NEET PG Pharmacology Sample Paper-6

Duration: 15 Minutes **Maximum Marks: 80**

Instructions

- This paper contains **20** Multiple Choice Questions.
- Each correct answer carries **+4** mark. Incorrect answer: **−1** marks. Only **one** correct option.
- Unattempted questions carry **0** marks.
- Use of mobile phones, smartwatches, or any electronic gadgets is strictly prohibited.

Q1. A 29-year-old male with an open fracture of the femur following a motor vehicle accident is scheduled for emergency orthopedic surgery. He has a known history of severe pseudocholinesterase deficiency. Which of the following neuromuscular blocking agents would undergo completely normal metabolic elimination and be safest to use for rapid sequence intubation in this patient?

(A) Succinylcholine

(B) Mivacurium

(C) Rocuronium

(D) Cisatracurium

Q2. A 58-year-old patient diagnosed with treatment-resistant schizophrenia is started on clozapine therapy. To minimize the risk of severe drug-induced agranulocytosis, regular monitoring of the absolute neutrophil count (ANC) is mandatory. Which of the following cytochrome P450 enzymes is primarily responsible for the metabolic clearance of clozapine, and would pose a high risk of toxicity if inhibited by a co-administered drug?

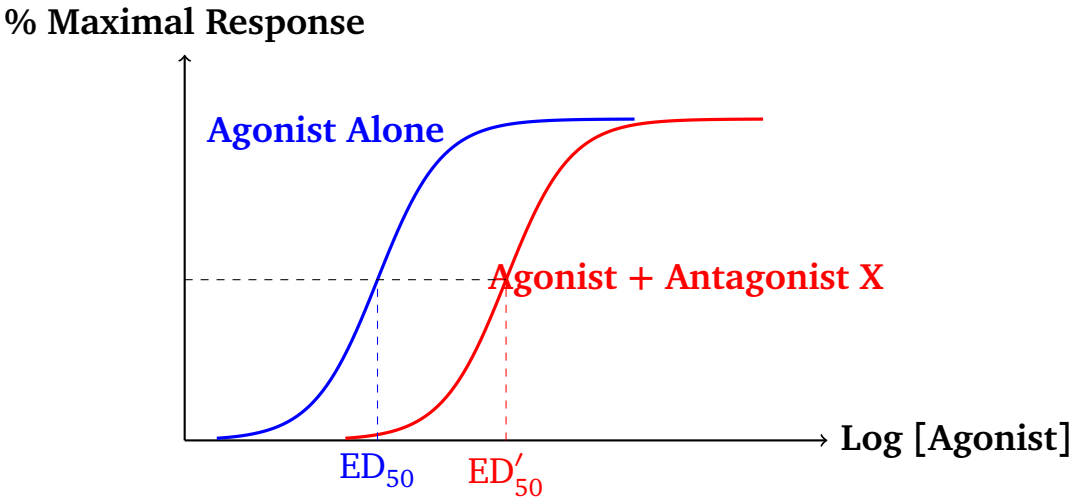
(A) CYP2D6

(B) CYP1A2

(C) CYP2C9

(D) CYP2E1

Q3. During a clinical study investigating a novel competitive antagonist (X) against an established full agonist (Y), researchers analyze the alterations in the log dose-response curve. The dynamic changes in receptor occupancy and shifting efficacy can be modeled by the structural distribution shown below:



Based on this pharmacological profile, what happens to the apparent affinity (K_m) and maximal efficacy (V_{max}) of the agonist Y in the presence of antagonist X?

(A) K_m increases, V_{max} decreases

(B) K_m remains unchanged, V_{max} decreases

(C) K_m increases, V_{max} remains unchanged

(D) K_m decreases, V_{max} increases

Q4. A 64-year-old female with a history of chronic plaque psoriasis is treated with an oral systemic medication. She presents to the emergency room with significant oral ulcerations, severe leukopenia, and a macrocytic anemia. Suspecting acute toxicity due to an inhibition of dihydrofolate reductase, the resident orders an immediate antidote. Which agent should be administered to bypass the blocked metabolic step directly?

(A) Folic acid

(B) Leucovorin (Folinic acid)

(C) Cyanocobalamin

(D) Pyridoxine

Q5. An 8-year-old boy is brought to the pediatric emergency care unit after accidentally ingesting an unknown insecticide from his grandfather's farm shed. He exhibits severe miosis, generalized muscle fasciculations, bradycardia, hypersalivation, and wet lung sounds on auscultation. A clinical decision is made to administer a drug that can reactivate the phosphorylated acetylcholinesterase enzyme before aging occurs. What is the molecular target site of this specific reactivator?

(A) Anionic site of acetylcholinesterase

(B) Esteratic site of acetylcholinesterase

(C) Allosteric peripheral anionic site

(D) Presynaptic choline transporter (ChT)

Q6. A 52-year-old post-menopausal woman with severe osteoporosis is being evaluated for pharmacological management. The physician wants to prescribe a selective estrogen receptor modulator (SERM) that effectively increases bone mineral density and lowers the risk of invasive breast cancer, without carrying an increased risk of inducing endometrial hyperplasia or carcinoma. Which of the following agents matches this profile?

(A) Tamoxifen

(B) Raloxifene

(C) Clomiphene

(D) Toremifene

Q7. A 34-year-old female presents with persistent high-grade fever, productive cough, and rust-colored sputum. Sputum culture confirms infection with

penicillin-resistant *Streptococcus pneumoniae*. The minimum inhibitory concentration (MIC) values indicate susceptibility to late-generation cephalosporins. Which of the following alterations in the bacterial cell wall synthesis machinery is the primary mechanism driving resistance to penicillin in this strain?

(A) Production of plasmid-mediated TEM-1 beta-lactamase

(B) Structural modification of Penicillin-Binding Proteins (PBPs)

(C) Downregulation of OmpF porin channels

(D) Active efflux via a proton-motive force pump

Q8. A 28-year-old pregnant woman in her third trimester requires active management for an acute deep vein thrombosis (DVT) in her left lower extremity. Which of the following anticoagulants is the safest choice for this patient because it does not cross the placental barrier and avoids embryopathy?

(A) Warfarin

(B) Enoxaparin

(C) Rivaroxaban

(D) Dabigatran

Q9. A 33-year-old male with a history of major depressive disorder is brought to the urgent care clinic presenting with hyperthermia, autonomic instability (tachycardia, diaphoresis, labile blood pressure), severe hyperreflexia, and spontaneous ocular clonus. His current medication regimen includes phenelzine, and he recently started an over-the-counter medication for a severe dry cough. Which cough suppressant is the most likely trigger for this life-threatening drug-drug interaction?

(A) Dextromethorphan

(B) Codeine

(C) Benzonatate

(D) Guaifenesin

Q10. A 50-year-old male presents with acute, excruciating pain, redness, and swelling in his right first metatarsophalangeal joint. A diagnosis of acute gouty arthritis is established. The patient has a background history of chronic kidney disease (Stage 3b). Which of the following first-line agents for acute gout should be completely avoided or heavily dose-restricted due to its high risk of systemic accumulation and serious bone marrow toxicity in renal impairment?

(A) Indomethacin

(B) Colchicine

(C) Naproxen

(D) Methylprednisolone

Q11. A 42-year-old patient with human immunodeficiency virus (HIV) infection is found to have a low CD4+ T-cell count and a high viral load despite being on a standard antiretroviral regimen. Genotypic sequencing reveals mutations conferring high-level resistance to nucleoside reverse transcriptase inhibitors (NRTIs). The physician decides to switch the patient to a regimen containing a viral integrase strand transfer inhibitor (INSTI). Which of the following drugs belongs to this therapeutic class?

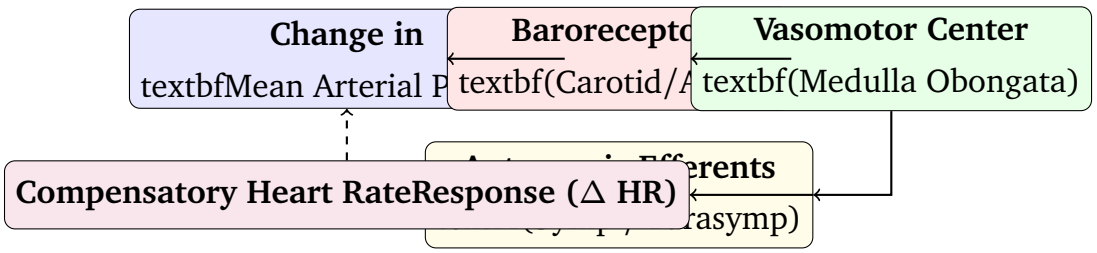
(A) Maraviroc

(B) Dolutegravir

(C) Etravirine

(D) Atazanavir

Q12. A pharmacology research group investigates the autonomic feedback pathways regulating heart rate. They map out the systemic baroreceptor reflex loop using a neural flow framework to analyze the compensatory bradycardia or tachycardia responses following intravenous drug infusions:



During an experimental trial, an intravenous bolus of a selective α_1-adrenergic receptor agonist is administered. Which of the following options correctly describes the primary direct vascular effect and the subsequent reflex autonomic adjustment observed in this loop?

(A) Vasodilation and reflex tachycardia via increased sympathetic tone

(B) Vasoconstriction and reflex bradycardia via increased vagal tone

(C) Vasoconstriction and reflex tachycardia via sympathetic activation

(D) Vasodilation and reflex bradycardia via vagal withdrawl

Q13. A 67-year-old male with a history of long-standing hypertension and Type 2 diabetes mellitus presents with a dry, hacking, non-productive cough that has persisted for three weeks. He denies any fever, rhinorrhea, or history of asthma. His current medication list includes metformin, atorvastatin, amlodipine, and enalapril. What endogenous substance is accumulating in the respiratory tract to cause this specific adverse drug reaction?

(A) Angiotensin II

(B) Bradykinin

(C) Aldosterone

(D) Renin

Q14. A neonate born at 31 weeks of gestation develops severe respiratory distress syndrome due to a lack of endogenous surfactant. The infant is intubated, and intratracheal surfactant replacement therapy is initiated. Which of the following compounds is the principal phospholipid component of natural pulmonary surfactant responsible for reducing alveolar surface tension?

(A) Dipalmitoylphosphatidylcholine

(B) Phosphatidylglycerol

(C) Sphingomyelin

(D) Phosphatidylinositol

Q15. A 58-year-old male undergoing intensive chemotherapy for acute myeloid leukemia develops a high-grade fever (38.9°C) accompanied by profound neutropenia (ANC $= 250/\mu$L). Intravenous empiric antimicrobial therapy must be initiated immediately to cover pseudomonal infections. Which of the following antipseudomonal beta-lactam antibiotics is structurally a monobactam and can be safely given even if the patient has a confirmed history of anaphylaxis to penicillin?

(A) Piperacillin

(B) Ceftazidime

(C) Aztreonam

(D) Meropenem

Q16. A 24-year-old graduate student presents with excessive daytime sleepiness that significantly interferes with her academic performance. She describes episodes where she suddenly loses muscle tone in her jaw and knees when laughing heartily. Polysomnography confirms a diagnosis of narcolepsy. Which of the following wakefulness-promoting agents, which acts primarily as a selective dopamine reuptake inhibitor with low abuse liability, is considered a first-line option?

(A) Methylphenidate

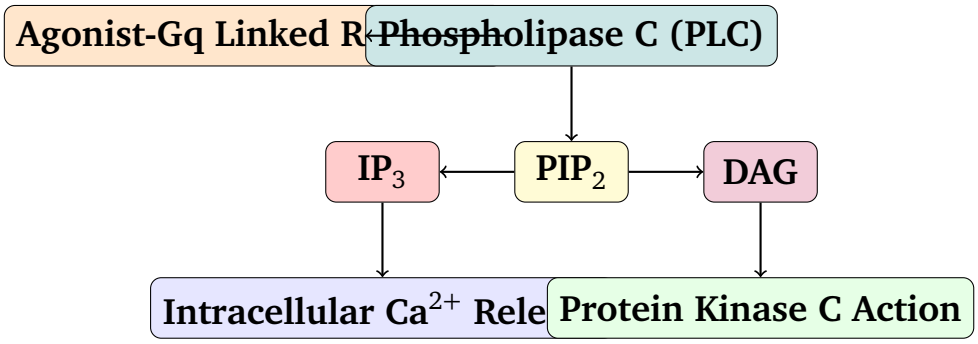
(B) Modafinil

(C) Dextroamphetamine

(D) Diazepam

Q17. A pharmaceutical researcher is evaluating the receptor binding profile and downstream second messenger pathways of a newly synthesized drug designed to treat refractory glaucoma. The drug works by lowering intraocular

pressure via targeted dynamic interactions within the ciliary muscle epithelium. The signaling cascade is mapped as follows:



Which of the following endogenous autonomic receptors naturally utilizes the exact intracellular signaling configuration depicted above?

(A) α_2-Adrenergic receptor

(B) β_1-Adrenergic receptor

(C) M_2 Muscarinic receptor

(D) M_3 Muscarinic receptor

Q18. A 39-year-old male traveler presents with a 3-day history of high fever, chills, severe headache, and cyclic diaphoresis. A peripheral blood smear reveals ring-form trophozoites and schizonts of *Plasmodium vivax*. He is treated successfully with a course of chloroquine. To prevent a relapse of malaria caused by the activation of dormant hypnozoites in the liver, which of the following drugs must also be prescribed, provided the patient is screened and found negative for G6PD deficiency?

(A) Quinine

(B) Artesunate

(C) Primaquine

(D) Lumefantrine

Q19. A 72-year-old male with a history of severe chronic obstructive pulmonary disease (COPD) and chronic atrial fibrillation is admitted with worsening dyspnea and productive cough. He is started on intravenous aminophylline and an empiric course of oral ciprofloxacin for a suspected lower respiratory

tract infection. Within 48 hours, the patient develops severe agitation, persistent nausea, and runs of non-sustained ventricular tachycardia. His serum theophylline level is found to be toxic ($32\,\mu g/mL$). What is the mechanism underlying this clinical event?

(A) Displacement of aminophylline from plasma protein binding sites by ciprofloxacin

(B) Pharmacodynamic antagonism at the adenosine receptor level

(C) Inhibition of hepatic CYP1A2-mediated metabolism of theophylline by ciprofloxacin

(D) Induction of renal excretion pathways by aminophylline

Q20. A 23-year-old female presents to the outpatient clinic with a 2-week history of heat intolerance, palpitations, weight loss despite an increased appetite, and a diffuse, non-tender enlargement of her thyroid gland. Laboratory evaluation confirms primary hyperthyroidism (Graves' disease). The physician decides to initiate antithyroid pharmacotherapy. Which of the following agents is preferred in this patient because it has a lower risk of severe hepatotoxicity compared to propylthiouracil, assuming she is not in her first trimester of pregnancy?

(A) Methimazole

(B) Potassium iodide

(C) Radioactive Iodine-131

(D) Propranolol

Detailed Solutions

Q1.

Solution

Concept:

Pseudocholinesterase deficiency is an inherited or acquired condition that impairs the body's ability to metabolize esters. Drugs that rely on plasma cholinesterase for degradation will cause prolonged neuromuscular blockade. Safe management requires alternative agents metabolized via independent pathways.

Solution:

(a) Succinylcholine and mivacurium are rapidly broken down by plasma pseudocholinesterase. In patients with a deficiency, their clearance is profoundly delayed, causing prolonged, life-threatening paralysis and apnea.

(b) Rocuronium is an amino-steroid non-depolarizing agent that undergoes hepatic elimination and renal excretion. It does not depend on pseudocholinesterase for clearance, making its metabolic pathway completely normal in this patient.

(c) Cisatracurium undergoes spontaneous degradative breakdown in plasma via Hofmann elimination and ester hydrolysis, which is also independent of pseudocholinesterase.

(d) While both rocuronium and cisatracurium are safe from a metabolic standpoint, rocuronium is clinically preferred and universally indicated for rapid sequence intubation due to its rapid onset of action, matching the speed needed for emergency trauma surgeries.

Final Answer: Rocuronium

Answer: (C)

Go Back to Question 1

Q2.

Solution

Concept:

Clozapine is an atypical antipsychotic highly effective for treatment-resistant schizophrenia. Its clinical use is strictly monitored due to severe risks like agranulocytosis. It undergoes extensive hepatic biotransformation, primarily via specific cytochrome P450 pathways, where drug interactions can significantly impact systemic toxicity.

Solution:

(a) The primary enzyme responsible for the major metabolic clearance of clozapine is Cytochrome P450 1A2 (CYP1A2), converting it into its metabolites.

(b) Concomitant administration of drugs that inhibit CYP1A2, such as ciprofloxacin or fluvoxamine, dramatically decreases clozapine clearance, causing elevated plasma concentrations and toxic side effects.

(c) Conversely, inducers of CYP1A2 like tobacco smoke accelerate its metabolism, reducing clinical efficacy.

(d) Other enzymes like CYP2D6, CYP2C9, and CYP2E1 play minor or negligible roles in clozapine's primary clearance cascade. Therefore, understanding the dominant role of CYP1A2 is critical for preventing drug-induced toxicities and managing life-threatening adverse profiles like agranulocytosis.

Final Answer: CYP1A2

Answer: (B)

Go Back to Question 2

Q3.

Solution

Concept:

Receptor pharmacology analyzes how drugs alter dose-response relationships. Competitive antagonists interact reversibly at the same orthosteric binding site as full agonists. Their effects can be overcome by increasing the concentration of the agonist, characteristically shifting the potency metrics without reducing overall max tissue responses.

Solution:

(a) A competitive antagonist binds reversibly to the active site. Increasing the agonist concentration displaces the antagonist, allowing the system to achieve maximum biological effect.

(b) Consequently, the maximal efficacy (V_{max}) of the agonist remains completely unchanged, as evidenced by the parallel curve reaching the original plateau.

(c) However, because higher agonist concentrations are required to achieve the same given fractional response, the log dose-response curve shifts parallelly to the right.

(d) This rightward shift indicates that the effective dose required to produce 50% of the maximal response (ED_{50}) increases, which directly translates to a decrease in the apparent affinity (K_m) of the agonist for the target receptor. Thus, K_m increases while V_{max} is preserved.

Final Answer: K_m increases, V_{max} remains unchanged

Answer: (C)

Go Back to Question 3

Q4.

Solution

Concept:

Methotrexate is a systemic antifolate medication used for severe plaque psoriasis. It competitively inhibits dihydrofolate reductase (DHFR), blocking the conversion of dihydrofolate to tetrahydrofolate. Toxicity manifests as severe mucosal ulcerations, myelosuppression, and macrocytic anemia due to impaired nucleotide synthesis.

Solution:

(a) Methotrexate toxicity causes a functional deficiency of reduced folate types essential for purine and thymidylate synthesis.

(b) Standard folic acid administration is ineffective because it requires active reduction by the inhibited DHFR enzyme to become biochemically functional.

(c) Leucovorin, also known as folinic acid, is a reduced form of folate (5-formyltetrahydrofolate) that does not require reduction by DHFR. It directly replenishes the intracellular reduced folate pool, effectively bypassing the metabolic block.

(d) Cyanocobalamin (Vitamin B12) and pyridoxine (Vitamin B6) are involved in separate biochemical pathways and cannot resolve the acute purine synthesis arrest caused by DHFR inhibition, leaving leucovorin rescue as the gold-standard antidote.

Final Answer: Leucovorin (Folinic acid)

Answer: (B)

Go Back to Question 4

Q5.

Solution

Concept:

Organophosphate insecticides inhibit acetylcholinesterase (AChE) by phosphorylating its active core, causing a toxic accumulation of acetylcholine. This presents with severe toxidromes including miosis, fasciculations, and bradycardia. Treatment requires oxime reactivators that break the covalent organophosphate-enzyme bond before permanent aging occurs.

Solution:

(a) The active center of acetylcholinesterase features two principal binding locations: an anionic site that anchors the quaternary ammonium of acetylcholine and an esteratic site containing a serine residue responsible for ester hydrolysis.

(b) Organophosphates exert toxicity by transferring a phosphate group specifically to the serine residue within the esteratic site, rendering the enzyme inactive.

(c) Pralidoxime acts as a specific enzyme reactivator by binding to the adjacent anionic site of the enzyme.

(d) From this position, its oxime group nucleophilically attacks the phosphoryl group attached to the serine at the esteratic site. This removes the phosphate, successfully restoring the catalytic function of AChE, making the esteratic site the targeted site of reactivation.

Final Answer: Esteratic site of acetylcholinesterase

Answer: (B)

Go Back to Question 5

Q6.

Solution

Concept:

Selective Estrogen Receptor Modulators (SERMs) display tissue-specific pharmacology, acting as estrogen receptor agonists in some tissues and antagonists in others. In managing post-menopausal osteoporosis, the ideal SERM provides estrogenic benefits to bone tissue while neutralizing proliferative actions in breast and endometrial tissues.

Solution:

(a) Tamoxifen acts as an estrogen agonist in bone tissue and an antagonist in breast tissue, reducing invasive breast cancer risk. However, it acts as an agonist in the endometrium, significantly increasing endometrial hyperplasia and carcinoma risks.

(b) Raloxifene maintains a favorable profile by acting as an estrogen agonist in bone tissue to preserve mineral density, while exhibiting clear antagonist activity in both breast and endometrial tissues.

(c) Consequently, raloxifene lowers the risk of invasive breast cancer without inducing endometrial proliferation or uterine malignancies.

(d) Toremifene behaves similarly to tamoxifen with dangerous stimulatory endometrial effects, whereas clomiphene is primarily utilized for ovulation induction and does not possess this specific dual therapeutic profile.

Final Answer: Raloxifene

Answer: (B)

Go Back to Question 6

Q7.

Solution

Concept:

Beta-lactam resistance in bacterial pathogens involves distinct genetic and structural mechanisms. In Gram-positive organisms like *Streptococcus pneumoniae*, resistance profiles develop via transformational recombination of cell wall synthesis enzymes rather than plasmid-mediated beta-lactamase production or outer membrane alterations.

Solution:

(a) *Streptococcus pneumoniae* is a Gram-positive bacterium, meaning it lacks an outer membrane. Therefore, resistance mechanisms involving porin down-regulation (like OmpF channels) are structurally impossible.

(b) Unlike many Gram-negative organisms or staphylococci, clinical resistance to penicillin in *Streptococcus pneumoniae* is not driven by the production of TEM-1 beta-lactamases or efflux pumps.

(c) Instead, it is mediated by the acquisition of mosaic genes via transformation, leading to structural modifications of Penicillin-Binding Proteins (PBPs).

(d) These altered PBPs exhibit a significantly reduced binding affinity for penicillin, allowing cell wall cross-linking to persist despite therapeutic drug concentrations. This modification leaves late-generation cephalosporins as the primary effective treatment.

Final Answer: Structural modification of Penicillin-Binding Proteins (PBPs)

Answer: (B)

Go Back to Question 7

Q8.

Solution

Concept:

Anticoagulant choice during pregnancy is strictly governed by placental permeability and teratogenic risk profiles. Managing deep vein thrombosis (DVT) in pregnant patients requires selecting an agent with high molecular weight or specific structural properties that prevent cross-placental passage and fetal exposure.

Solution:

(a) Warfarin is a low-molecular-weight lipophilic molecule that readily crosses the placental barrier, causing fetal warfarin syndrome, nasal hypoplasia, CNS abnormalities, and severe embryopathy.

(b) Direct oral anticoagulants (DOACs) like rivaroxaban and dabigatran also pass through the placenta and lack sufficient safety data, making them contraindicated during pregnancy.

(c) Enoxaparin is a low-molecular-weight heparin (LMWH). Despite the name, its large, highly charged polysaccharide structure prevents it from crossing the placental barrier.

(d) Because enoxaparin cannot reach the fetal circulation, it carries no risk of causing teratogenicity or fetal hemorrhage. It remains the gold-standard therapeutic recommendation for acute DVT management throughout all trimesters of pregnancy.

Final Answer: Enoxaparin

Answer: (B)

Go Back to Question 8

Q9.

Solution

Concept:

Serotonin syndrome is a life-threatening complication arising from drug-drug interactions that elevate synaptic serotonin levels. Combining a monoamine oxidase inhibitor (MAOI), which blocks serotonin degradation, with an over-the-counter medication that inhibits serotonin reuptake can trigger severe autonomic, somatic, and cognitive symptoms.

Solution:

(a) Phenelzine is a potent, irreversible monoamine oxidase inhibitor that significantly increases baseline intracellular stores of serotonin within presynaptic terminals.

(b) Dextromethorphan is a widely used over-the-counter antitussive that acts additionally as a non-selective serotonin reuptake inhibitor (SRI).

(c) When dextromethorphan is introduced to a patient taking phenelzine, the combined block of serotonin reuptake and clearance leads to an acute hyper-accumulation of serotonin in the synaptic cleft.

(d) This triggers severe serotonin syndrome, marked by hyperthermia, autonomic instability, hyperreflexia, and clonus. Codeine, benzonatate, and guaifenesin lack significant serotonergic reuptake inhibition activity and do not precipitate this toxic cross-reaction.

Final Answer: Dextromethorphan

Answer: (A)

Go Back to Question 9

Q10.

Solution

Concept:

Managing acute gouty arthritis in patients with impaired renal function requires careful evaluation of drug clearance profiles. Accumulation of narrow therapeutic index anti-gout medications due to reduced glomerular filtration risks profound systemic toxicities, particularly severe myelosuppression and cytopenias.

Solution:

(a) Nonsteroidal anti-inflammatory drugs (NSAIDs) like indomethacin and naproxen can worsen renal function but do not carry a primary risk of systemic accumulation-induced bone marrow aplasia.

(b) Corticosteroids like methylprednisolone are safe alternatives in renal insufficiency and do not undergo hazardous accumulation in Stage 3b chronic kidney disease.

(c) Colchicine relieves gouty inflammation by binding to tubulin and inhibiting microtubule polymerization in neutrophils. It is cleared via renal and hepatic pathways.

(d) In severe renal impairment, colchicine clearance drops significantly, leading to toxic systemic accumulation. This disrupts microtubule dynamics in rapidly dividing cells, causing catastrophic bone marrow toxicity, agranulocytosis, and aplastic anemia, meaning it must be avoided or severely restricted.

Final Answer: Colchicine

Answer: (B)

Go Back to Question 10

Q11.

Solution

Concept:

Antiretroviral therapy (ART) for Human Immunodeficiency Virus (HIV) aims to suppress viral replication by targeting specific stages of the viral lifecycle. When a patient develops high-level resistance to Nucleoside Reverse Transcriptase Inhibitors (NRTIs), alternative therapeutic classes must be integrated. Integrase Strand Transfer Inhibitors (INSTIs) block the covalent insertion of viral DNA into the host genome.

Solution:

(a) Maraviroc is a chemokine receptor antagonist that binds selectively to the host CCR5 co-receptor, preventing viral entry rather than inhibiting intracellular replication machinery.

(b) Etravirine is a second-generation Non-Nucleoside Reverse Transcriptase Inhibitor (NNRTI) that binds directly to the reverse transcriptase enzyme, rendering it ineffective via non-competitive inhibition.

(c) Atazanavir belongs to the protease inhibitor class, which prevents the proteolytic cleavage of viral polyproteins, leaving newborn virions immature and non-infectious.

(d) Dolutegravir is a potent, late-generation Integrase Strand Transfer Inhibitor (INSTI) that binds catalytic sites of the HIV integrase enzyme, preventing viral strand transfer and integration into host chromosomal DNA.

Final Answer: Dolutegravir

Answer: (B)

Go Back to Question 11

Q12.

Solution

Concept:

The baroreceptor reflex loop is a major homeostatic mechanism regulating blood pressure. Autonomic efferent adjustments respond to mechanoreceptor firing in the carotid sinus and aortic arch. Introducing a selective alpha-1 adrenergic agonist alters systemic vascular resistance, triggering predictable, compensatory cardiovascular reflex arcs via autonomic feedback pathways.

Solution:

(a) A selective α_1-adrenergic receptor agonist causes contraction of vascular smooth muscle, leading directly to widespread systemic vasoconstriction and a rise in mean arterial pressure.

(b) This acute elevation in blood pressure increases stretch on high-pressure baroreceptors located within the carotid sinus and the aortic arch, enhancing their afferent firing rate.

(c) Afferent signals travel via the glossopharyngeal and vagus nerves to the nucleus tractus solitarius in the medulla oblongata, which serves as the principal central integration center.

(d) The vasomotor center responds by augmenting efferent parasympathetic (vagal) outflow while simultaneously inhibiting sympathetic discharge. Increased vagal tone slows the sinoatrial node, resulting in a compensatory reflex bradycardia.

Final Answer: Vasoconstriction and reflex bradycardia via increased vagal tone

Answer: (B)

Go Back to Question 12

Q13.

Solution

Concept:

Angiotensin-converting enzyme (ACE) inhibitors are foundational agents in managing hypertension and diabetic nephropathy. However, they are frequently associated with a dry, hacking cough. This class-specific adverse effect is driven by the inhibition of metabolic breakdown pathways for inflammatory peptides within the respiratory tract tissue.

Solution:

(a) Enalapril is an ACE inhibitor that blocks the conversion of angiotensin I to angiotensin II, lowering systemic blood pressure and reducing intrarenal efferent arteriolar resistance.

(b) Angiotensin-converting enzyme is structurally identical to kininase II, an enzyme responsible for the metabolic degradation of bradykinin and substance P.

(c) By inhibiting this degradative pathway, enalapril causes a localized accumulation of bradykinin and substance P within the upper respiratory tract.

(d) Higher bradykinin levels sensitize afferent C-fibers, promote local synthesis of pro-inflammatory prostaglandins, and trigger localized airway edema. This non-allergic tissue irritation manifests clinically as a persistent, non-productive cough that requires discontinuation of the drug.

Final Answer: Bradykinin

Answer: (B)

Go Back to Question 13

Q14.

Solution

Concept:

Neonatal respiratory distress syndrome (RDS) in premature infants results from architectural instability caused by a lack of endogenous pulmonary surfactant. Natural pulmonary surfactant is a complex mixture of phospholipids, neutral lipids, and specific proteins that prevents alveolar collapse at end-expiration by lowering surface tension.

Solution:

(a) Natural pulmonary surfactant contains approximately 90

(b) Dipalmitoylphosphatidylcholine (DPPC), also known as lecithin, is the single most abundant phospholipid compound present in natural surfactant.

(c) The amphipathic structure of DPPC permits it to align effectively at the air-water interface of the alveoli, where it significantly lowers surface tension during respiration.

(d) Phosphatidylglycerol, phosphatidylinositol, and sphingomyelin are essential minor components, but they do not match the quantitative abundance or primary physical surface-active properties of DPPC.

Final Answer: Dipalmitoylphosphatidylcholine

Answer: (A)

Go Back to Question 14

Q15.

Solution

Concept:

Empiric management of febrile neutropenia requires immediate, bactericidal covered protection against virulent pathogens like *Pseudomonas aeruginosa*. For patients with a documented history of Type I penicillin anaphylaxis, monobactam antibiotics provide a unique, non-cross-reactive solution due to their modified core ring chemistry.

Solution:

(a) Piperacillin, ceftazidime, and meropenem are effective antipseudomonal agents, but they contain structural components that can carry cross-reactivity risks in individuals with severe penicillin allergies.

(b) Aztreonam is a unique monobactam antibiotic, meaning its core beta-lactam structure is monocyclic and lacks a fused adjacent ring.

(c) This unique monocyclic architecture prevents cross-allergenicity with penicillins and cephalosporins, allowing safe use during severe penicillin hypersensitivity.

(d) Aztreonam binds preferentially to penicillin-binding protein 3 of aerobic Gram-negative bacilli, disrupting cell wall synthesis and offering strong bactericidal coverage against *Pseudomonas aeruginosa*.

Final Answer: Aztreonam

Answer: (C)

Go Back to Question 15

Q16.

Solution

Concept:

Narcolepsy is a chronic neurological disorder characterized by dysregulated sleep-wake cycles, excessive daytime sleepiness, and cataplexy. First-line pharmacological management requires agents that enhance vigilance and wakefulness via targeted actions on catecholaminergic neurotransmission while demonstrating a favorable safety and low dependence profile.

Solution:

(a) Methylphenidate and dextroamphetamine are powerful sympathomimetic central nervous system stimulants that block norepinephrine and dopamine transporters, but they carry a high risk of abuse and schedule controls.

(b) Diazepam is a benzodiazepine that enhances GABAergic inhibitory neurotransmission, which increases somnolence and worsens narcoleptic symptoms.

(c) Modafinil is a first-line wakefulness-promoting agent that acts primarily as a selective dopamine reuptake inhibitor, increasing synaptic dopamine levels in cortical regions.

(d) Modafinil lacks the prominent reinforcing properties seen with traditional amphetamines, leading to a low abuse liability profile while successfully reducing daytime sleepiness and managing narcolepsy.

Final Answer: Modafinil

Answer: (B)

Go Back to Question 16

Q17.

Solution

Concept:

Autonomic neurotransmission utilizes specific G-protein coupled receptor pathways to modulate intracellular secondary messengers. Understanding the distinction between G_s, G_i, and G_q signaling pathways helps predict the physiological downstream effects of endogenous neurotransmitters on smooth muscle and glandular epithelium.

Solution:

(a) The provided schematic shows a classic G_q-coupled signaling cascade where receptor activation triggers phospholipase C to cleave PIP_2 into IP_3 and DAG, releasing calcium and activating protein kinase C.

(b) The α_2-adrenergic and M_2 muscarinic receptors are coupled to G_i proteins, which act to inhibit adenylyl cyclase and decrease intracellular cyclic AMP levels.

(c) The β_1-adrenergic receptor is coupled to a G_s protein, which stimulates adenylyl cyclase and raises intracellular cyclic AMP concentrations.

(d) The M_3 muscarinic receptor is naturally coupled to G_q proteins, utilizing this exact phospholipase C and calcium mobilization pathway to mediate ciliary muscle contraction.

Final Answer: M_3 Muscarinic receptor

Answer: (D)

Go Back to Question 17

Q18.

Solution

Concept:

The lifecycle of *Plasmodium vivax* and *Plasmodium ovale* features a dormant intrahepatic stage known as a hypnozoite. Standard blood schizonticides like chloroquine clear active erythrocytic parasites but fail to eradicate these latent hepatic reservoirs, requiring secondary targeted anti-relapse therapy.

Solution:

(a) Chloroquine, quinine, artesunate, and lumefantrine are potent blood schizonticides that eliminate asexual erythrocytic parasites to resolve acute clinical malaria symptoms.

(b) However, none of these erythrocytic agents can cross into or clear latent intrahepatic hypnozoites, leaving the patient at high risk for clinical relapse.

(c) Primaquine is an 8-aminoquinoline antimalarial that specifically targets and destroys tissue hypnozoites within hepatic cells, preventing disease recurrence.

(d) Before starting primaquine, patients must be screened for glucose-6-phosphate dehydrogenase (G6PD) deficiency, as the drug can induce severe, acute hemolytic anemia in individuals lacking this protective enzyme.

Final Answer: Primaquine

Answer: (C)

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Q19.

Solution

Concept:

Theophylline is a methylxanthine bronchodilator with a narrow therapeutic index, metabolized heavily by hepatic microsomal oxidation pathways. Co-administration of antimicrobial agents that inhibit these specific cytochrome P450 isoenzymes can impair clearance, causing rapid drug accumulation and severe toxicity.

Solution:

(a) Theophylline undergoes extensive hepatic biotransformation, where Cytochrome P450 1A2 (CYP1A2) serves as the primary metabolic clearing pathway.

(b) Ciprofloxacin is a fluoroquinolone antibiotic that acts as a potent inhibitor of hepatic CYP1A2.

(c) Concomitant use of ciprofloxacin blocks the metabolic degradation of theophylline, causing its serum levels to rise into toxic ranges.

(d) This accumulation triggers severe toxicity, manifesting as central nervous system overstimulation and fatal cardiac arrhythmias like ventricular tachycardia, driven by adenosine receptor antagonism and phosphodiesterase inhibition.

Final Answer: Inhibition of hepatic CYP1A2-mediated metabolism of theophylline by ciprofloxacin

Answer: (C)

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Q20.

Solution

Concept:

Thionamide pharmacotherapy is a first-line approach for Graves' hyperthyroidism, working by inhibiting thyroid peroxidase to block iodination and coupling reactions. Choosing between available thionamides involves evaluating their unique organ toxicity risk profiles and tailoring treatment to the patient's physiological status.

Solution:

(a) Propylthiouracil (PTU) and methimazole are both effective thionamides, but PTU carries a boxed warning for severe, sudden, and sometimes fatal fulminant hepatic failure.

(b) Methimazole is preferred in non-pregnant adults due to its longer half-life, superior adherence profile, and significantly lower risk of severe hepatotoxicity.

(c) While methimazole can cause cholestatic jaundice, it rarely induces the severe hepatic necrosis seen with PTU therapy.

(d) Potassium iodide and radioactive iodine are not first-line thionamides, and propranolol is used only for rapid symptomatic control of hyperadrenergic features rather than modifying thyroid hormone synthesis.

Final Answer: Methimazole

Answer: (A)

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Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	C	2	B	3	C	4	B	5	B
6	B	7	B	8	B	9	A	10	B
11	B	12	B	13	B	14	A	15	C
16	B	17	D	18	C	19	C	20	A