedule J: Drugs prohibited from manufacture/sale (e.g., unsafe).
• Schedule X: Strictly controlled narcotics/psychotropics with special record-keeping, not this label.

QUESTION

How much drug will be left after 6 hours if a patient is given a dose of 200 mg and 75 mg is excreted in 90 minutes. (Assume that the drug follows first-order kinetics)?

A 50 mg

B **30.5 mg** CORRECT

C 12.5 mg

D 6.25 mg

OK RIGHT ANSWER

B. 30.5 mg

WHY THIS IS RIGHT

The drug follows first-order kinetics, so a constant fraction is eliminated per unit time.
In 90 minutes (1.5 h), 75 mg is excreted from 200 mg, so 125 mg remains.
Fraction remaining after 1.5 h = 125/200 = 0.625.
Six hours equals four intervals of 1.5 hours.
Amount remaining after 6 h = $200 \times (0.625)^4 \approx 200 \times 0.153 = 30.5$ mg.

Why other options are incorrect:
• 50 mg: Assumes linear (zero-order) elimination.
• 12.5 mg: Overestimates elimination.
• 6.25 mg: Represents excessive half-life-based reduction, not supported by data.

QUESTION

A 45-year-old male weighing 80kgs was started on a drug with Vd =10 L and exhibits first-order elimination kinetics. The rate of drug clearance is calculated to be 6.93 L/hr. It is determined that repeating the second dose when 75% of the drug is eliminated will minimize toxicity. Based on these research findings, which of the following is the most appropriate dosing interval for this drug?

A 1 hour

B **2 hours** CORRECT

C 1 day

D 12 hours

OK RIGHT ANSWER

B. 2 hours

WHY THIS IS RIGHT

For first-order drugs, the fraction remaining depends on half-lives. “75% eliminated” means 25% remains, which equals 2 half-lives (since $0.5^2 = 0.25$). First find half-life:
$t½ = 0.693 \times Vd / Cl = 0.693 \times 10\ L / 6.93\ L/hr = 1$ hour.
Therefore, dosing interval = $2 \times t½ = 2$ hours to allow 75% elimination before the next dose, minimizing accumulation and toxicity.

Why others are incorrect:
• 1 hour: only 1 half-life -> \~50% eliminated.
• 12 hours / 1 day: far too long -> subtherapeutic levels.

QUESTION

A 65-year-old patient with a history of coronary artery disease is prescribed clopidogrel (Plavix) and omeprazole (Prilosec) for concurrent use. Which of the following statements regarding this drug combination is most accurate?

A. The combination of clopidogrel and omeprazole is safe and does not require any monitoring

B. Omeprazole may enhance the antiplatelet effects of clopidogrel, leading to improved cardiovascular outcomes

C. **Omeprazole inhibits the activity of CYP2C19, potentially reducing the effectiveness of clopidogrel and increasing the risk of cardiovascular events** CORRECT

D. Clopidogrel increases the metabolism of omeprazole, leading to decreased therapeutic levels of omeprazole in the body

RIGHT ANSWER

OK **C. Omeprazole inhibits the activity of CYP2C19, potentially reducing the effectiveness of clopidogrel and increasing the risk of cardiovascular events**

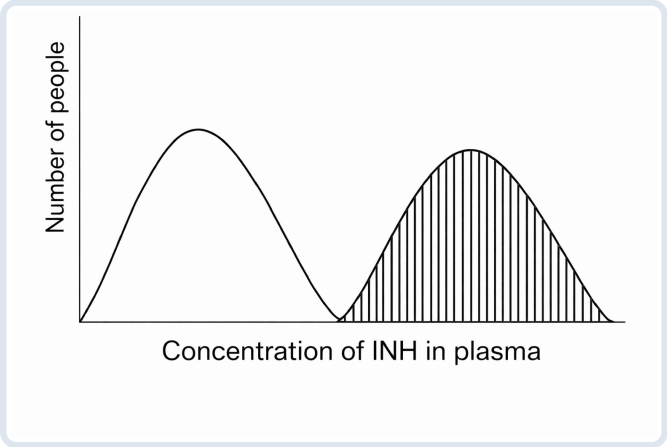
WHY THIS IS RIGHT

Clopidogrel is a prodrug that requires hepatic activation primarily by CYP2C19 to form its active antiplatelet metabolite. Omeprazole is a CYP2C19 inhibitor, which can reduce clopidogrel activation, leading to decreased antiplatelet effectand a higher risk of stent thrombosis and cardiovascular events. Hence, their concurrent use is generally avoided or requires careful consideration.

Why other options are incorrect:
• Safe without monitoring: Incorrect due to a well-known interaction.
• Enhances antiplatelet effect: Opposite of the true effect.
• Clopidogrel increases omeprazole metabolism: No such interaction exists.

QUESTION

A group of researchers is studying the variability in drug metabolism across individuals using isoniazid. Variation in which of the following metabolic processes is the most likely explanation for this observation?



A Methylation

B Hydrolysis

C **Acetylation** CORRECT

D Glucuronidation

RIGHT ANSWER

OK

C. Acetylation

WHY THIS IS RIGHT

isoniazid shows marked interindividual variability in metabolism due to genetic differences in N-acetyltransferase-2 (NAT2) activity. Individuals are classified as slow or fast acetylators, which affects isoniazid plasma levels, efficacy, and risk of adverse effects such as peripheral neuropathy and hepatotoxicity. This classic pharmacogenetic variation is best explained by differences in acetylation capacity.

Why other options are incorrect:
• Methylation: Not a major pathway for isoniazid metabolism.
• Hydrolysis: Plays no significant role in isoniazid clearance.
• Glucuronidation: Important for many drugs but not responsible for isoniazid variability.

QUESTION

Which of the following pairs of drugs act on the same receptor but have opposite effects?

A Somatostatin and octreotide

B Thioridazine and haloperidol

C Pioglitazone and gemfibrozil

D **Diazoxide and nateglinide** CORRECT

OK RIGHT ANSWER

D. Diazoxide and nateglinide

WHY THIS IS RIGHT

Diazoxide and nateglinide act on the ATP-sensitive potassium (K_ATP) channel in pancreatic β-cells but produce opposite effects. Diazoxide opens K_ATP channels, causing membrane hyperpolarization, reduced calcium influx, and inhibition of insulin release-used in hyperinsulinemic hypoglycemia. In contrast, nateglinide closes K_ATP channels, leading to depolarization, calcium entry, and increased insulin secretion for type 2 diabetes management.

Why other options are incorrect:
• Somatostatin & octreotide: Both are agonists at somatostatin receptors with similar effects.
• Thioridazine & haloperidol: Both are dopamine D□ antagonists.
• Pioglitazone & gemfibrozil: Act on different receptors (PPAR-γ vs PPAR-α).

QUESTION

A new antibiotic developed for the treatment of infections caused by resistant gram-positive cocci has a volume of distribution of 11L. It is eliminated by first-order kinetics and has a half-life of 10 hours. If given by a continuous infusion, approximately how much time would it require for the drug to achieve a 95% plasma steady state concentration?

A 10 hours

B 20 hours

C 30 hours

D **40 hours** CORRECT

OK RIGHT ANSWER

D. 40 hours

WHY THIS IS RIGHT

For drugs eliminated by first-order kinetics, the time to reach steady state during continuous infusion depends only on the half-life, not on dose or volume of distribution. Approximately 95% of steady-state concentration is achieved after about 4-5 half-lives.
Given a half-life of 10 hours, 4 half-lives = 40 hours, which corresponds closely to 95% steady state. Thus, the drug will effectively reach near-maximal steady plasma levels by around 40 hours.

Why other options are incorrect:
• 10 hours: Only one half-life (\~50% steady state).
• 20 hours: Two half-lives (\~75% steady state).
• 30 hours: Three half-lives (\~87.5% steady state), still below 95%.

QUESTION

A manufacturer substitutes a drug partly by another drug and doesn't add it in the label. What is this type of drug called?

A Misbranded drug

B Adulterated drug

C Unethical drug

D **Spurious drug** CORRECT

RIGHT ANSWER

OK **D. Spurious drug**

WHY THIS IS RIGHT

A spurious drug is one that is substituted wholly or partly by another drug or substance and this substitution is not declared on the label, making the product deceptive in identity and composition. Such drugs may falsely represent their contents, strength, or manufacturer, and are treated as counterfeit products under drug laws. The key feature is intentional substitution with misrepresentation, which fits this scenario precisely.

Why other options are incorrect:
• Misbranded drug: Involves labeling errors or misleading claims without actual substitution.
• Adulterated drug: Refers to contamination or quality compromise, not identity deception.
• Unethical drug: Not a recognized legal classification in pharmacovigilance or drug law.

QUESTION

Which of the following drugs does not increase the risk of bleeding in patients undergoing warfarin therapy?

A Cimetidine

B **Carbamazepine** CORRECT

C Isoniazid

D Amiodarone

RIGHT ANSWER

OK **B. Carbamazepine**

WHY THIS IS RIGHT

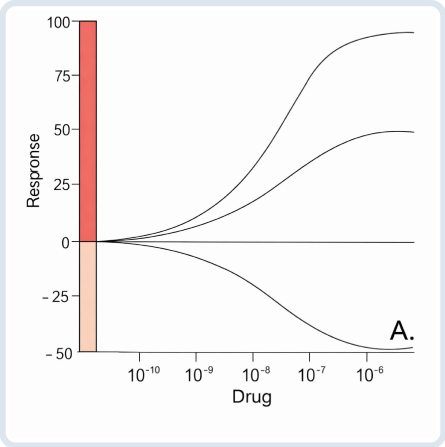
Warfarin is an anticoagulant that is metabolized primarily by the CYP2C9 enzyme. The risk of bleeding with warfarin therapy is influenced by drugs that either inhibit or induce the metabolism of warfarin.

Carbamazepine, is an enzyme inducer. It induces CYP450 enzymes, including CYP2C9, which increases the metabolism of warfarin, leading to reduced plasma warfarin levels and a decreased anticoagulant effect. Therefore, carbamazepine does not increase the risk of bleeding; instead, it can reduce the effectiveness of warfarin.

Why other options are incorrect:
• Cimetidine: CYP450 inhibitor -> decreases warfarin metabolism -> ↑ INR and bleeding.
• Isoniazid: Enzyme inhibitor -> potentiates warfarin effect -> bleeding risk.
• Amiodarone: Strong CYP inhibitor -> significantly increases INR and bleeding risk.

QUESTION

Match the following drugs-receptor combination appropriate for A



A Salbutamol at beta-2 receptor

B Naloxone at opioid receptors

C Buprenorphine at opioid receptor

D **Carboline at benzodiazepine site of GABA-A receptor** CORRECT

RIGHT ANSWER

OK

D. Carboline at benzodiazepine site of GABA-A receptor

WHY THIS IS RIGHT

β-Carbolines act as inverse agonists at the benzodiazepine site of the GABA-A receptor. Unlike benzodiazepines, which enhance GABA-mediated chloride influx, β-carbolines reduce basal GABA activity, producing effects opposite to benzodiazepines such as anxiety, seizures, and insomnia. Hence, when option A refers to an inverse agonist, the correct drug-receptor pairing is carboline at the BZD site of the GABA-A receptor.

Why other options are incorrect:
• Salbutamol at β□ receptor: A full agonist, not an inverse agonist.
• Naloxone at opioid receptor: A pure antagonist with zero intrinsic activity.
• Buprenorphine at opioid receptor: A partial agonist, not an inverse agonist.

QUESTION

Oral digoxin is being given to a 65-year-old male as a maintenance dose. A target plasma concentration of 0.70 ng/ mL is decided upon. The digoxin clearance for this particular individual is calculated as 60 mL/min. Knowing that the oral bioavailability of digoxin is 70%, what is the dosing rate in this patient?

A 40 ng/min

B **60 ng/min** CORRECT

C 70 ng/min

D 80 ng/min

OK RIGHT ANSWER

B. 60 ng/min

WHY THIS IS RIGHT

Maintenance dosing rate is calculated using the formula:
Dosing rate = (Target steady-state concentration × Clearance) / Bioavailability.
Here, Css = 0.70 ng/mL, clearance = 60 mL/min, and oral bioavailability (F) = 0.7.
Thus: (0.70 × 60) / 0.7 = 42 / 0.7 = 60 ng/min.
This is the required dosing rate to maintain the desired plasma digoxin concentration at steady state.

Why other options are incorrect:
• 40 ng/min: Fails to account for oral bioavailability.
• 70 ng/min & 80 ng/min: Overestimate dose and risk digoxin toxicity.

QUESTION

Healthy adult volunteers are enrolled in a phase I clinical trial investigating the properties of a newly developed oral antimicrobial agent. While reviewing the data, the researchers note that the drug's half-life seems to vary amongst the study participants. An increase in which of the following pharmacologic parameters is most likely responsible for the longer half-life seen in certain individuals?

A Drug glucuronidation

B Glomerular filtration rate

C Oral bioavailability

D **Volume of distribution** CORRECT

OK RIGHT ANSWER

D. Volume of distribution

WHY THIS IS RIGHT

Drug half-life is determined by the equation $t_{1/2} = 0.693 \times$ (Volume of distribution / Clearance). An increased volume of distribution (Vd) means the drug is more extensively distributed into tissues rather than remaining in plasma. This creates a larger drug reservoir, from which the drug returns slowly to the circulation for elimination, thereby prolonging the half-life even if clearance remains unchanged. Interindividual differences in body composition or tissue binding commonly explain such variability.

Why other options are incorrect:
• Drug glucuronidation: Increases clearance -> shortens half-life.
• Glomerular filtration rate: Higher GFR increases elimination -> shorter half-life.
• Oral bioavailability: Affects amount absorbed, not half-life.

QUESTION

A 65-year-old man is brought to the emergency department after developing sudden-onset right-side weakness and difficulty speaking. He has a history of paroxysmal atrial fibrillation and has been taking warfarin for the past several years with a stable prothrombin time. His wife adds that he started taking a new drug 2 weeks ago, but she does not remember its name. Transesophageal echocardiogram reveals a small thrombus in the left atrium. This patient most likely started taking which of the following drugs recently?

A Amiodarone

B Cimetidine

C Ciprofloxacin

D **St John's wort** CORRECT

OK RIGHT ANSWER

D. St John's wort

WHY THIS IS RIGHT

Despite a previously stable prothrombin time, this patient developed a left atrial thrombus and ischemic stroke, indicating reduced anticoagulant effect of warfarin. St John’s wort is a potent CYP450 (especially CYP2C9 and CYP3A4) enzyme inducer, which increases warfarin metabolism, lowers INR, and predisposes to thromboembolism. This interaction is a classic cause of warfarin failure despite compliance.

Why other options are incorrect:
• Amiodarone: CYP inhibitor; increases warfarin effect -> bleeding risk.
• Cimetidine: Enzyme inhibitor; raises INR.
• Ciprofloxacin: Inhibits warfarin metabolism, increasing bleeding risk, not thrombosis.

QUESTION

Omidenepag approved for treatment of

A Pulmonary hypertension

B **Glaucoma** CORRECT

C Cutaneous T cell lymphoma

D Thrombocytopenia

RIGHT ANSWER

OK **B. Glaucoma**

WHY THIS IS RIGHT

Omidenepag is a selective prostaglandin EP2 receptor agonist approved for the treatment of open-angle glaucoma and ocular hypertension. It lowers intraocular pressure by increasing uveoscleral outflow without acting on FP receptors, distinguishing it from traditional prostaglandin analogs like latanoprost. Because of this selective mechanism, it has a different side-effect profile, with less risk of prostaglandin-associated cosmetic changes.

Why other options are incorrect:
• Pulmonary hypertension: Treated with prostacyclin analogs and endothelin antagonists, not EP2 agonists.
• Cutaneous T-cell lymphoma: Treated with agents like bexarotene or HDAC inhibitors.
• Thrombocytopenia: Managed with thrombopoietin receptor agonists, not prostaglandins.

QUESTION

Which of the following adverse effects is associated with long-term prophylactic use of nitrofurantoin?

A Tendon rupture

B **Pulmonary fibrosis** CORRECT

C QT prolongation

D Optic neuritis

OK RIGHT ANSWER

B. Pulmonary fibrosis

WHY THIS IS RIGHT

Long-term prophylactic use of nitrofurantoin, especially for recurrent urinary tract infections, is classically associated with chronic pulmonary toxicity, leading to interstitial pneumonitis and pulmonary fibrosis. This occurs due to oxidative lung injury and immune-mediated mechanisms, typically after months to years of continuous therapy. Elderly patients are particularly susceptible, and symptoms include progressive dyspnea and dry cough.

Why other options are incorrect:
• Tendon rupture: Seen with fluoroquinolones, not nitrofurantoin.
• QT prolongation: Associated with macrolides and fluoroquinolones.
• Optic neuritis: Classically linked to ethambutol.

QUESTION

An elderly patient with overactive bladder develops confusion and memory issues after starting therapy. Which alternative drug would be least likely to worsen cognition?

A Oxybutynin

B Tolterodine

C **Trospium** CORRECT

D Solifenacin

RIGHT ANSWER

OK

C. Trospium

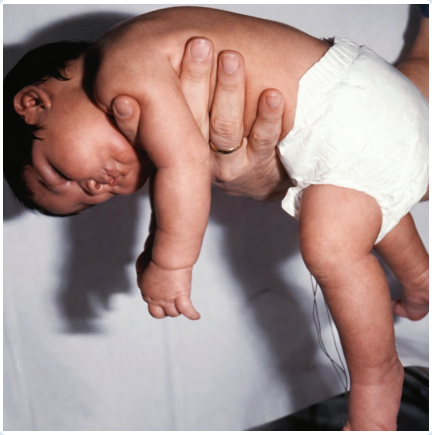
WHY THIS IS RIGHT

Trospium is a quaternary ammonium antimuscarinic that poorly crosses the blood-brain barrier, making it least likely to cause central anticholinergic effects such as confusion and memory impairment in elderly patients. Hence, it is preferred in overactive bladder when cognitive safety is a concern.

Why other options are incorrect:
• Oxybutynin: Tertiary amine with high CNS penetration; commonly causes confusion.
• Tolterodine: Tertiary amine; can cross the BBB and impair cognition.
• Solifenacin: M3-selective but still a tertiary amine with potential CNS effects.

QUESTION

Which drug when given in 3rd trimester to pregnant mother would result in following finding in her newborn?



A **Diazepam** CORRECT

B Promethazine

C Labetalol

D Paracetamol

OK RIGHT ANSWER

A. Diazepam

WHY THIS IS RIGHT

Diazepam given in the 3rd trimester crosses the placenta and accumulates in the fetus due to its long half-life.
Neonatal exposure leads to “floppy infant syndrome”, characterized by hypotonia, lethargy, poor suck, hypothermia, and respiratory depression.
The image of a baby hanging limply from the mother’s hand classically represents marked hypotonia, which is a hallmark of late-pregnancy benzodiazepine exposure.

Why other options are incorrect:
• Promethazine: May cause sedation but does not classically cause floppy infant syndrome.
• Labetalol: Causes neonatal bradycardia or hypoglycemia, not hypotonia.
• Paracetamol: Considered safe in pregnancy; no neonatal hypotonia association.

QUESTION

A 29-year-old male diagnosed with schizophrenia has been stable on risperidone for the past few months. Recently, he began experiencing dystonia, for which he was started on benztropine 2 mg twice daily by his psychiatrist. A week later, he presents to the emergency department with complaints of dry mouth, blurred vision, confusion and urinary retention. On examination, he is disoriented, tachycardic, and febrile. Pupils are dilated and bowel sounds are absent. Which of the following is the most appropriate next step in the management of this patient?

A. Discontinue risperidone and initiate lorazepam for possible neuroleptic malignant syndrome (NMS)

B. Administer intravenous dantrolene and initiate cooling measures for suspected serotonin syndrome

C. **Start physostigmine with continuous cardiac monitoring for anticholinergic toxicity due to benztropine overdose** CORRECT

D. Administer activated charcoal and IV fluids; observe the patient as symptoms are likely self-limiting

RIGHT ANSWER

OK **C. Start physostigmine with continuous cardiac monitoring for anticholinergic toxicity due to benztropine overdose**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

What is the preferred pre-operative drug for the management of hypertension in a patient with pheochromocytoma?

A Telmisartan

B **Phenoxybenzamine** CORRECT

C Prazosin

D Amlodipine

RIGHT ANSWER

OK **B. Phenoxybenzamine**

WHY THIS IS RIGHT

Pheochromocytoma = Catecholamine storm
Tumor of adrenal medulla -> excess:
• Epinephrine
• Norepinephrine

Tumor -> ↑ catecholamines
↓
α1 stimulation -> vasoconstriction
↓
Severe hypertension
Pheochromocytoma requires preoperative alpha blockade to prevent hypertensive crises. Phenoxybenzamine is an irreversible nonselective α-blocker that provides sustained control of catecholamine effects. It is started before surgery and followed by β-blockade if needed. Long-acting alpha blockade is the key preparation step.

Drug	Type	Role in Pheochromocytoma
Phenoxybenzamine	Non-selective α-blocker	□ GOLD STANDARD
Prazosin	α1 selective	Alternative
Propranolol	β-blocker	Only after α-blockade
CCBs	Vasodilators	Adjunct

QUESTION

A 34-year-old woman with a history of allergic rhinitis has been taking cetirizine for symptom control. She presents with a complaint of excessive daytime sleepiness, which she attributes to her medication. Which of the following would be the best alternative in this patient?

A Diphenhydramine

B **Fexofenadine** CORRECT

C Promethazine

D Chlorpheniramine

OK RIGHT ANSWER

B. Fexofenadine

WHY THIS IS RIGHT

Drug	Feature
Diphenhydramine	Strong sedation
Promethazine	Very sedating
Chlorpheniramine	Moderate sedation

Allergy -> Histamine release
↓
H1 receptor activation
↓
Symptoms (sneezing, rhinorrhea)
↓
Fexofenadine blocks H1
↓
Relief WITHOUT sedation
Daytime sleepiness from cetirizine reflects CNS penetration of some second-generation antihistamines. Fexofenadine has minimal blood-brain barrier entry and is essentially non-sedating. First-generation agents like diphenhydramine, promethazine, and chlorpheniramine are more sedating. Hence fexofenadine is the preferred alternative.

Drug	Generation	Sedation	BBB Penetration
Diphenhydramine	1st	High	High
Promethazine	1st	Very high	High
Chlorpheniramine	1st	Moderate	High
Cetirizine	2nd	Mild	Low
Fexofenadine	2nd	Minimal	Very low

QUESTION

A 12-year-old boy is brought to the emergency department after accidental ingestion of tricyclic antidepressant medications. He presents with palpitations, dry mouth, mydriasis, and tachycardia. Which of the following is the most appropriate antidote in this scenario?

A Lithium

B **Physostigmine** CORRECT

C Naloxone

D Flumazenil

RIGHT ANSWER

OK **B. Physostigmine**

WHY THIS IS RIGHT

Tricyclic antidepressants (e.g., imipramine, amitriptyline)
They block multiple receptors:
• Muscarinic -> anticholinergic effects
• α1 -> hypotension
• H1 -> sedation

Tricyclic antidepressant overdose produces a strong anticholinergic toxidrome with mydriasis, tachycardia, and dry mouth. Physostigmine is a centrally acting acetylcholinesterase inhibitor that reverses anticholinergic toxicity. It crosses the blood-brain barrier and restores cholinergic transmission. Naloxone and flumazenil act on opioid and benzodiazepine receptors respectively.

Poison	Antidote
Opioids	Naloxone
Benzodiazepines	Flumazenil
Anticholinergic	Physostigmine
TCA cardiotoxicity	Sodium bicarbonate

QUESTION

A patient comes to the casualty with organophosphate poisoning. He was started on atropine infusion and pralidoxime. After 2 hours, the patient had a sudden rise in temperature. What is the likely cause of fever?

A **Atropine toxicity** CORRECT

B Side effect of pralidoxime

C Due to organophosphate poisoning

D Idiopathic

RIGHT ANSWER

OK **A. Atropine toxicity**

WHY THIS IS RIGHT

Organophosphate (OP) poisoning
• Inhibits acetylcholinesterase
• -> ↑ Acetylcholine
• -> Cholinergic excess

Drug	Role
Atropine	Blocks muscarinic effects
Pralidoxime	Reactivates AChE

High-dose atropine can produce anticholinergic toxicity, causing hyperthermia from impaired sweating. This results in hot, dry skin and sudden temperature rise.
Atropine
↓
Blocks sweating (M3 receptors)
↓
No heat loss via evaporation
↓
Heat accumulation
↓
Hyperthermia (fever)
Pralidoxime does not typically cause fever. The hyperthermia reflects atropine overdose during organophosphate management.

Feature	OP Poisoning	Atropine Toxicity
Sweating	↑	Absent
Temperature	Normal/low	↑ (fever)
Secretions	↑	↓
Pupils	Constricted	Dilated

QUESTION

A chronic smoker was on nicotine replacement therapy and clonidine tablets for smoking de-addiction. He stopped taking clonidine tablets and now presents with a headache. What is the reason behind this condition?

A Postural hypotension

B Receptor upregulation

C **Rebound hypertension** CORRECT

D Receptor hypersensitivity

RIGHT ANSWER

OK

C. Rebound hypertension

WHY THIS IS RIGHT

Mechanism of Clonidine
• α□ receptor agonist (central action)
• Acts in brainstem -> ↓ sympathetic outflow

Clonidine is an α2-agonist that suppresses sympathetic outflow. Abrupt withdrawal causes rebound sympathetic overactivity, leading to severe hypertension and headache.
Clonidine
↓
Stimulates α2 receptors (presynaptic)
↓
↓ Norepinephrine release
↓
↓ Sympathetic tone
↓
↓ Blood pressure
Chronic use causes receptor adaptation, so sudden stoppage unmasks excess catecholamine release (What happens when you STOP clonidine suddenly? The system “snaps back” like a stretched rubber band)
This classic withdrawal effect is termed rebound hypertension.

QUESTION

Which of the following is the most appropriate treatment for an overactive bladder in a patient with dementia?

A **Tolterodine** CORRECT

B Tiotropium

C Telenzepine

D Tropicamide

RIGHT ANSWER

OK **A. Tolterodine**

WHY THIS IS RIGHT

Parasympathetic (ACh)
↓
M3 receptor activation
↓
Detrusor contraction
↓
Urination
Overactive bladder is treated with antimuscarinic agents that reduce detrusor overactivity. Tolterodine has relative bladder selectivity and less CNS penetration, making it safer in dementia. Tiotropium is for COPD, tropicamide is ophthalmic, and telenzepine is gastric selective. Tolterodine provides urinary control with fewer cognitive effects.
B) Tiotropium
• Used in:
• COPD

Acts on bronchial smooth muscle

C) Telenzepine
• M1 selective
• Used for:
• Gastric acid secretion

No role in bladder

D) Tropicamide
• Used in:
• Eye (mydriasis)

No urinary role

Drug	Feature
Tolterodine	□ Preferred
Oxybutynin	More CNS effects □
Darifenacin	M3 selective □
Solifenacin	M3 selective □

QUESTION

A patient is found on a railway track with hot dry skin, raised temperature, dilated pupils, slurred speech, and a staggering gait. Which of the following is the most likely cause?

- A **Dhatura poisoning** — CORRECT
- B Alcohol intoxication
- C Opioid overdose
- D Cocaine poisoning

RIGHT ANSWER

OK **A. Dhatura poisoning**

WHY THIS IS RIGHT

Dhatura (Datura stramonium)
A plant packed with anticholinergic alkaloids:
• Atropine
• Scopolamine
• Hyoscyamine

Hot dry skin, hyperthermia, mydriasis, slurred speech, and ataxia indicate an anticholinergic toxidrome. Dhatura contains atropine-like alkaloids causing central and peripheral antimuscarinic effects. Classic features are “hot as a hare, dry as a bone, blind as a bat.”
Opioids cause miosis and respiratory depression, not dryness or hyperthermia.

Feature	Anticholinergic	Opioid	Cocaine
Skin	Dry	Normal	Sweaty
Pupils	Dilated	Pinpoint	Dilated
Temp	↑	↓/normal	↑
CNS	Delirium	Depressed	Agitated

QUESTION

Which of the following neurotransmitters is released from SA node in response to increase in blood pressure?

A **Acetylcholine** CORRECT

B Dopamine

C Epinephrine

D Nor Epinephrine

OK RIGHT ANSWER

A. Acetylcholine

WHY THIS IS RIGHT

When BP rises, the body tries to slow the heart down
An increase in blood pressure activates the baroreceptor reflex, increasing vagal output to the heart. Vagal stimulation releases acetylcholine at the SA node, slowing heart rate.
↑ Blood pressure
↓
Baroreceptors activated (carotid sinus, aortic arch)
↓
Signals to medulla
↓
↑ Parasympathetic (vagal) output
↓
Acetylcholine released at SA node
↓
↓ Heart rate (bradycardia)
This parasympathetic response reduces cardiac output and helps normalize blood pressure. The SA node is primarily controlled by cholinergic vagal input in this reflex.

System	Neurotransmitter	Effect on Heart
Parasympathetic	Acetylcholine	↓ HR
Sympathetic	Norepinephrine	↑ HR

QUESTION

Which of the following statements regarding the causes of fall in diastolic blood pressure is correct?
1. Loss of sympathetic outflow in the lumbar region
2. Antagonism of beta-1 adrenergic receptors
3. Blockade of alpha-1 adrenergic receptors
4. Antagonism of beta-2 adrenergic receptors

A 1, 2 and 3

B **1 and 3** CORRECT

C 1, 2, 3 and 4

D 3 and 4

OK RIGHT ANSWER

B. 1 and 3

WHY THIS IS RIGHT

Diastolic BP is mainly governed by Systemic Vascular Resistance (SVR)
Not cardiac output.
Diastolic pressure mainly reflects peripheral vascular resistance. Loss of lumbar sympathetic outflow and alpha-1 blockade cause vasodilation and reduced resistance, lowering diastolic BP. Beta-1 antagonism primarily reduces heart rate and contractility, affecting systolic more than diastolic pressure. Beta-2 blockade causes vasoconstriction, which would increase diastolic pressure, not decrease it.

Receptor	Action	Effect on DBP
α1	Vasoconstriction	↑ DBP
β1	↑ CO	↑ SBP mainly
β2	Vasodilation	↓ DBP
β2 blockade	Vasoconstriction	↑ DBP

QUESTION

What is the main role of Phase 1 in xenobiotic metabolism?

A To make the compound more permeable across membranes

B To extract functional proteins through oxidase/reductase reactions

C **To introduce or expose functional groups for further metabolism** CORRECT

D To directly conjugate the compound and facilitate excretion

RIGHT ANSWER

OK **C. To introduce or expose functional groups for further metabolism**

WHY THIS IS RIGHT

Phase I reactions (oxidation, reduction, hydrolysis) introduce or expose polar functional groups on xenobiotics. These reactions are mainly mediated by CYP450 enzymes. The purpose is to prepare the molecule for Phase II conjugation. Thus, Phase I increases chemical reactivity for further metabolism.

Reaction	Example
Oxidation	CYP450 mediated
Reduction	Nitro -> amine
Hydrolysis	Ester breakdown

Feature	Phase I	Phase II
Function	Functionalization	Conjugation
Reaction	Oxidation, reduction	Glucuronidation, sulfation
Enzymes	CYP450	Transferases
Result	Slightly polar	Highly water-soluble

QUESTION

A 58-year-old man presents with septic shock. He is febrile, tachycardic, and has a blood pressure of 80/50 mmHg despite adequate fluid resuscitation with IV crystalloids. His urine output is low, and he is drowsy. What is the next best step in management?

A Administer dopamine

B **Administer norepinephrine** CORRECT

C Administer hydrocortisone

D Administer vasopressin

OK RIGHT ANSWER

B. Administer norepinephrine

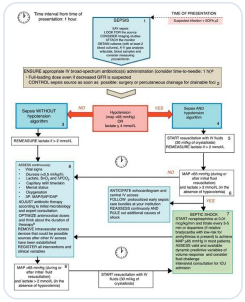
WHY THIS IS RIGHT

This patient has:
• Persistent hypotension (80/50 mmHg) despite adequate fluids
• Organ dysfunction -> low urine output, altered sensorium

This fulfills criteria for septic shock

Next best step:
• Start vasopressor therapy immediately
• First-line vasopressor = Norepinephrine
• Strong α□ agonist -> vasoconstriction -> ↑ SVR -> ↑ BP
• Mild β□ effect -> maintains cardiac output
• Best survival benefit + lowest arrhythmia risk

Other options:
• Hydrocortisone -> used *only if refractory shock* (after vasopressors)
• Vasopressin -> *add-on*, not first-line



QUESTION

Which of the following statements is true regarding the pharmacological relationship between imipramine and diphenhydramine?

A Imipramine inhibits diphenhydramine's action

B Diphenhydramine inhibits imipramine's action

C **Both block muscarinic receptors** CORRECT

D Both are norepinephrine reuptake inhibitors

OK RIGHT ANSWER

C. Both block muscarinic receptors

WHY THIS IS RIGHT

• Imipramine -> Tricyclic antidepressant (TCA)
• Diphenhydramine -> 1st generation antihistamine

Different primary roles, but both carry a strong anticholinergic personality
Imipramine (a TCA) and diphenhydramine (a first-generation antihistamine) both have strong antimuscarinic activity. This shared action explains common side effects like dry mouth, blurred vision, and urinary retention.

Feature	Imipramine	Diphenhydramine
Class	TCA	1st gen antihistamine
Main action	NE/5-HT reuptake inhibition	H1 blockade
Muscarinic block	□ Yes	□ Yes
Sedation	Moderate	Strong
Anticholinergic effects	□ Prominent	□ Prominent

Diphenhydramine is not a norepinephrine reuptake inhibitor, and neither drug blocks the other's action. Their pharmacologic overlap is anticholinergic blockade.

QUESTION

A 62-year-old male with a history of coronary heart disease (CHD) and renal failure was admitted to the ICU with elevated blood pressure (BP 190/110 mmHg) and declining urine output. The intensivist administers fenoldopam intravenously. How does this drug help in improving renal function in this patient?

A **D1 agonism** — CORRECT

B D2 antagonism

C Alpha antagonism

D Muscarinic antagonism

RIGHT ANSWER

OK

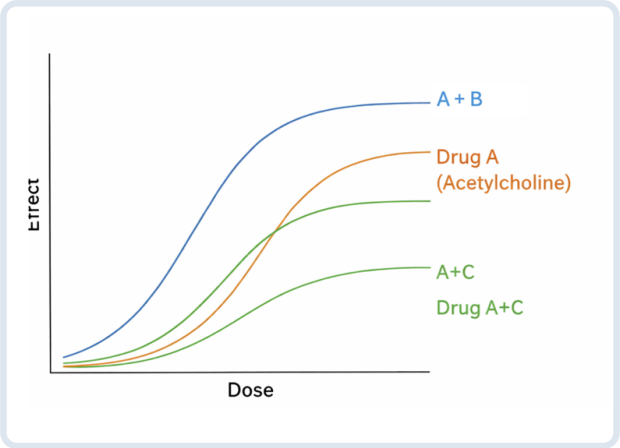
A. D1 agonism

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

In the given graph of effect vs dose, the orange line represents drug A which is acetylcholine. Identify drug B.



A **Physostigmine** CORRECT

B Bethanechol

C Atropine

D Adrenaline

OK RIGHT ANSWER

A. Physostigmine

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

Which of the following statements regarding Dronabinol are true?
1. It is used in AIDS-related weight loss
2. It acts on the CB1 receptor
3. It is used in chronic pain management
4. It is excreted unchanged in faeces

A **1 and 2** CORRECT

B 1, 2 and 3

C 2, 3 and 4

D 1, 2 and 4

OK RIGHT ANSWER

A. 1 and 2

WHY THIS IS RIGHT

Target: CB1 receptors (primarily CNS)
Action: Inhibits neurotransmitter release (via ↓ cAMP, ↓ Ca²□ influx)
Modulates:
• Appetite
• Nausea
• Pain perception (limited clinical role)

Dronabinol is a synthetic THC used for AIDS-related anorexia/cachexia and chemotherapy-induced nausea. It acts mainly via central CB1 cannabinoid receptors to stimulate appetite. It is not a standard drug for chronic pain management. The drug undergoes hepatic metabolism rather than being excreted unchanged in feces.

Feature	CB1	CB2
Location	CNS (brain)	Immune cells
Function	Appetite, mood, nausea	Immune modulation
Target of Dronabinol	□ Yes	□ No

QUESTION

A 45 year old male with erectile dysfunction was prescribed sildenafil. He develops bluish-green vision. Inhibition of which enzyme is the cause?

A Phosphodiesterase 3

B Phosphodiesterase 5

C **Phosphodiesterase 6** CORRECT

D Phosphodiesterase 2

RIGHT ANSWER

OK

C. Phosphodiesterase 6

WHY THIS IS RIGHT

Sildenafil primarily inhibits phosphodiesterase-5 (PDE-5) to treat erectile dysfunction. However, it also weakly inhibits phosphodiesterase-6 (PDE-6), an enzyme present in retinal photoreceptors involved in phototransduction. Inhibition of PDE-6 alters retinal cGMP levels, leading to bluish-green color vision (cyanopsia), a characteristic visual side effect of sildenafil.

Why other options are incorrect:
• PDE-3: Involved in cardiac contractility and platelet function.
• PDE-5: Desired target for erection, not responsible for visual disturbance.
• PDE-2: Has no role in retinal phototransduction.

QUESTION

Which of the following is used to monitor PCM toxicity?

A **Rumack Matthew** CORRECT

B Matzke

C Hartford

D Naranjo

RIGHT ANSWER

OK **A. Rumack Matthew**

WHY THIS IS RIGHT

The Rumack-Matthew nomogram is used to assess and monitor paracetamol (PCM/acetaminophen) toxicityfollowing acute overdose. It plots serum paracetamol concentration versus time since ingestion (4-24 hours) to predict the risk of hepatotoxicity and guide the need for N-acetylcysteine (NAC) therapy. It is applicable only for single, acute ingestions with a known time of intake.

Why other options are incorrect:
• Matzke: Dosing adjustment in renal impairment (e.g., aminoglycosides).
• Hartford: Once-daily aminoglycoside dosing nomogram.
• Naranjo: Assesses probability of adverse drug reactions, not toxicity monitoring.

QUESTION

Which of the following organs can differentiate between the action of epinephrine and nor-epinephrine:

A Atrioventricular node

B Iris of the eye

C **Bronchus** CORRECT

D Renal juxtaglomerular apparatus

OK RIGHT ANSWER

C. Bronchus

WHY THIS IS RIGHT

The bronchus can differentiate between epinephrine and norepinephrine because bronchial smooth muscle expresses predominantly β□-adrenergic receptors. Epinephrine has strong β□ agonist activity and causes bronchodilation, whereas norepinephrine has minimal β□ action and therefore produces little to no bronchodilation. This functional difference allows clear distinction between their effects in the bronchial tree.

Why other options are incorrect:
• Atrioventricular node: Both act via β□ receptors, increasing conduction similarly.
• Iris of the eye: Both cause mydriasis via α□ receptors.
• Renal JGA: Both stimulate β□ receptors, increasing renin release similarly

QUESTION

Which of the following is incorrect about the mechanism of action of carvedilol?

A Calcium channel antagonism

B Beta-1 receptor antagonism

C **Alpha-2 receptor antagonism** CORRECT

D Beta-2 receptor antagonism

RIGHT ANSWER

OK

C. Alpha-2 receptor antagonism

WHY THIS IS RIGHT

Carvedilol is a non-selective β-blocker with additional α□-adrenergic antagonism, leading to reduced heart rate, myocardial contractility, and peripheral vasodilation. It also exhibits mild calcium channel-blocking and antioxidant properties, contributing to its benefit in heart failure and hypertension. However, carvedilol does not block α□ receptors; α□ antagonism would increase norepinephrine release, which is opposite to its therapeutic profile.

Why other options are incorrect:
• Calcium channel antagonism: Mildly present as an ancillary effect.
• Beta-1 receptor antagonism: Core action reducing heart rate and contractility.
• Beta-2 receptor antagonism: Present due to non-selective β-blockade.

QUESTION

Which of the following is true regarding nicotine substitution therapy?

A Preferably given by gastrointestinal route

B Varenicline comes with a black box warning of cardiovascular death

C **There should be a 15-minute gap between nicotine gum and coffee/soda/acidic food as they decrease its absorption** CORRECT

D Nicotine chewing gum is better for constant use as it gives 25% higher blood level than lozenges

RIGHT ANSWER

OK **C. There should be a 15-minute gap between nicotine gum and coffee/soda/acidic food as they decrease its absorption**

WHY THIS IS RIGHT

Nicotine replacement therapy (NRT) is an effective method to assist in smoking cessation. It is used to relieve craving for tobacco by substituting nicotine in tobacco with nicotine in safer forms. Various preparations include nicotine gums, nicotine lozenges, nicotine patch, nicotine spray and nicotine inhaler. Nicotine gum's absorption can be affected by acidic beverages, so it's advised to avoid them shortly before or while using the gum.

A) Incorrect. NRT is not given via the gastrointestinal route. Nicotine in NRT is absorbed either through buccal mucosa or skin.

B) False. Varenicline doesn’t come with this black box warning.

C) True. Acidic beverages can interfere with nicotine gum's absorption.

D) Incorrect. Choice between gum and lozenges depends on individual preference, not blood levels.

QUESTION

A patient with narcolepsy has been prescribed tiprolisant. How does this drug act?

A H1 receptor agonism

B H1 receptor inverse agonism

C H3 receptor agonism

D **H3 receptor inverse agonism** CORRECT

RIGHT ANSWER

OK

D. H3 receptor inverse agonism

WHY THIS IS RIGHT

Tiprolisant (pitolisant) treats narcolepsy by acting as an inverse agonist at central H3 histamine receptors. H3 receptors are presynaptic autoreceptors that normally inhibit histamine release. Inverse agonism blocks this inhibitory tone, increasing histamine release in the brain, thereby promoting wakefulness and reducing excessive daytime sleepiness in narcolepsy.

Why other options are incorrect:
• H1 receptor agonism: Would promote wakefulness but is not the mechanism of pitolisant.
• H1 receptor inverse agonism: Causes sedation, opposite of desired effect.
• H3 receptor agonism: Would further suppress histamine release and worsen sleepiness.

QUESTION

A 60-year-old male patient, a known case of pancreatic carcinoma came to the hospital with complaints of loss of appetite for 3 months. Which of the following drugs is best suited to be prescribed in this patient to increase appetite?

A Loratidine

B Azelastine

C Cetirizine

D **Cyproheptadine** CORRECT

RIGHT ANSWER

OK

D. Cyproheptadine

WHY THIS IS RIGHT

Cyproheptadine is a first-generation H□ antihistamine with additional antiserotonergic (5-HT□) activity, which stimulates appetite and promotes weight gain. It is commonly used as an appetite stimulant in cancer-related anorexia and cachexia, including pancreatic carcinoma. Its central effects make it effective in improving appetite and food intake in chronic illness.

Why other options are incorrect:
• Loratidine: Second-generation antihistamine; minimal CNS effects and no appetite stimulation.
• Azelastine: Intranasal antihistamine for allergic rhinitis; not used systemically for appetite.
• Cetirizine: Second-generation H□ blocker with minimal sedative or appetite-stimulating effect.

QUESTION

The following table gives BP values before and after administration of drug 'X'. What would be the probable mechanism of action of drug 'X'?

Drug X		
SBP	120	150
DBP	80	68
HR	72	86

A α1 agonist and β1 agonist

B M2 agonist and M3 agonist

C α 1 antagonist and β2 agonist

D **β1 agonist and β2 agonist** CORRECT

OK RIGHT ANSWER

D. β1 agonist and β2 agonist

WHY THIS IS RIGHT

The drug's likely mechanism of action is β1 and β2 agonism.
Due to its action on β1 receptors, there is an increase in the HR of the patient and also of the SBP (Increase in HR leads to an increase in the CO upto a certain extent). The agonistic action on the β2 receptors causes vasodilation and leads to a fall in the DBP.

QUESTION

All the following are signs of amitriptyline toxicity except:

A Coma and shock

B Hot dry skin

C **Pinpoint pupil** CORRECT

D Hypotension

RIGHT ANSWER

OK

C. Pinpoint pupil

WHY THIS IS RIGHT

Amitriptyline is a tricyclic antidepressant (TCA) with prominent anticholinergic, α□-blocking, and quinidine-like (Na□ channel-blocking) effects. In toxicity, patients classically develop coma, seizures, hypotension, and shock due to CNS depression and myocardial sodium channel blockade. Hot, dry skin occurs from anticholinergic inhibition of sweating, and pupils are typically dilated (mydriasis), not constricted.

Why other options are incorrect:
• Coma and shock: Common in severe TCA overdose due to CNS and cardiac toxicity.
• Hot dry skin: Typical anticholinergic feature of TCAs.
• Hypotension: Due to α□-blockade and myocardial depression.

QUESTION

Which of the following is the most appropriate treatment for COPD in a patient with dementia?

A Tolterodine

B **Tiotropium** CORRECT

C Telenzepine

D Tropicamide

RIGHT ANSWER

OK **B. Tiotropium**

WHY THIS IS RIGHT

Tiotropium is a long-acting inhaled antimuscarinic (LAMA) used in COPD. It is a quaternary ammonium compound, so it has minimal systemic absorption and poor blood-brain barrier penetration, making it least likely to worsen cognition in patients with dementia. Hence, it is the preferred anticholinergic bronchodilator in elderly patients with cognitive impairment.

Why other options are incorrect:
• Tolterodine: Used for overactive bladder; central anticholinergic effects can worsen dementia.
• Telenzepine: A centrally acting antimuscarinic; not used in COPD.
• Tropicamide: Short-acting ophthalmic antimuscarinic for eye exams, no role in COPD

QUESTION

A 58-year-old man with advanced Parkinson disease experiences sudden “off” episodes despite optimal oral levodopa therapy. He is prescribed a subcutaneous rescue medication for rapid relief of these episodes. Shortly after administration in the clinic, he develops severe hypotension and loss of consciousness. On reviewing his medication list, it is noted that he recently received an antiemetic for nausea. Which of the following antiemetics most likely precipitated this adverse reaction when co-administered with the rescue drug?

A Metoclopramide

B Domperidone

C **Ondansetron** CORRECT

D Prochlorperazine

OK RIGHT ANSWER

C. Ondansetron

WHY THIS IS RIGHT

Apomorphine is a dopamine agonist used for acute “off” episodes in Parkinson disease
Concomitant use with 5-HT□ antagonists (ondansetron) is contraindicated
Combination can cause:
Profound hypotension
Syncope
Loss of consciousness

Why other options are incorrect:
• Metoclopramide: Dopamine antagonist; worsens Parkinson symptoms but not severe hypotension.
• Domperidone: Preferred antiemetic with apomorphine; does not cross BBB.
• Prochlorperazine: Dopamine antagonist causing EPS, not this interaction.

QUESTION

A 20-year-old male is brought to the emergency department with amphetamine overdose. He presents with severe hypertension and cardiac arrhythmia. Which of the following drugs is most appropriate to treat the cardiovascular manifestations in this patient?

A Metoprolol

B Prazosin

C **Labetalol** CORRECT

D Nebivolol

OK RIGHT ANSWER

C. Labetalol

WHY THIS IS RIGHT

Amphetamine overdose -> ↑ sympathetic activity
↑ α□ -> vasoconstriction -> hypertension
↑ β□ -> tachycardia, arrhythmia
↑ β□ effects also present
Pure β-blockers (e.g., metoprolol, nebivolol) are contraindicated
Cause unopposed α-adrenergic stimulation
Can worsen hypertension and vasoconstriction
Labetalol blocks:
α□ receptors -> reduces vasoconstriction
β□ receptors -> controls tachycardia & arrhythmia

Hence, labetalol is the drug of choice among the options.

Why other options are incorrect:
• Metoprolol: A selective β□-blocker; may cause unopposed α-stimulation, worsening hypertension.
• Prazosin: Pure α□-blocker; does not adequately control tachyarrhythmias.
• Nebivolol: β□-selective with NO release, but still risks unopposed α effects in sympathomimetic overdose.

QUESTION

PIPE therapy used in erectile dysfunction involves intracavernosal injection of:

A Propranolol

B Phenylephrine

C **Phentolamine** CORRECT

D Prazosin

RIGHT ANSWER

OK

C. Phentolamine

WHY THIS IS RIGHT

PIPE therapy (Papaverine, Isoproterenol, Phentolamine, and Prostaglandin E□) is used in erectile dysfunction to induce erection via intracavernosal vasodilation. Phentolamine is a non-selective α-adrenergic blocker that inhibits sympathetic vasoconstriction in penile vessels, thereby enhancing blood flow and facilitating erection.

Why other options are incorrect:
• Propranolol: A β-blocker that can worsen erectile dysfunction.
• Phenylephrine: An α□-agonist causing vasoconstriction; used for priapism treatment.
• Prazosin: An α□-blocker but not used intracavernosally in PIPE therapy.

QUESTION

A 58-year-old woman with urge urinary incontinence is started on a drug that relaxes detrusor smooth muscle independent of antimuscarinic action. Which of the following drugs was most likely prescribed?

A Solifenacin

B Tolterodine

C **Flavoxate** CORRECT

D Darifenacin

RIGHT ANSWER

OK

C. Flavoxate

WHY THIS IS RIGHT

Flavoxate is a direct smooth muscle relaxant used in urge urinary incontinence. It relaxes the detrusor muscle independent of antimuscarinic (M3) receptor blockade, likely via phosphodiesterase inhibition and local anesthetic action. Hence, it reduces bladder spasms without typical anticholinergic side effects, making it distinct from standard antimuscarinic drugs.

Why other options are incorrect:
• Solifenacin: A selective M3 antimuscarinic that reduces detrusor contractions via receptor blockade.
• Tolterodine: A nonselective antimuscarinic acting on bladder muscarinic receptors.
• Darifenacin: Highly selective M3 receptor antagonist, not a direct smooth muscle relaxant.

QUESTION

A 55-year-old male undergoing chemotherapy presents with progressive shortness of breath, dry cough, and fatigue over the past 2 weeks. He is afebrile and denies chest pain or sputum production. On examination, fine basal crepitations are heard. Chest X-ray reveals bilateral reticulonodular infiltrates. High-resolution CT (HRCT) of the chest confirms interstitial lung disease. Pulmonary function tests show a restrictive pattern with FEV1 = 61% of predicted and normal FEV1/FVC ratio. Notably, there is no evidence of bone marrow suppression. Which of the following drugs is most likely responsible for his condition?

A Cisplatin

B **Bleomycin** CORRECT

C Cyclophosphamide

D Vincristine

OK RIGHT ANSWER

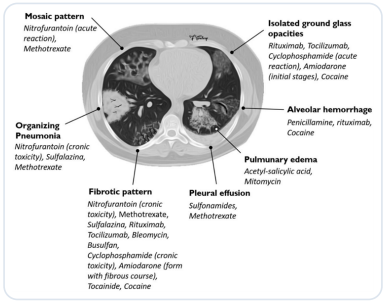
B. Bleomycin

WHY THIS IS RIGHT

Bleomycin causes dose-dependent pulmonary fibrosis presenting with dry cough, dyspnea, and restrictive lung disease. HRCT showing interstitial infiltrates with preserved marrow function is characteristic.

Bleomycin
↓
Free radical formation
↓
Lung tissue damage
↓
Interstitial inflammation
↓
Fibrosis -> restrictive lung disease
Unlike many chemotherapeutics, bleomycin has minimal bone marrow suppression. Pulmonary toxicity is its hallmark adverse effect.

Drug	Toxicity
Bleomycin	Pulmonary fibrosis
Cisplatin	Nephrotoxicity, ototoxicity
Cyclophosphamide	Hemorrhagic cystitis
Vincristine	Peripheral neuropathy



QUESTION

Methotrexate is a chemotherapeutic agent used in the treatment of breast cancer. It works by inhibiting the synthesis of which nucleotide?

A AMP

B CMP

C UMP

D **TMP** CORRECT

OK RIGHT ANSWER

D. TMP

WHY THIS IS RIGHT

A classic antimetabolite that sabotages DNA synthesis right at its biochemical roots.

Methotrexate inhibits dihydrofolate reductase, depleting tetrahydrofolate needed for thymidylate synthesis. This blocks conversion of dUMP to dTMP, impairing DNA replication. Rapidly dividing cancer cells are most affected. Inhibition of thymidine synthesis is the key cytotoxic mechanism.

QUESTION

Which drug is used to prevent cardiotoxicity induced by daunorubicin?

A Aprepitant

B **Dexrazoxane** CORRECT

C Amifostine

D Dexamethasone

RIGHT ANSWER

OK **B. Dexrazoxane**

WHY THIS IS RIGHT

Anthracycline (daunorubicin)
↓
Binds iron -> forms free radicals
↓
Oxidative damage
↓
Myocardial cell injury
↓
Dilated cardiomyopathy
Daunorubicin causes dose-dependent anthracycline cardiotoxicity via free radical-mediated myocardial injury. Dexrazoxane is an iron-chelating agent that reduces reactive oxygen species formation in cardiac tissue. It is used prophylactically to protect against anthracycline-induced cardiomyopathy. Cardiac protection is its key clinical role.

Toxicity	Drug
Anthracycline cardiotoxicity	Dexrazoxane
Cisplatin nephrotoxicity	Amifostine
Hemorrhagic cystitis (cyclophosphamide)	Mesna
Methotrexate toxicity	Leucovorin

QUESTION

All of the following drugs act as HER-1 inhibitors except

A Cetuximab

B Necitumumab

C **Pertuzumab** CORRECT

D Panitumumab

RIGHT ANSWER

OK

C. Pertuzumab

WHY THIS IS RIGHT

Drug	Target	Key Use
Cetuximab	EGFR (HER-1)	Colorectal cancer, head & neck cancer
Necitumumab	EGFR (HER-1)	Metastatic squamous NSCLC
Pertuzumab	HER-2	HER-2 positive breast cancer
Panitumumab	EGFR (HER-1)	Metastatic colorectal cancer

QUESTION

A 52-year-old female presents with metastatic breast cancer. She has previously undergone treatment with surgery, radiation, and multiple lines of chemotherapy, but her disease has progressed. Genetic testing reveals a BRCA1 mutation, indicating an impaired DNA repair mechanism. Her cancer is classified as HER2-negative and hormone receptor-positive. Given the genetic findings, her oncologist considers using a therapy that targets the PARP enzyme to exploit the DNA repair vulnerability of her cancer cells. Which of the following drugs would be appropriate to prescribe as a PARP inhibitor in this patient?

A Palbociclib

B **Rucaparib** CORRECT

C Vorinostat

D Ustekinumab

RIGHT ANSWER

OK **B. Rucaparib**

WHY THIS IS RIGHT

BRCA mutation = defective DNA repair system
Normal cells repair DNA via 2 major pathways:
• BRCA (Homologous recombination) -> repairs double-strand breaks
• PARP pathway -> repairs single-strand breaks

BRCA-mutated tumors have defective homologous recombination repair and are sensitive to PARP inhibition via synthetic lethality. Rucaparib is a PARP inhibitor that blocks DNA repair, causing tumor cell death. Palbociclib is a CDK4/6 inhibitor, vorinostat is an HDAC inhibitor, and ustekinumab is an immunologic agent. Targeting PARP exploits the DNA repair defect in BRCA cancers.

Drug	Target	Use
Rucaparib	PARP	BRCA cancers
Palbociclib	CDK4/6	Breast cancer
Trastuzumab	HER2	HER2+ cancer
Imatinib	BCR-ABL	CML

QUESTION

Which of the following statements regarding thalidomide is false?

A **It acts as an antimetabolite** CORRECT

B It is a teratogenic drug

C It is used in type 2 lepra reaction (ENL)

D It is used in relapsing multiple myeloma

RIGHT ANSWER

OK **A. It acts as an antimetabolite**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

Which of the following drugs acts on VEGF (Vascular Endothelial Growth Factor)?

A Pembrolizumab

B Ipilimumab

C **Ramucirumab** CORRECT

D Dostarlimab

RIGHT ANSWER

OK

C. Ramucirumab

WHY THIS IS RIGHT

Tumors are greedy, to survive, they stimulate new blood vessel formation (angiogenesis) using: VEGF (Vascular Endothelial Growth Factor)

Ramucirumab is a monoclonal antibody against VEGF receptor-2, blocking VEGF-mediated angiogenesis. It inhibits tumor blood vessel formation and growth. Pembrolizumab, ipilimumab, and dostarlimab are immune checkpoint inhibitors targeting PD-1 or CTLA-4, not VEGF.

Option	Drug	Target	Why Wrong
A	Pembrolizumab	PD-1	Immune checkpoint inhibitor
B	Ipilimumab	CTLA-4	T-cell activation ↑
D	Dostarlimab	PD-1	Used in MSI-high tumors

Anti-angiogenic action is the key distinguishing feature of ramucirumab.

QUESTION

All of the following cause pleural fibrosis except?

A **Metformin** CORRECT

B Methysergide

C Bleomycin

D Nitrofurantoin

OK RIGHT ANSWER

A. Metformin

WHY THIS IS RIGHT

Pleural fibrosis is a known adverse effect of several drugs due to chronic inflammatory or fibrotic reactions in lung and pleural tissue. Methysergide can cause pleuropulmonary fibrosis with long-term use due to serotonergic stimulation of fibroblasts. Bleomycin induces pulmonary and pleural fibrosis via free radical-mediated lung injury. Nitrofurantoin, especially with prolonged use, is associated with chronic interstitial lung disease and pleural fibrosis.

Metformin, however, is an oral antidiabetic drug that does not cause pulmonary or pleural fibrosis and has no fibrogenic pulmonary toxicity.

Why other options are incorrect:
• Methysergide: Causes pleuropulmonary fibrosis.
• Bleomycin: Classic cause of lung and pleural fibrosis.
• Nitrofurantoin: Long-term use causes chronic pulmonary fibrosis.

QUESTION

Match the incorrect pair of drugs and enzyme/reaction they inhibit:

A Azathiprine: PRPP synthetase

B Mycophenolate: IMP -> AMP conversion

C Lefluomide: Carbamoyl phosphate -> Orotic acid

D **5FU: Dihydrofolate reductase** CORRECT

RIGHT ANSWER

OK

D. 5FU: Dihydrofolate reductase

WHY THIS IS RIGHT

5-Fluorouracil (5-FU) inhibits thymidylate synthase, not dihydrofolate reductase (DHFR). It forms a stable ternary complex with thymidylate synthase and reduced folate, blocking dTMP synthesis and thereby inhibiting DNA synthesis. DHFR inhibition is the mechanism of methotrexate, not 5-FU.

Why other options are incorrect:
• Azathioprine : PRPP synthetase - Azathioprine (->6-MP) inhibits de novo purine synthesis via PRPP-dependent steps; this pairing is acceptable in exam context.
• Mycophenolate : IMP -> AMP conversion - Mycophenolate inhibits IMP dehydrogenase, blocking guanine nucleotide synthesis.
• Leflunomide : Carbamoyl phosphate -> orotic acid - Leflunomide inhibits dihydroorotate dehydrogenase in pyrimidine synthesis.

QUESTION

Two antineoplastic drugs are shown to inhibit intracellular thymidylate formation. The chemotherapeutic effect of drug X can be overcome by Ns-formyl-tetrahydrofolate supplementation, but that of drug Y is not affected. The drugs described in this scenario are most likely which of the following?

A X: Cytarabine Y:Gemcitabine

B X: Fluorouracil Y: Leucovorin

C X: Fludarabine Y: Methotrexate

D **X: Methotrexate Y: Fluorouracil** CORRECT

OK RIGHT ANSWER

D. X: Methotrexate Y: Fluorouracil

WHY THIS IS RIGHT

Methotrexate inhibits dihydrofolate reductase (DHFR), leading to depletion of reduced folate and decreased thymidylate (dTMP) synthesis. Its cytotoxic effect can be overcome by N^5-formyl-tetrahydrofolate (leucovorin/folinic acid) rescue, which bypasses DHFR and restores thymidylate synthesis.
Fluorouracil (5-FU) inhibits thymidylate synthase directly by forming a stable ternary complex with the enzyme and reduced folate. Leucovorin actually enhances 5-FU toxicity by stabilizing this complex and therefore does not reverse its effect.

Why other options are incorrect:
• Cytarabine/Gemcitabine: Primarily inhibit DNA polymerase, not folate pathways.
• Fluorouracil/Leucovorin: Leucovorin potentiates, not rescues, 5-FU.
• Fludarabine/Methotrexate: Fludarabine is a purine analog; methotrexate is folate-dependent but pairing is incorrect.

QUESTION

A 35-year-old man underwent an allogeneic hematopoietic stem cell transplant for aplastic anemia. Eight months later, he develops progressive cutaneous sclerosis, oral lichenoid lesions, and restrictive lung disease. He has an inadequate response to systemic corticosteroids and calcineurin inhibitors. A newer oral agent approved specifically for steroid-refractory chronic graft-versus-host disease is initiated. Which of the following drugs is most appropriate in this patient?

A Ruxolitinib

B Ibrutinib

C **Belumosudil** CORRECT

D Sirolimus

RIGHT ANSWER

OK **C. Belumosudil**

WHY THIS IS RIGHT

Belumosudil is a ROCK2 inhibitor
Approved for chronic graft-versus-host disease (cGVHD)
Used in patients after failure of ≥2 prior lines of systemic therapy
Acts by:
↓ Pro-fibrotic signaling
Modulating Th17 / Treg balance
Given orally

Why other options are incorrect:
• Ruxolitinib: A JAK1/2 inhibitor mainly approved for steroid-refractory acute GVHD.
• Ibrutinib: A BTK inhibitor approved for cGVHD but less specific for fibrotic manifestations.
• Sirolimus: An mTOR inhibitor used as immunosuppression, not a newer targeted therapy for steroid-refractory cGVHD.

QUESTION

A 26-year-old male presents to the hospital with fever and arthritis. Echocardiography confirms the presence of carditis in this patient. He gives a past history of myocardial infarction and peptic ulcer disease. Which of the following is the most appropriate anti-inflammatory agent in this patient?

A Diclofenac 50 mg TDS

B Naproxen 250 mg BD

C **Celecoxib 200 mg BD** CORRECT

D Ibuprofen 400 mg TDS

OK RIGHT ANSWER

C. Celecoxib 200 mg BD

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

Determine the total body clearance for a drug in a 70 kg male patient. The drug has an elimination half-life of 3 hours with an apparent volume of distribution of 100 ml/kg.

A 23 ml/h

B 230 ml/h

C **1617 ml/h** CORRECT

D 16 ml/h

RIGHT ANSWER

OK

C. 1617 ml/h

WHY THIS IS RIGHT

Total Vd = 100 ml/kg × 70 kg = 7000 ml.

Clearance = (0.693 × Vd) / t½ = (0.693 × 7000) / 3 ≈ 1617 ml/h.
This uses the standard clearance-half-life relationship. Hence total body clearance ≈ 1617 ml/h.

$$Cl = \frac{0.693 \times V_d}{t_{1/2}}$$

QUESTION

Which of the following drugs is not likely to cause a pleural pathology?

A **Metformin** CORRECT

B Methysergide

C Bleomycin

D Nitrofurantoin

RIGHT ANSWER

OK

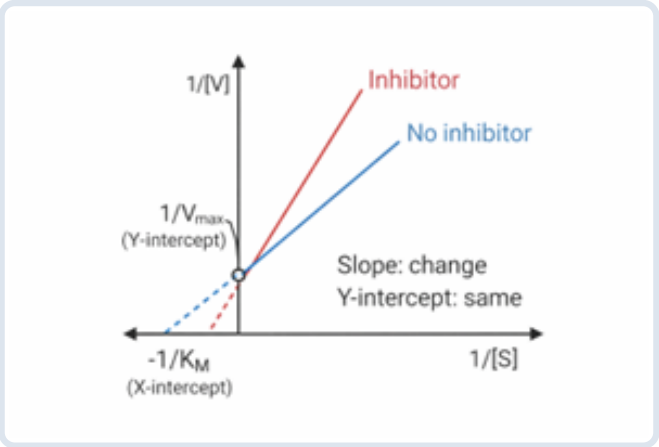
A. Metformin

WHY THIS IS RIGHT

Drug-induced pleural disease
Several drugs cause pleuropulmonary fibrosis or pleural reactions, including bleomycin, methysergide, and nitrofurantoin. These are classic causes of drug-induced pleural pathology. Metformin has no known association with pleural disease. Hence metformin is the exception.

QUESTION

Identify the type of inhibition from the given graph.



A **Competitive** CORRECT

B Non-competitive

C Un-competitive

D Allosteric

RIGHT ANSWER

OK **A. Competitive**

WHY THIS IS RIGHT

In competitive inhibition graphs (Michaelis-Menten or dose-response):
• Curve shifts RIGHT
• Same maximum effect (Emax)
• Increased Km (↓ affinity)

Drug (agonist) + receptor
↓
Competitive inhibitor present
↓
Competes for SAME site
↓
Need more agonist to achieve same effect
↓
Right shift in curve
The Lineweaver-Burk plot shows the same Y-intercept (1/Vmax) with a steeper slope in the presence of inhibitor. This indicates Vmax is unchanged while Km increases. Such a pattern is characteristic of competitive inhibition. Competitive inhibitors shift the X-intercept without altering Vmax.

Type	Km	Vmax	Graph Shift
Competitive	↑	Same	Right shift
Non-competitive	Same	↓	↓ height
Uncompetitive	↓	↓	Parallel shift

QUESTION

Lignocaine is used in a loading dose for the treatment of arrhythmias. The loading dose of this drug depends upon which of the following factors?

A Clearance

B **Volume of distribution** CORRECT

C Half-life

D Bioavailability

OK RIGHT ANSWER

B. Volume of distribution

WHY THIS IS RIGHT

Goal: Achieve target plasma concentration
↓
Drug distributes in body compartments
↓
Extent depends on Vd
↓
Higher Vd -> higher loading dose
Loading dose is determined by the amount of drug needed to rapidly achieve the target plasma concentration. It depends primarily on the volume of distribution, which reflects how extensively the drug distributes into tissues. Clearance and half-life influence maintenance dosing, not the loading dose. Hence Vd is the key determinant.

$$\text{Loading Dose} = \frac{C_p \times V_d}{F}$$

QUESTION

Concerning the mechanism of action of various antimicrobial drugs, which of the following statements is FALSE?

A **Linezolid: Binds to 30S ribosome and inhibits protein synthesis.** CORRECT

B Rifampicin: Inhibits DNA-dependent RNA polymerase.

C Aztreonam: Inhibits cell wall synthesis.

D Daptomycin: Causes depolarization of membranes.

RIGHT ANSWER

OK **A. Linezolid: Binds to 30S ribosome and inhibits protein synthesis.**

WHY THIS IS RIGHT

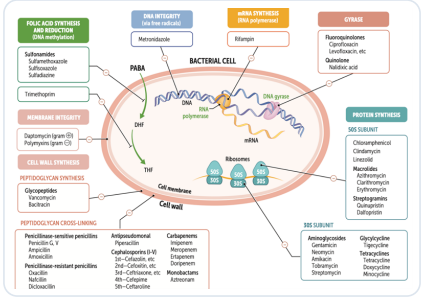
A) Linezolid: Binds to 30S

ribosome □ (FALSE)
• Linezolid is an oxazolidinone.
• It binds to the 50S ribosomal subunit, not 30S.
• Mechanism: prevents formation of initiation complex (70S).
• □□ Hence, statement is incorrect.

B) Rifampicin: Inhibits DNA-dependent RNA polymerase □
• Rifampicin blocks bacterial RNA synthesis by inhibiting DNA-dependent RNA polymerase.
• □□ Correct.

C) Aztreonam: Inhibits cell wall synthesis □
• Aztreonam is a monobactam (β-lactam).
• Acts on penicillin-binding proteins (PBPs) -> inhibits peptidoglycan cross-linking.
• □□ Correct.

D) Daptomycin: Causes membrane depolarization □
• Daptomycin inserts into bacterial membrane -> rapid depolarization -> cell death.
• □□ Correct.



QUESTION

5. Which of the following is a suicide inhibitor?
1. Nor-ethindrone
2. Ritonavir
3. Spironolactone
4. Benzbromarone

A 1, 2 and 3

B **1 and 3** CORRECT

C 2, 3 and 4

D 1, 2, 3 and 4

OK RIGHT ANSWER
B. 1 and 3

WHY THIS IS RIGHT

Enzyme + drug (looks like substrate)
↓
Drug gets activated inside enzyme
↓
Forms covalent bond
↓
Enzyme permanently inactivated
A suicide inhibitor irreversibly inactivates its target enzyme during metabolism. Norethindrone and spironolactone form reactive intermediates that permanently inhibit steroid-metabolizing CYP enzymes. Ritonavir is a potent but reversible CYP inhibitor, and benzbromarone acts mainly as a uricosuric drug. Hence only 1 and 3 are classic mechanism-based inhibitors.

QUESTION

A young male weighing 46 kg was treated with a drug. It has a Vd of 6 L/kg and clearance of 4.6 L/hour. What is the approximate half-life of this drug?

A 0.9 hours

B **40 hours** CORRECT

C 0.5 hours

D 20 hours

OK RIGHT ANSWER

B. 40 hours

WHY THIS IS RIGHT

Given:
• Vd = 6 L/kg
• Weight = 46 kg

Total Vd = 6 L/kg × 46 kg = 276 L.
Half-life t½ = 0.693 × Vd / Clearance = 0.693 × 276 / 4.6 ≈ 0.693 × 60 ≈ 41.6 hours.
Thus the approximate half-life is \~40 hours.
This uses the standard pharmacokinetic half-life equation.

Factor	Effect
Vd ↑	Drug stored in tissues -> longer half-life
Clearance ↓	Drug removed slowly -> longer half-life

$$t_{1/2} = \frac{0.693 \times V_d}{Cl}$$

QUESTION

All of the following statements are true regarding thiopentone except?

A It is highly lipid-soluble drug

B It is quickly concentrated in the brain after IV administration

C It shows the phenomenon of redistribution

D **It has a short duration of action due to rapid renal excretion** CORRECT

RIGHT ANSWER

OK

D. It has a short duration of action due to rapid renal excretion

WHY THIS IS RIGHT

Thiopentone (Thiopental)- An ultra-short-acting barbiturate IV anesthetic used for induction of anesthesia.

Thiopentone is highly lipid soluble and rapidly enters the brain after IV injection. Its brief anesthetic effect is due to redistribution from brain to muscle and fat, not elimination.
Metabolism occurs mainly in the liver, not rapid renal excretion. Hence the short duration is redistribution-dependent, making option D false.

QUESTION

Which of the following drugs is most teratogenic and strongly associated with neural tube defects (NTDs) when used during pregnancy?

A Lamotrigine

B Oxcarbazepine

C **Valproic acid** CORRECT

D Levetiracetam

OK RIGHT ANSWER

C. Valproic acid

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

Which pharmacokinetic parameters are used to determine the loading dose of an orally administered drug?
1. Volume of distribution (Vd)
2. Half-life
3. Plasma concentration
4. Bioavailability

A 1, 2 and 3

B Only 1

C **1, 3 and 4** CORRECT

D 1, 2, 3 and 4

OK RIGHT ANSWER
C. 1, 3 and 4

WHY THIS IS RIGHT

Loading dose = (Target plasma concentration × Volume of distribution) / Bioavailability.
Thus it depends on Vd, desired plasma concentration, and bioavailability for oral drugs.

Goal: Achieve target plasma concentration quickly
↓
Need to fill drug in body compartments
↓
Depends on:
Volume of distribution
Desired plasma concentration
Bioavailability (oral)
↓
Calculate loading dose
Half-life determines dosing interval and maintenance dose, not loading dose. Hence parameters 1, 3, and 4 are required.

$$\text{Loading Dose} = \frac{C_p \times V_d}{F}$$

QUESTION

Which of the following drug is associated with cholestasis without significant portal inflammation?

A Acetaminophen

B Amoxicillin-clavulanic acid

C Isoniazid

D **Oestrogen plus androgen** CORRECT

RIGHT ANSWER

OK

D. Oestrogen plus androgen

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

Match the following drugs with their respective Schedules:

Choose the option where the match for Morphine is correct

Drugs	Schedule Options
A. Hepatitis B vaccine	1. Schedule X
B. Veterinary drugs	2. Schedule H
C. Morphine	3. Schedule G
D. Insulin	4. Schedule Z

A A-3, B-1, C-2, D-4

B A-4, B-1, C-3, D-2

C A-2, B-1, C-4, D-3

D **A-3, B-4, C-1, D-2** CORRECT

OK RIGHT ANSWER

D. A-3, B-4, C-1, D-2

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1c” here and does not include a written explanation for this item.

QUESTION

A 30-year-old lady who is a known case of seizures was on carbamazepine. She was prescribed erythromycin for her recent URTI. A few days later, she developed ataxia. What is the most likely cause of her symptoms?

A **Carbamazepine toxicity** CORRECT

B Erythromycin toxicity

C Rebound epileptic episode

D Increased hepatic metabolism

RIGHT ANSWER

OK

A. Carbamazepine toxicity

WHY THIS IS RIGHT

Carbamazepine -> metabolized by CYP3A4 -> normal levels
Erythromycin inhibits CYP3A4, the main enzyme responsible for carbamazepine metabolism. This raises serum carbamazepine levels, causing toxicity with neurologic signs like ataxia and diplopia. The interaction is due to reduced hepatic clearance, not increased metabolism. Drug-drug inhibition leading to accumulation explains the symptoms.
B) Erythromycin toxicity
• Causes GI upset, QT prolongation
• NOT ataxia

C) Rebound epileptic episode
• Would cause seizures, not ataxia

D) Increased hepatic metabolism
• Opposite happened!
• Metabolism is inhibited, not increased

QUESTION

A 29-year-old male diagnosed with schizophrenia has been stable on risperidone for the past few months. Recently, he began experiencing dystonia, for which he was started on benztropine 2 mg twice daily by his psychiatrist. A week later, he presents to the emergency department with complaints of dry mouth, blurred vision, confusion and urinary retention. On examination, he is disoriented, tachycardic, and febrile. Pupils are dilated and bowel sounds are absent. Which of the following is the most appropriate next step in the management of this patient?

A Discontinue risperidone and initiate lorazepam for possible neuroleptic malignant syndrome (NMS)

B Administer intravenous dantrolene and initiate cooling measures for suspected serotonin syndrome

C **Start physostigmine with continuous cardiac monitoring for anticholinergic toxicity due to benztropine overdose** CORRECT

D Administer activated charcoal and IV fluids; observe the patient as symptoms are likely self-limiting

RIGHT ANSWER

OK **C. Start physostigmine with continuous cardiac monitoring for anticholinergic toxicity due to benztropine overdose**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1d” here and does not include a written explanation for this item.

QUESTION

What is the preferred pre-operative drug for the management of hypertension in a patient with pheochromocytoma?

A Telmisartan

B **Phenoxybenzamine** CORRECT

C Prazosin

D Amlodipine

OK RIGHT ANSWER

B. Phenoxybenzamine

WHY THIS IS RIGHT

Pheochromocytoma = Catecholamine storm
Tumor of adrenal medulla -> excess:
• Epinephrine
• Norepinephrine

Tumor -> ↑ catecholamines
↓
α1 stimulation -> vasoconstriction
↓
Severe hypertension
Pheochromocytoma requires preoperative alpha blockade to prevent hypertensive crises. Phenoxybenzamine is an irreversible nonselective α-blocker that provides sustained control of catecholamine effects. It is started before surgery and followed by β-blockade if needed. Long-acting alpha blockade is the key preparation step.

Drug	Type	Role in Pheochromocytoma
Phenoxybenzamine	Non-selective α-blocker	□ GOLD STANDARD
Prazosin	α1 selective	Alternative
Propranolol	β-blocker	Only after α-blockade
CCBs	Vasodilators	Adjunct

QUESTION

A 34-year-old woman with a history of allergic rhinitis has been taking cetirizine for symptom control. She presents with a complaint of excessive daytime sleepiness, which she attributes to her medication. Which of the following would be the best alternative in this patient?

A Diphenhydramine

B **Fexofenadine** CORRECT

C Promethazine

D Chlorpheniramine

OK RIGHT ANSWER

B. Fexofenadine

WHY THIS IS RIGHT

Drug	Feature
Diphenhydramine	Strong sedation
Promethazine	Very sedating
Chlorpheniramine	Moderate sedation

Allergy -> Histamine release
↓
H1 receptor activation
↓
Symptoms (sneezing, rhinorrhea)
↓
Fexofenadine blocks H1
↓
Relief WITHOUT sedation
Daytime sleepiness from cetirizine reflects CNS penetration of some second-generation antihistamines. Fexofenadine has minimal blood-brain barrier entry and is essentially non-sedating. First-generation agents like diphenhydramine, promethazine, and chlorpheniramine are more sedating. Hence fexofenadine is the preferred alternative.

Drug	Generation	Sedation	BBB Penetration
Diphenhydramine	1st	High	High
Promethazine	1st	Very high	High
Chlorpheniramine	1st	Moderate	High
Cetirizine	2nd	Mild	Low
Fexofenadine	2nd	Minimal	Very low

QUESTION

A 12-year-old boy is brought to the emergency department after accidental ingestion of tricyclic antidepressant medications. He presents with palpitations, dry mouth, mydriasis, and tachycardia. Which of the following is the most appropriate antidote in this scenario?

A Lithium

B **Physostigmine** CORRECT

C Naloxone

D Flumazenil

RIGHT ANSWER

OK **B. Physostigmine**

WHY THIS IS RIGHT

Tricyclic antidepressants (e.g., imipramine, amitriptyline)
They block multiple receptors:
• Muscarinic -> anticholinergic effects
• α1 -> hypotension
• H1 -> sedation

Tricyclic antidepressant overdose produces a strong anticholinergic toxidrome with mydriasis, tachycardia, and dry mouth. Physostigmine is a centrally acting acetylcholinesterase inhibitor that reverses anticholinergic toxicity. It crosses the blood-brain barrier and restores cholinergic transmission. Naloxone and flumazenil act on opioid and benzodiazepine receptors respectively.

Poison	Antidote
Opioids	Naloxone
Benzodiazepines	Flumazenil
Anticholinergic	Physostigmine
TCA cardiotoxicity	Sodium bicarbonate

QUESTION

A patient comes to the casualty with organophosphate poisoning. He was started on atropine infusion and pralidoxime. After 2 hours, the patient had a sudden rise in temperature. What is the likely cause of fever?

A **Atropine toxicity** CORRECT

B Side effect of pralidoxime

C Due to organophosphate poisoning

D Idiopathic

RIGHT ANSWER

OK **A. Atropine toxicity**

WHY THIS IS RIGHT

Organophosphate (OP) poisoning
• Inhibits acetylcholinesterase
• -> ↑ Acetylcholine
• -> Cholinergic excess

Drug	Role
Atropine	Blocks muscarinic effects
Pralidoxime	Reactivates AChE

High-dose atropine can produce anticholinergic toxicity, causing hyperthermia from impaired sweating. This results in hot, dry skin and sudden temperature rise.
Atropine
↓
Blocks sweating (M3 receptors)
↓
No heat loss via evaporation
↓
Heat accumulation
↓
Hyperthermia (fever)
Pralidoxime does not typically cause fever. The hyperthermia reflects atropine overdose during organophosphate management.

Feature	OP Poisoning	Atropine Toxicity
Sweating	↑	Absent
Temperature	Normal/low	↑ (fever)
Secretions	↑	↓
Pupils	Constricted	Dilated

QUESTION

A chronic smoker was on nicotine replacement therapy and clonidine tablets for smoking de-addiction. He stopped taking clonidine tablets and now presents with a headache. What is the reason behind this condition?

A Postural hypotension

B Receptor upregulation

C **Rebound hypertension** CORRECT

D Receptor hypersensitivity

RIGHT ANSWER

OK

C. Rebound hypertension

WHY THIS IS RIGHT

Mechanism of Clonidine
• α□ receptor agonist (central action)
• Acts in brainstem -> ↓ sympathetic outflow

Clonidine is an α2-agonist that suppresses sympathetic outflow. Abrupt withdrawal causes rebound sympathetic overactivity, leading to severe hypertension and headache.
Clonidine
↓
Stimulates α2 receptors (presynaptic)
↓
↓ Norepinephrine release
↓
↓ Sympathetic tone
↓
↓ Blood pressure
Chronic use causes receptor adaptation, so sudden stoppage unmasks excess catecholamine release (What happens when you STOP clonidine suddenly? The system “snaps back” like a stretched rubber band)
This classic withdrawal effect is termed rebound hypertension.

QUESTION

Which of the following is the most appropriate treatment for an overactive bladder in a patient with dementia?

A **Tolterodine** CORRECT

B Tiotropium

C Telenzepine

D Tropicamide

RIGHT ANSWER

OK **A. Tolterodine**

WHY THIS IS RIGHT

Parasympathetic (ACh)
↓
M3 receptor activation
↓
Detrusor contraction
↓
Urination
Overactive bladder is treated with antimuscarinic agents that reduce detrusor overactivity. Tolterodine has relative bladder selectivity and less CNS penetration, making it safer in dementia. Tiotropium is for COPD, tropicamide is ophthalmic, and telenzepine is gastric selective. Tolterodine provides urinary control with fewer cognitive effects.
B) Tiotropium
• Used in:
• COPD

Acts on bronchial smooth muscle

C) Telenzepine
• M1 selective
• Used for:
• Gastric acid secretion

No role in bladder

D) Tropicamide
• Used in:
• Eye (mydriasis)

No urinary role

Drug	Feature
Tolterodine	□ Preferred
Oxybutynin	More CNS effects □
Darifenacin	M3 selective □
Solifenacin	M3 selective □

QUESTION

A patient is found on a railway track with hot dry skin, raised temperature, dilated pupils, slurred speech, and a staggering gait. Which of the following is the most likely cause?

A **Dhatura poisoning** — CORRECT

B Alcohol intoxication

C Opioid overdose

D Cocaine poisoning

RIGHT ANSWER

OK **A. Dhatura poisoning**

WHY THIS IS RIGHT

Dhatura (Datura stramonium)
A plant packed with anticholinergic alkaloids:
• Atropine
• Scopolamine
• Hyoscyamine

Hot dry skin, hyperthermia, mydriasis, slurred speech, and ataxia indicate an anticholinergic toxidrome. Dhatura contains atropine-like alkaloids causing central and peripheral antimuscarinic effects. Classic features are “hot as a hare, dry as a bone, blind as a bat.”
Opioids cause miosis and respiratory depression, not dryness or hyperthermia.

Feature	Anticholinergic	Opioid	Cocaine
Skin	Dry	Normal	Sweaty
Pupils	Dilated	Pinpoint	Dilated
Temp	↑	↓/normal	↑
CNS	Delirium	Depressed	Agitated

QUESTION

Which of the following neurotransmitters is released from SA node in response to increase in blood pressure?

A **Acetylcholine** CORRECT

B Dopamine

C Epinephrine

D Nor Epinephrine

OK RIGHT ANSWER

A. Acetylcholine

WHY THIS IS RIGHT

When BP rises, the body tries to slow the heart down
An increase in blood pressure activates the baroreceptor reflex, increasing vagal output to the heart. Vagal stimulation releases acetylcholine at the SA node, slowing heart rate.
↑ Blood pressure
↓
Baroreceptors activated (carotid sinus, aortic arch)
↓
Signals to medulla
↓
↑ Parasympathetic (vagal) output
↓
Acetylcholine released at SA node
↓
↓ Heart rate (bradycardia)
This parasympathetic response reduces cardiac output and helps normalize blood pressure. The SA node is primarily controlled by cholinergic vagal input in this reflex.

System	Neurotransmitter	Effect on Heart
Parasympathetic	Acetylcholine	↓ HR
Sympathetic	Norepinephrine	↑ HR

QUESTION

Which of the following statements regarding the causes of fall in diastolic blood pressure is correct?
1. Loss of sympathetic outflow in the lumbar region
2. Antagonism of beta-1 adrenergic receptors
3. Blockade of alpha-1 adrenergic receptors
4. Antagonism of beta-2 adrenergic receptors

A 1, 2 and 3

B **1 and 3** CORRECT

C 1, 2, 3 and 4

D 3 and 4

OK RIGHT ANSWER

B. 1 and 3

WHY THIS IS RIGHT

Diastolic BP is mainly governed by Systemic Vascular Resistance (SVR)
Not cardiac output.
Diastolic pressure mainly reflects peripheral vascular resistance. Loss of lumbar sympathetic outflow and alpha-1 blockade cause vasodilation and reduced resistance, lowering diastolic BP. Beta-1 antagonism primarily reduces heart rate and contractility, affecting systolic more than diastolic pressure. Beta-2 blockade causes vasoconstriction, which would increase diastolic pressure, not decrease it.

Receptor	Action	Effect on DBP
α1	Vasoconstriction	↑ DBP
β1	↑ CO	↑ SBP mainly
β2	Vasodilation	↓ DBP
β2 blockade	Vasoconstriction	↑ DBP

QUESTION

What is the main role of Phase 1 in xenobiotic metabolism?

A To make the compound more permeable across membranes

B To extract functional proteins through oxidase/reductase reactions

C **To introduce or expose functional groups for further metabolism** CORRECT

D To directly conjugate the compound and facilitate excretion

RIGHT ANSWER

OK

C. To introduce or expose functional groups for further metabolism

WHY THIS IS RIGHT

Phase I reactions (oxidation, reduction, hydrolysis) introduce or expose polar functional groups on xenobiotics. These reactions are mainly mediated by CYP450 enzymes. The purpose is to prepare the molecule for Phase II conjugation. Thus, Phase I increases chemical reactivity for further metabolism.

Reaction	Example
Oxidation	CYP450 mediated
Reduction	Nitro -> amine
Hydrolysis	Ester breakdown

Feature	Phase I	Phase II
Function	Functionalization	Conjugation
Reaction	Oxidation, reduction	Glucuronidation, sulfation
Enzymes	CYP450	Transferases
Result	Slightly polar	Highly water-soluble

QUESTION

A 58-year-old man presents with septic shock. He is febrile, tachycardic, and has a blood pressure of 80/50 mmHg despite adequate fluid resuscitation with IV crystalloids. His urine output is low, and he is drowsy. What is the next best step in management?

A Administer dopamine

B **Administer norepinephrine** CORRECT

C Administer hydrocortisone

D Administer vasopressin

OK RIGHT ANSWER

B. Administer norepinephrine

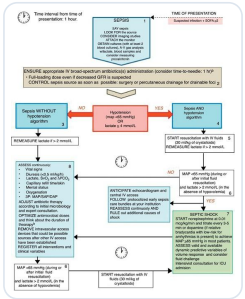
WHY THIS IS RIGHT

This patient has:
• Persistent hypotension (80/50 mmHg) despite adequate fluids
• Organ dysfunction -> low urine output, altered sensorium

This fulfills criteria for septic shock

Next best step:
• Start vasopressor therapy immediately
• First-line vasopressor = Norepinephrine
• Strong α□ agonist -> vasoconstriction -> ↑ SVR -> ↑ BP
• Mild β□ effect -> maintains cardiac output
• Best survival benefit + lowest arrhythmia risk

Other options:
• Hydrocortisone -> used *only if refractory shock* (after vasopressors)
• Vasopressin -> *add-on*, not first-line



QUESTION

Which of the following statements is true regarding the pharmacological relationship between imipramine and diphenhydramine?

A Imipramine inhibits diphenhydramine's action

B Diphenhydramine inhibits imipramine's action

C **Both block muscarinic receptors** CORRECT

D Both are norepinephrine reuptake inhibitors

OK RIGHT ANSWER

C. Both block muscarinic receptors

WHY THIS IS RIGHT

• Imipramine -> Tricyclic antidepressant (TCA)
• Diphenhydramine -> 1st generation antihistamine

Different primary roles, but both carry a strong anticholinergic personality
Imipramine (a TCA) and diphenhydramine (a first-generation antihistamine) both have strong antimuscarinic activity. This shared action explains common side effects like dry mouth, blurred vision, and urinary retention.

Feature	Imipramine	Diphenhydramine
Class	TCA	1st gen antihistamine
Main action	NE/5-HT reuptake inhibition	H1 blockade
Muscarinic block	□ Yes	□ Yes
Sedation	Moderate	Strong
Anticholinergic effects	□ Prominent	□ Prominent

Diphenhydramine is not a norepinephrine reuptake inhibitor, and neither drug blocks the other’s action. Their pharmacologic overlap is anticholinergic blockade.

QUESTION

A 62-year-old male with a history of coronary heart disease (CHD) and renal failure was admitted to the ICU with elevated blood pressure (BP 190/110 mmHg) and declining urine output. The intensivist administers fenoldopam intravenously. How does this drug help in improving renal function in this patient?

A **D1 agonism** CORRECT

B D2 antagonism

C Alpha antagonism

D Muscarinic antagonism

RIGHT ANSWER

OK

A. D1 agonism

WHY THIS IS RIGHT

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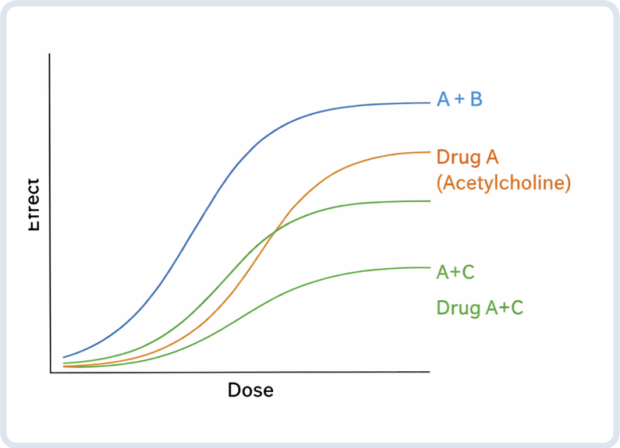
QUESTION

In the given graph of effect vs dose, the orange line represents drug A which is acetylcholine. Identify drug B.



A **Physostigmine** CORRECT

B Bethanechol

C Atropine

D Adrenaline

RIGHT ANSWER

OK **A. Physostigmine**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1f” here and does not include a written explanation for this item.

QUESTION

Which of the following statements regarding Dronabinol are true?
1. It is used in AIDS-related weight loss
2. It acts on the CB1 receptor
3. It is used in chronic pain management
4. It is excreted unchanged in faeces

A **1 and 2** CORRECT

B 1, 2 and 3

C 2, 3 and 4

D 1, 2 and 4

OK RIGHT ANSWER

A. 1 and 2

WHY THIS IS RIGHT

Target: CB1 receptors (primarily CNS)
Action: Inhibits neurotransmitter release (via ↓ cAMP, ↓ Ca²□ influx)
Modulates:
• Appetite
• Nausea
• Pain perception (limited clinical role)

Dronabinol is a synthetic THC used for AIDS-related anorexia/cachexia and chemotherapy-induced nausea. It acts mainly via central CB1 cannabinoid receptors to stimulate appetite. It is not a standard drug for chronic pain management. The drug undergoes hepatic metabolism rather than being excreted unchanged in feces.

Feature	CB1	CB2
Location	CNS (brain)	Immune cells
Function	Appetite, mood, nausea	Immune modulation
Target of Dronabinol	□ Yes	□ No

QUESTION

A 55-year-old male undergoing chemotherapy presents with progressive shortness of breath, dry cough, and fatigue over the past 2 weeks. He is afebrile and denies chest pain or sputum production. On examination, fine basal crepitations are heard. Chest X-ray reveals bilateral reticulonodular infiltrates. High-resolution CT (HRCT) of the chest confirms interstitial lung disease. Pulmonary function tests show a restrictive pattern with FEV1 = 61% of predicted and normal FEV1/FVC ratio. Notably, there is no evidence of bone marrow suppression. Which of the following drugs is most likely responsible for his condition?

A Cisplatin

B **Bleomycin** CORRECT

C Cyclophosphamide

D Vincristine

OK RIGHT ANSWER

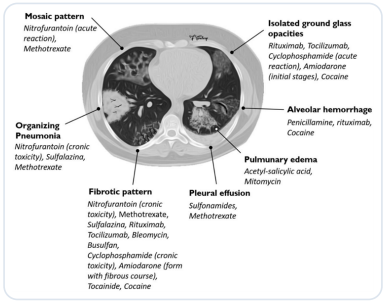
B. Bleomycin

WHY THIS IS RIGHT

Bleomycin causes dose-dependent pulmonary fibrosis presenting with dry cough, dyspnea, and restrictive lung disease. HRCT showing interstitial infiltrates with preserved marrow function is characteristic.

Bleomycin
↓
Free radical formation
↓
Lung tissue damage
↓
Interstitial inflammation
↓
Fibrosis -> restrictive lung disease
Unlike many chemotherapeutics, bleomycin has minimal bone marrow suppression. Pulmonary toxicity is its hallmark adverse effect.

Drug	Toxicity
Bleomycin	Pulmonary fibrosis
Cisplatin	Nephrotoxicity, ototoxicity
Cyclophosphamide	Hemorrhagic cystitis
Vincristine	Peripheral neuropathy



QUESTION

Methotrexate is a chemotherapeutic agent used in the treatment of breast cancer. It works by inhibiting the synthesis of which nucleotide?

A AMP

B CMP

C UMP

D **TMP** CORRECT

OK RIGHT ANSWER

D. TMP

WHY THIS IS RIGHT

A classic antimetabolite that sabotages DNA synthesis right at its biochemical roots.

Methotrexate inhibits dihydrofolate reductase, depleting tetrahydrofolate needed for thymidylate synthesis. This blocks conversion of dUMP to dTMP, impairing DNA replication. Rapidly dividing cancer cells are most affected. Inhibition of thymidine synthesis is the key cytotoxic mechanism.

QUESTION

Which drug is used to prevent cardiotoxicity induced by daunorubicin?

A Aprepitant

B **Dexrazoxane** CORRECT

C Amifostine

D Dexamethasone

RIGHT ANSWER

OK **B. Dexrazoxane**

WHY THIS IS RIGHT

Anthracycline (daunorubicin)
↓
Binds iron -> forms free radicals
↓
Oxidative damage
↓
Myocardial cell injury
↓
Dilated cardiomyopathy
Daunorubicin causes dose-dependent anthracycline cardiotoxicity via free radical-mediated myocardial injury. Dexrazoxane is an iron-chelating agent that reduces reactive oxygen species formation in cardiac tissue. It is used prophylactically to protect against anthracycline-induced cardiomyopathy. Cardiac protection is its key clinical role.

Toxicity	Drug
Anthracycline cardiotoxicity	Dexrazoxane
Cisplatin nephrotoxicity	Amifostine
Hemorrhagic cystitis (cyclophosphamide)	Mesna
Methotrexate toxicity	Leucovorin

QUESTION

All of the following drugs act as HER-1 inhibitors except

A Cetuximab

B Necitumumab

C **Pertuzumab** CORRECT

D Panitumumab

RIGHT ANSWER

OK

C. Pertuzumab

WHY THIS IS RIGHT

Drug	Target	Key Use
Cetuximab	EGFR (HER-1)	Colorectal cancer, head & neck cancer
Necitumumab	EGFR (HER-1)	Metastatic squamous NSCLC
Pertuzumab	HER-2	HER-2 positive breast cancer
Panitumumab	EGFR (HER-1)	Metastatic colorectal cancer

QUESTION

A 52-year-old female presents with metastatic breast cancer. She has previously undergone treatment with surgery, radiation, and multiple lines of chemotherapy, but her disease has progressed. Genetic testing reveals a BRCA1 mutation, indicating an impaired DNA repair mechanism. Her cancer is classified as HER2-negative and hormone receptor-positive. Given the genetic findings, her oncologist considers using a therapy that targets the PARP enzyme to exploit the DNA repair vulnerability of her cancer cells. Which of the following drugs would be appropriate to prescribe as a PARP inhibitor in this patient?

A Palbociclib

B **Rucaparib** CORRECT

C Vorinostat

D Ustekinumab

RIGHT ANSWER

OK

B. Rucaparib

WHY THIS IS RIGHT

BRCA mutation = defective DNA repair system
Normal cells repair DNA via 2 major pathways:
• BRCA (Homologous recombination) -> repairs double-strand breaks
• PARP pathway -> repairs single-strand breaks

BRCA-mutated tumors have defective homologous recombination repair and are sensitive to PARP inhibition via synthetic lethality. Rucaparib is a PARP inhibitor that blocks DNA repair, causing tumor cell death. Palbociclib is a CDK4/6 inhibitor, vorinostat is an HDAC inhibitor, and ustekinumab is an immunologic agent. Targeting PARP exploits the DNA repair defect in BRCA cancers.

Drug	Target	Use
Rucaparib	PARP	BRCA cancers
Palbociclib	CDK4/6	Breast cancer
Trastuzumab	HER2	HER2+ cancer
Imatinib	BCR-ABL	CML

QUESTION

Which of the following statements regarding thalidomide is false?

A **It acts as an antimetabolite** — CORRECT

B It is a teratogenic drug

C It is used in type 2 lepra reaction (ENL)

D It is used in relapsing multiple myeloma

RIGHT ANSWER

OK **A. It acts as an antimetabolite**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$20” here and does not include a written explanation for this item.

QUESTION

Which of the following drugs acts on VEGF (Vascular Endothelial Growth Factor)?

A Pembrolizumab

B Ipilimumab

C **Ramucirumab** CORRECT

D Dostarlimab

RIGHT ANSWER

OK

C. Ramucirumab

WHY THIS IS RIGHT

Tumors are greedy, to survive, they stimulate new blood vessel formation (angiogenesis) using: VEGF (Vascular Endothelial Growth Factor)

Ramucirumab is a monoclonal antibody against VEGF receptor-2, blocking VEGF-mediated angiogenesis. It inhibits tumor blood vessel formation and growth. Pembrolizumab, ipilimumab, and dostarlimab are immune checkpoint inhibitors targeting PD-1 or CTLA-4, not VEGF.

Option	Drug	Target	Why Wrong
A	Pembrolizumab	PD-1	Immune checkpoint inhibitor
B	Ipilimumab	CTLA-4	T-cell activation ↑
D	Dostarlimab	PD-1	Used in MSI-high tumors

Anti-angiogenic action is the key distinguishing feature of ramucirumab.