

INI CET Pharmacology

Sample Paper – 8

Duration: 14 Minutes

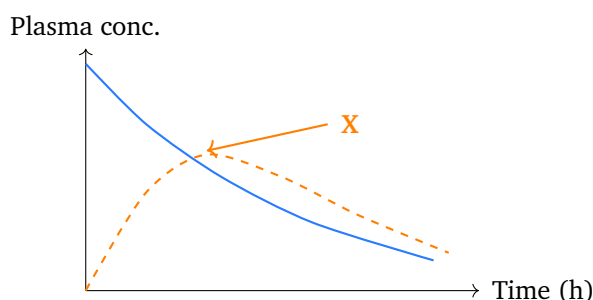
Maximum Marks: 15

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 clinical fact.
- 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 compares plasma drug concentration after two routes of administration. The dashed curve marked **X**, which starts from zero, rises to a delayed peak and then falls, represents drug given by which route?



(A) Intravenous bolus



- (B) Oral
- (C) A drug that is not absorbed at all
- (D) Direct intra-arterial injection

Q2. Two brands of the same drug are said to be bioequivalent when they:

- (A) Contain exactly the same inactive excipients
- (B) Are manufactured by the same pharmaceutical company
- (C) Cost the same amount per tablet
- (D) Show no significant difference in the rate and extent of absorption (similar C_{max} and AUC)

Q3. When a drug crosses a membrane by simple passive diffusion following first-order kinetics, the rate of absorption is:

- (A) Directly proportional to the concentration gradient across the membrane
- (B) Constant and completely independent of concentration
- (C) Dependent on a saturable carrier protein
- (D) Always faster than any intravenous route

Autonomic Nervous System

Q4. Clonidine, a centrally acting antihypertensive, lowers blood pressure mainly by:

- (A) Stimulating central alpha-2 adrenergic receptors and reducing sympathetic outflow
- (B) Blocking peripheral alpha-1 adrenergic receptors
- (C) Selectively blocking beta-1 receptors in the heart
- (D) Directly relaxing vascular smooth muscle through nitric oxide

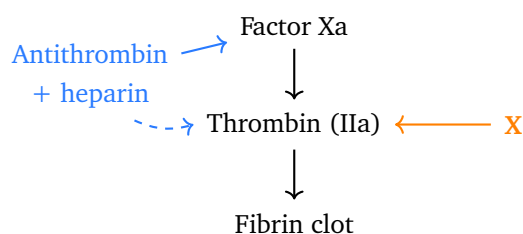
Q5. Which centrally acting sympatholytic is regarded as a well-tried, preferred antihypertensive for use during pregnancy?



- (A) Clonidine
- (B) Methyldopa
- (C) Reserpine
- (D) Sodium nitroprusside

Cardiovascular and Renal Pharmacology

Q6. In the coagulation schematic, the factor marked **X** (thrombin, IIa) is strongly inactivated when heparin binds antithrombin. Heparin therefore acts as an anticoagulant by:



- (A) Activating antithrombin III, which then inhibits thrombin and factor Xa
 - (B) Antagonising the vitamin K dependent clotting factors
 - (C) Irreversibly inhibiting platelet cyclo-oxygenase
 - (D) Dissolving fibrin that has already formed
- Q7.** Oral warfarin therapy is monitored using which laboratory test?
- (A) Activated partial thromboplastin time (aPTT)
 - (B) Bleeding time
 - (C) Prothrombin time expressed as the INR
 - (D) Platelet count
- Q8.** The specific antibody antidote used to rapidly reverse dabigatran, a direct thrombin inhibitor of the DOAC class, is:
- (A) Protamine sulphate
 - (B) Vitamin K



- (C) Fresh frozen plasma alone
- (D) Idarucizumab

Central Nervous System Pharmacology

- Q9.** Levodopa is almost always combined with carbidopa in the treatment of Parkinson's disease because carbidopa:
- (A) Itself crosses the blood-brain barrier and is converted to dopamine centrally
 - (B) Acts directly as a dopamine receptor agonist
 - (C) Blocks the central breakdown of dopamine by monoamine oxidase
 - (D) Inhibits peripheral dopa decarboxylase, so more levodopa reaches the brain with fewer peripheral side effects
- Q10.** After years of levodopa therapy, a patient develops abrupt, unpredictable swings between mobility and sudden immobility. This complication is known as the:
- (A) On-off phenomenon
 - (B) First-dose hypotension
 - (C) Serotonin syndrome
 - (D) Neuroleptic malignant syndrome

Chemotherapy

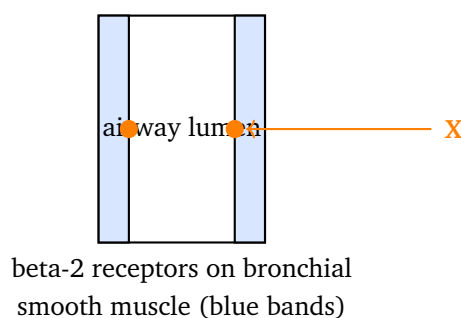
- Q11.** A patient taking metronidazole develops flushing, throbbing headache, nausea and palpitations shortly after drinking alcohol. This disulfiram-like reaction occurs because metronidazole:
- (A) Increases the rate of alcohol absorption from the gut
 - (B) Blocks central dopamine receptors
 - (C) Inhibits aldehyde dehydrogenase, so acetaldehyde accumulates
 - (D) Displaces alcohol from plasma protein binding sites



- Q12.** Which drug is a broad-spectrum anthelmintic that acts by binding parasitic beta-tubulin and blocking the worm's glucose uptake?
- (A) Metronidazole
 - (B) Chloroquine
 - (C) Albendazole
 - (D) Amphotericin B
- Q13.** Metronidazole is regarded as a drug of choice for which of the following infections?
- (A) Falciparum malaria
 - (B) Amoebiasis and giardiasis
 - (C) Roundworm (Ascaris) infestation
 - (D) Tuberculosis

Autacoids, Endocrine and Miscellaneous

- Q14.** In the airway schematic, the drug marked X binds the beta-2 receptors on bronchial smooth muscle (blue bands) and produces rapid bronchodilation, acting as a quick reliever in an acute asthma attack. This drug is:



- (A) Inhaled budesonide
- (B) Salbutamol
- (C) Montelukast
- (D) Sodium cromoglycate



- Q15.** Which of the following is used mainly as a long-term controller (preventer) rather than an acute reliever in the management of bronchial asthma?
- (A) Salbutamol
 - (B) Injectable adrenaline
 - (C) Inhaled corticosteroid such as budesonide
 - (D) Ipratropium bromide given for sudden breathlessness



Detailed Solutions**Q1.****Solution**

Concept – shape of the concentration-time curve: The route controls how fast drug enters blood.

Step 1 – read curve X: Curve X begins at zero, meaning no drug is in blood at time zero.

Step 2 – interpret the rise: It climbs slowly to a delayed peak, because the drug must first be absorbed across the gut wall.

Step 3 – match the route: A lag, a gentle absorption phase and a delayed peak are the signature of the **oral** route.

Why the other options are wrong:

- A, intravenous bolus: gives the solid curve that starts at the highest value at time zero and only falls.
- C, not absorbed: the curve would stay flat at zero.
- D, intra-arterial: also delivers drug directly into blood, without an absorption lag.

Final Answer: Oral route $\Rightarrow$ **B**

Answer: (B) [Go Back to Q1](#)

Q2.**Solution**

Concept – meaning of bioequivalence: It compares how two products behave in the body.

Step 1 – define the term: Two brands are **bioequivalent** when they deliver the same active drug at the same rate and to the same extent.

Step 2 – name the measures: This is judged by comparing peak concentration (C_{max}) and total exposure (area under the curve, AUC).

Step 3 – conclude: If C_{max} and AUC show no significant difference, the products are bioequivalent.

Why the other options are wrong:

- A, same excipients: bioequivalent brands often use different excipients.



- B, same company: the manufacturer is irrelevant to bioequivalence.
- C, same cost: price has nothing to do with drug behaviour in the body.

Final Answer: Similar rate and extent of absorption $\Rightarrow$ D

Answer: (D) [Go Back to Q2](#)

Q3.

Solution

Concept – first-order absorption: Passive diffusion depends on the gradient.

Step 1 – state the rule: In first-order kinetics, the rate of transfer is **proportional to the concentration gradient** across the membrane.

Step 2 – explain: A larger difference in concentration between the two sides drives a faster rate of diffusion.

Step 3 – note the fraction: A constant fraction, not a constant amount, is absorbed per unit time.

Why the other options are wrong:

- B, constant rate: that describes zero-order kinetics, not first-order.
- C, saturable carrier: describes carrier-mediated (facilitated or active) transport.
- D, faster than intravenous: impossible, since the intravenous route places drug straight into blood.

Final Answer: Proportional to the concentration gradient $\Rightarrow$ A

Answer: (A) [Go Back to Q3](#)

Q4.

Solution

Concept – centrally acting sympatholytics: Clonidine works inside the brainstem.

Step 1 – name the target: Clonidine stimulates **central alpha-2 adrenergic receptors** in the vasomotor centre.

Step 2 – trace the effect: Alpha-2 stimulation reduces the sympathetic outflow leaving the brainstem.

Step 3 – result: Less sympathetic drive lowers heart rate, cardiac output and



peripheral resistance, so blood pressure falls.

Why the other options are wrong:

- B, peripheral alpha-1 block: that is the action of prazosin, not clonidine.
- C, beta-1 block: that describes atenolol and similar beta-blockers.
- D, direct nitric oxide: that is the mechanism of nitrates and nitroprusside.

Final Answer: Central alpha-2 stimulation $\Rightarrow$ A

Answer: (A) [Go Back to Q4](#)

Q5.

Solution

Concept – antihypertensive in pregnancy: Safety data guide the choice.

Step 1 – name the drug: **Methyldopa**, an alpha-2 agonist converted centrally to methylnoradrenaline, is a long-used, well-tried antihypertensive in pregnancy.

Step 2 – reason: It has the longest safety record and does not harm fetal growth.

Why the other options are wrong:

- A, clonidine: effective but limited by rebound hypertension and less pregnancy data.
- C, reserpine: causes depression and nasal stuffiness, and is largely obsolete.
- D, sodium nitroprusside: reserved for hypertensive emergencies and risks fetal cyanide toxicity.

Final Answer: Methyldopa $\Rightarrow$ B

Answer: (B) [Go Back to Q5](#)

Q6.

Solution

Concept – mechanism of heparin: Heparin needs a natural cofactor.

Step 1 – read the schematic: Heparin binds **antithrombin III** and greatly speeds its inactivation of thrombin (the factor marked X) and factor Xa.

Step 2 – consequence: With thrombin blocked, fibrinogen is not converted to fibrin, so no clot forms.

Step 3 – clinical link: This action is immediate and is monitored by the aPTT.



Why the other options are wrong:

- B, vitamin K antagonism: that is the mechanism of warfarin.
- C, platelet cyclo-oxygenase: that describes aspirin.
- D, dissolving formed fibrin: that is fibrinolysis by streptokinase or alteplase.

Final Answer: Activates antithrombin III $\Rightarrow$ **A**

Answer: (A) [Go Back to Q6](#)

Q7.

Solution

Concept – monitoring anticoagulants: Each drug has its own test.

Step 1 – name the test: Warfarin is monitored by the **prothrombin time, reported as the INR**.

Step 2 – reason: Warfarin lowers the vitamin K dependent factors (II, VII, IX, X), and the prothrombin time is most sensitive to factor VII in the extrinsic pathway.

Step 3 – target: A typical therapeutic INR is about 2 to 3.

Why the other options are wrong:

- A, aPTT: this is used to monitor unfractionated heparin.
- B, bleeding time: reflects platelet function, not warfarin effect.
- D, platelet count: unaffected by warfarin.

Final Answer: Prothrombin time / INR $\Rightarrow$ **C**

Answer: (C) [Go Back to Q7](#)

Q8.

Solution

Concept – reversing a DOAC: Each anticoagulant has a specific antidote.

Step 1 – match dabigatran: Dabigatran is a direct thrombin inhibitor, and its specific reversal agent is **idarucizumab**, a monoclonal antibody fragment.

Step 2 – action: Idarucizumab binds dabigatran tightly and neutralises its anticoagulant effect within minutes.

Why the other options are wrong:

- A, protamine sulphate: reverses heparin, not dabigatran.



- B, vitamin K: reverses warfarin.
- C, fresh frozen plasma alone: a nonspecific measure, not the specific antidote.

Final Answer: Idarucizumab ⇒ D

Answer: (D) [Go Back to Q8](#)

Q9.

Solution

Concept – why carbidopa is added: The problem is peripheral conversion.

Step 1 – state the issue: Given alone, most levodopa is converted to dopamine in the periphery by dopa decarboxylase, causing nausea and hypotension and leaving little to reach the brain.

Step 2 – name carbidopa's action: Carbidopa **inhibits peripheral dopa decarboxylase** but does not cross the blood-brain barrier.

Step 3 – result: More levodopa survives to enter the brain, where it is converted to dopamine, and peripheral side effects fall.

Why the other options are wrong:

- A, crosses into brain: carbidopa deliberately does not cross the blood-brain barrier.
- B, dopamine agonist: that describes drugs such as bromocriptine or pramipexole.
- C, blocks central MAO: that is the action of selegiline.

Final Answer: Inhibits peripheral dopa decarboxylase ⇒ D

Answer: (D) [Go Back to Q9](#)

Q10.

Solution

Concept – long-term levodopa complications: Chronic therapy brings response fluctuations.

Step 1 – name the phenomenon: Abrupt swings between mobility (on) and sudden immobility (off) are the **on-off phenomenon**.

Step 2 – explain: It reflects fluctuating brain dopamine as the disease advances



and levodopa's short action becomes unpredictable.

Why the other options are wrong:

- B, first-dose hypotension: an early effect of alpha-blockers such as prazosin.
- C, serotonin syndrome: caused by excess serotonergic drugs, not levodopa.
- D, neuroleptic malignant syndrome: linked to dopamine-blocking antipsychotics or abrupt levodopa withdrawal.

Final Answer: On-off phenomenon $\Rightarrow$ A

Answer: (A) [Go Back to Q10](#)

Q11.

Solution

Concept – metronidazole and alcohol: The interaction mimics disulfiram.

Step 1 – name the enzyme: Metronidazole **inhibits aldehyde dehydrogenase**, the enzyme that clears acetaldehyde.

Step 2 – trace the reaction: When alcohol is taken, acetaldehyde accumulates and causes flushing, headache, vomiting and palpitations.

Step 3 – advice: Patients are warned to avoid alcohol during, and for a few days after, metronidazole.

Why the other options are wrong:

- A, faster alcohol absorption: not the mechanism of the reaction.
- B, dopamine block: unrelated to the alcohol interaction.
- D, protein displacement: does not explain acetaldehyde build-up.

Final Answer: Inhibits aldehyde dehydrogenase $\Rightarrow$ C

Answer: (C) [Go Back to Q11](#)

Q12.

Solution

Concept – anthelmintic mechanism: Benzimidazoles target the worm's cytoskeleton.

Step 1 – name the drug: **Albendazole** is a broad-spectrum benzimidazole anthelmintic.



Step 2 – state the mechanism: It binds parasitic beta-tubulin, blocks microtubule assembly and prevents the worm's glucose uptake, starving it of energy.

Why the other options are wrong:

- A, metronidazole: an antiprotozoal and antianaerobic, not an anthelmintic.
- B, chloroquine: an antimalarial.
- D, amphotericin B: an antifungal.

Final Answer: Albendazole ⇒

Answer: (C) [Go Back to Q12](#)

Q13.

Solution

Concept – clinical uses of metronidazole: It targets anaerobes and certain protozoa.

Step 1 – match the infection: Metronidazole is a drug of choice for **amoebiasis and giardiasis**, as well as trichomoniasis and anaerobic infections.

Step 2 – explain: Its nitro group is reduced inside the parasite to reactive intermediates that damage microbial DNA.

Why the other options are wrong:

- A, falciparum malaria: treated with artemisinin combinations, not metronidazole.
- C, roundworm: treated with albendazole or mebendazole.
- D, tuberculosis: treated with rifampicin, isoniazid and companions.

Final Answer: Amoebiasis and giardiasis ⇒

Answer: (B) [Go Back to Q13](#)

Q14.

Solution

Concept – the quick reliever in asthma: Beta-2 agonists open the airway fast.

Step 1 – identify X: The drug marked X binds **beta-2 receptors** on bronchial smooth muscle.

Step 2 – trace the effect: Beta-2 stimulation raises cyclic AMP, relaxes the smooth



muscle and rapidly widens the airway.

Step 3 – name the drug: A short-acting beta-2 agonist that does this is **salbutamol**, the classic reliever in an acute attack.

Why the other options are wrong:

- A, budesonide: an inhaled corticosteroid used as a controller, with a slow anti-inflammatory action.
- C, montelukast: a leukotriene antagonist used for prevention, not quick relief.
- D, sodium cromoglycate: a mast-cell stabiliser used prophylactically.

Final Answer: Salbutamol ⇒

Answer: (B) [Go Back to Q14](#)

Q15.

Solution

Concept – reliever versus controller: Controllers reduce underlying inflammation.

Step 1 – name the controller: An **inhaled corticosteroid** such as budesonide is the mainstay long-term controller in asthma.

Step 2 – explain: It suppresses airway inflammation over days to weeks, cutting the frequency and severity of attacks, but does not relieve a sudden attack.

Why the other options are wrong:

- A, salbutamol: a short-acting reliever, not a controller.
- B, injectable adrenaline: an emergency rescue in severe attacks and anaphylaxis.
- D, ipratropium for sudden breathlessness: used as a reliever, particularly in COPD, not as the daily preventer.

Final Answer: Inhaled corticosteroid ⇒

Answer: (C) [Go Back to Q15](#)



Answer Key

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

