INI CET Pharmacology

Sample Paper – 1

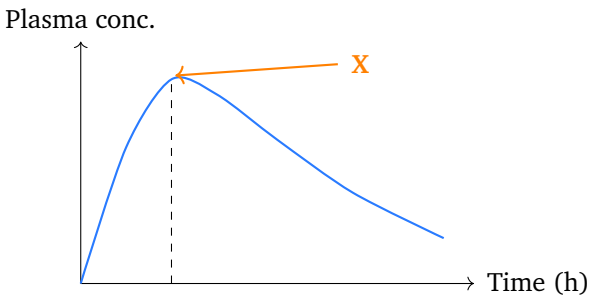
Duration: 14 Minutes **Maximum Marks: 15**

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 plasma drug concentration-time curve after a single oral dose is shown. The peak marked **X** represents:



(A) The area under the concentration-time curve (AUC)

(B) The elimination half-life

(C) The trough (minimum) plasma concentration

(D) The maximum (peak) plasma concentration (Cmax)

Q2. Which drug undergoes extensive first-pass hepatic metabolism, greatly reducing its oral bioavailability?

(A) Propranolol

(B) Atenolol

(C) Digoxin

(D) Gentamicin

Q3. Which route of drug administration gives 100 per cent bioavailability by definition, because the whole dose reaches the systemic circulation?

(A) Oral

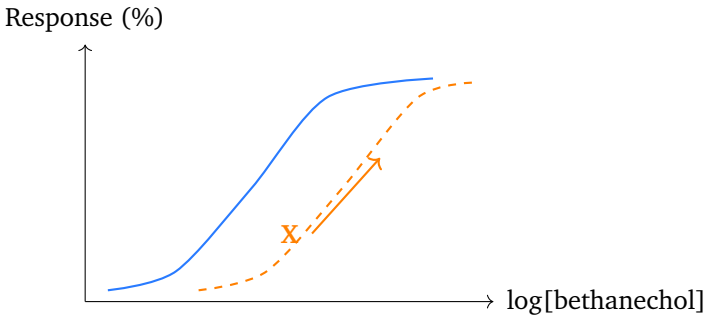
(B) Intravenous

(C) Subcutaneous

(D) Intramuscular

Autonomic Nervous System

Q4. The solid curve is the log dose-response to the muscarinic agonist bethanechol. Adding drug X shifts it to the parallel dashed curve (marked **X**) with the same maximum response. Drug X is acting as:



(A) A competitive (reversible) antagonist at the muscarinic receptor

(B) A non-competitive (irreversible) antagonist

(C) A full agonist

(D) An enzyme inducer

Q5. Bethanechol is a muscarinic agonist resistant to cholinesterase. Its main clinical use is to treat:

(A) Postoperative non-obstructive urinary retention and bladder atony

(B) Bronchial asthma

(C) Peptic ulcer disease

(D) Essential hypertension

Cardiovascular and Renal Pharmacology

Q6. ACE inhibitors such as enalapril lower blood pressure mainly by:

(A) Blocking angiotensin II AT1 receptors

(B) Blocking beta-1 adrenergic receptors

(C) Inhibiting conversion of angiotensin I to angiotensin II

(D) Blocking L-type calcium channels

Q7. A dry, persistent cough is a characteristic adverse effect of ACE inhibitors. It is attributed to accumulation of:

(A) Angiotensin II

(B) Bradykinin

(C) Histamine

(D) Renin

Q8. Losartan (an angiotensin receptor blocker) differs from enalapril in that it:

(A) Inhibits ACE more potently than enalapril

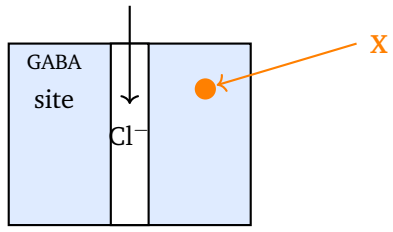
(B) Blocks the angiotensin II AT1 receptor and does not raise bradykinin, so cough is uncommon

(C) Is a beta-adrenergic blocker

(D) Causes cough more often than enalapril

Central Nervous System Pharmacology

Q9. The schematic shows the GABA-A receptor chloride channel. Benzodiazepines bind at the modulatory site marked **X** and:



GABA-A receptor Cl^- channel

(A) Increase the duration of chloride channel opening

(B) Open the chloride channel even without GABA

(C) Physically block the chloride channel pore

(D) Increase the frequency of chloride channel opening in the presence of GABA

Q10. Flumazenil is used clinically as:

(A) A benzodiazepine agonist for chronic anxiety

(B) An antidote for opioid overdose

(C) A first-line anticonvulsant

(D) A competitive benzodiazepine-receptor antagonist that reverses benzodiazepine overdose

Chemotherapy

Q11. Penicillins and cephalosporins (beta-lactam antibiotics) are bactericidal because they:

(A) Inhibit protein synthesis at the 30S ribosomal subunit

(B) Inhibit bacterial cell wall synthesis by binding penicillin-binding proteins (transpeptidases)

(C) Inhibit DNA gyrase (topoisomerase)

(D) Inhibit bacterial folate synthesis

Q12. Which agent is a fourth-generation cephalosporin with useful activity against Pseudomonas aeruginosa?

(A) Cefazolin

(B) Cefuroxime

(C) Cefepime

(D) Cephalexin

Q13. The most common mechanism of bacterial resistance to penicillins is:

(A) Production of beta-lactamase (penicillinase) enzymes that hydrolyse the beta-lactam ring

(B) Increased drug efflux pumps

(C) Reduced bacterial folate synthesis

(D) Alteration of the ribosomal target

Autacoids, Endocrine and Miscellaneous

Q14. Non-selective NSAIDs such as aspirin and ibuprofen produce their analgesic and anti-inflammatory effect mainly by:

(A) Inhibiting lipoxygenase

(B) Blocking histamine H1 receptors

(C) Inhibiting cyclo-oxygenase (COX) and reducing prostaglandin synthesis

(D) Inhibiting phospholipase A2

Q15. The specific antidote for paracetamol (acetaminophen) overdose, which replenishes hepatic glutathione, is:

(A) Naloxone

(B) Flumazenil

(C) N-acetylcysteine

(D) Atropine

Detailed Solutions

Q1.

Solution

Concept – reading the concentration-time curve: After an oral dose the plasma level rises, peaks, then falls.

Step 1 – locate the peak: The highest point of the curve, marked **X**, is the peak plasma level.

Step 2 – name it: This peak is the **maximum plasma concentration (Cmax)**; the time to reach it is the Tmax.

Why the other options are wrong:

- A, AUC: the whole area under the curve, a measure of total exposure, not a single peak.
- B, half-life: the time for concentration to halve, read from the falling limb.
- C, trough: the lowest level, not the peak.

Final Answer: Maximum plasma concentration (Cmax) $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q1

Q2.

Solution

Concept – first-pass metabolism: An orally absorbed drug passes through the liver before reaching the systemic circulation.

Step 1 – meaning of high first-pass: Extensive hepatic metabolism removes much of the dose on the first pass, lowering oral bioavailability.

Step 2 – match the drug: **Propranolol** is highly lipid soluble and is heavily metabolised in the liver, so its oral bioavailability is low and variable.

Why the other options are wrong:

- B, atenolol: water soluble, poorly metabolised, excreted renally, low first-pass.
- C, digoxin: mainly excreted unchanged by the kidney.
- D, gentamicin: not given orally for systemic effect; it is given parenterally and is not metabolised in the liver.

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

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – bioavailability by route: Bioavailability is the fraction of a dose reaching the systemic circulation unchanged.

Step 1 – the reference route: The **intravenous** route places the drug directly into the blood.

Step 2 – consequence: There is no absorption barrier and no first-pass loss, so bioavailability is defined as 100 per cent (F = 1).

Why the other options are wrong:

- A, oral: reduced by incomplete absorption and first-pass metabolism.
- C and D, subcutaneous and intramuscular: usually high but not defined as 100 per cent, since absorption from the site can be incomplete or slow.

Final Answer: Intravenous ⇒ B

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – antagonism on a dose-response curve: The shape of the shift reveals the type of antagonist.

Step 1 – read the graph: The dashed curve is shifted parallel to the right but reaches the **same maximum** response.

Step 2 – interpret: A parallel rightward shift with an unchanged maximum, that can be overcome by more agonist, is the signature of a **competitive (reversible) antagonist**, such as atropine at the muscarinic receptor.

Why the other options are wrong:

- B, non-competitive antagonist: lowers the maximum response, which does not happen here.
- C, full agonist: would add to the response, not shift the curve rightward.
- D, enzyme inducer: alters metabolism, not the receptor dose-response relationship shown.

Final Answer: Competitive antagonist ⇒ A

Answer: (A) Go Back to Q4

Q5.

Solution

Concept – direct muscarinic agonists: Bethanechol stimulates muscarinic receptors and resists cholinesterase.

Step 1 – target organs: It selectively stimulates smooth muscle of the bladder and gut, promoting emptying.

Step 2 – clinical use: Its main use is **postoperative non-obstructive urinary retention and bladder atony** (and paralytic ileus).

Why the other options are wrong:

- B, asthma: muscarinic agonists cause bronchoconstriction and are contraindicated.
- C, peptic ulcer: cholinergic stimulation raises acid, so it is avoided.
- D, hypertension: bethanechol is not an antihypertensive.

Final Answer: Postoperative urinary retention ⇒ A

Answer: (A) Go Back to Q5

Q6.

Solution

Concept – the renin-angiotensin system: Angiotensin-converting enzyme (ACE) makes the vasoconstrictor angiotensin II.

Step 1 – action of enalapril: ACE inhibitors **inhibit conversion of angiotensin I to angiotensin II**.

Step 2 – result: Less angiotensin II means vasodilatation and less aldosterone, lowering blood pressure.

Why the other options are wrong:

- A, AT1 receptor block: that is the mechanism of ARBs (e.g. losartan), not ACE inhibitors.
- B, beta-1 block: the mechanism of beta blockers.
- D, calcium channel block: the mechanism of drugs like amlodipine.

Final Answer: Inhibiting angiotensin I to II conversion ⇒ C

Answer: (C) Go Back to Q6

Q7.

Solution

Concept – ACE and bradykinin: ACE also degrades bradykinin.

Step 1 – effect of blocking ACE: When ACE is inhibited, **bradykinin** is not broken down and accumulates.

Step 2 – clinical link: Raised bradykinin (and substance P) in the airways provokes the characteristic dry cough, and can cause angioedema.

Why the other options are wrong:

- A, angiotensin II: its level falls, not rises, with an ACE inhibitor.
- C, histamine: not the mediator of ACE-inhibitor cough.
- D, renin: rises by feedback but does not cause the cough.

Final Answer: Bradykinin ⇒ B

Answer: (B) Go Back to Q7

Q8.

Solution

Concept – ARBs versus ACE inhibitors: Both block the renin-angiotensin system but at different points.

Step 1 – action of losartan: Losartan **blocks the angiotensin II AT1 receptor** directly.

Step 2 – practical difference: Because it does not inhibit ACE, bradykinin is not raised, so the dry cough (and angioedema) seen with ACE inhibitors is uncommon.

Why the other options are wrong:

- A, inhibits ACE: losartan does not act on ACE at all.
- C, beta blocker: wrong drug class.
- D, more cough: the opposite is true; ARBs cause less cough than ACE inhibitors.

Final Answer: Blocks AT1 receptor, cough uncommon ⇒ B

Answer: (B) Go Back to Q8

Q9.

Solution

Concept – the GABA-A receptor: It is a ligand-gated chloride channel modulated at several sites.

Step 1 – benzodiazepine site X: Benzodiazepines bind the modulatory site (marked X) distinct from the GABA site.

Step 2 – mechanism: They **increase the frequency of chloride channel opening** but only when GABA is present, enhancing inhibition (hyperpolarisation).

Why the other options are wrong:

- A, increase duration of opening: that is the action of barbiturates.
- B, open without GABA: barbiturates can at high dose; benzodiazepines cannot, which is why they are safer in overdose.
- C, block the pore: benzodiazepines potentiate, not block, the channel.

Final Answer: Increase frequency of Cl^- channel opening $\Rightarrow$ D

Answer: (D) Go Back to Q9

Q10.

Solution

Concept – reversing benzodiazepines: An antagonist at the benzodiazepine site can reverse their effect.

Step 1 – identify flumazenil: Flumazenil is a **competitive benzodiazepine-receptor antagonist**.

Step 2 – use: It reverses benzodiazepine-induced sedation and is used in benzodiazepine overdose (with caution, as it can precipitate seizures in dependent patients).

Why the other options are wrong:

- A, agonist for anxiety: flumazenil is an antagonist, not an anxiolytic.
- B, opioid antidote: that is naloxone.
- C, anticonvulsant: flumazenil can actually provoke seizures, so it is not used to treat them.

Final Answer: Benzodiazepine-receptor antagonist ⇒ D

Answer: (D) Go Back to Q10

Q11.

Solution

Concept – beta-lactam mechanism: The beta-lactam ring targets bacterial cell wall synthesis.

Step 1 – the target: Penicillins and cephalosporins bind **penicillin-binding proteins (transpeptidases)**.

Step 2 – consequence: Cross-linking of peptidoglycan is blocked, the cell wall weakens, and the bacterium lyses (bactericidal).

Why the other options are wrong:

- A, 30S protein synthesis: the mechanism of aminoglycosides and tetracyclines.
- C, DNA gyrase: the mechanism of fluoroquinolones.
- D, folate synthesis: the mechanism of sulfonamides and trimethoprim.

Final Answer: Inhibit cell wall synthesis via PBPs ⇒ B

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – cephalosporin generations: Each generation changes the spectrum of activity.

Step 1 – identify the fourth generation: **Cefepime** is a fourth-generation cephalosporin.

Step 2 – spectrum: It has broad activity, including anti-pseudomonal cover, and good stability against many beta-lactamases.

Why the other options are wrong:

- A, cefazolin: first generation.
- B, cefuroxime: second generation.
- D, cephalexin: first generation, oral.

Final Answer: Cefepime ⇒ C

Answer: (C) Go Back to Q12

Q13.

Solution

Concept – resistance to penicillins: Bacteria most often disarm the drug enzymatically.

Step 1 – the main mechanism: Bacteria produce **beta-lactamase (penicillinase)** enzymes.

Step 2 – effect: These enzymes hydrolyse the beta-lactam ring, inactivating the antibiotic; this is why inhibitors like clavulanic acid are added.

Why the other options are wrong:

- B, efflux pumps: contribute to resistance to some drugs but are not the main penicillin mechanism.
- C, reduced folate: unrelated to beta-lactams.
- D, altered ribosome: relevant to protein-synthesis inhibitors, not cell wall agents.

Final Answer: Beta-lactamase production $\Rightarrow$ A

Answer: (A) Go Back to Q13

Q14.

Solution

Concept – how NSAIDs work: Prostaglandins mediate pain, fever and inflammation.

Step 1 – the enzyme target: Aspirin and ibuprofen **inhibit cyclo-oxygenase (COX)**, both COX-1 and COX-2.

Step 2 – consequence: Less prostaglandin production gives analgesic, antipyretic and anti-inflammatory effects; COX-1 block also explains gastric irritation.

Why the other options are wrong:

- A, lipoxygenase: not the main NSAID target; it makes leukotrienes.
- B, H1 receptors: the mechanism of antihistamines.
- D, phospholipase A2: inhibited by corticosteroids, not NSAIDs.

Final Answer: Inhibiting cyclo-oxygenase (COX) $\Rightarrow$ C

Answer: (C) Go Back to Q14

Q15.

Solution

Concept – paracetamol toxicity: Overdose forms the toxic metabolite NAPQI, which depletes glutathione.

Step 1 – the antidote: **N-acetylcysteine** replenishes hepatic glutathione and detoxifies NAPQI.

Step 2 – clinical use: Given early, it prevents the hepatic necrosis of paracetamol poisoning.

Why the other options are wrong:

- A, naloxone: antidote for opioid overdose.
- B, flumazenil: antidote for benzodiazepine overdose.
- D, atropine: antidote for organophosphate (muscarinic) poisoning.

Final Answer: N-acetylcysteine $\Rightarrow$ C

Answer: (C) Go Back to Q15

Answer Key

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