Crown to Cortex

Pharmacology

Antiepileptic Drugs

The Unhackables Medical

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How to read this topic

Antiepileptic Drugs is a high-yield Pharmacology topic for NEET PG and INI-CET. The safest preparation approach is to organize it by mechanism, classification, prototype drugs, indications, adverse effects, contraindications, interactions, and emergency use. This PDF is designed as a compact final-revision note, not a textbook chapter.

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
Na channel blockers	phenytoin, carbamazepine, lamotrigine, lacosamide
Ca channel drugs	ethosuximide T-type, gabapentin/pregabalin alpha2delta
GABA enhancers	benzodiazepines, barbiturates, vigabatrin, tiagabine
Broad spectrum	valproate, levetiracetam, topiramate, lamotrigine
Special	ethosuximide for absence

Prototype drug map

Prototype	What to remember
Valproate	broad spectrum; hepatotoxicity, pancreatitis, teratogenic neural tube defects
Phenytoin	gingival hyperplasia, hirsutism, ataxia, CYP induction, fetal hydantoin
Carbamazepine	trigeminal neuralgia; SIADH, agranulocytosis, HLA risk
Ethosuximide	absence seizures; GI upset
Levetiracetam	few interactions; behavioral adverse effects

Mechanism to clinical use

1. Na channel blockers

Mechanism anchor: phenytoin, carbamazepine, lamotrigine, lacosamide. 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. Ca channel drugs

Mechanism anchor: ethosuximide T-type, gabapentin/pregabalin alpha2delta. 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. GABA enhancers

Mechanism anchor: benzodiazepines, barbiturates, vigabatrin, tiagabine. 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. Broad spectrum

Mechanism anchor: valproate, levetiracetam, topiramate, lamotrigine. 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.

5. Special

Mechanism anchor: ethosuximide for absence. 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
Valproate	broad spectrum	Know preferred indication	Know signature adverse effect
Phenytoin	gingival hyperplasia, hirsutism, ataxia, CYP induction, fetal hydantoin	Know preferred indication	Know signature adverse effect
Carbamazepine	trigeminal neuralgia	Know preferred indication	Know signature adverse effect
Ethosuximide	absence seizures	Know preferred indication	Know signature adverse effect
Levetiracetam	few interactions	Know preferred indication	Know signature adverse effect

Clinical edges

- Status epilepticus: benzodiazepine first, then fosphenytoin/valproate/levetiracetam, then anesthesia
- Pregnancy: avoid valproate when possible; folate; monotherapy lowest effective dose
- Monitoring: phenytoin nonlinear kinetics; carbamazepine CBC/sodium
- Rash: lamotrigine and carbamazepine can cause SJS/TEN
- For Antiepileptic Drugs, start every clinical question by identifying the syndrome, patient risk factors, organ function, pregnancy status, and interacting drugs.
- Prototype drugs are more important than long drug lists; know one clean example for each mechanism.
- Adverse-effect signatures often identify the drug even when the stem hides the class name.
- When two drugs look similar, compare onset, route, elimination, monitoring, and toxicity.

Adverse effects and contraindication logic

Valproate

Expected exam cue: broad spectrum; hepatotoxicity, pancreatitis, teratogenic neural tube defects. 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.

Phenytoin

Expected exam cue: gingival hyperplasia, hirsutism, ataxia, CYP induction, fetal hydantoin. 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.

Carbamazepine

Expected exam cue: trigeminal neuralgia; SIADH, agranulocytosis, HLA risk. 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.

Ethosuximide

Expected exam cue: absence seizures; GI upset. 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.

Levetiracetam

Expected exam cue: few interactions; behavioral adverse effects. 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

- Do not choose a drug only because it belongs to the right class; contraindications can reverse the answer.
- Do not ignore renal or hepatic impairment in dosing questions.
- Drug interactions are commonly tested through enzyme induction, enzyme inhibition, additive toxicity, or pharmacodynamic opposition.
- Emergency therapy depends on speed and route, not only mechanism.
- In Antiepileptic Drugs, 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?	Valproate, Phenytoin, Carbamazepine, Ethosuximide
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
Na channel blockers	phenytoin, carbamazepine, lamotrigine, lacosamide
Ca channel drugs	ethosuximide T-type, gabapentin/pregabalin alpha2delta
GABA enhancers	benzodiazepines, barbiturates, vigabatrin, tiagabine
Broad spectrum	valproate, levetiracetam, topiramate, lamotrigine
Special	ethosuximide for absence
Valproate	broad spectrum; hepatotoxicity, pancreatitis, teratogenic neural tube defects
Phenytoin	gingival hyperplasia, hirsutism, ataxia, CYP induction, fetal hydantoin
Carbamazepine	trigeminal neuralgia; SIADH, agranulocytosis, HLA risk
Ethosuximide	absence seizures; GI upset
Levetiracetam	few interactions; behavioral adverse effects
Target	Ion channels, transporters, GPCRs, and enzymes dominate CNS pharmacology.

How this helps in Antiepileptic Drugs: 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
Valproate vs Phenytoin	Valproate is recalled by: broad spectrum; hepatotoxicity, pancreatitis, teratogenic neural tube defects. Phenytoin is recalled by: gingival hyperplasia, hirsutism, ataxia, CYP induction, fetal hydantoin.	Indication, toxicity pattern, route/onset, or contraindication hidden in the stem.
Carbamazepine vs Ethosuximide	Carbamazepine is recalled by: trigeminal neuralgia; SIADH, agranulocytosis, HLA risk. Ethosuximide is recalled by: absence seizures; GI upset.	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
Valproate	Link the prototype clue to organ toxicity, laboratory change, ECG change, bleeding, CNS depression, allergy, or pregnancy risk. Cue: broad spectrum; hepatotoxicity, pancreatitis, teratogenic neural tube defects	Stop/avoid the drug if the stem contains the danger sign; choose antidote or safer alternative when asked.
Phenytoin	Link the prototype clue to organ toxicity, laboratory change, ECG change, bleeding, CNS depression, allergy, or pregnancy risk. Cue: gingival hyperplasia, hirsutism, ataxia, CYP induction, fetal hydantoin	Stop/avoid the drug if the stem contains the danger sign; choose antidote or safer alternative when asked.
Carbamazepine	Link the prototype clue to organ toxicity, laboratory change, ECG change, bleeding, CNS depression, allergy, or pregnancy risk. Cue: trigeminal neuralgia; SIADH, agranulocytosis, HLA risk	Stop/avoid the drug if the stem contains the danger sign; choose antidote or safer alternative when asked.
Ethosuximide	Link the prototype clue to organ toxicity, laboratory change, ECG change, bleeding, CNS depression, allergy, or pregnancy risk. Cue: absence seizures; GI upset	Stop/avoid the drug if the stem contains the danger sign; choose antidote or safer alternative when asked.
Levetiracetam	Link the prototype clue to organ toxicity, laboratory change, ECG change, bleeding, CNS depression, allergy, or pregnancy risk. Cue: few interactions; behavioral adverse effects	Stop/avoid the drug if the stem contains the danger sign; choose antidote or safer alternative when asked.
Antiepileptic Drugs	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.
Valproate	broad spectrum; hepatotoxicity, pancreatitis, teratogenic neural tube defects	Ask: where is this drug dangerous?
Phenytoin	gingival hyperplasia, hirsutism, ataxia, CYP induction, fetal hydantoin	Ask: where is this drug dangerous?
Carbamazepine	trigeminal neuralgia; SIADH, agranulocytosis, HLA risk	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.
Valproate	broad spectrum; hepatotoxicity, pancreatitis, teratogenic neural tube defects	Check whether the vignette adds another drug that amplifies toxicity or reduces benefit.
Phenytoin	gingival hyperplasia, hirsutism, ataxia, CYP induction, fetal hydantoin	Check whether the vignette adds another drug that amplifies toxicity or reduces benefit.
Carbamazepine	trigeminal neuralgia; SIADH, agranulocytosis, HLA risk	Check whether the vignette adds another drug that amplifies toxicity or reduces benefit.
Ethosuximide	absence seizures; GI upset	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.
Valproate	Monitoring depends on the toxicity implied by its mechanism and elimination.	broad spectrum; hepatotoxicity, pancreatitis, teratogenic neural tube defects
Phenytoin	Monitoring depends on the toxicity implied by its mechanism and elimination.	gingival hyperplasia, hirsutism, ataxia, CYP induction, fetal hydantoin
Carbamazepine	Monitoring depends on the toxicity implied by its mechanism and elimination.	trigeminal neuralgia; SIADH, agranulocytosis, HLA risk

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Emergency decision table

Emergency scenario	First pharmacology decision	Common mistake
Shock/anaphylaxis/severe acute state	Choose route and onset before elegance of mechanism.	Choosing an oral chronic drug for an emergency.
Poisoning/toxicity	Stabilize airway, breathing, circulation, then antidote if indicated.	Giving antidote while ignoring supportive care.
Severe infection or organ-threatening disease	Start rational empirical therapy promptly, then narrow when data arrives.	Waiting for perfect information in an unstable patient.
Withdrawal or rebound	Recognize dependence physiology and taper/replace appropriately.	Abruptly stopping clonidine, beta blockers, steroids, opioids, alcohol, or benzodiazepines.
Antiepileptic Drugs: Valproate	broad spectrum; hepatotoxicity, pancreatitis, teratogenic neural tube defects	Wrong route, delayed onset, or ignored contraindication.
Antiepileptic Drugs: Phenytoin	gingival hyperplasia, hirsutism, ataxia, CYP induction, fetal hydantoin	Wrong route, delayed onset, or ignored contraindication.
Antiepileptic Drugs: Carbamazepine	trigeminal neuralgia; SIADH, agranulocytosis, HLA risk	Wrong route, delayed onset, or ignored contraindication.
Antiepileptic Drugs: Ethosuximide	absence seizures; GI upset	Wrong route, delayed onset, or ignored contraindication.

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INI-CET stem decoding

Stem clue	What it is trying to test	Answer strategy
Age, pregnancy, renal/liver disease	Safety filter rather than diagnosis.	Eliminate unsafe drugs first.
New symptom after drug start	Adverse-effect signature.	Name the drug from toxicity.
Drug added recently	Interaction question.	Check CYP, QT, bleeding, CNS depression, nephrotoxicity.
Emergency wording	Route/onset question.	Prefer fast, titratable, evidence-based acute therapy.
Chronic prevention wording	Outcome benefit question.	Prefer disease-modifying therapy over only symptomatic relief.
Na channel blockers	phenytoin, carbamazepine, lamotrigine, lacosamide	Place this under Antiepileptic Drugs, then compare with nearby alternatives.
Ca channel drugs	ethosuximide T-type, gabapentin/pregabalin alpha2delta	Place this under Antiepileptic Drugs, then compare with nearby alternatives.
GABA enhancers	benzodiazepines, barbiturates, vigabatrin, tiagabine	Place this under Antiepileptic Drugs, then compare with nearby alternatives.
Broad spectrum	valproate, levetiracetam, topiramate, lamotrigine	Place this under Antiepileptic Drugs, then compare with nearby alternatives.

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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.