INI CET Pharmacology

Sample Paper – 7

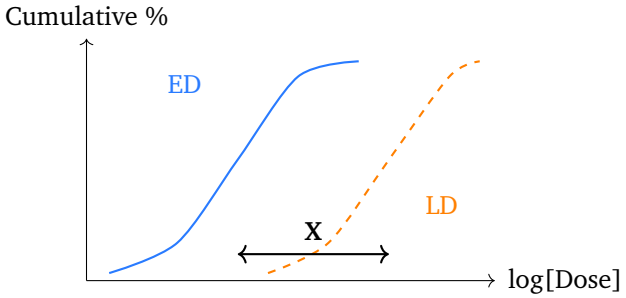
Duration: 14 Minutes **Maximum Marks: 15**

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 figure shows two quantal dose-response curves for a drug: the therapeutic effect (solid) and the lethal effect (dashed). The therapeutic index, related to the separation marked **X**, is defined as:



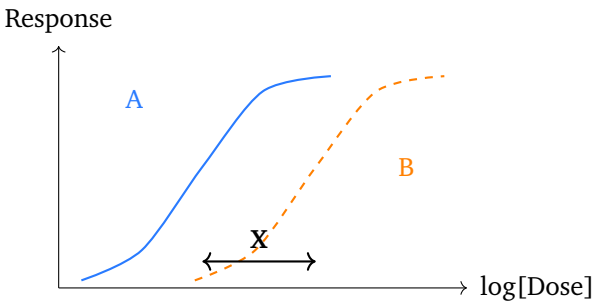
(A) ED50 / LD50

(B) LD50 – ED50

(C) ED50 × LD50

(D) LD50 / ED50

Q2. The graded dose-response curves of two drugs acting on the same receptor are shown. Drug A lies to the left of drug B (leftward shift marked **X**), while both reach the same maximum response. This means:



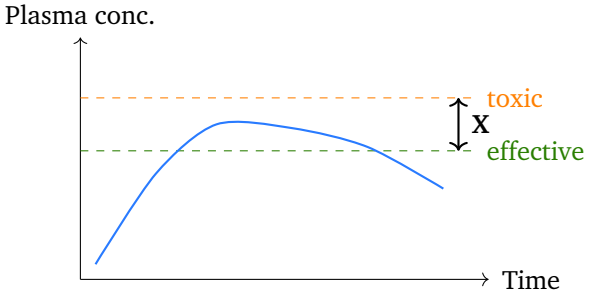
(A) Drug A is more potent than drug B

(B) Drug A is less potent than drug B

(C) Drug A has lower efficacy than drug B

(D) Drug A has higher efficacy than drug B

Q3. The plasma concentration-time curve shows a drug whose therapeutic band lies just below its toxic threshold, giving the very narrow therapeutic window marked **X**. Which of the following drugs is the classic example of such a narrow therapeutic index?



(A) Penicillin

(B) Amoxicillin

(C) Digoxin

(D) Paracetamol

Autonomic Nervous System

Q4. Succinylcholine, a depolarising neuromuscular blocker, produces its phase I block at the motor end-plate by:

(A) Persistent depolarisation of the motor end-plate

(B) Competitive antagonism at nicotinic receptors

(C) Inhibiting the release of acetylcholine from the nerve terminal

(D) Reversibly inhibiting acetylcholinesterase

Q5. The most feared adverse effect of succinylcholine in a patient with extensive burns or a crush injury is:

(A) Life-threatening hyperkalaemia

(B) Marked hypokalaemia

(C) Bronchodilation

(D) Hypoglycaemia

Cardiovascular and Renal Pharmacology

Q6. Statins (for example atorvastatin) lower plasma LDL cholesterol mainly by inhibiting which enzyme in the hepatic cholesterol synthesis pathway?

(A) Lipoprotein lipase

(B) Acetyl-CoA carboxylase

(C) HMG-CoA reductase

(D) Squalene epoxidase

Q7. The most serious muscle-related adverse effect of statin therapy, marked by severe myopathy with a very high creatine kinase and risk of acute kidney injury, is:

(A) Acute gouty arthritis

(B) Rhabdomyolysis

(C) Aplastic anaemia

(D) Pulmonary fibrosis

Q8. Which lipid-lowering drug acts mainly by inhibiting the intestinal absorption of cholesterol at the brush-border NPC1L1 transporter?

(A) Atorvastatin

(B) Gemfibrozil

(C) Niacin

(D) Ezetimibe

Central Nervous System Pharmacology

Q9. Morphine produces its analgesic and euphoric effects mainly by acting as an agonist at which receptor?

(A) GABA-A receptor

(B) Mu (μ) opioid receptor

(C) NMDA glutamate receptor

(D) Dopamine D2 receptor

Q10. A patient with opioid overdose develops pinpoint pupils and severe respiratory depression. The specific antidote that rapidly reverses this is:

(A) Flumazenil

(B) Atropine

(C) Naloxone

(D) Neostigmine

Chemotherapy

Q11. Chloroquine kills the intra-erythrocytic malaria parasite mainly by:

(A) Inhibiting dihydrofolate reductase

(B) Blocking the mitochondrial electron transport chain

(C) Inhibiting haem polymerisation, so toxic free haem accumulates

(D) Inhibiting bacterial DNA gyrase

Q12. The recommended first-line treatment for uncomplicated *Plasmodium falciparum* malaria is:

(A) Artemisinin-based combination therapy (ACT)

(B) Chloroquine monotherapy

(C) Primaquine alone

(D) Doxycycline alone

Q13. Which antimalarial drug is most likely to cause acute haemolytic anaemia in a patient with glucose-6-phosphate dehydrogenase (G6PD) deficiency?

(A) Chloroquine

(B) Artemisinin

(C) Quinine

(D) Primaquine

Autacoids, Endocrine and Miscellaneous

Q14. Which prostaglandin (PGE1) analogue is used as an oxytocic to prevent and treat postpartum haemorrhage (PPH)?

(A) Ritodrine

(B) Misoprostol

(C) Nifedipine

(D) Salbutamol

Q15. Which drug is used as a tocolytic to suppress uterine contractions and delay premature labour?

(A) Oxytocin

(B) Nifedipine

(C) Misoprostol

(D) Ergometrine

Detailed Solutions

Q1.

Solution

Concept – therapeutic index (TI): The TI measures the margin of safety of a drug from its quantal dose-response data.

Step 1 – read the two curves: The solid curve plots the cumulative percentage showing the therapeutic effect (ED); the dashed curve plots the percentage showing the lethal effect (LD).

Step 2 – take the two midpoints: ED50 is the dose that is effective in 50% of the population; LD50 is the dose that is lethal in 50% of the population. The separation **X** between them reflects safety.

Step 3 – write the ratio: Therapeutic index = LD50 / ED50. A larger ratio means a wider safety margin.

Why the other options are wrong:

- A, ED50/LD50: the inverted ratio, which would fall (not rise) with greater safety.
- B, LD50 – ED50: a difference, not the standard index.
- C, ED50 × LD50: a product with no pharmacological meaning.

Final Answer: LD50 / ED50 ⇒ D

Answer: (D) Go Back to Q1

Q2.

Solution

Concept – potency versus efficacy: Position on the dose axis reflects potency; height of the plateau reflects efficacy.

Step 1 – read the leftward shift: Drug A reaches any given response at a lower dose than drug B, so its curve sits to the left (shift **X**).

Step 2 – interpret potency: A drug that produces the same effect at a smaller dose is **more potent**. Hence drug A is more potent than drug B.

Step 3 – check efficacy: Both curves reach the same maximum response, so their efficacy is equal; only potency differs.

Why the other options are wrong:

- B, less potent: the leftward drug is the more potent one.
- C and D, efficacy claims: efficacy is the plateau height, which is identical here.

Final Answer: Drug A is more potent than drug B $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – narrow therapeutic index drugs: A narrow window means the effective and toxic plasma levels lie close together.

Step 1 – read the figure: The therapeutic band sits just below the toxic threshold, so the gap **X** is very small; small dose changes can cause toxicity.

Step 2 – match the drug: Digoxin is a classic narrow-therapeutic-index drug and needs plasma level monitoring.

Step 3 – confirm: Its toxic range (arrhythmias, nausea, visual changes) is close to the therapeutic range, unlike the penicillins.

Why the other options are wrong:

- A and B, penicillin and amoxicillin: very wide safety margins.
- D, paracetamol: safe at therapeutic doses; toxicity needs a large overdose.

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

Answer: (C) Go Back to Q3

Q4.

Solution

Concept – depolarising blockade: Succinylcholine acts like acetylcholine but is broken down slowly.

Step 1 – name the mechanism: It binds nicotinic receptors and produces **persistent depolarisation** of the motor end-plate (phase I block).

Step 2 – consequence: The end-plate stays depolarised, the sodium channels cannot reset, and the muscle cannot respond; a brief fasciculation is followed by flaccid paralysis.

Step 3 – reversal note: Because the block is depolarising, an anticholinesterase

such as neostigmine does not reverse it (it can worsen phase I block), unlike the competitive non-depolarisers.

Why the other options are wrong:

- B, competitive antagonism: the mechanism of non-depolarisers (d-tubocurarine, vecuronium), reversible by neostigmine.
- C, blocking acetylcholine release: mechanism of botulinum toxin.
- D, inhibiting acetylcholinesterase: mechanism of neostigmine, not succinylcholine.

Final Answer: Persistent depolarisation of the motor end-plate $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q4

Q5.

Solution

Concept – succinylcholine adverse effects: Depolarisation drives potassium out of muscle cells.

Step 1 – name the danger: In burns, crush injury or prolonged immobility, extra-junctional receptors multiply, so succinylcholine causes a large potassium efflux and **life-threatening hyperkalaemia**.

Step 2 – consequence: The rise in serum potassium can trigger cardiac arrhythmias and arrest.

Step 3 – related caution: Succinylcholine is also a trigger of malignant hyperthermia in susceptible patients.

Why the other options are wrong:

- B, hypokalaemia: the opposite of what occurs.
- C, bronchodilation: not a feature; histamine release may instead cause bronchospasm.
- D, hypoglycaemia: unrelated to the drug.

Final Answer: Life-threatening hyperkalaemia $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q5

Q6.

Solution

Concept – statin mechanism: Statins act at the rate-limiting step of cholesterol synthesis.

Step 1 – name the enzyme: They competitively inhibit **HMG-CoA reductase**, which converts HMG-CoA to mevalonate.

Step 2 – downstream effect: Falling hepatic cholesterol up-regulates LDL receptors, so more LDL is cleared from the blood.

Step 3 – result: Plasma LDL cholesterol falls, reducing cardiovascular risk.

Why the other options are wrong:

- A, lipoprotein lipase: hydrolyses triglycerides; activated by fibrates, not blocked by statins.
- B, acetyl-CoA carboxylase: a fatty-acid synthesis enzyme, not the statin target.
- D, squalene epoxidase: the target of antifungal terbinafine.

Final Answer: HMG-CoA reductase $\Rightarrow$ C

Answer: (C) Go Back to Q6

Q7.

Solution

Concept – statin muscle toxicity: Statins can injure skeletal muscle, from mild aches to severe breakdown.

Step 1 – name the severe form: Rhabdomyolysis is the dangerous end of the spectrum, with muscle breakdown, a very high creatine kinase and myoglobinuria.

Step 2 – consequence: Released myoglobin can precipitate in renal tubules and cause acute kidney injury.

Step 3 – risk factors: Risk rises with high doses and with drugs that raise statin levels (for example fibrates such as gemfibrozil).

Why the other options are wrong:

- A, gout: not a statin effect.
- C, aplastic anaemia: not associated with statins.
- D, pulmonary fibrosis: a toxicity of drugs like amiodarone and bleomycin, not statins.

Final Answer: Rhabdomyolysis $\Rightarrow$ B

Answer: (B) Go Back to Q7

Q8.

Solution

Concept – lipid-lowering drug classes: Each class acts at a different point in cholesterol handling.

Step 1 – match the mechanism: Ezetimibe blocks the NPC1L1 transporter in the intestinal brush border, reducing dietary and biliary cholesterol absorption.

Step 2 – use: It is often added to a statin for extra LDL lowering.

Step 3 – contrast: This is a different site from the statins, which act inside the liver.

Why the other options are wrong:

- A, atorvastatin: inhibits HMG-CoA reductase in the liver.
- B, gemfibrozil: a fibrate that activates PPAR-alpha to lower triglycerides.
- C, niacin: reduces hepatic VLDL secretion and raises HDL.

Final Answer: Ezetimibe $\Rightarrow$ D

Answer: (D) Go Back to Q8

Q9.

Solution

Concept – opioid receptors: Morphine and related opioids act on G-protein coupled opioid receptors.

Step 1 – name the receptor: Morphine is a strong agonist at the **mu (μ) opioid receptor**.

Step 2 – mechanism: Mu activation opens potassium channels and closes calcium channels, reducing neuronal firing and neurotransmitter release along the pain pathway.

Step 3 – effects: This gives analgesia, euphoria, respiratory depression, miosis and constipation.

Why the other options are wrong:

- A, GABA-A: the target of benzodiazepines and barbiturates.

- C, NMDA: blocked by ketamine, not the main morphine site.
- D, dopamine D2: relevant to antipsychotics, not morphine analgesia.

Final Answer: Mu opioid receptor ⇒ B

Answer: (B) Go Back to Q9

Q10.

Solution

Concept – opioid overdose antidote: Opioid toxicity is reversed by blocking the opioid receptor.

Step 1 – read the picture: Pinpoint pupils with respiratory depression is the classic opioid overdose triad (with coma).

Step 2 – name the antidote: Naloxone, a pure opioid receptor antagonist, rapidly displaces the opioid and restores breathing.

Step 3 – caution: Its short duration may need repeat dosing, and it can precipitate acute withdrawal in dependent patients.

Why the other options are wrong:

- A, flumazenil: reverses benzodiazepines, not opioids.
- B, atropine: an antimuscarinic, used in organophosphate or bradycardia, not opioid overdose.
- D, neostigmine: an anticholinesterase, used to reverse non-depolarising blockers.

Final Answer: Naloxone ⇒ C

Answer: (C) Go Back to Q10

Q11.

Solution

Concept – chloroquine action: The parasite digests haemoglobin and must detoxify the released haem.

Step 1 – name the mechanism: Chloroquine concentrates in the parasite food vacuole and **inhibits haem polymerisation**, blocking the conversion of toxic free haem into inert hemozoin.

Step 2 – consequence: Toxic free haem accumulates and kills the intra-

erythrocytic parasite.

Step 3 – resistance note: Resistant *P. falciparum* pumps chloroquine out of the vacuole (PfCRT transporter), lowering its concentration at the target.

Why the other options are wrong:

- A, dihydrofolate reductase: the target of pyrimethamine and proguanil.
- B, mitochondrial electron transport: the target of atovaquone.
- D, DNA gyrase: the target of fluoroquinolone antibiotics.

Final Answer: Inhibiting haem polymerisation ⇒ C

Answer: (C) Go Back to Q11

Q12.

Solution

Concept – first-line falciparum treatment: Combinations delay resistance and clear parasites quickly.

Step 1 – name the regimen: Uncomplicated *P. falciparum* malaria is treated with **artemisinin-based combination therapy (ACT)**.

Step 2 – rationale: The fast-acting artemisinin rapidly reduces the parasite load, while a longer-acting partner drug clears the remainder and protects against resistance.

Step 3 – practice point: Monotherapy is avoided because it promotes artemisinin resistance.

Why the other options are wrong:

- B, chloroquine monotherapy: widespread falciparum resistance makes it unreliable.
- C, primaquine alone: used for radical cure of liver hypnozoites, not blood-stage falciparum.
- D, doxycycline alone: only a slow-acting partner or prophylactic, not first-line therapy.

Final Answer: Artemisinin-based combination therapy (ACT) ⇒ A

Answer: (A) Go Back to Q12

Q13.

Solution

Concept – G6PD deficiency and oxidant drugs: G6PD-deficient red cells cannot handle oxidative stress.

Step 1 – name the drug: Primaquine is a strong oxidant and causes acute haemolytic anaemia in G6PD deficiency.

Step 2 – mechanism: Without enough NADPH the cells cannot regenerate glutathione, so oxidised haemoglobin precipitates and red cells lyse.

Step 3 – practice point: Screen for G6PD deficiency before giving primaquine for radical cure of vivax malaria.

Why the other options are wrong:

- A, chloroquine: not a typical trigger of G6PD haemolysis at usual doses.
- B, artemisinin: not the classic oxidant offender here.
- C, quinine: mainly causes cinchonism and rarely blackwater fever, not the classic G6PD trigger.

Final Answer: Primaquine $\Rightarrow$ D

Answer: (D) Go Back to Q13

Q14.

Solution

Concept – prostaglandin oxytocics: Some prostaglandin analogues contract the uterus.

Step 1 – name the drug: Misoprostol, a PGE1 analogue, contracts uterine smooth muscle and is used to prevent and treat postpartum haemorrhage (PPH).

Step 2 – advantage: It is cheap, heat-stable and can be given orally or rectally, useful where injectable oxytocin storage is difficult.

Step 3 – other uses: It is also used for medical abortion (with mifepristone) and to protect the gastric mucosa.

Why the other options are wrong:

- A, ritodrine: a beta-2 agonist tocolytic that relaxes the uterus, the opposite action.
- C, nifedipine: a calcium-channel blocker used as a tocolytic, not an oxytocic.
- D, salbutamol: a beta-2 agonist, relaxes the uterus.

Final Answer: Misoprostol $\Rightarrow$ B

Answer: (B) Go Back to Q14

Q15.

Solution

Concept – tocolytics: Tocolytics relax the uterus to delay preterm labour.

Step 1 – name the drug: Nifedipine, a calcium-channel blocker, is a widely used tocolytic that suppresses uterine contractions.

Step 2 – mechanism: By blocking L-type calcium channels it reduces calcium entry into uterine smooth muscle, so contractions weaken.

Step 3 – purpose: Buying 48 hours allows corticosteroids for fetal lung maturity and transfer to a unit with neonatal care.

Why the other options are wrong:

- A, oxytocin: stimulates contractions (used to induce or augment labour).
- C, misoprostol: a prostaglandin oxytocic that contracts the uterus.
- D, ergometrine: an oxytocic used for postpartum haemorrhage.

Final Answer: Nifedipine $\Rightarrow$ B

Answer: (B) Go Back to Q15

Answer Key

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