INI CET Pharmacology

Sample Paper – 4

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 of the following is a potent inducer of hepatic cytochrome P450 enzymes, and can therefore reduce the plasma levels and efficacy of oral contraceptives?

(A) Cimetidine

(B) Ketoconazole

(C) Rifampicin

(D) Erythromycin

Q2. A patient stabilised on warfarin develops bleeding after starting a new drug that inhibits cytochrome P450, raising the plasma warfarin level. Which drug is the likely inhibitor?

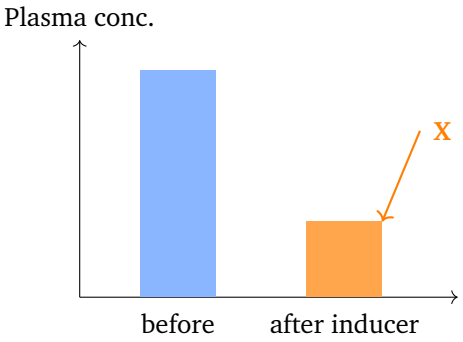
(A) Phenobarbitone

(B) Carbamazepine

(C) Cimetidine

(D) Rifampicin

Q3. The bar diagram compares the steady-state plasma concentration of a drug before and after adding an enzyme inducer. The fall in concentration marked **X** is caused by:



(A) Increased hepatic metabolism of the drug

(B) Reduced renal clearance of the drug

(C) Increased plasma protein binding

(D) Slower gastric absorption

Autonomic Nervous System

Q4. Prazosin lowers blood pressure and relieves the symptoms of benign prostatic hyperplasia by selectively blocking which receptor?

(A) Beta-1 adrenergic receptor

(B) Beta-2 adrenergic receptor

(C) Alpha-1 adrenergic receptor

(D) Muscarinic (M3) receptor

Q5. Which drug is a long-acting, irreversible (non-competitive) alpha-adrenergic blocker used for preoperative preparation of a patient with pheochromocytoma?

(A) Atenolol

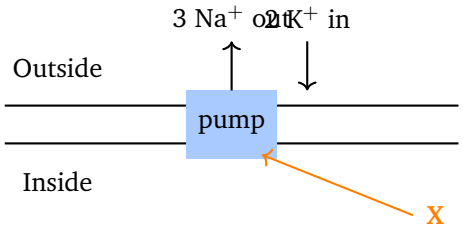
(B) Prazosin

(C) Clonidine

(D) Phenoxybenzamine

Cardiovascular and Renal Pharmacology

Q6. The membrane schematic shows the ion pump (marked **X**) that digoxin inhibits to produce its positive inotropic effect. Digoxin acts by inhibiting:



(A) Na^+/K^+-ATPase

(B) Voltage-gated calcium channels

(C) Beta-1 adrenergic receptors

(D) Phosphodiesterase-3

Q7. Which of the following is a characteristic electrocardiographic feature of the therapeutic (digitalis) effect of digoxin?

(A) Tall peaked T waves

(B) Sagging (scooped) ST-segment depression

(C) Prolonged QT interval

(D) Delta wave

Q8. Which electrolyte disturbance most strongly predisposes a patient on digoxin to digitalis toxicity and arrhythmias?

(A) Hyperkalaemia

(B) Hypokalaemia

(C) Hypernatraemia

(D) Hyperphosphataemia

Central Nervous System Pharmacology

Q9. A characteristic adverse effect seen with long-term phenytoin therapy is:

(A) Gingival hyperplasia

(B) Weight loss

(C) Deafness

(D) Cataract

Q10. Which drug is the classical drug of choice for uncomplicated absence (petit mal) seizures?

(A) Ethosuximide

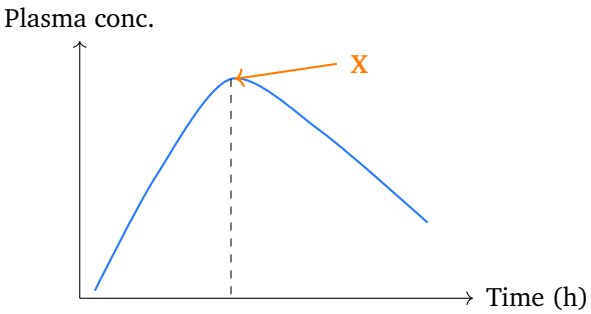
(B) Phenytoin

(C) Carbamazepine

(D) Phenobarbitone

Chemotherapy

Q11. The plasma concentration-time curve of an orally administered antitubercular drug is shown. The point marked **X**, where the plasma level is highest, occurs at a time called:



(A) Half-life

(B) Steady-state time

(C) Elimination time

(D) Tmax (time of peak concentration)

Q12. Peripheral neuropathy caused by isoniazid can be prevented by the routine co-administration of:

(A) Folic acid

(B) Pyridoxine (vitamin B6)

(C) Cyanocobalamin (vitamin B12)

(D) Thiamine (vitamin B1)

Q13. Which antitubercular drug characteristically causes optic neuritis with loss of red-green colour discrimination?

(A) Isoniazid

(B) Rifampicin

(C) Ethambutol

(D) Pyrazinamide

Autacoids, Endocrine and Miscellaneous

Q14. Prolonged systemic glucocorticoid (corticosteroid) therapy characteristically produces which of the following adverse effects?

(A) Weight loss with hyperpigmentation

(B) Cushingoid features (moon face, truncal obesity, striae)

(C) Hypoglycaemia

(D) Hypotension

Q15. Abrupt withdrawal of long-term high-dose glucocorticoid therapy is dangerous because it may precipitate:

(A) Thyroid storm

(B) Hypertensive crisis

(C) Cushing's syndrome

(D) Acute adrenal insufficiency (Addisonian crisis)

Detailed Solutions

Q1.

Solution

Concept – enzyme induction: Some drugs increase the synthesis of hepatic cytochrome P450 enzymes, speeding up the metabolism of co-administered drugs.

Step 1 – identify the inducer: Rifampicin is one of the most powerful CYP450 inducers used clinically.

Step 2 – clinical consequence: By accelerating oestrogen and progestin metabolism, rifampicin lowers oral-contraceptive levels and can cause contraceptive failure.

Why the other options are wrong:

- A, cimetidine: an enzyme inhibitor, not an inducer.
- B, ketoconazole: a potent CYP3A4 inhibitor.
- D, erythromycin: also a CYP3A4 inhibitor.

Final Answer: Rifampicin ⇒ C

Answer: (C) Go Back to Q1

Q2.

Solution

Concept – enzyme inhibition: An inhibitor of CYP450 slows the metabolism of another drug, raising its plasma level and effect.

Step 1 – identify the inhibitor: Cimetidine is a classic CYP450 inhibitor.

Step 2 – consequence with warfarin: Reduced warfarin metabolism raises its plasma level, increasing the anticoagulant effect and the risk of bleeding.

Why the other options are wrong:

- A, phenobarbitone: an enzyme inducer.
- B, carbamazepine: an enzyme inducer.
- D, rifampicin: an enzyme inducer, which would lower warfarin levels instead.

Final Answer: Cimetidine ⇒ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – reading the induction bar diagram: The taller bar is the drug level before, and the shorter bar (marked X) is the level after an inducer is added.

Step 1 – interpret the fall: An enzyme inducer raises the amount of CYP450 available, so the drug is broken down faster.

Step 2 – link to the graph: Faster breakdown means less drug remains in plasma, which is why bar X is lower. This reflects **increased hepatic metabolism**.

Why the other options are wrong:

- B, reduced renal clearance: this would raise, not lower, plasma levels.
- C, increased protein binding: does not reduce total steady-state concentration in this way.
- D, slower gastric absorption: affects the rate of absorption, not steady-state metabolism.

Final Answer: Increased hepatic metabolism $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q3

Q4.

Solution

Concept – alpha blockers: Alpha-1 receptors mediate vascular smooth-muscle contraction and prostatic/urethral tone.

Step 1 – name the target: Prazosin selectively blocks the **alpha-1 adrenergic receptor**.

Step 2 – dual benefit: Vascular alpha-1 blockade lowers blood pressure, while prostatic alpha-1 blockade relaxes the bladder neck and eases urinary flow in BPH.

Why the other options are wrong:

- A and B, beta receptors: blocked by beta blockers, not prazosin.
- D, muscarinic M3: blocked by antimuscarinics, unrelated to prazosin.

Final Answer: Alpha-1 adrenergic receptor $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q4

Q5.

Solution

Concept – pheochromocytoma preparation: An adrenaline-secreting tumour needs stable alpha blockade before surgery.

Step 1 – name the drug: Phenoxybenzamine is a long-acting, irreversible (non-competitive) alpha blocker.

Step 2 – why it fits: Its irreversible binding gives durable blockade that resists surges of catecholamines during tumour handling; alpha blockade is started before any beta blocker.

Why the other options are wrong:

- A, atenolol: a beta-1 blocker, not alpha.
- B, prazosin: a competitive (reversible) alpha-1 blocker; usable but not irreversible.
- C, clonidine: a central alpha-2 agonist, not a peripheral alpha blocker.

Final Answer: Phenoxybenzamine $\Rightarrow$ D

Answer: (D) Go Back to Q5

Q6.

Solution

Concept – reading the pump schematic: The membrane protein that moves 3 Na^+ out and 2 K^+ in is the sodium-potassium pump.

Step 1 – identify X: The structure marked X is the **Na^+/K^+-ATPase**, the target of digoxin.

Step 2 – mechanism of inotropy: Inhibiting this pump raises intracellular Na^+, which slows the Na^+/Ca^{2+} exchanger, so intracellular Ca^{2+} rises and contractility increases.

Why the other options are wrong:

- B, calcium channels: blocked by verapamil-type drugs, not digoxin.
- C, beta-1 receptors: the target of dobutamine and beta blockers.
- D, phosphodiesterase-3: inhibited by milrinone, a different inotrope.

Final Answer: Na^+/K^+-ATPase $\Rightarrow$ A

Answer: (A) Go Back to Q6

Q7.

Solution

Concept – digitalis ECG effect: Therapeutic digoxin changes repolarisation in a typical way.

Step 1 – name the feature: The characteristic sign is **sagging (scooped) ST-segment depression**, often described as the reverse-tick or Salvador Dali sign.

Step 2 – extra features: Flattened or inverted T waves and a shortened QT interval may also accompany the digitalis effect.

Why the other options are wrong:

- A, tall peaked T waves: a feature of hyperkalaemia.
- C, prolonged QT: digoxin actually shortens QT.
- D, delta wave: seen in Wolff-Parkinson-White pre-excitation.

Final Answer: Sagging ST-segment depression $\Rightarrow$ B

Answer: (B) Go Back to Q7

Q8.

Solution

Concept – factors worsening digoxin toxicity: Digoxin and potassium compete for the same site on the Na^+/K^+-ATPase.

Step 1 – name the disturbance: Hypokalaemia increases digoxin binding to the pump and predisposes to toxicity.

Step 2 – clinical note: This matters most in patients on diuretics, which cause potassium loss; monitoring and replacing potassium reduces the risk.

Why the other options are wrong:

- A, hyperkalaemia: high potassium competes with digoxin and reduces its binding (though severe acute toxicity itself raises potassium).
- C and D, hypernatraemia and hyperphosphataemia: not classical precipitants of digitalis toxicity.

Final Answer: Hypokalaemia $\Rightarrow$ B

Answer: (B) Go Back to Q8

Q9.

Solution

Concept – phenytoin adverse effects: Chronic phenytoin has several distinctive toxicities.

Step 1 – name the effect: Gingival hyperplasia (overgrowth of the gums) is the classic long-term effect.

Step 2 – other clues: Phenytoin can also cause hirsutism, coarsening of facial features, megaloblastic anaemia and cerebellar signs at high levels.

Why the other options are wrong:

- B, weight loss: not typical; valproate tends to cause weight gain instead.
- C, deafness: not a recognised phenytoin effect.
- D, cataract: not associated with phenytoin.

Final Answer: Gingival hyperplasia $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – drug of choice by seizure type: Absence seizures respond to drugs that block thalamic T-type calcium currents.

Step 1 – name the drug: Ethosuximide is the classical first choice for pure (uncomplicated) absence seizures.

Step 2 – alternative: Valproate is preferred when absence coexists with generalised tonic-clonic seizures, since ethosuximide does not cover those.

Why the other options are wrong:

- B and C, phenytoin and carbamazepine: can worsen absence seizures.
- D, phenobarbitone: not a first-line agent for absence seizures.

Final Answer: Ethosuximide $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – reading the concentration-time curve: After an oral dose the plasma level rises to a peak and then falls as elimination takes over.

Step 1 – identify X: Point X is the highest point of the curve, so it marks the peak plasma concentration (Cmax).

Step 2 – name the time: The time at which this peak occurs is the **Tmax**, shown by the dashed line dropping to the time axis.

Why the other options are wrong:

- A, half-life: the time for the concentration to fall by half, read on the falling limb.
- B, steady-state time: reached only after repeated dosing, not from a single dose.
- C, elimination time: not the point of peak concentration.

Final Answer: Tmax $\Rightarrow$ D

Answer: (D) Go Back to Q11

Q12.

Solution

Concept – isoniazid and pyridoxine: Isoniazid interferes with pyridoxine metabolism, causing a functional B6 deficiency.

Step 1 – name the preventive: Pyridoxine (vitamin B6) is co-administered to prevent isoniazid-induced peripheral neuropathy.

Step 2 – who needs it most: Malnourished patients, diabetics, pregnant women, alcoholics and slow acetylators are at highest risk and benefit most.

Why the other options are wrong:

- A, folic acid: prevents methotrexate toxicity, not INH neuropathy.
- C, vitamin B12: used for pernicious anaemia, not this indication.
- D, thiamine: prevents Wernicke encephalopathy, not INH neuropathy.

Final Answer: Pyridoxine (vitamin B6) $\Rightarrow$ B

Answer: (B) Go Back to Q12

Q13.

Solution

Concept – antitubercular toxicities: Each first-line drug carries a signature adverse effect.

Step 1 – match optic neuritis: Ethambutol causes dose-dependent optic (retrobulbar) neuritis with loss of red-green colour vision.

Step 2 – monitoring: Visual acuity and colour vision are checked before and during therapy, and the drug is stopped if changes appear.

Why the other options are wrong:

- A, isoniazid: causes peripheral neuropathy and hepatitis.
- B, rifampicin: causes orange-red body secretions and hepatotoxicity.
- D, pyrazinamide: causes hyperuricaemia and hepatotoxicity.

Final Answer: Ethambutol $\Rightarrow$ C

Answer: (C) Go Back to Q13

Q14.

Solution

Concept – glucocorticoid adverse effects: Chronic excess of steroid mimics endogenous cortisol excess.

Step 1 – name the pattern: Long-term therapy produces **Cushingoid features**: moon face, truncal obesity, buffalo hump and purple striae.

Step 2 – other effects: Hyperglycaemia, hypertension, osteoporosis, immunosuppression and thinning of skin are also seen.

Why the other options are wrong:

- A, weight loss with hyperpigmentation: features of adrenal insufficiency, the opposite state.
- C, hypoglycaemia: steroids raise, not lower, blood glucose.
- D, hypotension: steroids tend to cause hypertension through mineralocorticoid activity.

Final Answer: Cushingoid features $\Rightarrow$ B

Answer: (B) Go Back to Q14

Q15.

Solution

Concept – HPA axis suppression: Prolonged exogenous steroids suppress the hypothalamic-pituitary-adrenal axis, so the adrenal glands stop making enough cortisol.

Step 1 – name the danger: Sudden stopping leaves the body unable to make cortisol, precipitating **acute adrenal insufficiency (Addisonian crisis)**.

Step 2 – prevention: Long-term steroids must be tapered gradually, and doses are increased during stress such as surgery or infection.

Why the other options are wrong:

- A, thyroid storm: relates to thyroid, not steroid withdrawal.
- B, hypertensive crisis: not the typical withdrawal event; steroids more often cause hypertension while being taken.
- C, Cushing's syndrome: a result of steroid excess, not withdrawal.

Final Answer: Acute adrenal insufficiency $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q15

Answer Key

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