INI CET Pharmacology

Sample Paper – 3

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. Which statement correctly describes first-order elimination kinetics?

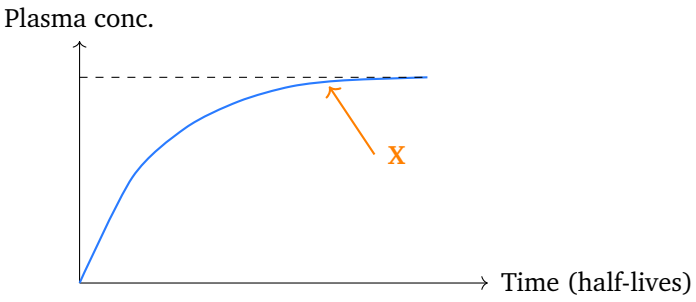
(A) A constant fraction of the drug present is eliminated per unit time

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

(C) The rate of elimination is independent of plasma concentration

(D) The elimination half-life increases as the dose rises

Q2. The graph shows plasma concentration rising during a constant-rate infusion until it levels off at the plateau marked **X** (steady state). Approximately how many elimination half-lives are needed to reach that steady state?



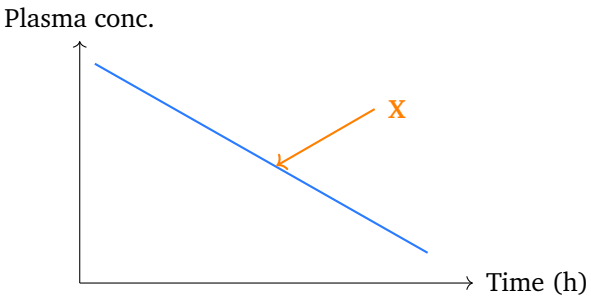
(A) About 1 half-life

(B) About 2 half-lives

(C) About 3 half-lives

(D) About 4 to 5 half-lives

Q3. The graph shows a drug whose plasma concentration falls along a straight line (marked **X**), meaning a fixed amount is cleared per hour regardless of concentration. Which drug shows this zero-order (saturation) kinetics at therapeutic doses?



(A) Penicillin

(B) Paracetamol (therapeutic dose)

(C) Phenytoin

(D) Digoxin

Autonomic Nervous System

Q4. Atropine produces its pharmacological effects by:

(A) Stimulating muscarinic receptors

(B) Irreversibly inhibiting acetylcholinesterase

(C) Competitively blocking muscarinic (M) cholinergic receptors

(D) Blocking nicotinic receptors at the neuromuscular junction

Q5. A child who has ingested atropine-containing berries is brought with the classic anticholinergic toxidrome. Which set of features is expected?

(A) Constricted pupils, salivation, sweating and bradycardia

(B) Dry mouth, dilated pupils, hot flushed dry skin, tachycardia and delirium

(C) Bronchospasm, lacrimation and diarrhoea

(D) Muscle fasciculations with excessive secretions

Cardiovascular and Renal Pharmacology

Q6. Furosemide, a loop diuretic, inhibits the Na-K-2Cl cotransporter located in the:

(A) Proximal convoluted tubule

(B) Thick ascending limb of the loop of Henle

(C) Distal convoluted tubule

(D) Cortical collecting duct

Q7. Which electrolyte disturbance is a characteristic adverse effect of thiazide diuretics?

(A) Hyperkalaemia

(B) Hyperphosphataemia

(C) Hypokalaemia

(D) Hypermagnesaemia

Q8. Spironolactone is a potassium-sparing diuretic. Because it antagonises aldosterone at the collecting duct, it is especially useful in:

(A) Nephrogenic diabetes insipidus

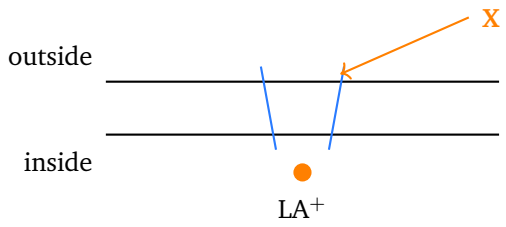
(B) Acute gouty arthritis

(C) Hypokalaemic periodic paralysis

(D) Primary hyperaldosteronism (Conn syndrome) and heart failure

Central Nervous System Pharmacology

Q9. The schematic shows a nerve-membrane sodium channel (marked **X**) with a local anaesthetic molecule approaching it. Local anaesthetics such as lignocaine act by:



(A) Opening potassium channels to hyperpolarise the membrane

(B) Blocking voltage-gated Na+ channels from the inner (cytoplasmic) side of the membrane

(C) Blocking voltage-gated calcium channels at the nerve terminal

(D) Enhancing GABA-mediated chloride entry

Q10. Which of the following is an amide-linked local anaesthetic?

(A) Lignocaine (lidocaine)

(B) Procaine

(C) Cocaine

(D) Benzocaine

Chemotherapy

Q11. Fluoroquinolones such as ciprofloxacin exert their bactericidal action by inhibiting:

(A) Bacterial cell-wall (peptidoglycan) synthesis

(B) DNA gyrase (topoisomerase II) and topoisomerase IV

(C) Protein synthesis at the 30S ribosomal subunit

(D) The 50S ribosomal subunit

Q12. Which adverse effect is specifically associated with fluoroquinolone therapy, particularly in the elderly and in patients on corticosteroids?

(A) Tendinitis and tendon (Achilles) rupture

(B) Grey baby syndrome

(C) Irreversible ototoxicity

(D) Dose-independent aplastic anaemia

Q13. Sulfonamides such as sulfamethoxazole exert their antibacterial action by:

(A) Inhibiting DNA gyrase

(B) Inhibiting bacterial cell-wall synthesis

(C) Binding the 50S ribosomal subunit

(D) Competitively inhibiting dihydropteroate synthase, blocking folic acid synthesis

Autacoids, Endocrine and Miscellaneous

Q14. Ondansetron controls chemotherapy-induced nausea and vomiting mainly by blocking which receptor?

(A) 5-HT3 (serotonin) receptor

(B) D2 dopamine receptor

(C) H1 histamine receptor

(D) M1 muscarinic receptor

Q15. Metoclopramide acts as a prokinetic and antiemetic drug mainly by:

(A) Blocking H2 histamine receptors on parietal cells

(B) Stimulating muscarinic receptors in the gut wall

(C) Blocking central and peripheral D2 dopamine receptors (with 5-HT4 agonism)

(D) Irreversibly blocking 5-HT3 receptors in the gut only

Detailed Solutions

Q1.

Solution

Concept – order of elimination: Kinetics describe how elimination rate relates to drug concentration.

Step 1 – define first-order: In **first-order kinetics** a **constant fraction** of the drug present is removed per unit time.

Step 2 – consequence: Because a fixed percentage is cleared, the absolute rate falls as concentration falls, and the half-life stays constant.

Why the other options are wrong:

- B and C, constant amount and rate independent of concentration: these describe zero-order kinetics.
- D, half-life rising with dose: seen in zero-order (saturation) kinetics, not first-order.

Final Answer: Constant fraction eliminated per unit time ⇒ A

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – reaching steady state: On a constant-rate infusion, plasma level climbs and plateaus.

Step 1 – read the curve: The plateau marked **X** is steady state, where input rate equals elimination rate.

Step 2 – count half-lives: Accumulation reaches about 94% at 4 half-lives and about 97% at 5, so steady state is taken as **4 to 5 half-lives**.

Why the other options are wrong:

- A, 1 half-life: only about 50% of steady state.
- B, 2 half-lives: about 75%.
- C, 3 half-lives: about 87.5%, still short of the plateau.

Final Answer: About 4 to 5 half-lives ⇒ D

Answer: (D) Go Back to Q2

Q3.

Solution

Concept – zero-order (saturation) kinetics: When elimination enzymes are saturated, a fixed amount is cleared per unit time, giving a straight-line decline.

Step 1 – read the graph: The straight line marked **X** shows a constant amount removed per hour, the signature of zero-order kinetics.

Step 2 – match the drug: Phenytoin shows dose-dependent (saturation) kinetics at therapeutic levels, so small dose increases cause large rises in plasma level. Ethanol and high-dose aspirin behave similarly.

Why the other options are wrong:

- A, penicillin: eliminated by first-order renal excretion.
- B, paracetamol at therapeutic dose: first-order; saturation only appears in overdose.
- D, digoxin: first-order elimination.

Final Answer: Phenytoin $\Rightarrow$ C

Answer: (C) Go Back to Q3

Q4.

Solution

Concept – antimuscarinic action: Atropine is the prototype muscarinic antagonist.

Step 1 – name the mechanism: Atropine **competitively blocks muscarinic (M) receptors**, opposing acetylcholine at parasympathetic effector sites.

Step 2 – effects: This raises heart rate, dries secretions, dilates pupils and relaxes gut and bladder.

Why the other options are wrong:

- A, stimulating muscarinic receptors: that is a muscarinic agonist (e.g. pilocarpine).
- B, inhibiting acetylcholinesterase: that is a cholinergic (anticholinesterase) action.
- D, blocking nicotinic receptors: that is the action of neuromuscular blockers, not atropine.

Final Answer: Competitive muscarinic blockade $\Rightarrow$ C

Answer: (C) Go Back to Q4

Q5.

Solution

Concept – anticholinergic toxidrome: Atropine toxicity is remembered as "dry as a bone, red as a beet, hot as a hare, blind as a bat, mad as a hatter".

Step 1 – list the features: Dry mouth, dilated pupils, hot flushed dry skin, tachycardia and delirium together define the picture.

Step 2 – treatment: Severe cases are reversed with physostigmine, a centrally acting anticholinesterase.

Why the other options are wrong:

- A, C and D: constricted pupils, salivation, sweating, bronchospasm, diarrhoea and fasciculations are cholinergic (muscarinic/nicotinic excess) features, the opposite of atropine toxicity.

Final Answer: Dry, flushed, dilated pupils, tachycardia, delirium ⇒ B

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – site of loop diuretic action: Each diuretic class targets a specific nephron segment.

Step 1 – locate furosemide: Furosemide inhibits the **Na-K-2Cl cotransporter** in the **thick ascending limb of the loop of Henle**.

Step 2 – effect: Blocking this transporter gives powerful natriuresis and diuresis and abolishes the medullary concentration gradient.

Why the other options are wrong:

- A, proximal tubule: site of carbonic anhydrase inhibitors (acetazolamide).
- C, distal convoluted tubule: site of thiazides.
- D, collecting duct: site of potassium-sparing diuretics.

Final Answer: Thick ascending limb of the loop of Henle ⇒ B

Answer: (B) Go Back to Q6

Q7.

Solution

Concept – thiazide adverse effects: Thiazides raise distal sodium delivery and increase potassium loss.

Step 1 – name the disturbance: Hypokalaemia is a characteristic adverse effect, from enhanced potassium secretion in the collecting duct.

Step 2 – other metabolic effects: Thiazides also cause hyperglycaemia, hyperuricaemia, hypercalcaemia and hyponatraemia.

Why the other options are wrong:

- A, hyperkalaemia: caused by potassium-sparing diuretics, not thiazides.
- B and D, hyperphosphataemia and hypermagnesaemia: not typical thiazide effects (thiazides tend to cause hypomagnesaemia).

Final Answer: Hypokalaemia ⇒ C

Answer: (C) Go Back to Q7

Q8.

Solution

Concept – aldosterone antagonism: Spironolactone competitively blocks the mineralocorticoid (aldosterone) receptor.

Step 1 – match the indication: Blocking aldosterone makes it the drug of choice in **primary hyperaldosteronism (Conn syndrome)** and a proven agent in **heart failure**.

Step 2 – effect: It conserves potassium while promoting sodium and water loss.

Why the other options are wrong:

- A, nephrogenic diabetes insipidus: paradoxically treated with thiazides, not spironolactone.
- B, acute gout: diuretics can precipitate gout, not treat it.
- C, hypokalaemic periodic paralysis: managed with potassium and other measures, not primarily spironolactone.

Final Answer: Primary hyperaldosteronism and heart failure ⇒ D

Answer: (D) Go Back to Q8

Q9.

Solution

Concept – local anaesthetic mechanism: Local anaesthetics stop nerve conduction by acting on sodium channels.

Step 1 – identify X and the action: X is the voltage-gated **Na+ channel**. The ionised drug (LA^+) enters the axon and **blocks the Na+ channel from the inner (cytoplasmic) side**, preventing depolarisation.

Step 2 – result: No action potential is generated, so pain conduction is blocked; the block is use-dependent (favours open/inactivated channels).

Why the other options are wrong:

- A, opening K channels: not the mechanism of local anaesthetics.
- C, calcium-channel block: not the primary action.
- D, GABA-mediated chloride entry: mechanism of benzodiazepines and barbiturates.

Final Answer: Block of voltage-gated Na+ channels from the inner side $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q9

Q10.

Solution

Concept – amide vs ester local anaesthetics: The linkage between the aromatic and amine ends defines the class.

Step 1 – identify the amide: Lignocaine (lidocaine) is an amide-linked agent (amides have two "i" in the name, e.g. lignocaine, bupivacaine).

Step 2 – significance: Amides are metabolised in the liver and rarely cause allergy; esters are hydrolysed by plasma esterases and more often allergenic.

Why the other options are wrong:

- B, procaine: ester-linked.
- C, cocaine: ester-linked.
- D, benzocaine: ester-linked.

Final Answer: Lignocaine (lidocaine) $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – fluoroquinolone mechanism: Quinolones target the enzymes that manage bacterial DNA supercoiling.

Step 1 – name the targets: Ciprofloxacin inhibits **DNA gyrase (topoisomerase II)** in Gram-negatives and **topoisomerase IV** in Gram-positives.

Step 2 – result: Blocking these enzymes prevents DNA replication and is bactericidal.

Why the other options are wrong:

- A, cell-wall synthesis: mechanism of beta-lactams and vancomycin.
- C, 30S subunit: mechanism of aminoglycosides and tetracyclines.
- D, 50S subunit: mechanism of macrolides and chloramphenicol.

Final Answer: DNA gyrase and topoisomerase IV $\Rightarrow$ B

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – fluoroquinolone toxicity: This class carries a specific musculoskeletal warning.

Step 1 – name the effect: Tendinitis and tendon (Achilles) rupture is a recognised fluoroquinolone adverse effect, more likely in the elderly and in patients on corticosteroids. QT prolongation and cartilage damage in the young are also class concerns.

Step 2 – caution: For this reason quinolones are generally avoided in children and pregnancy.

Why the other options are wrong:

- B, grey baby syndrome: caused by chloramphenicol.
- C, ototoxicity: caused by aminoglycosides.
- D, aplastic anaemia: caused by chloramphenicol.

Final Answer: Tendinitis and tendon rupture $\Rightarrow$ A

Answer: (A) Go Back to Q12

Q13.

Solution

Concept – sulfonamide mechanism: Sulfonamides are structural analogues of PABA.

Step 1 – name the target: Sulfonamides **competitively inhibit dihydropteroate synthase**, blocking incorporation of PABA into folic acid.

Step 2 – result: Bacteria cannot synthesise folate; combining with trimethoprim (a dihydrofolate reductase inhibitor) gives sequential blockade and a bactericidal effect.

Why the other options are wrong:

- A, DNA gyrase: target of quinolones.
- B, cell-wall synthesis: target of beta-lactams.
- C, 50S subunit: target of macrolides.

Final Answer: Inhibition of dihydropteroate synthase (folate synthesis) $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q13

Q14.

Solution

Concept – 5-HT3 antagonists: Serotonin released from gut enterochromaffin cells drives chemotherapy-induced vomiting.

Step 1 – name the receptor: Ondansetron blocks the **5-HT3 (serotonin) receptor** on vagal afferents and in the chemoreceptor trigger zone.

Step 2 – use: It is a first-line antiemetic for chemotherapy-induced and post-operative nausea and vomiting.

Why the other options are wrong:

- B, D2 dopamine: blocked by metoclopramide and antipsychotics.
- C, H1 histamine: blocked by drugs used for motion sickness (e.g. promethazine).
- D, M1 muscarinic: blocked by hyoscine for motion sickness.

Final Answer: 5-HT3 receptor $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q14

Q15.

Solution

Concept – prokinetic antiemetics: Metoclopramide works mainly through dopamine blockade.

Step 1 – name the mechanism: It **blocks central and peripheral D2 dopamine receptors**, with 5-HT4 agonist activity that speeds gastric emptying; it is both antiemetic and prokinetic.

Step 2 – caution: Central D2 blockade can cause extrapyramidal reactions (acute dystonia), especially in the young.

Why the other options are wrong:

- A, H2 blockade: mechanism of ranitidine, an acid suppressant, not a prokinetic.
- B, muscarinic stimulation: not the mechanism of metoclopramide.
- D, 5-HT3 block only: describes ondansetron, which is not prokinetic.

Final Answer: Central and peripheral D2 dopamine blockade $\Rightarrow$ C

Answer: (C) Go Back to Q15

Answer Key

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