# INI CET Pharmacology

## Sample Paper – 9

**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 drug, mechanism or agent.
- Attempt this practice paper in one timed sitting of about **14 minutes**, the pace the real computer-based test demands.

## General Pharmacology

**Q1.** Clearance of a drug is best defined as:

(A) The fraction of the administered dose removed unchanged in urine

(B) The volume of plasma completely cleared of the drug per unit time

(C) The time taken for the plasma concentration to fall by one half

(D) The total amount of drug eliminated after a single dose

**Q2.** Only the free (unbound) fraction of a drug is available for glomerular filtration. Which renal process can additionally clear a drug that is largely protein-bound in plasma?

(A) Passive reabsorption in the distal tubule

(B) Glomerular filtration acting alone

(C) Countercurrent multiplication in the loop of Henle

(D) Active tubular secretion in the proximal tubule

**Q3.** Aspirin (a weak acid) overdose is managed with urinary alkalinisation using sodium bicarbonate. The reason this enhances elimination is that in alkaline urine the drug is:

(A) Converted to a more lipid-soluble form that diffuses out faster

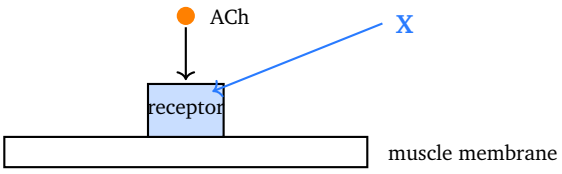
(B) Reabsorbed more readily across the tubular membrane

(C) Ionised and trapped in the tubular lumen, reducing reabsorption

(D) Filtered less because alkalinisation lowers the glomerular filtration rate

## Autonomic Nervous System

**Q4.** The receptor marked **X** on the skeletal-muscle membrane of the neuromuscular junction, opened by acetylcholine and blocked by tubocurarine, is a:




(A) Muscarinic M3 receptor

(B) Nicotinic (Nm) acetylcholine receptor

(C) Beta-2 adrenergic receptor

(D) GABA-A receptor

**Q5.** The drug of choice for long-term symptomatic treatment of myasthenia gravis, given orally for its longer duration of action, is:

(A) Edrophonium

(B) Pyridostigmine

(C) Atropine

(D) Tubocurarine

## Cardiovascular and Renal Pharmacology

**Q6.** Low-dose aspirin produces its antiplatelet effect chiefly by:

(A) Blocking the platelet ADP (P2Y12) receptor

(B) Blocking the platelet glycoprotein IIb/IIIa receptor

(C) Activating antithrombin III

(D) Irreversibly inhibiting platelet cyclo-oxygenase, lowering thromboxane A2

**Q7.** Which antiplatelet drug acts by irreversibly blocking the platelet ADP (P2Y12) receptor?

(A) Clopidogrel

(B) Abciximab

(C) Aspirin

(D) Streptokinase

**Q8.** Which drug is a fibrinolytic (thrombolytic) that activates plasminogen to dissolve an established thrombus in acute myocardial infarction, its main hazard being bleeding?

(A) Heparin

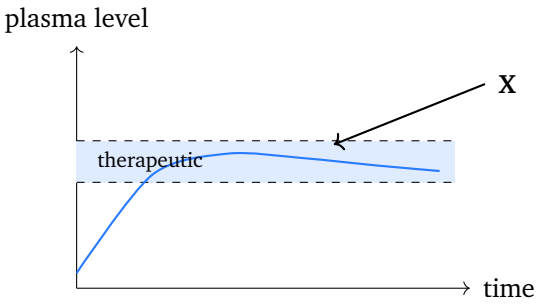
(B) Clopidogrel

(C) Alteplase (tissue plasminogen activator)

(D) Warfarin

## Central Nervous System Pharmacology

**Q9.** The plasma concentration-time graph shows a drug whose curve sits inside a very narrow therapeutic band (marked **X**); toxicity appears just above the upper line, so plasma level monitoring is mandatory. This mood stabiliser is:




(A) Lithium

(B) Diazepam

(C) Fluoxetine

(D) Amoxicillin

**Q10.** A patient on lithium develops coarse tremor and ataxia (early toxicity), and lithium must be stopped. Which of the following is an alternative mood stabiliser that can be substituted?

(A) Haloperidol

(B) Fluoxetine

(C) Propranolol

(D) Valproate (sodium valproate)

## Chemotherapy

**Q11.** Cyclophosphamide, an alkylating agent, can cause haemorrhagic cystitis through its metabolite acrolein. Which agent is co-administered to prevent this bladder toxicity?

(A) Mesna

(B) Folinic acid (leucovorin)

(C) Allopurinol

(D) Ondansetron

**Q12.** Methotrexate exerts its cytotoxic antimetabolite action mainly by inhibiting:

(A) DNA topoisomerase II

(B) Ribonucleotide reductase

(C) Dihydrofolate reductase

(D) Microtubule assembly

**Q13.** Which anticancer drug is a pyrimidine analogue (antimetabolite) whose active metabolite inhibits thymidylate synthase?

(A) 5-Fluorouracil

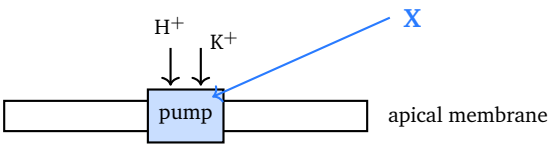
(B) Cyclophosphamide

(C) Vincristine

(D) Doxorubicin

## Autacoids, Endocrine and Miscellaneous

**Q14.** The parietal-cell enzyme marked **X** in the diagram, irreversibly inhibited by omeprazole to switch off gastric acid secretion, is the:




(A) Carbonic anhydrase

(B) H+/K+-ATPase (the proton pump)

(C) Na+/K+-ATPase

(D) Histamine H2 receptor

**Q15.** Which of the following is a histamine H2-receptor antagonist used to reduce gastric acid secretion in peptic ulcer disease?

(A) Omeprazole

(B) Misoprostol

(C) Ranitidine

(D) Sucralfate

## Detailed Solutions

**Q1.**

**Solution**

**Concept – what clearance measures:** Clearance is a rate of removal expressed as a volume, not an amount.

**Step 1 – state the definition:** Clearance is the **volume of plasma completely cleared of the drug per unit time** (units such as mL/min).

**Step 2 – link to elimination:** Rate of elimination equals clearance multiplied by the plasma concentration, so clearance governs the maintenance dose.

**Why the other options are wrong:**

- A, urinary fraction: describes renal recovery, not the clearance concept.
- C, time to halve: that is the half-life, a separate parameter.
- D, total amount eliminated: an amount, whereas clearance is a volume per time.

**Final Answer:** Volume of plasma cleared per unit time $\Rightarrow$ $\boxed{\text{B}}$

**Answer: (B)** Go Back to Q1

**Q2.**

**Solution**

**Concept – how the kidney handles drugs:** Renal drug removal uses filtration, secretion and reabsorption.

**Step 1 – limit of filtration:** Only free drug is filtered at the glomerulus, so a heavily protein-bound drug is filtered poorly.

**Step 2 – the extra route: Active tubular secretion** in the proximal tubule pumps drug (including protein-bound drug, as free drug is stripped off) into the lumen, adding to clearance.

**Why the other options are wrong:**

- A, distal reabsorption: returns drug to blood, lowering clearance.
- B, filtration alone: cannot clear the bound fraction.
- C, countercurrent multiplication: concentrates urine, it is not a drug-clearing route.

**Final Answer:** Active proximal tubular secretion $\Rightarrow$ $\boxed{\text{D}}$

**Answer: (D)** Go Back to Q2

## Q3.

### Solution

**Concept – ion trapping:** A drug is reabsorbed only in its unionised (lipid-soluble) form.

**Step 1 – aspirin is a weak acid:** In alkaline urine a weak acid loses a proton and becomes **ionised**.

**Step 2 – trapping:** The charged, water-soluble form cannot diffuse back across the tubule, so it is trapped in the lumen and excreted faster.

**Why the other options are wrong:**

- A, more lipid-soluble: alkalinisation does the opposite for a weak acid.
- B, more reabsorbed: trapping reduces reabsorption.
- D, lower filtration: alkalinisation does not meaningfully reduce the filtration rate here.

**Final Answer:** Ionised and trapped in the lumen $\Rightarrow$ C

**Answer: (C)** Go Back to Q3

## Q4.

### Solution

**Concept – the neuromuscular junction receptor:** Skeletal muscle carries a specific cholinergic receptor.

**Step 1 – identify X:** Acetylcholine opens the **nicotinic (Nm) acetylcholine receptor**, a ligand-gated cation channel on the muscle end-plate.

**Step 2 – pharmacology:** Non-depolarising blockers such as tubocurarine antagonise it, causing muscle relaxation.

**Why the other options are wrong:**

- A, M3 muscarinic: a G-protein receptor on glands and smooth muscle, not the NMJ.
- C, beta-2 adrenergic: an adrenoceptor, not activated by ACh.
- D, GABA-A: an inhibitory chloride channel in the CNS.

**Final Answer:** Nicotinic (Nm) acetylcholine receptor $\Rightarrow$ B

**Answer: (B)** Go Back to Q4

## Q5.

### Solution

**Concept – treating myasthenia gravis:** The aim is sustained acetylcholinesterase inhibition at the NMJ.

**Step 1 – name the maintenance drug:** **Pyridostigmine**, an oral anti-cholinesterase with a longer duration, is the drug of choice for long-term symptom control.

**Step 2 – role of edrophonium:** The ultra-short-acting edrophonium is used only for the diagnostic Tensilon test and to separate myasthenic from cholinergic crisis, not for maintenance.

**Why the other options are wrong:**

- A, edrophonium: too short-acting for treatment.
- C, atropine: an antimuscarinic, it would worsen weakness.
- D, tubocurarine: a neuromuscular blocker, contraindicated.

**Final Answer:** Pyridostigmine $\Rightarrow$ B

**Answer: (B)** Go Back to Q5

## Q6.

### Solution

**Concept – aspirin as an antiplatelet:** Platelets depend on thromboxane A2 for aggregation.

**Step 1 – the mechanism:** Low-dose aspirin **irreversibly acetylates platelet cyclo-oxygenase (COX-1)**, cutting thromboxane A2 synthesis.

**Step 2 – consequence:** Because platelets cannot make new enzyme, the effect lasts the platelet lifespan (about 7 to 10 days).

**Why the other options are wrong:**

- A, P2Y12 block: that is clopidogrel.
- B, GPIIb/IIIa block: that is abciximab and its class.
- C, antithrombin III: that is heparin.

**Final Answer:** Irreversible COX inhibition lowering thromboxane A2 $\Rightarrow$ D

**Answer: (D)** Go Back to Q6

## Q7.

### Solution

**Concept – ADP receptor blockers:** The P2Y12 receptor drives ADP-mediated platelet activation.

**Step 1 – name the drug: Clopidogrel**, a thienopyridine prodrug, irreversibly blocks the platelet P2Y12 (ADP) receptor.

**Step 2 – use:** It is combined with aspirin as dual antiplatelet therapy after coronary stenting.

**Why the other options are wrong:**

- B, abciximab: a GPIIb/IIIa inhibitor, not an ADP blocker.
- C, aspirin: acts through COX, not the ADP receptor.
- D, streptokinase: a thrombolytic, not an antiplatelet.

**Final Answer:** Clopidogrel $\Rightarrow$ A

**Answer: (A)** Go Back to Q7

## Q8.

### Solution

**Concept – thrombolytics:** Fibrinolytics dissolve a clot that has already formed.

**Step 1 – name the drug: Alteplase** (recombinant tissue plasminogen activator) converts plasminogen to plasmin, which lyses fibrin.

**Step 2 – risk and contrast:** Bleeding is the main hazard. Streptokinase is an older thrombolytic; both differ from anticoagulants and antiplatelets, which only prevent clot growth.

**Why the other options are wrong:**

- A, heparin: an anticoagulant, it does not lyse existing clot.
- B, clopidogrel: an antiplatelet.
- D, warfarin: an oral anticoagulant, not a thrombolytic.

**Final Answer:** Alteplase (tPA) $\Rightarrow$ C

**Answer: (C)** Go Back to Q8

## Q9.

**Solution**

**Concept – narrow therapeutic index:** Some drugs sit in a tight band between effect and toxicity.

**Step 1 – read the graph:** The curve hugging the narrow band X, with toxicity just above it, describes **lithium** (target roughly 0.6 to 1.2 mmol/L).

**Step 2 – implication:** Regular plasma level monitoring is mandatory; toxicity brings tremor, ataxia and confusion.

**Why the other options are wrong:**

- B, diazepam: a wide safety margin, no routine level monitoring.
- C, fluoxetine: an SSRI, not level-monitored this way.
- D, amoxicillin: a safe antibiotic with a broad window.

**Final Answer:** Lithium $\Rightarrow$ A

**Answer: (A)** Go Back to Q9

## Q10.

**Solution**

**Concept – alternative mood stabilisers:** When lithium cannot be used, other agents stabilise mood.

**Step 1 – name the substitute: Valproate (sodium valproate)** is an established alternative mood stabiliser in bipolar disorder.

**Step 2 – context:** Coarse tremor and ataxia signal early lithium toxicity, so switching to valproate (or carbamazepine) is appropriate.

**Why the other options are wrong:**

- A, haloperidol: an antipsychotic for acute mania, not a first-line maintenance mood stabiliser.
- B, fluoxetine: an antidepressant that can precipitate mania if used alone.
- C, propranolol: only treats the tremor symptom, not the mood disorder.

**Final Answer:** Valproate $\Rightarrow$ D

**Answer: (D)** Go Back to Q10

## Q11.

**Solution**

**Concept – protecting the bladder from alkylators:** Cyclophosphamide releases acrolein, which injures urothelium.

**Step 1 – name the protector:** **Mesna** (2-mercaptoethane sulfonate) binds acrolein in the urine, preventing haemorrhagic cystitis.

**Step 2 – support:** Adequate hydration further dilutes the toxic metabolite.

**Why the other options are wrong:**

- B, folinic acid: rescues cells from methotrexate, not from acrolein.
- C, allopurinol: prevents tumour-lysis hyperuricaemia, not cystitis.
- D, ondansetron: controls chemotherapy nausea, not bladder toxicity.

**Final Answer:** Mesna $\Rightarrow$ $\boxed{\text{A}}$

**Answer: (A)** Go Back to Q11

## Q12.

**Solution**

**Concept – antifolate action:** Folate is needed to make thymidine and purines.

**Step 1 – the target:** Methotrexate inhibits **dihydrofolate reductase**, blocking regeneration of tetrahydrofolate and halting DNA synthesis.

**Step 2 – rescue:** Folinic acid (leucovorin) bypasses the block to rescue normal cells after high-dose therapy.

**Why the other options are wrong:**

- A, topoisomerase II: inhibited by etoposide and anthracyclines.
- B, ribonucleotide reductase: inhibited by hydroxyurea.
- D, microtubules: the target of vinca alkaloids and taxanes.

**Final Answer:** Dihydrofolate reductase $\Rightarrow$ $\boxed{\text{C}}$

**Answer: (C)** Go Back to Q12

## Q13.

**Solution**

**Concept – antimetabolite analogues:** Pyrimidine and purine analogues block nucleotide synthesis.

**Step 1 – name the drug: 5-Fluorouracil**, a pyrimidine analogue, is converted to a metabolite that inhibits thymidylate synthase, starving cells of thymidine.

**Step 2 – companion:** 6-Mercaptopurine is the analogous purine antimetabolite, illustrating the same class.

**Why the other options are wrong:**

- B, cyclophosphamide: an alkylating agent.
- C, vincristine: a microtubule inhibitor.
- D, doxorubicin: an anthracycline that intercalates DNA and blocks topoisomerase II.

**Final Answer:** 5-Fluorouracil $\Rightarrow$ A

**Answer: (A)** Go Back to Q13

## Q14.

**Solution**

**Concept – the gastric proton pump:** The final step of acid secretion is a membrane pump.

**Step 1 – identify X:** Omeprazole irreversibly inhibits the parietal-cell **H+/K+-ATPase (proton pump)**, the enzyme that secretes H+ into the stomach.

**Step 2 – effect:** Blocking the final common step gives powerful, long-lasting acid suppression.

**Why the other options are wrong:**

- A, carbonic anhydrase: supplies H+ but is not the drug target here.
- C, Na+/K+-ATPase: a basolateral pump, not inhibited by omeprazole.
- D, H2 receptor: the target of ranitidine, not of a proton-pump inhibitor.

**Final Answer:** H+/K+-ATPase (proton pump) $\Rightarrow$ B

**Answer: (B)** Go Back to Q14

## Q15.

**Solution**

**Concept – classes of acid-lowering drugs:** H2 blockers and proton-pump inhibitors act at different points.

**Step 1 – name the H2 blocker: Ranitidine** (like famotidine) competitively blocks the parietal-cell histamine H2 receptor, reducing acid output.

**Step 2 – wider therapy:** For H. pylori-associated ulcers, a PPI plus two antibiotics (triple therapy) is used, showing how classes are combined.

**Why the other options are wrong:**

- A, omeprazole: a proton-pump inhibitor, not an H2 blocker.
- B, misoprostol: a prostaglandin analogue.
- D, sucralfate: a mucosal-coating protective agent.

**Final Answer:** Ranitidine $\Rightarrow$ C

**Answer: (C)** Go Back to Q15

# Answer Key

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