Crown to Cortex

Pharmacology

NSAIDs

The Unhackables Medical

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How to read this topic

NSAIDs inhibit cyclooxygenase pathways and reduce prostaglandin synthesis. They are tested for analgesic, antipyretic, anti-inflammatory, antiplatelet effects, COX selectivity, gastric and renal toxicity, aspirin poisoning, pregnancy cautions, and paracetamol toxicity.

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
Salicylates	aspirin
Propionic acids	ibuprofen, naproxen
Acetic acids	diclofenac, indomethacin, ketorolac
Oxicams	piroxicam, meloxicam
COX-2 selective	celecoxib, etoricoxib
Para-aminophenol	paracetamol

Prototype drug map

Prototype	What to remember
Aspirin	irreversible antiplatelet; ACS, stroke prevention, Kawasaki disease
Indomethacin	PDA closure; high toxicity
Ketorolac	short-term severe pain only
Celecoxib	lower GI ulcer risk but thrombotic caution
Paracetamol	NAC-responsive hepatotoxic overdose

Mechanism to clinical use

1. Salicylates

Mechanism anchor: aspirin. 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. Propionic acids

Mechanism anchor: ibuprofen, naproxen. 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. Acetic acids

Mechanism anchor: diclofenac, indomethacin, ketorolac. 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. Oxicams

Mechanism anchor: piroxicam, meloxicam. 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. COX-2 selective

Mechanism anchor: celecoxib, etoricoxib. 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.

6. Para-aminophenol

Mechanism anchor: paracetamol. 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
Aspirin	irreversible antiplatelet	Know preferred indication	Know signature adverse effect
Indomethacin	PDA closure	Know preferred indication	Know signature adverse effect
Ketorolac	short-term severe pain only	Know preferred indication	Know signature adverse effect
Celecoxib	lower GI ulcer risk but thrombotic caution	Know preferred indication	Know signature adverse effect
Paracetamol	NAC-responsive hepatotoxic overdose	Know preferred indication	Know signature adverse effect

Clinical edges

- Renal: afferent constriction, salt-water retention, hyperkalemia, papillary necrosis
- GI: ulcer, bleeding; PPI/misoprostol protection in high-risk patients
- Respiratory: aspirin-exacerbated respiratory disease due to leukotriene excess
- Pregnancy: avoid late pregnancy due ductus closure and renal effects
- NSAIDs can precipitate renal failure by blocking prostaglandin-mediated afferent arteriolar dilation.
- Avoid NSAIDs late in pregnancy due to premature ductus closure.
- Aspirin-exacerbated respiratory disease is due to leukotriene shunting.
- N-acetylcysteine replenishes glutathione in paracetamol poisoning.

Adverse effects and contraindication logic

Aspirin

Expected exam cue: irreversible antiplatelet; ACS, stroke prevention, Kawasaki disease. 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.

Indomethacin

Expected exam cue: PDA closure; high toxicity. 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.

Ketorolac

Expected exam cue: short-term severe pain only. 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.

Celecoxib

Expected exam cue: lower GI ulcer risk but thrombotic caution. 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.

Paracetamol

Expected exam cue: NAC-responsive hepatotoxic overdose. 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

- Paracetamol is weak anti-inflammatory.
- Aspirin is irreversible; most other NSAIDs are reversible.
- COX-2 selective drugs spare platelets less completely and may increase thrombotic risk.
- NSAIDs can reduce antihypertensive efficacy.
- In NSAIDs, 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?	Aspirin, Indomethacin, Ketorolac, Celecoxib
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
Salicylates	aspirin
Propionic acids	ibuprofen, naproxen
Acetic acids	diclofenac, indomethacin, ketorolac
Oxicams	piroxicam, meloxicam
COX-2 selective	celecoxib, etoricoxib
Para-aminophenol	paracetamol
Aspirin	irreversible antiplatelet; ACS, stroke prevention, Kawasaki disease
Indomethacin	PDA closure; high toxicity
Ketorolac	short-term severe pain only
Celecoxib	lower GI ulcer risk but thrombotic caution
Paracetamol	NAC-responsive hepatotoxic overdose

How this helps in NSAIDs: 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
Aspirin vs Indomethacin	Aspirin is recalled by: irreversible antiplatelet; ACS, stroke prevention, Kawasaki disease. Indomethacin is recalled by: PDA closure; high toxicity.	Indication, toxicity pattern, route/onset, or contraindication hidden in the stem.
Ketorolac vs Celecoxib	Ketorolac is recalled by: short-term severe pain only. Celecoxib is recalled by: lower GI ulcer risk but thrombotic caution.	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
Aspirin	Link the prototype clue to organ toxicity, laboratory change, ECG change, bleeding, CNS depression, allergy, or pregnancy risk. Cue: irreversible antiplatelet; ACS, stroke prevention, Kawasaki disease	Stop/avoid the drug if the stem contains the danger sign; choose antidote or safer alternative when asked.
Indomethacin	Link the prototype clue to organ toxicity, laboratory change, ECG change, bleeding, CNS depression, allergy, or pregnancy risk. Cue: PDA closure; high toxicity	Stop/avoid the drug if the stem contains the danger sign; choose antidote or safer alternative when asked.
Ketorolac	Link the prototype clue to organ toxicity, laboratory change, ECG change, bleeding, CNS depression, allergy, or pregnancy risk. Cue: short-term severe pain only	Stop/avoid the drug if the stem contains the danger sign; choose antidote or safer alternative when asked.
Celecoxib	Link the prototype clue to organ toxicity, laboratory change, ECG change, bleeding, CNS depression, allergy, or pregnancy risk. Cue: lower GI ulcer risk but thrombotic caution	Stop/avoid the drug if the stem contains the danger sign; choose antidote or safer alternative when asked.
Paracetamol	Link the prototype clue to organ toxicity, laboratory change, ECG change, bleeding, CNS depression, allergy, or pregnancy risk. Cue: NAC-responsive hepatotoxic overdose	Stop/avoid the drug if the stem contains the danger sign; choose antidote or safer alternative when asked.
NSAIDs	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.
Aspirin	irreversible antiplatelet; ACS, stroke prevention, Kawasaki disease	Ask: where is this drug dangerous?
Indomethacin	PDA closure; high toxicity	Ask: where is this drug dangerous?
Ketorolac	short-term severe pain only	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.
Aspirin	irreversible antiplatelet; ACS, stroke prevention, Kawasaki disease	Check whether the vignette adds another drug that amplifies toxicity or reduces benefit.
Indomethacin	PDA closure; high toxicity	Check whether the vignette adds another drug that amplifies toxicity or reduces benefit.
Ketorolac	short-term severe pain only	Check whether the vignette adds another drug that amplifies toxicity or reduces benefit.
Celecoxib	lower GI ulcer risk but thrombotic caution	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.
Aspirin	Monitoring depends on the toxicity implied by its mechanism and elimination.	irreversible antiplatelet; ACS, stroke prevention, Kawasaki disease
Indomethacin	Monitoring depends on the toxicity implied by its mechanism and elimination.	PDA closure; high toxicity
Ketorolac	Monitoring depends on the toxicity implied by its mechanism and elimination.	short-term severe pain only

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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.
NSAIDs: Aspirin	irreversible antiplatelet; ACS, stroke prevention, Kawasaki disease	Wrong route, delayed onset, or ignored contraindication.
NSAIDs: Indomethacin	PDA closure; high toxicity	Wrong route, delayed onset, or ignored contraindication.
NSAIDs: Ketorolac	short-term severe pain only	Wrong route, delayed onset, or ignored contraindication.
NSAIDs: Celecoxib	lower GI ulcer risk but thrombotic caution	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.
Salicylates	aspirin	Place this under NSAIDs, then compare with nearby alternatives.
Propionic acids	ibuprofen, naproxen	Place this under NSAIDs, then compare with nearby alternatives.
Acetic acids	diclofenac, indomethacin, ketorolac	Place this under NSAIDs, then compare with nearby alternatives.
Oxicams	piroxicam, meloxicam	Place this under NSAIDs, 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.