INI CET Pharmacology

Sample Paper – 10

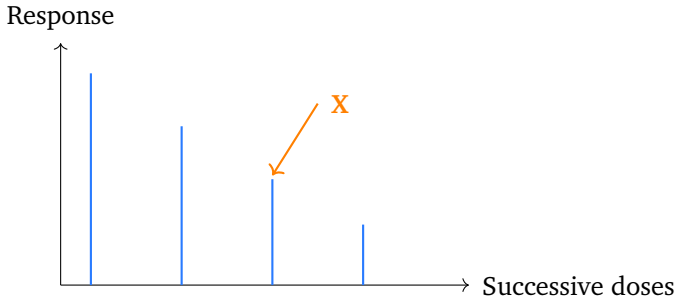
Duration: 14 Minutes **Maximum Marks: 15**

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 covering drug action, therapeutics and adverse effects.
- 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 a progressively falling response to successive equal doses of a drug given over days to weeks (marked **X**). This gradual reduction in effect on repeated administration is best termed:



(A) Idiosyncrasy

(B) Drug antagonism

(C) Hypersensitivity

(D) Tolerance

Q2. Repeated doses of ephedrine or nitrates given at short intervals produce a rapid loss of response, largely from depletion of stored mediator. This quick fall-off in effect is called:

(A) Cumulative toxicity

(B) Tachyphylaxis

(C) Cross-tolerance

(D) Physical dependence

Q3. A patient inactivates isoniazid slowly because of a genetically determined low activity of N-acetyltransferase, raising the risk of peripheral neuropathy. This individual is best described as a:

(A) Rapid acetylator

(B) G6PD-deficient individual

(C) Slow acetylator

(D) Pseudocholinesterase-deficient individual

Autonomic Nervous System

Q4. A farm worker presents with a cholinergic crisis after organophosphate exposure: salivation, lacrimation, bronchorrhea, bronchospasm, miosis and bradycardia. The drug given first to reverse these muscarinic features is:

(A) Atropine

(B) Pralidoxime

(C) Neostigmine

(D) Physostigmine

Q5. In the same organophosphate poisoning, pralidoxime (2-PAM) is added to relieve the muscle weakness. Its mechanism, which must be used early before "aging" of the enzyme, is:

(A) Direct blockade of muscarinic receptors

(B) Further inhibition of acetylcholinesterase

(C) Reactivation of acetylcholinesterase by removing the bound organophosphate

(D) Stimulation of nicotinic receptors at the muscle end plate

Cardiovascular and Renal Pharmacology

Q6. Which fixed-dose angiotensin receptor-neprilysin inhibitor (ARNI), shown to reduce mortality in heart failure with reduced ejection fraction, combines a neprilysin inhibitor with an angiotensin receptor blocker?

(A) Enalapril with hydrochlorothiazide

(B) Amlodipine with atenolol

(C) Sacubitril with valsartan

(D) Digoxin with furosemide

Q7. Which drug class, first introduced as an oral antidiabetic, now improves survival and reduces hospitalisation in heart failure with reduced ejection fraction (for example dapagliflozin)?

(A) Sulfonylureas

(B) Biguanides

(C) Thiazolidinediones

(D) SGLT2 inhibitors

Q8. The potassium-sparing diuretic that antagonises aldosterone at the mineralocorticoid receptor and improves survival in heart failure (but may cause gynaecomastia) is:

(A) Furosemide

(B) Spironolactone

(C) Mannitol

(D) Acetazolamide

Central Nervous System Pharmacology

Q9. Barbiturates produce CNS depression with a low margin of safety. Their principal molecular action at the GABA-A receptor is to:

(A) Prolong the duration of opening of the GABA-A chloride channel

(B) Block NMDA glutamate receptors

(C) Antagonise the GABA-A receptor

(D) Inhibit the reuptake of dopamine

Q10. Amphetamine acts as a CNS stimulant chiefly by which mechanism at central adrenergic and dopaminergic nerve terminals?

(A) Blockade of adenosine receptors

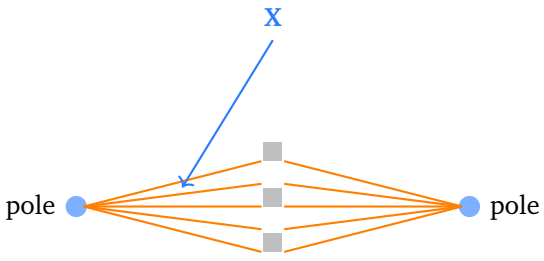
(B) Inhibition of phosphodiesterase

(C) Direct activation of GABA-A receptors

(D) Increased release of noradrenaline and dopamine from nerve terminals

Chemotherapy

Q11. The schematic shows the mitotic spindle, with microtubules running from the two poles to the metaphase chromosomes (marked **X**). Vinca alkaloids such as vincristine act on these microtubules by:



(A) Inhibiting microtubule polymerisation and preventing spindle assembly

(B) Stabilising microtubules and preventing their disassembly

(C) Cross-linking DNA strands

(D) Inhibiting dihydrofolate reductase

Q12. Trastuzumab, a monoclonal antibody used in breast cancer as targeted therapy, exerts its action against which molecular target?

(A) BCR-ABL tyrosine kinase

(B) HER2 (ErbB2) receptor

(C) Vascular endothelial growth factor (VEGF)

(D) CD20 antigen on B cells

Q13. A frequent mechanism of multidrug resistance in tumour cells is increased efflux of chemotherapeutic drugs out of the cell, mediated mainly by:

(A) The P-glycoprotein (MDR1) efflux pump

(B) Increased intracellular drug accumulation

(C) Loss of DNA repair enzymes

(D) Increased topoisomerase activity

Autacoids, Endocrine and Miscellaneous

Q14. Which haematinic, needed for DNA synthesis, causes a megaloblastic anaemia accompanied by neurological signs such as subacute combined degeneration of the cord when it is deficient?

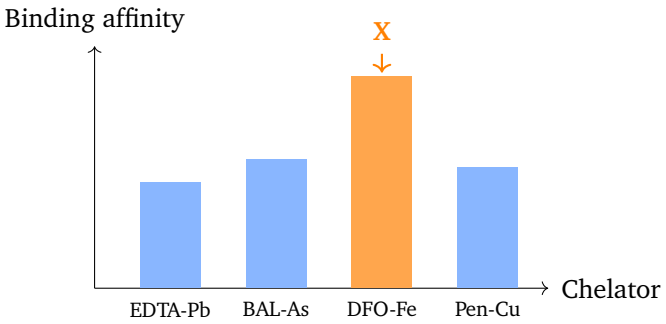
(A) Iron

(B) Vitamin B12 (cyanocobalamin)

(C) Vitamin C (ascorbic acid)

(D) Folic acid

Q15. The bar chart pairs chelating agents with the heavy metal each binds best. The chelator of choice for acute iron poisoning (marked **X**) is:



(A) Calcium disodium edetate (EDTA)

(B) Dimercaprol (BAL)

(C) Desferrioxamine

(D) Penicillamine

Detailed Solutions

Q1.

Solution

Concept – reduced drug response on repeated dosing: Several distinct terms describe a fall in effect.

Step 1 – read the graph: The response falls gradually across successive doses given over days to weeks, not within minutes.

Step 2 – name it: A slow, progressive decrease in effect on repeated administration is **tolerance**.

Why the other options are wrong:

- A, idiosyncrasy: a genetically abnormal, unexpected response in an individual, not a gradual decline.
- B, drug antagonism: one drug opposing another, not repeated dosing of the same drug.
- C, hypersensitivity: an allergic (immune) reaction, not a diminishing effect.

Final Answer: Tolerance $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q1

Q2.

Solution

Concept – rapid loss of response: A quick fall-off differs from slow tolerance.

Step 1 – note the speed: The response is lost rapidly over a few closely spaced doses.

Step 2 – name it: Tachyphylaxis is the rapid decrease in response, classically with indirectly acting sympathomimetics like ephedrine (depletion of noradrenaline stores) and with nitrates.

Why the other options are wrong:

- A, cumulative toxicity: build-up of a slowly eliminated drug, not loss of effect.
- C, cross-tolerance: reduced response to a related drug of the same class.
- D, physical dependence: an adaptive state revealed by a withdrawal syndrome.

Final Answer: Tachyphylaxis ⇒ B

Answer: (B) Go Back to Q2

Q3.

Solution

Concept – pharmacogenetic variation: Genes alter how a drug is handled.

Step 1 – identify the enzyme: Isoniazid is inactivated by hepatic **N-acetyltransferase**, whose activity is genetically polymorphic.

Step 2 – classify the patient: Low enzyme activity means slow inactivation, so the patient is a **slow acetylator** and is prone to peripheral neuropathy (prevented by pyridoxine).

Why the other options are wrong:

- A, rapid acetylator: metabolises isoniazid quickly, the opposite phenotype.
- B, G6PD deficiency: causes haemolysis with oxidant drugs (primaquine, dapsone), not slow isoniazid metabolism.
- D, pseudocholinesterase deficiency: prolongs the action of succinylcholine, not isoniazid.

Final Answer: Slow acetylator ⇒ C

Answer: (C) Go Back to Q3

Q4.

Solution

Concept – organophosphate poisoning: Excess acetylcholine drives a cholinergic crisis.

Step 1 – target the muscarinic signs: Salivation, bronchorrhea, bronchospasm, miosis and bradycardia are muscarinic effects.

Step 2 – choose the drug: Atropine, a competitive muscarinic antagonist, is given first and titrated to dry secretions and a clear chest.

Why the other options are wrong:

- B, pralidoxime: reactivates the enzyme and helps nicotinic (muscle) signs, but does not directly block muscarinic receptors.
- C, neostigmine and D, physostigmine: anticholinesterases that would worsen

the poisoning.

Final Answer: Atropine $\Rightarrow$ A

Answer: (A) Go Back to Q4

Q5.

Solution

Concept – cholinesterase reactivation: Pralidoxime works at the enzyme itself.

Step 1 – state the mechanism: Pralidoxime (2-PAM) has a high affinity for the phosphorus atom and **removes the organophosphate from acetylcholinesterase, reactivating the enzyme**.

Step 2 – timing: It must be given early, before "aging" (irreversible dealkylation of the enzyme-inhibitor bond) makes reactivation impossible; it especially relieves nicotinic muscle weakness.

Why the other options are wrong:

- A, muscarinic blockade: that is atropine's action, not pralidoxime's.
- B, further inhibition of the enzyme: the opposite of its effect.
- D, nicotinic stimulation: pralidoxime relieves, not stimulates, the depolarising block.

Final Answer: Reactivation of acetylcholinesterase before aging $\Rightarrow$ C

Answer: (C) Go Back to Q5

Q6.

Solution

Concept – ARNI in heart failure: A newer combination improves survival in HFrEF.

Step 1 – decode ARNI: An angiotensin receptor-neprilysin inhibitor pairs a neprilysin inhibitor (sacubitril) with an ARB (valsartan).

Step 2 – name it: The drug is **sacubitril-valsartan**; neprilysin inhibition raises beneficial natriuretic peptides while the ARB blocks angiotensin II.

Why the other options are wrong:

- A, enalapril-hydrochlorothiazide: an ACE inhibitor with a thiazide, not an ARNI.

- B, amlodipine-atenolol: a calcium blocker with a beta-blocker, an antihypertensive pair.
- D, digoxin-furosemide: symptomatic heart-failure drugs, not an ARNI and not mortality-reducing.

Final Answer: Sacubitril-valsartan ⇒ C

Answer: (C) Go Back to Q6

Q7.

Solution

Concept – antidiabetic class with heart-failure benefit: One class crossed over into cardiology.

Step 1 – identify the class: SGLT2 inhibitors (dapagliflozin, empagliflozin) block glucose reabsorption in the proximal tubule.

Step 2 – heart-failure evidence: Large trials show they cut heart-failure hospitalisation and mortality even in patients without diabetes.

Why the other options are wrong:

- A, sulfonylureas: increase insulin release; no proven heart-failure survival benefit.
- B, biguanides (metformin): first-line for glycaemia, not a heart-failure survival drug.
- C, thiazolidinediones: cause fluid retention and can worsen heart failure.

Final Answer: SGLT2 inhibitors ⇒ D

Answer: (D) Go Back to Q7

Q8.

Solution

Concept – aldosterone antagonism in heart failure: Blocking aldosterone improves outcomes.

Step 1 – name the drug: Spironolactone competitively blocks the mineralocorticoid (aldosterone) receptor in the distal nephron.

Step 2 – effects: It is potassium-sparing, reduces mortality in HFrEF, and may cause gynaecomastia through anti-androgen activity.

Why the other options are wrong:

- A, furosemide: a loop diuretic for congestion; it wastes potassium and is not an aldosterone antagonist.
- C, mannitol: an osmotic diuretic, contraindicated in heart failure.
- D, acetazolamide: a carbonic anhydrase inhibitor with weak, self-limiting diuresis.

Final Answer: Spironolactone $\Rightarrow$ B

Answer: (B) Go Back to Q8

Q9.

Solution

Concept – barbiturate action at GABA-A: Barbiturates enhance inhibitory GABA signalling.

Step 1 – state the action: Barbiturates **prolong the duration of opening** of the GABA-A chloride channel (benzodiazepines instead raise the frequency of opening).

Step 2 – clinical note: At higher doses they also open the channel directly, which explains their low margin of safety and dangerous respiratory depression in overdose.

Why the other options are wrong:

- B, NMDA blockade: mechanism of ketamine, not barbiturates.
- C, GABA-A antagonism: barbiturates enhance, not block, GABA.
- D, dopamine reuptake inhibition: a stimulant-type action, unrelated to barbiturates.

Final Answer: Prolong GABA-A chloride channel opening $\Rightarrow$ A

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – amphetamine as a CNS stimulant: Its action is on catecholamine terminals.

Step 1 – state the mechanism: Amphetamine **increases release of nora-**

drenaline and dopamine from nerve terminals (and also blocks their reuptake).

Step 2 – effects: This produces alertness, wakefulness, elevated mood and appetite suppression.

Why the other options are wrong:

- A, adenosine blockade: the mechanism of caffeine, not amphetamine.
- B, phosphodiesterase inhibition: how methylxanthines raise cyclic AMP, not amphetamine's main action.
- C, direct GABA-A activation: a depressant action, opposite to a stimulant.

Final Answer: Increased release of noradrenaline and dopamine ⇒ D

Answer: (D) Go Back to Q10

Q11.

Solution

Concept – vinca alkaloids and microtubules: Spindle microtubules are the drug target.

Step 1 – identify X: The marked structures are spindle microtubules assembled from tubulin.

Step 2 – state the action: Vinca alkaloids (vincristine, vinblastine) bind tubulin and **inhibit microtubule polymerisation**, so the spindle cannot form and the cell arrests in metaphase (M phase).

Why the other options are wrong:

- B, stabilising microtubules: that is the taxane mechanism (paclitaxel prevents disassembly), the opposite of vinca alkaloids.
- C, DNA cross-linking: the action of alkylating agents and platinum drugs.
- D, dihydrofolate reductase inhibition: the action of methotrexate.

Final Answer: Inhibiting microtubule polymerisation ⇒ A

Answer: (A) Go Back to Q11

Q12.

Solution

Concept – targeted monoclonal antibodies: Each antibody hits a defined target.

Step 1 – match trastuzumab: Trastuzumab is directed against the **HER2 (ErbB2) receptor**, overexpressed in about a fifth of breast cancers.

Step 2 – effect: Blocking HER2 signalling slows tumour growth; cardiotoxicity is a notable adverse effect.

Why the other options are wrong:

- A, BCR-ABL: the target of imatinib in chronic myeloid leukaemia.
- C, VEGF: the target of bevacizumab (anti-angiogenic).
- D, CD20: the target of rituximab in B-cell lymphomas.

Final Answer: HER2 (ErbB2) receptor $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q12

Q13.

Solution

Concept – multidrug resistance: Tumours pump drugs back out.

Step 1 – name the mechanism: The **P-glycoprotein (MDR1)** efflux pump is an ATP-dependent transporter that expels many anticancer drugs from the cell.

Step 2 – consequence: Lower intracellular drug levels give cross-resistance to several structurally unrelated agents.

Why the other options are wrong:

- B, increased accumulation: resistance lowers, not raises, intracellular drug.
- C, loss of DNA repair: this would make cells more sensitive to DNA-damaging drugs.
- D, increased topoisomerase activity: not the classic efflux mechanism of multidrug resistance.

Final Answer: P-glycoprotein (MDR1) efflux pump $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q13

Q14.

Solution

Concept – haematinics and megaloblastic anaemia: Two vitamins cause megaloblastic change; one adds neurology.

Step 1 – match the clue: Megaloblastic anaemia **with** neurological signs (subacute combined degeneration) points to **vitamin B12 (cyanocobalamin)** deficiency.

Step 2 – caution: Giving folate alone can correct the anaemia but let the neuropathy progress, so B12 status must be checked and replaced.

Why the other options are wrong:

- A, iron: causes microcytic hypochromic anaemia, not megaloblastic.
- C, vitamin C: deficiency causes scurvy, not megaloblastic anaemia.
- D, folic acid: causes megaloblastic anaemia but *without* the cord degeneration seen with B12 deficiency.

Final Answer: Vitamin B12 (cyanocobalamin) $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q14

Q15.

Solution

Concept – chelators and their metals: Each chelator matches a specific poisoning.

Step 1 – read the chart: The tallest bar, marked X, pairs **desferrioxamine** with iron (DFO-Fe).

Step 2 – clinical use: Desferrioxamine is the chelator of choice for acute iron poisoning and for iron overload from repeated transfusions.

Why the other options are wrong:

- A, calcium disodium edetate (EDTA): used for lead poisoning.
- B, dimercaprol (BAL): used for arsenic, mercury and gold poisoning.
- D, penicillamine: used for copper (Wilson's disease) and as a second-line lead chelator.

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

Answer: (C) Go Back to Q15

Answer Key

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