INI CET Pharmacology

Sample Paper – 2

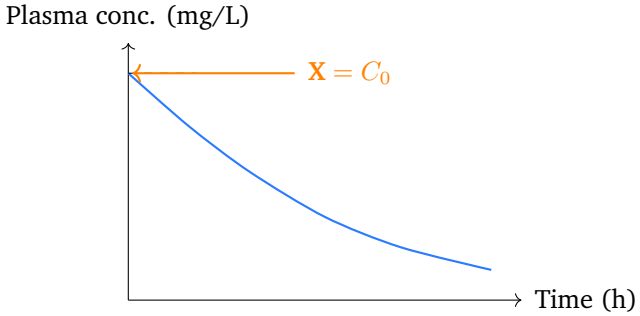
Duration: 14 Minutes **Maximum Marks: 15**

Instructions

- This paper contains **15** Multiple Choice Questions (Single Best Response), modelled on the Pharmacology content of **INI CET** (Institutes of National Importance Combined Entrance Test) conducted by AIIMS New Delhi.
- The **15-question** length matches the number of Pharmacology items you actually meet in the real 200-question paper.
- Each correct answer carries **+1 mark**. There is **negative marking of one-third (0.33) mark** for every incorrect answer; unattempted questions carry no penalty.
- Every question has **exactly one best answer**. Choose the single most appropriate option.
- Attempt this practice paper in one timed sitting of about **14 minutes**, the pace the real computer-based test demands.

General Pharmacology

Q1. The graph shows plasma concentration after a 500 mg intravenous bolus of a drug. The concentration extrapolated back to time zero (C_0, marked **X**) is 10 mg/L. The apparent volume of distribution (Vd) of this drug is:



(A) 5 L

(B) 25 L

(C) 50 L

(D) 500 L

Q2. Warfarin is highly bound to plasma albumin. Co-administration of which drug can displace warfarin from albumin, raising its free (active) fraction and the risk of bleeding?

(A) Digoxin

(B) Insulin

(C) Sulfonamides

(D) Gentamicin

Q3. Which of the following drugs is a classic example of very high (about 99%) binding to plasma proteins?

(A) Warfarin

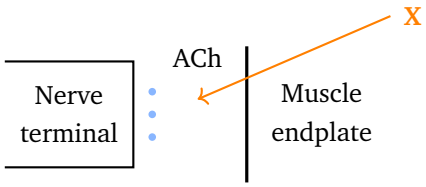
(B) Lithium

(C) Gentamicin

(D) Ethanol

Autonomic Nervous System

Q4. The schematic shows the neuromuscular junction. Neostigmine, used to reverse non-depolarising blockade and to treat myasthenia gravis, produces its effect at the cleft (marked **X**) by:



(A) Blocking nicotinic receptors on the endplate

(B) Preventing acetylcholine release from the nerve terminal

(C) Directly stimulating muscarinic receptors

(D) Inhibiting acetylcholinesterase, so acetylcholine accumulates in the cleft

Q5. Organophosphate insecticide poisoning causes a cholinergic crisis. Besides atropine, the specific agent that reactivates the phosphorylated (inhibited) acetylcholinesterase enzyme is:

(A) Neostigmine

(B) Physostigmine

(C) Edrophonium

(D) Pralidoxime (an oxime)

Cardiovascular and Renal Pharmacology

Q6. Which of the following is a non-dihydropyridine calcium channel blocker that acts mainly on the heart to slow atrioventricular (AV) conduction?

(A) Amlodipine

(B) Nifedipine

(C) Verapamil

(D) Felodipine

Q7. A characteristic adverse effect of dihydropyridine calcium channel blockers such as amlodipine is:

(A) Ankle (pedal) oedema

(B) Dry cough

(C) Hyperkalaemia

(D) Ototoxicity

Q8. Verapamil frequently causes which adverse effect because of its relaxing action on gastrointestinal smooth muscle?

(A) Diarrhoea

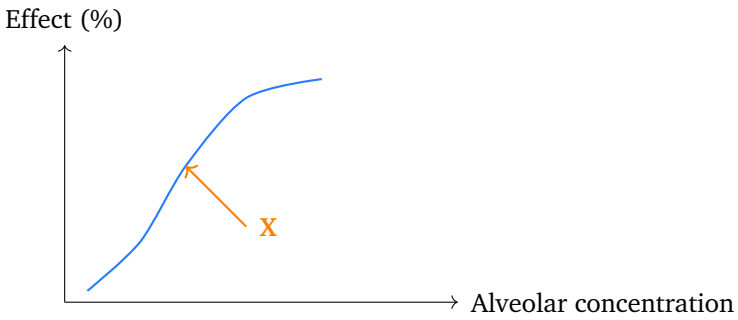
(B) Constipation

(C) Reflex tachycardia

(D) Hypokalaemia

Central Nervous System Pharmacology

Q9. Minimum alveolar concentration (MAC) measures the potency of an inhalational anaesthetic. The dose-response curve marked **X** lies to the left (a low MAC value). An agent with such a low MAC is:



(A) Less potent

(B) More potent

(C) Less lipid soluble

(D) More rapidly eliminated

Q10. Malignant hyperthermia, a life-threatening rise in temperature with muscle rigidity triggered by halothane and succinylcholine, is treated with the muscle relaxant:

(A) Propofol

(B) Thiopentone

(C) Dantrolene

(D) Atropine

Chemotherapy

Q11. Aminoglycosides such as gentamicin inhibit bacterial protein synthesis by binding irreversibly to which ribosomal subunit?

(A) 50S subunit

(B) 30S subunit

(C) 60S subunit

(D) 40S subunit

Q12. Apart from nephrotoxicity, the most characteristic dose-related toxicity of aminoglycosides is:

(A) Hepatotoxicity

(B) Ototoxicity

(C) Pulmonary fibrosis

(D) Grey baby syndrome

Q13. Doxycycline, a tetracycline that inhibits protein synthesis, is the drug of choice for which of the following infections?

(A) Tuberculosis

(B) Falciparum malaria

(C) Herpes simplex

(D) Rickettsial infections (e.g. scrub typhus)

Autacoids, Endocrine and Miscellaneous

Q14. Which first-generation H1 antihistamine is most likely to cause sedation because it readily crosses the blood-brain barrier?

(A) Diphenhydramine

(B) Fexofenadine

(C) Loratadine

(D) Cetirizine

Q15. A second-generation H1 antihistamine such as loratadine is most appropriately used to treat:

(A) Allergic rhinitis and urticaria

(B) Peptic ulcer disease

(C) Parkinson's disease

(D) Bacterial meningitis

Detailed Solutions

Q1.

Solution

Concept – apparent volume of distribution: Vd links the total amount of drug in the body to the plasma concentration.

Step 1 – write the formula: $Vd = \dfrac{\text{Dose (amount in body)}}{C_0 \text{ (plasma concentration at time zero)}}$.

Step 2 – read the values: Dose $= 500$ mg and $C_0 = 10$ mg/L (the extrapolated intercept marked X).

Step 3 – calculate: $Vd = \dfrac{500 \text{ mg}}{10 \text{ mg/L}} = 50$ L.

Why the other options are wrong:

- A, 5 L: this would need $C_0 = 100$ mg/L.
- B, 25 L: this would need $C_0 = 20$ mg/L.
- D, 500 L: this would need $C_0 = 1$ mg/L.

Final Answer: 50 L $\Rightarrow$ C

Answer: (C) Go Back to Q1

Q2.

Solution

Concept – displacement interactions: Only free (unbound) drug is pharmacologically active. Displacing a highly bound drug from albumin raises its free fraction.

Step 1 – identify the displacer: Sulfonamides compete with warfarin for albumin binding sites and displace it.

Step 2 – consequence: The increased free warfarin enhances the anticoagulant effect and the bleeding risk.

Why the other options are wrong:

- A, digoxin: minimally protein bound; does not displace warfarin this way.
- B, insulin: a peptide hormone, not an albumin-binding competitor.
- D, gentamicin: poorly protein bound and does not displace warfarin.

Final Answer: Sulfonamides $\Rightarrow$ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – extent of plasma protein binding: Some drugs are almost entirely albumin bound, which limits their free fraction and volume of distribution.

Step 1 – name the highly bound drug: Warfarin is bound about 99% to plasma albumin.

Step 2 – significance: Small changes in its binding produce large changes in free drug, so it is prone to displacement interactions.

Why the other options are wrong:

- B, lithium: an ion that is not protein bound.
- C, gentamicin: poorly protein bound (highly water soluble).
- D, ethanol: distributes in body water and is not significantly protein bound.

Final Answer: Warfarin $\Rightarrow$ A

Answer: (A) Go Back to Q3

Q4.

Solution

Concept – anticholinesterase action: Acetylcholinesterase in the synaptic cleft normally breaks down acetylcholine (ACh).

Step 1 – identify the mechanism at X: Neostigmine **inhibits acetylcholinesterase**, so ACh accumulates in the cleft and acts longer on the endplate receptors.

Step 2 – clinical use: More available ACh improves transmission, which reverses non-depolarising blockade and relieves myasthenia gravis.

Why the other options are wrong:

- A, blocking nicotinic receptors: that is the action of tubocurarine, the opposite effect.
- B, preventing ACh release: that is botulinum toxin, not neostigmine.
- C, direct muscarinic stimulation: describes agonists like pilocarpine, not an anticholinesterase.

Final Answer: Inhibiting acetylcholinesterase $\Rightarrow$ D

Answer: (D) Go Back to Q4

Q5.

Solution

Concept – organophosphate poisoning: Organophosphates irreversibly phosphorylate acetylcholinesterase, causing ACh excess (a cholinergic crisis).

Step 1 – name the reactivator: Pralidoxime, an oxime, reactivates the phosphorylated enzyme if given before "ageing" of the bond.

Step 2 – combined therapy: Atropine blocks muscarinic effects; pralidoxime restores enzyme activity, especially at the neuromuscular junction.

Why the other options are wrong:

- A and B, neostigmine and physostigmine: these are themselves anticholinesterases and would worsen the poisoning.
- C, edrophonium: a short-acting anticholinesterase used in diagnosis, not a reactivator.

Final Answer: Pralidoxime $\Rightarrow$ D

Answer: (D) Go Back to Q5

Q6.

Solution

Concept – classes of calcium channel blockers: Dihydropyridines act mainly on vascular smooth muscle; non-dihydropyridines act on the heart.

Step 1 – pick the non-dihydropyridine: Verapamil (a phenylalkylamine) acts on cardiac tissue, slowing AV nodal conduction and heart rate.

Step 2 – use: This makes it useful in supraventricular arrhythmias and angina.

Why the other options are wrong:

- A, amlodipine; B, nifedipine; D, felodipine: all dihydropyridines that act chiefly on blood vessels, not on AV conduction.

Final Answer: Verapamil $\Rightarrow$ C

Answer: (C) Go Back to Q6

Q7.

Solution

Concept – dihydropyridine adverse effects: These drugs cause marked arteriolar dilatation.

Step 1 – name the effect: Ankle (pedal) oedema results from precapillary arteriolar dilatation that raises capillary pressure.

Step 2 – note: It is not due to fluid overload, so diuretics help little; it improves on stopping or reducing the drug.

Why the other options are wrong:

- B, dry cough: typical of ACE inhibitors.
- C, hyperkalaemia: seen with potassium-sparing diuretics and ACE inhibitors.
- D, ototoxicity: a feature of aminoglycosides and loop diuretics.

Final Answer: Ankle (pedal) oedema $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – verapamil on smooth muscle: Verapamil relaxes gut smooth muscle by blocking calcium entry.

Step 1 – name the effect: Reduced colonic motility produces **constipation**, the commonest adverse effect of verapamil.

Step 2 – note: It is dose related and often needs dietary fibre or a dose reduction.

Why the other options are wrong:

- A, diarrhoea: opposite to the expected effect.
- C, reflex tachycardia: seen with dihydropyridines; verapamil slows the heart instead.
- D, hypokalaemia: a diuretic effect, not a verapamil effect.

Final Answer: Constipation $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q8

Q9.

Solution

Concept – MAC and potency: MAC is the alveolar concentration that stops movement in 50% of patients; a lower MAC means less drug is needed.

Step 1 – read the curve X: A curve shifted to the left (low MAC) means the effect is produced at a low concentration, so the agent is **more potent**.

Step 2 – correlation: Potency of inhalational agents parallels lipid solubility (Meyer-Overton), so more potent agents are more lipid soluble.

Why the other options are wrong:

- A, less potent: this would be a high MAC (curve shifted right).
- C, less lipid soluble: potent low-MAC agents are in fact more lipid soluble.
- D, more rapidly eliminated: elimination speed relates to blood solubility, not to MAC.

Final Answer: More potent $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q9

Q10.

Solution

Concept – malignant hyperthermia: This is a genetic disorder of the skeletal muscle ryanodine receptor, causing uncontrolled calcium release.

Step 1 – name the treatment: Dantrolene blocks calcium release from the sarcoplasmic reticulum and is the specific antidote.

Step 2 – supportive care: Stop the trigger, cool the patient, and give oxygen along with dantrolene.

Why the other options are wrong:

- A, propofol; B, thiopentone: intravenous anaesthetics, not treatments for the crisis.
- D, atropine: an antimuscarinic with no role in malignant hyperthermia.

Final Answer: Dantrolene $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q10

Q11.

Solution

Concept – protein-synthesis inhibitors: Different antibiotic classes bind different ribosomal subunits.

Step 1 – match aminoglycosides: Aminoglycosides bind the **30S subunit**, causing misreading of mRNA and blocking initiation.

Step 2 – effect: The action is bactericidal and concentration dependent.

Why the other options are wrong:

- A, 50S: the target of macrolides, chloramphenicol and clindamycin.
- C, 60S and D, 40S: these are eukaryotic ribosomal subunits, not bacterial.

Final Answer: 30S subunit $\Rightarrow$ B

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – aminoglycoside toxicity: The two dose-limiting toxicities are on the kidney and the ear.

Step 1 – name it: Ototoxicity (cochlear and vestibular damage) is the characteristic toxicity, and it can be irreversible.

Step 2 – prevention: Monitor drug levels and avoid combining with other ototoxic drugs such as loop diuretics.

Why the other options are wrong:

- A, hepatotoxicity: not typical of aminoglycosides.
- C, pulmonary fibrosis: a feature of bleomycin and amiodarone.
- D, grey baby syndrome: caused by chloramphenicol in neonates.

Final Answer: Ototoxicity $\Rightarrow$ B

Answer: (B) Go Back to Q12

Q13.

Solution

Concept – tetracycline uses: Doxycycline has broad activity, including intracellular organisms.

Step 1 – match the infection: Doxycycline is the drug of choice for **rickettsial infections** such as scrub typhus.

Step 2 – other uses: It also treats chlamydial infection, cholera, brucellosis and acne.

Why the other options are wrong:

- A, tuberculosis: treated with rifampicin, isoniazid and others.
- B, falciparum malaria: treated with artemisinin combinations (doxycycline is only an adjunct).
- C, herpes simplex: a viral infection treated with acyclovir, not an antibiotic.

Final Answer: Rickettsial infections $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q13

Q14.

Solution

Concept – generations of H1 antihistamines: First-generation agents cross the blood-brain barrier; second-generation agents do not.

Step 1 – name the sedating drug: Diphenhydramine is a first-generation H1 blocker that enters the brain and causes sedation.

Step 2 – note: This central action is used to advantage in motion sickness and as a sleep aid.

Why the other options are wrong:

- B, fexofenadine and C, loratadine: second-generation, non-sedating agents.
- D, cetirizine: second-generation and only mildly sedating, far less than diphenhydramine.

Final Answer: Diphenhydramine $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q14

Q15.

Solution

Concept – clinical use of H1 antihistamines: Second-generation H1 blockers control peripheral histamine-mediated allergy without sedation.

Step 1 – name the use: Loratadine is used for **allergic rhinitis and urticaria**.

Step 2 – advantage: It gives once-daily, non-drowsy relief, suiting patients who must stay alert.

Why the other options are wrong:

- B, peptic ulcer disease: needs H2 blockers (ranitidine) or proton pump inhibitors, not H1 blockers.
- C, Parkinson's disease: treated with levodopa and dopamine agonists.
- D, bacterial meningitis: requires antibiotics, not antihistamines.

Final Answer: Allergic rhinitis and urticaria $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q15

Answer Key

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