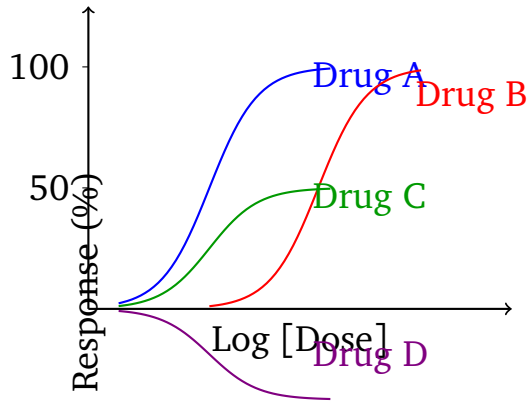
NEET PG Pharmacology Sample Paper-7

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. Consider the log dose-response curve plotted below representing four different drug candidates (A, B, C, and D) acting on the same receptor system:



Based on this pharmacological profile, which of the following statements is completely accurate?

(A) Drug A and Drug B have different maximal efficacies but identical potencies.

(B) Drug C exhibits the same intrinsic activity as Drug A but has lower potency.

(C) Drug D reduces constitutive receptor activity, acting as an inverse agonist with a negative intrinsic activity.

(D) Drug B is a competitive antagonist that shifts the curve of Drug A to the right.

Q2. A 45-year-old patient with severe chronic kidney disease (GFR = 15 mL/min) requires maintenance therapy with a drug that is predominantly eliminated by the kidneys. The drug has a normal half-life of 6 hours. Which parameter must be altered to maintain the same steady-state target plasma concentration without risking cumulative toxicity?

(A) Loading dose must be halved

(B) Maintenance dose rate must be reduced proportionally to the reduction in clearance

(C) Volume of distribution must be expanded artificially

(D) Bioavailability must be monitored via alternative routes of administration

Q3. An experimental drug is found to follow zero-order elimination kinetics at therapeutic plasma concentrations. Which of the following properties is characteristic of this drug's elimination profile?

(A) A constant fraction of the drug is eliminated per unit of time

(B) The clearance of the drug remains constant regardless of the plasma concentration

(C) The elimination half-life increases as the plasma concentration increases

(D) Steady-state concentration is achieved within 4 to 5 half-lives

Q4. A patient is given an intravenous bolus dose of 500 mg of a novel therapeutic agent. Immediate plasma concentration extrapolation to time zero is found to be 25 mg/L. What is the apparent volume of distribution (V_d) of this drug?

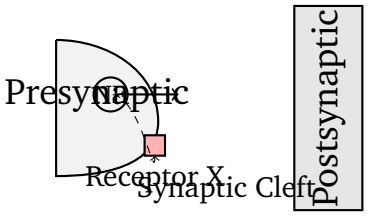
(A) 2 Liters

(B) 12.5 Liters

(C) 20 Liters

(D) 50 Liters

Q5. The schematic below represents a noradrenergic neuroeffector junction highlighting different physiological targets and autoreceptors:



Activation of "Receptor X" on the presynaptic terminal serves as a negative feedback mechanism to inhibit further release of Norepinephrine (NE). Which of the following drugs acts as a selective agonist at this specific "Receptor X"?

(A) Phenylephrine

(B) Clonidine

(C) Yohimbine

(D) Isoproterenol

Q6. A 62-year-old male presenting with acute angle-closure glaucoma requires immediate medical management to reduce intraocular pressure. Which of the following topical miotics acts by directly stimulating muscarinic receptors on the sphincter pupillae muscle, facilitating the drainage of aqueous humor through the trabecular meshwork?

(A) Atropine

(B) Pilocarpine

(C) Timolol

(D) Apraclonidine

Q7. During a surgical procedure, a neuromuscular blocking agent is administered to induce skeletal muscle relaxation. Later, Neostigmine is administered along with Glycopyrrolate to reverse the neuromuscular blockade. What is the primary purpose of co-administering Glycopyrrolate in this clinical scenario?

(A) To synergistically enhance nicotinic receptor activation at the motor endplate

(B) To block unwanted peripheral muscarinic side effects induced by Neostigmine

(C) To facilitate the central nervous system penetration of Neostigmine

(D) To induce profound pupillary constriction and improve visual recovery

Q8. A 35-year-old female presents to the emergency department with severe bradycardia, wheezing, and hypotension following an accidental overdose of Propropanolol. Which pharmacological antidote should be administered immediately due to its ability to stimulate the heart via pathways independent of beta-adrenergic receptors?

(A) Atropine sulfate

(B) Glucagon

(C) Epinephrine

(D) Calcium gluconate

Q9. A 28-year-old woman diagnosed with generalized anxiety disorder is prescribed a medication that acts as a selective partial agonist at the serotonin 5-HT_{1A} receptor. The drug takes approximately 2 weeks to show therapeutic benefits and lacks sedative, anticonvulsant, or muscle-relaxant properties. Identify the drug:

(A) Diazepam

(B) Buspirone

(C) Zolpidem

(D) Alprazolam

Q10. A patient with Parkinson's disease is undergoing long-term treatment with Levodopa-Carbidopa. Over time, the patient experiences severe "wearing-off" fluctuations. The neurologist decides to add Entacapone to the regimen. What is the mechanism of action of Entacapone?

(A) Central inhibition of Monoamine Oxidase-B (MAO-B)

(B) Peripheral inhibition of Catechol-O-Methyltransferase (COMT)

(C) Direct stimulation of postsynaptic Dopamine D2 receptors

(D) Blockade of central muscarinic receptors to restore cholinergic balance

Q11. An emergency room patient presents with severe respiratory depression, pinpoint pupils, and coma following a suspected heroin overdose. Intravenous Naloxone is immediately administered. Naloxone acts as an antagonist at which receptor subtype to reverse these life-threatening effects?

(A) Mu (μ) opioid receptor

(B) Kappa (κ) opioid receptor

(C) Delta (δ) opioid receptor

(D) GABA-A receptor complex

Q12. A 40-year-old male with chronic epilepsy is maintained on Phenytoin. He experiences an increase in seizure frequency, and a laboratory workup reveals sub-therapeutic plasma concentrations of Phenytoin. Further history reveals he recently started taking a drug for peptic ulcer disease that induced hepatic microsomal enzymes. Which of the following concurrent medications is responsible for lowering his Phenytoin levels?

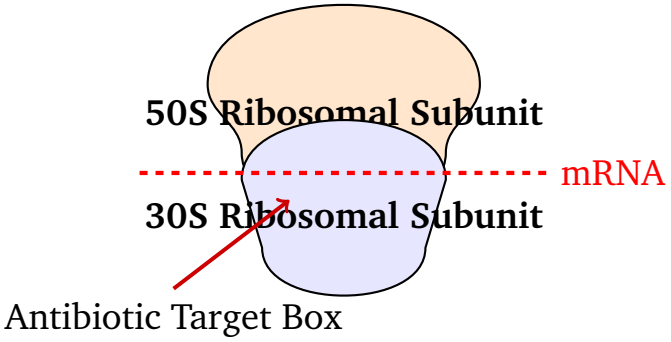
(A) Cimetidine

(B) Omeprazole

(C) Rifampin

(D) Phenobarbital

Q13. The diagram below illustrates the structural site of action of a class of antibiotics within the bacterial protein synthesis machinery:



An antibiotic class targets the A-site of the **30S ribosomal subunit**, causing misreading of the genetic code and premature termination of the peptide chain. Which antibiotic group matches this description and site of action?

(A) Macrolides (e.g., Azithromycin)

(B) Aminoglycosides (e.g., Gentamicin)

(C) Lincosamides (e.g., Clindamycin)

(D) Chloramphenicol

Q14. A 26-year-old male presents with an uncomplicated lower urinary tract infection. He is prescribed a combination of Sulfamethoxazole and Trimethoprim (Cotrimoxazole). This drug combination achieves sequential blockade of which vital bacterial metabolic pathway?

(A) Peptidoglycan cell wall cross-linking

(B) Tetrahydrofolate synthesis pathway

(C) Topoisomerase II and IV DNA replication

(D) Cell membrane phospholipid synthesis

Q15. A hospitalized patient develops severe pseudomembranous colitis secondary to Clostridioides difficile overgrowth following broad-spectrum antibiotic use. Which of the following is the preferred oral non-absorbable antibiotic agent recommended for the targeted treatment of this condition?

(A) Intravenous Vancomycin

(B) Oral Fidaxomicin or Oral Vancomycin

(C) Oral Metronidazole

(D) Oral Ciprofloxacin

Q16. A 32-year-old pregnant patient in her second trimester requires treatment for a respiratory tract infection. Which of the following class of antibiotics must be strictly avoided during pregnancy due to the high risk of causing permanent tooth discoloration and bone growth retardation in the developing fetus?

(A) Penicillins

(B) Macrolides

(C) Tetracyclines

(D) Cephalosporins

Q17. A patient undergoing treatment for pulmonary tuberculosis develops peripheral neuropathy as an adverse effect. This neurological complication is caused by a structural deficiency of Pyridoxine (Vitamin B6) induced by which component of the anti-tubercular regimen?

(A) Rifampin

(B) Isoniazid

(C) Ethambutol

(D) Pyrazinamide

Q18. A 68-year-old male with chronic heart failure is prescribed Digoxin. He presents to the clinic with anorexia, nausea, and changes in color vision (yellow-green halos around objects). A concurrent electrolyte imbalance is known to severely potentiate Digoxin cardiac toxicity. Which electrolyte abnormality is most dangerous in this scenario?

(A) Hyperkalemia

(B) Hypokalemia

(C) Hypernatremia

(D) Hypocalcemia

Q19. A 54-year-old postmenopausal woman with a history of deep vein thrombosis requires long-term management for type 2 diabetes mellitus. The physician wants to prescribe an oral hypoglycemic agent that acts by activating AMP-activated protein kinase (AMPK), reducing hepatic gluconeogenesis, and increasing peripheral glucose uptake without causing hypoglycemia. Choose the best drug:

(A) Glipizide

(B) Metformin

(C) Pioglitazone

(D) Repaglinide

Q20. A patient with severe essential hypertension is started on a therapeutic regimen that includes Hydralazine. Shortly after initiating therapy, the patient experiences significant reflex tachycardia and fluid retention. Which drug class should be added to counter these specific compensatory homeostatic responses?

(A) Loop diuretic + Beta-blocker

(B) Alpha-blocker + ACE inhibitor

(C) Calcium channel blocker + Nitrate

(D) Potassium-sparing diuretic + ARB

Detailed Solutions

Q1.

Solution

Concept:

Receptor pharmacology evaluates drug interactions via log dose-response curves. These curves illustrate key parameters including potency, maximal efficacy, and intrinsic activity. Potency is determined by the position along the dose axis, while efficacy reflects the ceiling of the response. Intrinsic activity defines the relative ability of a bound drug to initiate a cellular response compared to a full agonist.

Solution:

(a) Drug A and Drug B achieve the exact same maximum vertical response, demonstrating that they possess identical maximal efficacy. However, the curve for Drug A lies to the left of Drug B, meaning Drug A requires a lower concentration to achieve fifty percent of its maximal effect, making it more potent.

(b) Drug C reaches only half the maximal response compared to Drug A and Drug B despite sharing an identical half-maximal effective dose with Drug A. This profile characterizes Drug C as a partial agonist with lower intrinsic activity.

(c) Baseline or constitutive receptor activation occurs in the absolute absence of an ligand. Drug D suppresses this baseline level of signaling to a negative value below zero. This behavior is the hallmark of an inverse agonist, which possesses negative intrinsic activity.

(d) A competitive antagonist would shift the curve of an agonist to the right without reducing its maximal response, but it does not cause a downward baseline deviation on its own like Drug D does.

Final Answer: The curve represents an inverse agonist with negative intrinsic activity.

Answer: (C)

Go Back to Question 1

Q2.

Solution

Concept:

Maintenance dosing regimens aim to sustain a target steady-state drug concentration within the therapeutic window. This equilibrium is achieved when the rate of drug administration equals the rate of elimination. Clearance is the single most critical pharmacokinetic parameter governing maintenance dosage design, whereas the volume of distribution determines the initial loading dose requirements.

Solution:

(a) Renal clearance falls drastically in severe chronic kidney disease when the glomerular filtration rate drops to fifteen milliliters per minute. If a drug is cleared primarily by the kidneys, its overall systemic clearance is diminished in direct proportion to this renal impairment.

(b) The maintenance dose rate is mathematically defined as the product of the target steady-state plasma concentration and systemic clearance, divided by bioavailability. Therefore, any reduction in clearance necessitates a proportional decrease in the daily dosing rate.

(c) Failing to adjust the dosing rate downward results in drug accumulation because the rate of input exceeds the impaired rate of output, inevitably triggering severe systemic toxicity.

(d) The loading dose depends on the volume of distribution and the target concentration. Since the volume of distribution is generally unaltered by acute or chronic renal failure, changing the loading dose will not resolve long-term accumulation risks.

Final Answer: The maintenance dose rate must be adjusted in proportion to clearance.

Answer: (B)

Go Back to Question 2

Q3.

Solution

Concept:

Elimination kinetics dictate how drugs are cleared from the body over time. First-order kinetics involve elimination at a rate proportional to plasma concentration, characterized by a constant half-life. Zero-order kinetics occur when elimination pathways become saturated, meaning a constant absolute amount of drug is cleared per unit of time regardless of total systemic concentration.

Solution:

(a) In zero-order elimination, clearance is not constant because it decreases as the plasma concentration rises due to the saturation of metabolic enzymes or active transport mechanisms. This means the body eliminates a fixed number of milligrams every hour.

(b) Because the elimination mechanisms are operating at their absolute maximum capacity, the time required to eliminate half of the remaining systemic drug cannot stay fixed. The half-life becomes directly dependent on the initial concentration.

(c) As the total amount of drug in the plasma increases, the saturated elimination pathways take longer to process the larger pool, which directly causes the elimination half-life to lengthen as concentrations rise.

(d) True steady-state concentrations cannot be reliably predicted via standard half-life rules in zero-order kinetics because small dosage increments can cause disproportionate, dangerous elevations in plasma levels once saturation is reached.

Final Answer: The elimination half-life increases with higher plasma concentrations.

Answer: (C)

Go Back to Question 3

Q4.

Solution

Concept:

The apparent volume of distribution is a fundamental pharmacokinetic parameter that relates the total amount of drug in the body to its resulting concentration in the plasma. It reflects the extent to which a drug distributes into extravascular tissues versus remaining confined within the systemic circulation.

Solution:

(a) The calculation uses the relationship where the apparent volume of distribution equals the total administered dose divided by the plasma concentration extrapolated back to time zero. This represents the concentration before elimination begins.

(b) In this scenario, the total intravenous bolus dose administered is five hundred milligrams. The immediate plasma concentration at time zero is determined by laboratory measurement to be twenty-five milligrams per liter.

(c) Dividing five hundred milligrams by twenty-five milligrams per liter yields a value of twenty. The units resolve directly to liters, indicating that the drug occupies an apparent volume of twenty liters.

(d) This moderate volume suggests that the drug is not entirely restricted to the vascular compartment, which is about four liters, but instead distributes into extracellular fluid and intermediate tissue compartments without extreme intracellular binding.

Final Answer: The apparent volume of distribution is twenty liters.

Answer: (C)

Go Back to Question 4

Q5.

Solution

Concept:

Autonomic neurotransmission is tightly regulated by presynaptic autoreceptors that monitor neurotransmitter levels in the synaptic cleft. In noradrenergic junctions, activation of these presynaptic receptors initiates a negative feedback cascade that decreases further exocytosis of norepinephrine, preserving homeostatic balance and preventing excessive postsynaptic stimulation.

Solution:

(a) Receptor X is situated on the presynaptic sympathetic nerve terminal. Physiologically, this negative feedback loop is mediated specifically by alpha-two adrenergic receptors, which couple to inhibitory G-proteins to suppress adenyl cyclase activity and restrict calcium influx.

(b) Clonidine acts as a selective agonist at these central and peripheral presynaptic alpha-two receptors. By stimulating Receptor X, it suppresses further vesicular release of norepinephrine into the synaptic cleft, decreasing sympathetic tone.

(c) Phenylephrine is a selective alpha-one adrenergic agonist that acts primarily on postsynaptic membranes to induce smooth muscle contraction and vasoconstriction, rather than mediating presynaptic autoinhibition.

(d) Yohimbine functions as an alpha-two receptor antagonist, which blocks this negative feedback mechanism and actually increases norepinephrine release. Isoproterenol is a non-selective beta agonist with no affinity for alpha-two autoreceptors.

Final Answer: The drug that acts as a selective agonist at the autoreceptor is Clonidine.

Answer: (B)

Go Back to Question 5

Q6.

Solution

Concept:

Acute angle-closure glaucoma is an ocular emergency characterized by a rapid elevation in intraocular pressure due to mechanical obstruction of the trabecular meshwork. Emergency management requires rapid pupillary constriction to physically pull the iris away from the filtration angle, thereby restoring the physiological drainage of aqueous humor.

Solution:

(a) The sphincter pupillae muscle is controlled by parasympathetic cholinergic fibers acting on muscarinic M3 receptors. Stimulating these receptors induces miosis, flattening the iris and opening the iridocorneal angle.

(b) Pilocarpine is a direct-acting muscarinic cholinergic agonist. When applied topically to the eye, it directly binds and activates these M3 receptors on the sphincter muscle, causing profound miosis to alleviate mechanical angle closure.

(c) Atropine is a muscarinic antagonist that causes mydriasis and cycloplegia, which worsens angle-closure glaucoma by further crowding the filtration angle and is completely contraindicated.

(d) Timolol is a topical beta-blocker that reduces intraocular pressure by decreasing aqueous humor production from the ciliary epithelium. Apraclonidine is an alpha-two agonist that lowers pressure by reducing production and increasing uveoscleral outflow, but neither acts directly as a miotic.

Final Answer: The direct muscarinic agonist utilized for miosis is Pilocarpine.

Answer: (B)

Go Back to Question 6

Q7.

Solution

Concept:

Reversal of non-depolarizing neuromuscular blockade is achieved using acetylcholinesterase inhibitors. By inhibiting the breakdown of acetylcholine, these agents increase neurotransmitter availability at the nicotinic motor endplate to outcompete the blocker. However, they also cause generalized accumulation of acetylcholine at peripheral muscarinic sites.

Solution:

(a) Neostigmine is an acetylcholinesterase inhibitor that effectively reverses skeletal muscle relaxation. Unfortunately, its action is non-specific, leading to excessive muscarinic stimulation throughout the body, causing bradycardia, excessive salivation, bronchoconstriction, and hypermotility.

(b) Glycopyrrolate is a quaternary ammonium anticholinergic agent that functions as a competitive antagonist at muscarinic receptors. It is added to the reversal regimen specifically to counter these unwanted peripheral parasympathetic side effects.

(c) Because glycopyrrolate possesses a polar quaternary amine structure, it does not readily cross the blood-brain barrier. This prevents central anticholinergic toxicity while protecting peripheral organs like the heart and lungs.

(d) Atropine is an alternative muscarinic antagonist, but it crosses into the central nervous system. Glycopyrrolate is preferred when central sedation or tachycardia control requires a strictly peripheral antimuscarinic profile.

Final Answer: The drug is given to counter peripheral muscarinic side effects.

Answer: (B)

Go Back to Question 7

Q8.

Solution

Concept:

Beta-blocker toxicity presents with profound bradycardia, hypotension, and bronchospasm due to widespread blockade of myocardial beta-one and bronchial beta-two adrenergic receptors. When receptors are completely saturated or blocked by an antagonist, standard adrenergic agonists may prove ineffective at restoring normal cardiac output.

Solution:

(a) Severe propranolol overdose requires a pharmacological intervention that can bypass the blocked beta-adrenergic receptors entirely to stimulate intracellular cyclic adenosine monophosphate production within myocardial cells.

(b) Glucagon binds to specific, distinct G-protein-coupled glucagon receptors on the myocardium. This binding activates adenylate cyclase via a pathway completely independent of the beta-adrenergic receptor complex.

(c) The resulting rise in intracellular cyclic AMP activates protein kinase A, enhancing calcium influx through L-type calcium channels and increasing sarcoplasmic reticular calcium release, which exerts powerful positive inotropic and chronotropic effects.

(d) Epinephrine may fail to overcome the high receptor occupancy of a severe competitive overdose and can trigger dangerous alpha-one mediated vasoconstriction against a failing heart. Atropine treats vagal bradycardia but does not correct underlying myocardial depression.

Final Answer: The antidote that bypasses beta receptors to stimulate the heart is Glucagon.

Answer: (B)

Go Back to Question 8

Q9.

Solution

Concept:

Anxiolytic medications display distinct pharmacological profiles based on their receptor specificities. Classic agents like benzodiazepines modulate the gamma-aminobutyric acid system, providing immediate relief but carrying risks of sedation, muscle relaxation, tolerance, and physical dependence. Alternative pathways target serotonin receptors to manage chronic anxiety without sedative complications.

Solution:

(a) Buspirone represents a unique class of chronic anxiolytics. It operates primarily as a selective partial agonist at presynaptic and postsynaptic serotonin five-HT-one-A receptors, modulating serotonergic transmission over time.

(b) Unlike benzodiazepines, buspirone does not interact with GABA-A receptors. Consequently, it completely lacks sedative, hypnotic, anticonvulsant, or skeletal muscle relaxant properties, making it ideal for patients requiring unimpaired psychomotor function.

(c) A defining clinical characteristic of buspirone is its delayed onset of therapeutic action. It typically requires two to three weeks of continuous daily administration to achieve noticeable reductions in generalized anxiety symptoms.

(d) Diazepam and alprazolam are benzodiazepines that provide immediate sedation and carry dependency risks. Zolpidem is a non-benzodiazepine sedative-hypnotic used strictly for insomnia and is not indicated for generalized anxiety disorders.

Final Answer: The selective partial serotonin agonist used for anxiety is Buspirone.

Answer: (B)

Go Back to Question 9

Q10.

Solution

Concept:

Parkinson's disease management relies on restoring striatal dopaminergic tone. Levodopa is converted to dopamine centrally, but extensive peripheral metabolism limits its central bioavailability. Adjunctive therapies inhibit peripheral degradative enzymes to maximize the delivery of levodopa across the blood-brain barrier and smooth out motor fluctuations.

Solution:

(a) When carbidopa blocks peripheral dopa-decarboxylase, an alternative metabolic pathway mediated by catechol-O-methyltransferase becomes prominent, converting levodopa into three-O-methyldopa in peripheral tissues.

(b) Entacapone is a specific, reversible inhibitor of peripheral catechol-O-methyltransferase. By blocking this enzyme, it prevents the peripheral breakdown of levodopa, significantly extending its plasma half-life and therapeutic duration.

(c) This mechanical action increases the steady fraction of levodopa available to cross the blood-brain barrier, effectively reducing the disabling wearing-off phenomena and motor fluctuations experienced by patients on long-term therapy.

(d) Entacapone does not cross the blood-brain barrier to any significant degree, distinguishing it from tolcapone, which inhibits both central and peripheral COMT. It does not directly stimulate dopamine receptors or affect central monoamine oxidase-B activity.

Final Answer: The mechanism involves peripheral inhibition of the COMT enzyme.

Answer: (B)

Go Back to Question 10

Q11.

Solution

Concept:

Opioid overdose constitutes a major medical emergency characterized by a classic clinical triad of signs: respiratory depression, miosis, and a comatose state. Opioids produce these physiological actions by binding to specialized family members of G-protein coupled receptors located throughout the central and peripheral nervous systems.

Solution:

(a) The mu opioid receptor is the primary molecular target responsible for mediating the profound analgesic, sedative, and respiratory depressant properties of clinical and illicit opioids such as morphine, fentanyl, and heroin.

(b) Heroin undergoes rapid metabolic conversion into active morphine derivatives within the body, which then bind strongly to these central mu receptors, causing a dangerous drop in the brainstem response to carbon dioxide levels.

(c) Naloxone is a high-affinity, pure opioid receptor antagonist that physically displaces opioid molecules from the receptor sites due to its superior competitive binding properties, rapidly reversing the life-threatening respiratory depression.

(d) While Naloxone also possesses weak competitive affinity for kappa and delta opioid receptor subtypes, its clinical life-saving utility in restoring spontaneous respiration during an acute overdose is exclusively due to its blockade of mu receptors.

Final Answer: The life-threatening respiratory depression is reversed by blocking the Mu opioid receptor.

Answer: (A)

Go Back to Question 11

Q12.

Solution

Concept:

Phenytoin exhibits non-linear, saturable elimination kinetics at therapeutic concentrations, which renders its serum levels highly sensitive to changes in metabolic enzyme activity. Concomitant administration of drugs that influence the cytochrome P450 enzyme system can drastically alter Phenytoin clearance.

Solution:

(a) Phenytoin undergoes extensive hepatic metabolism primarily mediated by the cytochrome P450 enzymes CYP2C9 and CYP2C19. Any substance that significantly stimulates these specific isoenzymes will accelerate the breakdown of Phenytoin.

(b) Rifampin is an exceptionally potent inducer of multiple cytochrome P450 hepatic microsomal enzymes. When introduced into a patient's regimen, it drives an increase in enzyme synthesis, drastically accelerating the metabolic clearance of Phenytoin.

(c) This accelerated elimination reduces the steady-state plasma concentration of Phenytoin below the established therapeutic window of ten to twenty milligrams per liter, precipitating a breakthrough of breakthrough epileptic seizures.

(d) Other medications listed such as Cimetidine and Omeprazole act as classic enzyme inhibitors rather than inducers, which would increase rather than decrease Phenytoin levels, whereas Phenobarbital is an inducer but is not used for peptic ulcers.

Final Answer: The hepatic microsomal enzyme inducer responsible is Rifampin.

Answer: (C)

Go Back to Question 12

Q13.

Solution

Concept:

Bacterial protein synthesis is a major target for diverse antimicrobial drug classes. By exploiting structural differences between eukaryotic and prokaryotic ribosomal subunits, these therapeutic agents selectively disrupt translation blocks without causing direct toxicity to human host cells.

Solution:

(a) The prokaryotic ribosome consists of a large fifty-S subunit and a smaller thirty-S subunit. Different antibiotic classes bind to specific pockets on these subunits to block standard peptide elongation.

(b) Aminoglycosides, including Gentamicin and Amikacin, bind irreversibly to specific protein sites on the thirty-S ribosomal subunit. This specific binding alters the structural configuration of the aminoacyl tRNA recognition site.

(c) This structural distortion causes the ribosome to misread genetic code codons on the mRNA strand, leading to the insertion of improper amino acids and creating defective, non-functional proteins that compromise bacterial membrane integrity.

(d) Macrolides like Azithromycin, along with Lincosamides like Clindamycin and Chloramphenicol, exert their pharmacological effects by binding to the fifty-S ribosomal subunit to inhibit transpeptidation or translocation processes.

Final Answer: The antibiotic class targeting the thirty-S subunit to cause misreading is Aminoglycosides.

Answer: (B)

Go Back to Question 13

Q14.

Solution

Concept:

Bacterial survival requires the continuous endogenous synthesis of folic acid, which serves as an indispensable cofactor for the production of purines, pyrimidines, and essential structural amino acids necessary for DNA replication and cell division.

Solution:

(a) Unlike mammalian cells, which can actively absorb preformed dietary folates, most bacteria must synthesize folates de novo. Cotrimoxazole exploits this physiological dependency by combining two agents that target successive steps in the metabolic pathway.

(b) Sulfamethoxazole functions as a structural analogue of para-aminobenzoic acid, competitively inhibiting the enzyme dihydropteroate synthase to block the initial step in the incorporation of PABA into dihydropteroic acid.

(c) Trimethoprim binds to and selectively inhibits the subsequent enzyme, dihydrofolate reductase, preventing the chemical reduction of dihydrofolate into its active form, tetrahydrofolate, which stops essential single-carbon transfer reactions.

(d) This sequential dual blockade produces a powerful synergistic bactericidal effect against susceptible organisms, which is significantly more potent than the bacteriostatic action achieved by using either antimicrobial agent alone.

Final Answer: Cotrimoxazole achieves sequential blockade of the Tetrahydrofolate synthesis pathway.

Answer: (B)

Go Back to Question 14

Q15.

Solution

Concept:

Pseudomembranous colitis is caused by toxigenic strains of Clostridioides difficile multiplying rapidly within the colon, typically after broad-spectrum antibiotics eliminate normal protective gut flora. Effective therapy requires achieving high drug concentrations within the intestinal lumen.

Solution:

(a) Clostridioides difficile remains localized within the mucosa of the large intestine. Therefore, treating this infection effectively requires an antimicrobial agent that is present in high concentrations directly inside the bowel lumen.

(b) Oral Vancomycin is a large, highly polar glycopeptide molecule that cannot cross the gastrointestinal epithelium. When taken orally, it remains completely unabsorbed, ensuring maximum therapeutic availability throughout the colon.

(c) Oral Fidaxomicin is a narrow-spectrum macrocyclic antibiotic that is also minimally absorbed from the gut and is highly recommended due to its low recurrence rates and minimal disruption to residual healthy microflora.

(d) Intravenous Vancomycin is completely ineffective for this condition because it is not secreted into the lumen of the bowel from the bloodstream, while oral Metronidazole is no longer considered first-line therapy due to rising resistance.

Final Answer: The preferred non-absorbable agents are Oral Fidaxomicin or Oral Vancomycin.

Answer: (B)

Go Back to Question 15

Q16.

Solution

Concept:

Prescribing medications during pregnancy requires a careful evaluation of maternal therapeutic benefits against potential teratogenic risks to the developing fetus. Certain antibiotic groups readily cross the placental barrier and disrupt fetal osteogenesis.

Solution:

(a) Tetracyclines, such as Doxycycline and Minocycline, are broad-spectrum agents that readily cross the placenta and accumulate within fetal skeletal tissues due to their chemical properties as strong calcium chelators.

(b) When tetracyclines bind directly to calcium phosphate during periods of active calcification, they form a stable tetracycline-calcium orthophosphate complex that is deposited permanently into developing teeth and bones.

(c) This deposition causes permanent brownish-yellow discoloration of the deciduous teeth, enamel hypoplasia, and a temporary retardation of linear bone growth, making this class contraindicated during the second and third trimesters.

(d) Beta-lactams, such as Penicillins and Cephalosporins, along with Macrolides like Erythromycin, do not affect calcification pathways and are generally considered safe alternatives for treating infections during pregnancy.

Final Answer: The antibiotic class causing fetal tooth discoloration is Tetracyclines.

Answer: (C)

Go Back to Question 16

Q17.

Solution

Concept:

Standard combination chemotherapy regimens for pulmonary tuberculosis utilize multiple first-line agents to prevent the emergence of drug resistance. However, these individual components have distinct, predictable toxicity profiles that require targeted prophylactic monitoring.

Solution:

(a) Isoniazid is a cornerstone antitubercular drug that works by inhibiting mycolic acid synthesis. A well-known adverse effect of Isoniazid therapy is the development of a dose-dependent peripheral neuropathy.

(b) Mechanistically, Isoniazid chemically reacts with Pyridoxal phosphate to form a hydrazone complex, which accelerates the renal excretion of Pyridoxine, leading to a profound systemic deficiency of Vitamin B6.

(c) Pyridoxine is an essential cofactor for the synthesis of critical neurotransmitters, including gamma-aminobutyric acid. Its depletion results in structural axonal degeneration, presenting clinically as symmetrical paresthesias.

(d) This neurotoxic side effect can be completely prevented or reversed by co-administering supplemental Pyridoxine daily, a practice routinely recommended for high-risk individuals undergoing standard anti-tubercular therapy.

Final Answer: The anti-tubercular drug causing Pyridoxine deficiency is Isoniazid.

Answer: (B)

Go Back to Question 17

Q18.

Solution

Concept:

Digoxin is a cardiac glycoside with a narrow therapeutic index used in heart failure management. It works by reversibly inhibiting the membrane-bound sodium-potassium adenosine triphosphatase pump, which increases intracellular calcium to enhance myocardial contractility.

Solution:

(a) The sodium-potassium ATPase pump normally transports three sodium ions out of the cell in exchange for two potassium ions moving in. Extracellular potassium ions compete directly with Digoxin for the same binding site on this enzyme.

(b) When systemic extracellular potassium levels fall below normal values, there is less competition for these enzymatic binding sites, which allows Digoxin to bind more heavily and exert an exaggerated inhibitory effect.

(c) This unchecked inhibition stops pump activity, creating severe intracellular sodium and calcium imbalances that trigger dangerous delayed afterdepolarizations, leading to life-threatening ventricular arrhythmias.

(d) Hypokalemia also accentuates classic signs of systemic digoxin toxicity, such as gastrointestinal distress and xanthopsia, making close monitoring of serum potassium levels critical when patients are taking concurrent loop diuretics.

Final Answer: The electrolyte abnormality that severely potentiates toxicity is Hypokalemia.

Answer: (B)

Go Back to Question 18

Q19.

Solution

Concept:

Pharmacological choices for type 2 diabetes must address insulin resistance while minimizing macrovascular and thromboembolic clinical complications. Ideal baseline therapies lower blood glucose concentrations efficiently without inducing dangerous hypoglycemic episodes.

Solution:

(a) Metformin is a biguanide that stands as the foundational first-line agent for type 2 diabetes. Its cellular mechanism involves the activation of adenosine monophosphate-activated protein kinase within hepatocytes.

(b) Activating this kinase downregulates the transcription of key lipogenic and gluconeogenic enzymes, suppressing excessive hepatic glucose production, which is the primary driver of elevated fasting blood glucose levels.

(c) Metformin also enhances insulin sensitivity in skeletal muscle tissue, improving glucose uptake without stimulating direct insulin secretion from pancreatic beta cells, thereby eliminating the risk of clinical hypoglycemia.

(d) Sulfonylureas like Glipizide and Meglitinides like Repaglinide function as direct insulin secretagogues that carry a high risk of causing hypoglycemia, while Pioglitazone is associated with fluid retention risks.

Final Answer: The oral hypoglycemic agent that activates AMPK is Metformin.

Answer: (B)

Go Back to Question 19

Q20.

Solution

Concept:

Potent arterial vasodilators lower systemic vascular resistance to reduce blood pressure, but they also trigger strong compensatory homeostatic responses mediated by the sympathetic nervous system and the renin-angiotensin-aldosterone axis.

Solution:

(a) Hydralazine is a direct-acting smooth muscle relaxant that selectively dilates arterioles, causing a rapid drop in systemic vascular resistance. This drop is sensed by high-pressure baroreceptors in the carotid sinus and aortic arch.

(b) The resulting baroreceptor reflex triggers a surge in sympathetic outflow, causing significant reflex tachycardia and increasing myocardial oxygen demand, which can precipitate angina in patients with underlying coronary artery disease.

(c) Simultaneously, decreased renal perfusion pressure stimulates the release of renin, leading to aldosterone synthesis, which promotes significant sodium and water retention, presenting clinically as peripheral edema.

(d) To counter these predictable compensatory responses, a beta-blocker must be added to blunt the reflex tachycardia, alongside a loop diuretic to eliminate the retained fluid and maintain long-term antihypertensive efficacy.

Final Answer: A Loop diuretic and Beta-blocker must be added to counter the compensatory mechanisms.

Answer: (A)

Go Back to Question 20

Answer Key

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