INI CET Pharmacology

Sample Paper – 6

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. A drug that binds a receptor with affinity but has zero intrinsic activity, and shifts the agonist log dose-response curve to the right in a surmountable manner, is best described as a:

(A) Partial agonist

(B) Inverse agonist

(C) Full agonist

(D) Competitive antagonist

Q2. Which of the following acts as a partial agonist at the opioid mu receptor and is used in analgesia and de-addiction therapy?

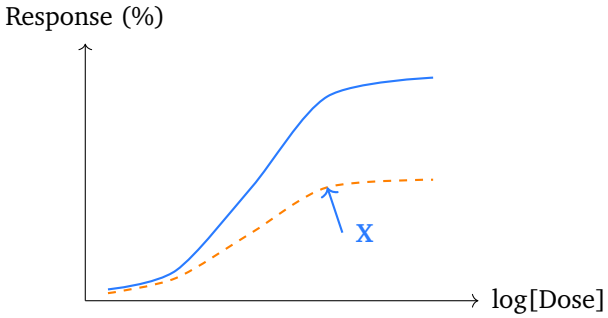
(A) Morphine

(B) Naloxone

(C) Buprenorphine

(D) Fentanyl

Q3. In the log dose-response curves shown, the dashed curve (marked **X**) reaches a lower maximal response than the full agonist even at receptor-saturating doses. A drug producing this curve has lower efficacy and is called a:



(A) Competitive antagonist

(B) Partial agonist

(C) Full agonist

(D) Inverse agonist

Autonomic Nervous System

Q4. Which selective beta-2 adrenergic agonist is used as an inhaled bronchodilator for acute relief in bronchial asthma?

(A) Adrenaline

(B) Dobutamine

(C) Salbutamol

(D) Phenylephrine

Q5. The drug of choice for acute anaphylactic shock, acting on alpha-1, beta-1 and beta-2 receptors, is:

(A) Adrenaline (epinephrine)

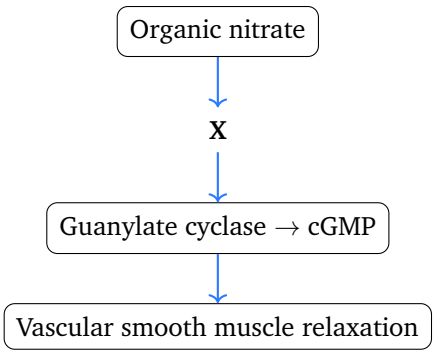
(B) Dobutamine

(C) Atropine

(D) Propranolol

Cardiovascular and Renal Pharmacology

Q6. The schematic shows how organic nitrates relieve angina. A nitrate releases the mediator marked **X**, which activates guanylate cyclase, raises cGMP and relaxes vascular smooth muscle. X is:



(A) Nitric oxide (NO)

(B) Calcium ion

(C) Adenosine

(D) Prostacyclin

Q7. Continuous round-the-clock exposure to organic nitrates causes loss of their antianginal effect (nitrate tolerance). This is best avoided by:

(A) Steadily increasing the dose

(B) Providing a daily nitrate-free interval of 8 to 10 hours

(C) Adding a PDE5 inhibitor

(D) Switching to the intravenous route

Q8. Organic nitrates are strictly contraindicated with which drug class, because the combination causes severe, potentially fatal hypotension?

(A) Beta blockers

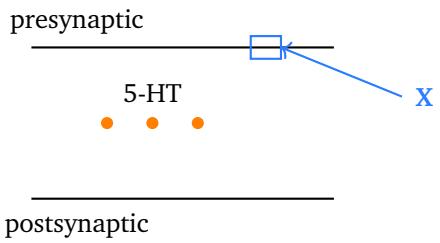
(B) ACE inhibitors

(C) Statins

(D) PDE5 inhibitors (e.g. sildenafil)

Central Nervous System Pharmacology

Q9. The schematic shows a serotonergic synapse. An SSRI such as fluoxetine acts by blocking the presynaptic protein marked **X**, thereby raising serotonin (5-HT) in the cleft. X is the:



(A) Postsynaptic 5-HT receptor

(B) Serotonin reuptake transporter (SERT)

(C) Monoamine oxidase enzyme

(D) Vesicular monoamine transporter

Q10. Which class of antidepressants is notorious for anticholinergic effects and dangerous cardiotoxicity (QRS-complex widening and arrhythmias) in overdose?

(A) Tricyclic antidepressants (TCAs)

(B) Selective serotonin reuptake inhibitors (SSRIs)

(C) Serotonin-noradrenaline reuptake inhibitors (SNRIs)

(D) Monoamine oxidase inhibitors (MAOIs)

Chemotherapy

Q11. Acyclovir is selectively activated only inside herpes-infected cells, after which it inhibits viral DNA polymerase. The viral enzyme that carries out its first (monophosphate) activation step is:

(A) Viral thymidine kinase

(B) Reverse transcriptase

(C) Viral protease

(D) Neuraminidase

Q12. Zidovudine, one of the earliest antiretroviral drugs used in HIV/AIDS, belongs to which class?

(A) Protease inhibitor

(B) Integrase strand transfer inhibitor

(C) Fusion inhibitor

(D) Nucleoside reverse transcriptase inhibitor (NRTI)

Q13. A characteristic dose-limiting toxicity of zidovudine is:

(A) Nephrotoxicity with crystalluria

(B) Optic neuritis

(C) Bone marrow suppression (anaemia and neutropenia)

(D) Haemorrhagic cystitis

Autacoids, Endocrine and Miscellaneous

Q14. Which antithyroid drug lowers thyroid hormone synthesis by inhibiting thyroid peroxidase, thereby blocking the organification of iodine and coupling of iodotyrosines?

(A) Levothyroxine

(B) Carbimazole

(C) Lugol's iodine

(D) Propranolol

Q15. In a thyroid storm, propylthiouracil is often preferred over carbimazole mainly because propylthiouracil additionally:

(A) Has fewer adverse effects

(B) Can be given as a single daily dose

(C) Blocks the peripheral conversion of T4 to the more active T3

(D) Is completely safe in pregnancy

Detailed Solutions

Q1.

Solution

Concept – classifying receptor ligands: Affinity is the ability to bind; intrinsic activity (efficacy) is the ability to activate.

Step 1 – read the description: The drug binds the receptor (has affinity) but produces no effect on its own (zero intrinsic activity).

Step 2 – read the curve effect: It shifts the agonist curve to the right and the shift is surmountable, that is, a high enough agonist dose still reaches the full maximum.

Step 3 – name it: Affinity but no activity, plus a surmountable right shift, defines a **competitive antagonist**.

Why the other options are wrong:

- A, partial agonist: has some intrinsic activity, not zero.
- B, inverse agonist: reduces the baseline (constitutive) activity below normal.
- C, full agonist: has maximal intrinsic activity.

Final Answer: Competitive antagonist $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q1

Q2.

Solution

Concept – partial agonists in practice: A partial agonist activates the receptor but produces only a submaximal effect.

Step 1 – scan the opioids: Among the choices, one is a classic mu-receptor partial agonist.

Step 2 – identify it: Buprenorphine is a partial agonist at the mu receptor; its ceiling effect on respiration makes it safer, and it is used in analgesia and opioid de-addiction.

Why the other options are wrong:

- A, morphine: a full mu agonist.
- B, naloxone: a pure mu antagonist used to reverse opioid overdose.
- D, fentanyl: a potent full mu agonist.

Final Answer: Buprenorphine ⇒ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – efficacy on a dose-response curve: The height of the plateau reflects efficacy (intrinsic activity).

Step 1 – read curve X: The dashed curve X plateaus below the full agonist even at saturating doses.

Step 2 – interpret: A lower ceiling despite full receptor occupancy means lower efficacy.

Step 3 – name it: A drug with affinity but only submaximal efficacy is a **partial agonist**.

Why the other options are wrong:

- A, competitive antagonist: produces no response of its own, so it would not generate a curve.
- C, full agonist: reaches the same maximum (the solid curve).
- D, inverse agonist: drives the response below baseline, not a reduced positive plateau.

Final Answer: Partial agonist ⇒ B

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – adrenergic receptor selectivity: Beta-2 receptors on bronchial smooth muscle cause bronchodilation when stimulated.

Step 1 – match the target: Asthma relief needs a selective beta-2 agonist to open airways with minimal cardiac effect.

Step 2 – identify it: Salbutamol is a selective short-acting beta-2 agonist, the standard reliever inhaler.

Why the other options are wrong:

- A, adrenaline: acts on alpha and all beta receptors, not selective.

- B, dobutamine: a beta-1 agonist used as a cardiac inotrope.
- D, phenylephrine: a selective alpha-1 agonist (vasoconstrictor, decongestant).

Final Answer: Salbutamol $\Rightarrow$ C

Answer: (C) Go Back to Q4

Q5.

Solution

Concept – drug of choice in anaphylaxis: Anaphylaxis needs a drug that raises blood pressure, supports the heart and opens airways at once.

Step 1 – pick the agent: Adrenaline (epinephrine) acts on alpha-1 (vasoconstriction), beta-1 (cardiac stimulation) and beta-2 (bronchodilation).

Step 2 – practical point: It is given intramuscularly (0.5 mg of 1:1000) into the anterolateral thigh without delay.

Why the other options are wrong:

- B, dobutamine: beta-1 inotrope only, no airway or alpha effect.
- C, atropine: an antimuscarinic used for bradycardia, not anaphylaxis.
- D, propranolol: a beta blocker, which would worsen anaphylaxis.

Final Answer: Adrenaline (epinephrine) $\Rightarrow$ A

Answer: (A) Go Back to Q5

Q6.

Solution

Concept – how nitrates work: Organic nitrates are prodrugs that release a signalling gas in vascular smooth muscle.

Step 1 – identify X: Nitrates are denitrated to release **nitric oxide (NO)**, the mediator marked X.

Step 2 – follow the pathway: NO activates guanylate cyclase, which raises cGMP; cGMP lowers intracellular calcium and relaxes smooth muscle, mainly veins, reducing preload and cardiac oxygen demand.

Why the other options are wrong:

- B, calcium: its fall (not release) mediates relaxation.

- C, adenosine: a different vasodilator, not the nitrate mediator.
- D, prostacyclin: works through cAMP, not the nitrate-cGMP pathway.

Final Answer: Nitric oxide (NO) $\Rightarrow$ A

Answer: (A) Go Back to Q6

Q7.

Solution

Concept – nitrate tolerance: Sustained nitrate levels blunt the response within a day.

Step 1 – state the fix: Tolerance is prevented by a daily **nitrate-free interval** of about 8 to 10 hours (usually overnight), letting the tissues recover.

Step 2 – apply it: Eccentric dosing or removing the patch at night keeps the drug effective.

Why the other options are wrong:

- A, increasing the dose: only deepens tolerance and adds side effects.
- C, adding a PDE5 inhibitor: dangerous and contraindicated with nitrates.
- D, intravenous route: continuous infusion actually promotes tolerance faster.

Final Answer: A daily nitrate-free interval $\Rightarrow$ B

Answer: (B) Go Back to Q7

Q8.

Solution

Concept – a dangerous drug interaction: Both nitrates and PDE5 inhibitors amplify the same NO-cGMP pathway.

Step 1 – name the class: PDE5 inhibitors such as sildenafil, tadalafil and vardenafil are contraindicated with nitrates.

Step 2 – explain the danger: Nitrates raise cGMP; PDE5 inhibitors block its breakdown. Together cGMP accumulates massively, causing profound, refractory hypotension.

Why the other options are wrong:

- A, beta blockers: often combined with nitrates safely in angina.
- B, ACE inhibitors: no such prohibition.

- C, statins: routinely co-prescribed in coronary disease.

Final Answer: PDE5 inhibitors $\Rightarrow$ D

Answer: (D) Go Back to Q8

Q9.

Solution

Concept – SSRI mechanism at the synapse: Serotonin action is ended by its reuptake back into the presynaptic terminal.

Step 1 – identify X: The presynaptic protein marked X is the **serotonin reuptake transporter (SERT)**.

Step 2 – link to the drug: Fluoxetine and other SSRIs block SERT, so serotonin stays longer in the cleft and postsynaptic signalling increases, lifting mood over weeks.

Why the other options are wrong:

- A, postsynaptic 5-HT receptor: the target of the released serotonin, not what the SSRI blocks.
- C, monoamine oxidase: the enzyme blocked by MAOIs, not SSRIs.
- D, vesicular monoamine transporter: loads serotonin into vesicles (target of reserpine).

Final Answer: Serotonin reuptake transporter (SERT) $\Rightarrow$ B

Answer: (B) Go Back to Q9

Q10.

Solution

Concept – antidepressant toxicity profiles: Each class has a signature adverse effect.

Step 1 – match the clues: Anticholinergic effects (dry mouth, blurred vision, constipation) plus cardiotoxicity with QRS widening point to **tricyclic antidepressants (TCAs)**.

Step 2 – mechanism of harm: TCAs block fast sodium channels in the myocardium, so overdose causes wide-QRS arrhythmias; sodium bicarbonate is the antidote.

Why the other options are wrong:

- B, SSRIs: much safer in overdose; main risk is serotonin syndrome.
- C, SNRIs: can raise blood pressure but lack marked TCA-type cardiotoxicity.
- D, MAOIs: cause the tyramine (cheese) reaction and hypertensive crisis, a different problem.

Final Answer: Tricyclic antidepressants (TCAs) $\Rightarrow$ A

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – selective activation of acyclovir: Acyclovir is a prodrug that becomes active only inside infected cells.

Step 1 – name the enzyme: The first phosphorylation is done by **viral thymidine kinase**, present only in herpes-infected cells.

Step 2 – follow the steps: Host kinases then add two more phosphates; acyclovir triphosphate inhibits viral DNA polymerase and acts as a chain terminator. This selectivity explains its low toxicity.

Why the other options are wrong:

- B, reverse transcriptase: an HIV enzyme, not involved in herpes.
- C, viral protease: matures HIV proteins, unrelated to acyclovir.
- D, neuraminidase: the target of oseltamivir in influenza.

Final Answer: Viral thymidine kinase $\Rightarrow$ A

Answer: (A) Go Back to Q11

Q12.

Solution

Concept – antiretroviral drug classes: HAART combines drugs from different classes targeting the HIV life cycle.

Step 1 – classify zidovudine: Zidovudine (AZT) is a **nucleoside reverse transcriptase inhibitor (NRTI)**.

Step 2 – mechanism: After phosphorylation it is incorporated into viral DNA and terminates the chain, blocking reverse transcription.

Why the other options are wrong:

- A, protease inhibitor: e.g. ritonavir, ends in "-navir".
- B, integrase inhibitor: e.g. raltegravir, ends in "-tegravir".
- C, fusion inhibitor: e.g. enfuvirtide, blocks viral entry.

Final Answer: Nucleoside reverse transcriptase inhibitor (NRTI) $\Rightarrow$ D

Answer: (D) Go Back to Q12

Q13.

Solution

Concept – signature toxicity of zidovudine: Each antiretroviral has a typical adverse effect that guides monitoring.

Step 1 – recall it: Zidovudine characteristically causes **bone marrow suppression**, producing anaemia and neutropenia.

Step 2 – clinical action: Regular blood counts are needed; severe anaemia may require dose reduction or a switch.

Why the other options are wrong:

- A, nephrotoxicity with crystalluria: typical of tenofovir and of indinavir, not zidovudine.
- B, optic neuritis: a feature of ethambutol (an antitubercular).
- D, haemorrhagic cystitis: caused by cyclophosphamide, not zidovudine.

Final Answer: Bone marrow suppression $\Rightarrow$ C

Answer: (C) Go Back to Q13

Q14.

Solution

Concept – how thioamides work: Thioamide antithyroid drugs act inside the thyroid gland on hormone synthesis.

Step 1 – name the drug: Carbimazole (and its active form methimazole) inhibits thyroid peroxidase.

Step 2 – mechanism: Blocking peroxidase stops iodine organification and the coupling of iodotyrosines, so less T3 and T4 are made. The effect is delayed because stored hormone must first be used up.

Why the other options are wrong:

- A, levothyroxine: synthetic T4 given as replacement in hypothyroidism, not an antithyroid drug.
- C, Lugol's iodine: high-dose iodine that briefly blocks hormone release (Wolff-Chaikoff effect), a different mechanism.
- D, propranolol: only controls adrenergic symptoms, it does not block synthesis.

Final Answer: Carbimazole $\Rightarrow$ B

Answer: (B) Go Back to Q14

Q15.

Solution

Concept – choosing a thioamide in thyroid storm: Thyroid storm needs a drug that lowers active hormone as fast as possible.

Step 1 – state the advantage: Propylthiouracil blocks the peripheral conversion of T4 to the more active T3, on top of inhibiting peroxidase.

Step 2 – why it matters: This extra action lowers active hormone faster, which is valuable in the emergency of a thyroid storm.

Why the other options are wrong:

- A, fewer adverse effects: false; propylthiouracil carries a higher risk of hepatotoxicity.
- B, single daily dose: false; it is short acting and given more often.
- D, safe in pregnancy: it is preferred only in the first trimester, not the reason it is used in storm.

Final Answer: Blocks peripheral T4 to T3 conversion $\Rightarrow$ C

Answer: (C) Go Back to Q15

Answer Key

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