Crown to Cortex

Pharmacology

Pharmacodynamics

The Unhackables Medical

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How to read this topic

Pharmacodynamics explains what a drug does to the body. NEET PG and INI-CET questions focus on receptor theory, dose-response curves, potency, efficacy, partial agonism, inverse agonism, antagonism, therapeutic index, tolerance, tachyphylaxis, and signal transduction.

Classify

Place the drug in the correct class before reading the clinical stem.

v

Mechanism

Convert receptor, enzyme, or pathway action into expected benefit.

v

Patient filter

Apply pregnancy, renal/hepatic disease, ECG, electrolyte, allergy, and interaction filters.

v

Exam answer

Choose the drug that fits the indication and avoids the hidden contraindication.

Classification map

Class / axis	High-yield details
Agonist	full, partial, inverse
Antagonist	competitive, irreversible, physiological, chemical
Receptors	ion channel, GPCR, enzyme-linked, nuclear
Dose response	graded and quantal curves

Prototype drug map

Prototype	What to remember
Partial agonist	buprenorphine, pindolol, aripiprazole
Inverse agonist	many H1 antihistamines, beta-carbolines at GABA-A
Competitive antagonist	naloxone, atropine, propranolol
Physiological antagonist	adrenaline versus histamine in bronchospasm

Mechanism to clinical use

1. Agonist

Mechanism anchor: full, partial, inverse. In NEET PG style questions, this becomes important when the stem asks for drug choice, mechanism of toxicity, resistance, organ-specific effect, or a contraindication. Always connect the class to the expected physiological change rather than memorizing the name alone.

Clinical conversion: ask whether the desired effect is immediate symptom relief, disease modification, prophylaxis, reversal of toxicity, or long-term prevention. The same class can be correct or wrong depending on timing, route, patient risk, and monitoring feasibility.

2. Antagonist

Mechanism anchor: competitive, irreversible, physiological, chemical. In NEET PG style questions, this becomes important when the stem asks for drug choice, mechanism of toxicity, resistance, organ-specific effect, or a contraindication. Always connect the class to the expected physiological change rather than memorizing the name alone.

Clinical conversion: ask whether the desired effect is immediate symptom relief, disease modification, prophylaxis, reversal of toxicity, or long-term prevention. The same class can be correct or wrong depending on timing, route, patient risk, and monitoring feasibility.

3. Receptors

Mechanism anchor: ion channel, GPCR, enzyme-linked, nuclear. In NEET PG style questions, this becomes important when the stem asks for drug choice, mechanism of toxicity, resistance, organ-specific effect, or a contraindication. Always connect the class to the expected physiological change rather than memorizing the name alone.

Clinical conversion: ask whether the desired effect is immediate symptom relief, disease modification, prophylaxis, reversal of toxicity, or long-term prevention. The same class can be correct or wrong depending on timing, route, patient risk, and monitoring feasibility.

4. Dose response

Mechanism anchor: graded and quantal curves. In NEET PG style questions, this becomes important when the stem asks for drug choice, mechanism of toxicity, resistance, organ-specific effect, or a contraindication. Always connect the class to the expected physiological change rather than memorizing the name alone.

Clinical conversion: ask whether the desired effect is immediate symptom relief, disease modification, prophylaxis, reversal of toxicity, or long-term prevention. The same class can be correct or wrong depending on timing, route, patient risk, and monitoring feasibility.

Drug signatures

Drug / class	Mechanism cue	Use cue	Toxicity cue
Partial agonist	buprenorphine, pindolol, aripiprazole	Know preferred indication	Know signature adverse effect
Inverse agonist	many H1 antihistamines, beta-carbolines at GABA-A	Know preferred indication	Know signature adverse effect
Competitive antagonist	naloxone, atropine, propranolol	Know preferred indication	Know signature adverse effect
Physiological antagonist	adrenaline versus histamine in bronchospasm	Know preferred indication	Know signature adverse effect

Clinical edges

- Therapeutic index: narrow for lithium, digoxin, warfarin, phenytoin, aminoglycosides
- Tolerance: opioids, nitrates, benzodiazepines
- Tachyphylaxis: ephedrine, tyramine, nitrates
- Rebound: beta blockers, clonidine, steroids
- Therapeutic index is TD50/ED50; low therapeutic index drugs need monitoring.
- Tachyphylaxis is rapid loss of response, classically seen with indirect sympathomimetics and nitrates.
- Spare receptors allow maximal response without full receptor occupancy.
- Up-regulation after chronic antagonist use can cause rebound after abrupt withdrawal.

Adverse effects and contraindication logic

Partial agonist

Expected exam cue: buprenorphine, pindolol, aripiprazole. When this drug or class appears in a clinical vignette, actively look for allergy, pregnancy risk, renal or hepatic impairment, ECG abnormality, electrolyte disturbance, bleeding risk, respiratory disease, CNS depression, or interacting medicines.

How to eliminate options: reject drugs that worsen the dominant clinical danger in the stem, even if their mechanism seems suitable. This is especially important in pharmacology questions where the wrong option is often a contraindicated first-line drug.

Inverse agonist

Expected exam cue: many H1 antihistamines, beta-carbolines at GABA-A. When this drug or class appears in a clinical vignette, actively look for allergy, pregnancy risk, renal or hepatic impairment, ECG abnormality, electrolyte disturbance, bleeding risk, respiratory disease, CNS depression, or interacting medicines.

How to eliminate options: reject drugs that worsen the dominant clinical danger in the stem, even if their mechanism seems suitable. This is especially important in pharmacology questions where the wrong option is often a contraindicated first-line drug.

Competitive antagonist

Expected exam cue: naloxone, atropine, propranolol. When this drug or class appears in a clinical vignette, actively look for allergy, pregnancy risk, renal or hepatic impairment, ECG abnormality, electrolyte disturbance, bleeding risk, respiratory disease, CNS depression, or interacting medicines.

How to eliminate options: reject drugs that worsen the dominant clinical danger in the stem, even if their mechanism seems suitable. This is especially important in pharmacology questions where the wrong option is often a contraindicated first-line drug.

Physiological antagonist

Expected exam cue: adrenaline versus histamine in bronchospasm. When this drug or class appears in a clinical vignette, actively look for allergy, pregnancy risk, renal or hepatic impairment, ECG abnormality, electrolyte disturbance, bleeding risk, respiratory disease, CNS depression, or interacting medicines.

How to eliminate options: reject drugs that worsen the dominant clinical danger in the stem, even if their mechanism seems suitable. This is especially important in pharmacology questions where the wrong option is often a contraindicated first-line drug.

Exam traps

- Potency does not mean clinical superiority; efficacy usually matters more for maximum response.
- Partial agonists can reduce response in the presence of a full agonist.
- Inverse agonists reduce constitutive receptor activity; they are not neutral antagonists.
- Tolerance may be pharmacokinetic, pharmacodynamic, or behavioral.
- In Pharmacodynamics, do not memorize a class without its route, onset, elimination, and monitoring.
- Toxicity questions often hide the drug name and reveal the answer through one adverse-effect signature.
- Contraindications are tested more often than rare mechanisms.
- A drug can be first-line in one patient and dangerous in another.

Last-day revision grid

Question	Answer to recall quickly
Best prototype?	Partial agonist, Inverse agonist, Competitive antagonist, Physiological antagonist
Most tested danger?	toxicity, contraindication, interaction, and monitoring
Emergency angle?	route, onset, antidote, supportive care
Do-not-miss filter?	pregnancy, renal/hepatic failure, ECG/electrolytes, bleeding or respiratory risk

High-yield definitions

Term	Definition / exam meaning
Agonist	full, partial, inverse
Antagonist	competitive, irreversible, physiological, chemical
Receptors	ion channel, GPCR, enzyme-linked, nuclear
Dose response	graded and quantal curves
Partial agonist	buprenorphine, pindolol, aripiprazole
Inverse agonist	many H1 antihistamines, beta-carbolines at GABA-A
Competitive antagonist	naloxone, atropine, propranolol
Physiological antagonist	adrenaline versus histamine in bronchospasm
Potency	Amount of drug required for a given effect; reflected by EC50.
Efficacy	Maximum effect a drug can produce; reflected by Emax.
Competitive antagonist	Shifts curve right; Emax preserved if enough agonist is added.

How this helps in Pharmacodynamics: this page is meant to convert memorized pharmacology into option elimination. Read the left column first, then force yourself to say the mechanism, clinical use, toxicity, and reason another option is wrong.

Drug-by-drug comparison

Comparison	How to separate them in an exam stem	Most useful discriminator
Partial agonist vs Inverse agonist	Partial agonist is recalled by: buprenorphine, pindolol, aripiprazole. Inverse agonist is recalled by: many H1 antihistamines, beta-carbolines at GABA-A.	Indication, toxicity pattern, route/onset, or contraindication hidden in the stem.
Competitive antagonist vs Physiological antagonist	Competitive antagonist is recalled by: naloxone, atropine, propranolol. Physiological antagonist is recalled by: adrenaline versus histamine in bronchospasm.	Indication, toxicity pattern, route/onset, or contraindication hidden in the stem.

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Toxicity signatures

Drug / class	Toxicity pattern to actively search for	Immediate exam response
Partial agonist	Link the prototype clue to organ toxicity, laboratory change, ECG change, bleeding, CNS depression, allergy, or pregnancy risk. Cue: buprenorphine, pindolol, aripiprazole	Stop/avoid the drug if the stem contains the danger sign; choose antidote or safer alternative when asked.
Inverse agonist	Link the prototype clue to organ toxicity, laboratory change, ECG change, bleeding, CNS depression, allergy, or pregnancy risk. Cue: many H1 antihistamines, beta-carbolines at GABA-A	Stop/avoid the drug if the stem contains the danger sign; choose antidote or safer alternative when asked.
Competitive antagonist	Link the prototype clue to organ toxicity, laboratory change, ECG change, bleeding, CNS depression, allergy, or pregnancy risk. Cue: naloxone, atropine, propranolol	Stop/avoid the drug if the stem contains the danger sign; choose antidote or safer alternative when asked.
Physiological antagonist	Link the prototype clue to organ toxicity, laboratory change, ECG change, bleeding, CNS depression, allergy, or pregnancy risk. Cue: adrenaline versus histamine in bronchospasm	Stop/avoid the drug if the stem contains the danger sign; choose antidote or safer alternative when asked.
Pharmacodynamics	Any severe allergy, organ failure, pregnancy risk, or dangerous interaction can override first-line status.	Do not pick a drug only because it is famous.

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Contraindication filters

Clinical filter	What it changes	Exam habit
Pregnancy/lactation	Avoid teratogenic, fetal-toxic, or neonatal-toxic drugs; prefer established safer options.	Always scan age/sex/history lines.
Renal impairment	Accumulation increases toxicity for renally cleared drugs; dose interval may need extension.	Look for creatinine, oliguria, CKD, elderly patient.
Hepatic disease	Reduced metabolism, low albumin, and bleeding risk can change drug choice.	Check jaundice, cirrhosis, INR, albumin.
ECG/electrolytes	QT prolongation, heart block, hypokalemia, and hyperkalemia decide many answers.	Never ignore ECG and potassium.
Respiratory disease	Bronchospasm or respiratory depression risk can make a familiar drug unsafe.	Asthma/COPD/sleep apnea are not decorative details.
Bleeding risk	Antiplatelets, anticoagulants, thrombolytics, NSAIDs, and marrow-toxic drugs need caution.	Check ulcer, surgery, stroke, platelets, INR.
Partial agonist	buprenorphine, pindolol, aripiprazole	Ask: where is this drug dangerous?
Inverse agonist	many H1 antihistamines, beta-carbolines at GABA-A	Ask: where is this drug dangerous?
Competitive antagonist	naloxone, atropine, propranolol	Ask: where is this drug dangerous?

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Drug interaction map

Interaction type	Mechanism	Common exam expression
CYP induction	Increases metabolism of substrate drugs and can cause treatment failure.	Rifampicin/carbamazepine/phenytoin reducing OCP, warfarin, antiretroviral, or steroid effect.
CYP inhibition	Raises substrate levels and toxicity.	Macrolide/azole/ritonavir/cimetidine/grapefruit toxicity stem.
Additive toxicity	Two drugs injure the same organ or pathway.	QT plus QT, bleeding plus bleeding, nephrotoxic plus nephrotoxic, CNS depressant plus CNS depressant.
Pharmacodynamic opposition	One drug blocks the desired effect of another.	NSAID reducing antihypertensive effect; beta blocker opposing beta agonist.
Partial agonist	buprenorphine, pindolol, aripiprazole	Check whether the vignette adds another drug that amplifies toxicity or reduces benefit.
Inverse agonist	many H1 antihistamines, beta-carbolines at GABA-A	Check whether the vignette adds another drug that amplifies toxicity or reduces benefit.
Competitive antagonist	naloxone, atropine, propranolol	Check whether the vignette adds another drug that amplifies toxicity or reduces benefit.
Physiological antagonist	adrenaline versus histamine in bronchospasm	Check whether the vignette adds another drug that amplifies toxicity or reduces benefit.

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Monitoring and dose adjustment

Monitoring target	Why it matters	What to remember
Clinical endpoint	Symptom relief or prevention outcome confirms benefit.	Pain, BP, seizure control, infection response, glucose, dyspnea, psychosis, bleeding.
Laboratory endpoint	Detects efficacy and silent toxicity.	Renal function, liver enzymes, CBC, electrolytes, coagulation, glucose, drug levels.
ECG	Many drugs alter conduction, QT, or rhythm.	QT prolongation, AV block, QRS widening, torsades risk.
Therapeutic drug monitoring	Needed when therapeutic window is narrow.	Lithium, digoxin, phenytoin, valproate, aminoglycosides, vancomycin, tacrolimus.
Partial agonist	Monitoring depends on the toxicity implied by its mechanism and elimination.	buprenorphine, pindolol, aripiprazole
Inverse agonist	Monitoring depends on the toxicity implied by its mechanism and elimination.	many H1 antihistamines, beta-carbolines at GABA-A
Competitive antagonist	Monitoring depends on the toxicity implied by its mechanism and elimination.	naloxone, atropine, propranolol

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Rapid pathway

Read the stem

Disease, severity, age, pregnancy, organ function, emergency status.

V

Name the class

Mechanism and prototype before option elimination.

V

Apply exclusions

Contraindications, interactions, and toxicity signatures.

V

Pick final answer

Most specific safe drug for that exact stem.