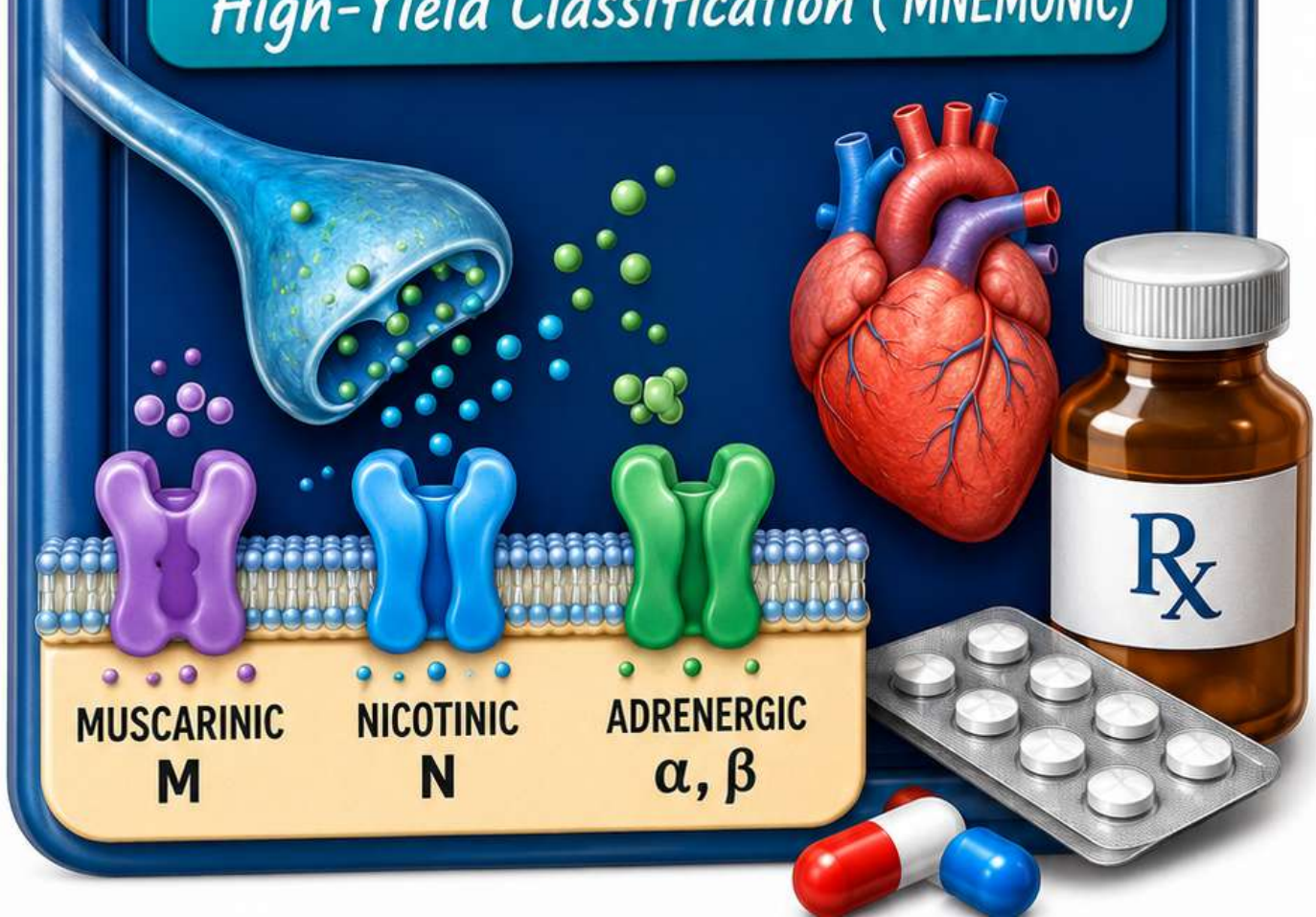
IMPORTANT DRUGS OF AUTONOMIC NERVOUS SYSTEM (ANS)

High-Yield Classification (MNEMONIC)



Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

TROPICAMIDE*

Muscarinic antagonist; causes mydriasis

1 DEFINITION

- Tropicamide is a short-acting antimuscarinic drug used mainly in ophthalmology.
- It blocks muscarinic receptors in the eye.
- Produces mydriasis and mild cycloplegia.

2 CLASSIFICATION

- **Class:** Anticholinergic / parasympatholytic.
- **Type:** Muscarinic receptor antagonist.
- **Use group:** Ophthalmic mydriatic.
- **Nature:** Short-acting topical ocular drug.

3 MECHANISM OF ACTION

- Competitively blocks muscarinic receptors, mainly M_3, in iris sphincter and ciliary muscle.
- Relaxes sphincter pupillae → mydriasis.
- Relaxes ciliary muscle → cycloplegia.
- Prevents parasympathetic constriction of pupil.
- Short duration compared with atropine.

4 PHARMACOLOGICAL EFFECTS

- Dilatation of pupil.
- Mild to moderate cycloplegia.
- Blurred near vision.
- Photophobia due to mydriasis.
- Slight rise in intraocular pressure in susceptible patients.

5 THERAPEUTIC USES

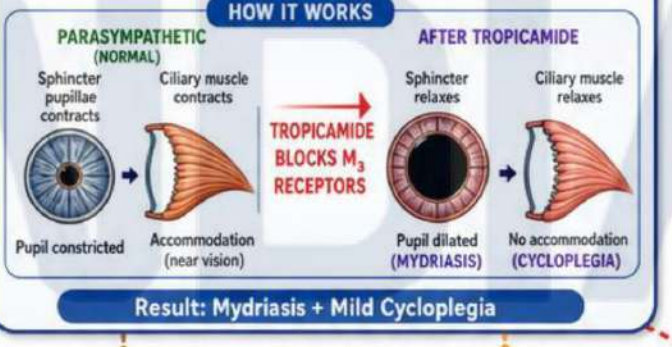
- Fundus examination.
- Diagnostic pupil dilatation.
- Cycloplegic refraction.
- Pre-operative dilatation.
- Occasionally in anterior uveitis to prevent synechiae.

6 ADVERSE EFFECTS

- Stinging or irritation in eye.
- Photophobia.
- Blurred vision.
- Dry mouth.
- Tachycardia (*rare systemic effect*).
- May precipitate acute angle-closure glaucoma.

7 CONTRAINDICATIONS

- Narrow-angle / angle-closure glaucoma.
- Hypersensitivity to tropicamide.
- Caution in elderly.
- Caution in infants / very young children.
- Use carefully in patients prone to raised IOP.



8 DRUG INTERACTIONS

- Additive anticholinergic effects with atropine, antihistamines, TCAs, antipsychotics.
- Sympathomimetics may further enhance mydriasis.
- Systemic absorption is usually low, but caution is advised.

9 IMPORTANT EXAMPLES

- Tropicamide eye drops 0.5%.
- Tropicamide eye drops 1%.
- Related ophthalmic antimuscarinics: atropine, cyclopentolate, homatropine.

★ **Highlight:** tropicamide is **shorter acting** than atropine.

10 MNEMONICS

"TROPI = Tiny Time Mydriatic"

- ✓ Short action.
- ✓ Tropicamide → dilates pupil quickly.
- ✓ Think: *"Tropicamide for testing the eye."*

11 EXAM POINTS

- ★ Short-acting muscarinic antagonist.
- ★ Causes mydriasis and cycloplegia.
- ★ Used for fundus examination.
- ★ Safer for routine eye exam than atropine because duration is shorter.
- ★ Can precipitate angle-closure glaucoma.

12 IMPORTANT QUESTIONS

1. Write the mechanism of action of tropicamide.
2. Why is tropicamide preferred over atropine for routine eye examination?
3. Mention therapeutic uses of tropicamide.
4. List adverse effects of topical antimuscarinic eye drops.
5. Differentiate tropicamide and atropine.

EXAM BOOSTER ★

High-Yield Fact:

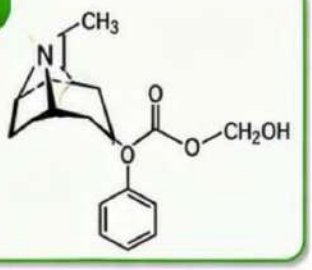
Tropicamide is a short-acting ophthalmic antimuscarinic used to produce rapid mydriasis for eye examination.

★ Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners ★

1 DEFINITION

- Naturally occurring belladonna alkaloid.
- Competitive, reversible muscarinic receptor antagonist.
- Parasympatholytic / antimuscarinic drug.

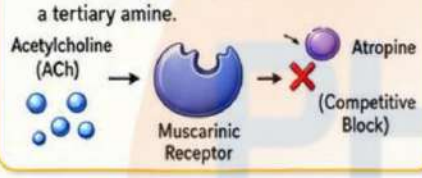
2 CLASSIFICATION

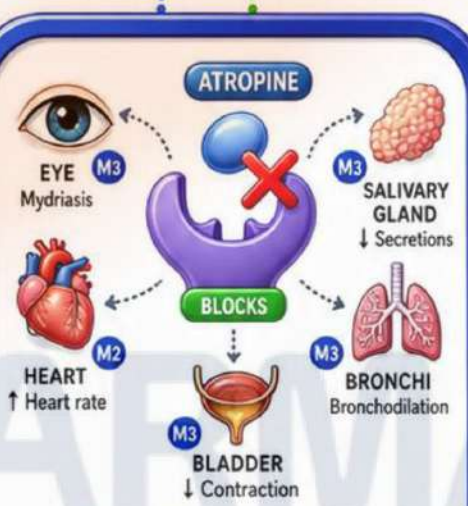


- Anticholinergic (antimuscarinic) drug.
- Tertiary amine.
- Prototype muscarinic blocker.
- Acts mainly on M1–M5 muscarinic receptors.

3 MECHANISM OF ACTION

- Competitively blocks muscarinic receptors.
- Inhibits actions of acetylcholine at parasympathetic neuroeffector sites.
- Reduces glandular secretion and smooth muscle tone.
- Causes mydriasis and cycloplegia in the eye.
- No nicotinic receptor blockade.
- Crosses BBB because it is a tertiary amine.





ATROPINE*

Competitive muscarinic receptor antagonist.

PHARMACOLOGICAL EFFECTS

- Mydriasis and cycloplegia.
- Tachycardia.
- Decreased salivary, bronchial, and sweat secretions.
- Bronchodilation (mild).
- Decreased GI motility and spasm.
- Relaxes bladder detrusor → urinary retention.

THERAPEUTIC USES

- Symptomatic sinus bradycardia.
- Pre-anesthetic medication to reduce secretions.
- Organophosphate poisoning (with pralidoxime).
- Ophthalmic mydriatic / cycloplegic use.
- Antispasmodic in GI tract.
- Mushroom muscarine poisoning.

ADVERSE EFFECTS

- Dry mouth.
- Blurred vision / photophobia.
- Tachycardia.
- Constipation.
- Urinary retention.
- Decreased sweating → fever / hot dry skin.
- CNS excitement, confusion, especially in high dose.

7 CONTRAINDICATIONS

- Angle-closure glaucoma.
- Prostatic hypertrophy / urinary retention.
- Paralytic ileus or pyloric obstruction.
- Tachyarrhythmias.
- Fever / hot environment with caution.
- Use cautiously in elderly patients.

8 DRUG INTERACTIONS

- Additive anticholinergic effects with antihistamines.
- Additive effects with tricyclic antidepressants and antipsychotics.
- Antagonizes cholinergic drugs / prokinetics.
- May delay gastric emptying and alter absorption of some oral drugs.

★ 9 IMPORTANT EXAMPLES

- Atropine sulfate.
- Related antimuscarinics:
  - Scopolamine
  - Homatropine
  - Tropicamide
  - Ipratropium

11 EXAM POINTS

- Prototype antimuscarinic drug.
- Tertiary amine → crosses BBB.
- Causes mydriasis, cycloplegia, tachycardia, and dry mouth.
- Used in organophosphorus poisoning.
- Does not block nicotinic receptors.
- Antidote for severe muscarinic excess.

EXAM BOOSTER

12 IMPORTANT QUESTIONS

Q. What type of drug is atropine?
A. Competitive antimuscarinic.
Q. Major use in poisoning?
A. Organophosphate poisoning.
Q. Effect on pupil?
A. Mydriasis with cycloplegia.
Q. Does it cross BBB?
A. Yes, tertiary amine.
Q. Major contraindication?
A. Angle-closure glaucoma.

10 MNEMONICS

H ot as a hare
D - ry as a bone
B - lind as a bat
R ed as a beet
M ad as a hatter

Atropine = dries secretions + dilates pupil + raises heart rate.

Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

1 Definition

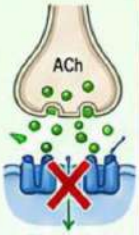
- Tertiary antimuscarinic drug
- Natural belladonna alkaloid
- Crosses blood-brain barrier

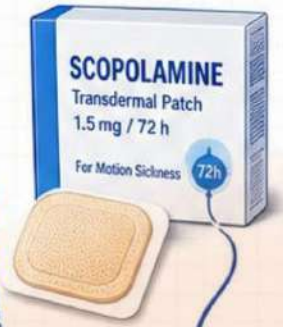
2 Classification

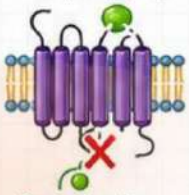
- Parasympatholytic / anticholinergic
- Muscarinic receptor blocker
- Tertiary amine tropane alkaloid

3 Mechanism of Action

- Competitive muscarinic receptor antagonist
- Blocks cholinergic transmission in vestibular pathways
- Depresses vomiting center input
- Reduces salivary and GI secretions







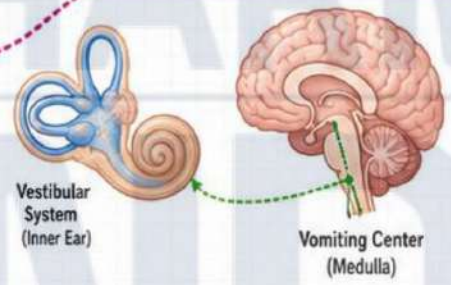
Scopolamine* – Muscarinic blocker; prevents motion sickness

4 Pharmacological Effects

- Antiemetic
- Antisialagogue
- Sedative / CNS depressant
- Mydriasis and cycloplegia
- ↓ GI motility

5 Therapeutic Uses

- Prevention of motion sickness
- Postoperative nausea and vomiting
- Pre-anaesthetic medication
- Excess salivation control



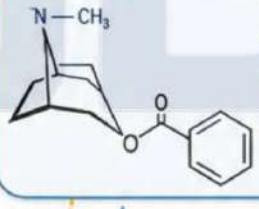
6 Adverse Effects

- Dry mouth
- Blurred vision
- Drowsiness
- Constipation
- Urinary retention
- Tachycardia
- Confusion at high dose

7 Contraindications

- Angle-closure glaucoma
- Prostatic hypertrophy
- Paralytic ileus
- Caution in elderly

Chemical Structure of Scopolamine



- Tropane alkaloid
- Tertiary amine (N–CH_3)
- Lipid soluble → crosses BBB

8 Drug Interactions

- Additive anticholinergic effects with antihistamines
- Additive with TCAs and antipsychotics
- Alcohol /CNS depressants ↑ sedation

9 Important Examples

- Scopolamine hydrobromide
- Transdermal scopolamine patch
- Related antimuscarinics: atropine, homatropine

10 Mnemonics

- "Sco-PATCH-alamine" = patch for travel sickness
- "Sea trip = Scopolamine"

11 Exam Points

- ★ Best antimuscarinic for motion sickness
- ★ Tertiary amine → enters CNS
- ★ Commonly used as transdermal patch
- ★ More sedative than atropine

Learn • Understand • Remember • Excel

12 Important Questions

Q: Why is scopolamine preferred in motion sickness?
A: It acts on vestibular pathways and vomiting center in CNS and is more sedative → effective in motion sickness.

Q: Why does it cause sedation?
A: It crosses the blood-brain barrier and depresses CNS activity.

Q: Name one common dosage form.
A: Transdermal patch (1.5 mg / 72 h).

High Yield Remember Smartly!

Key Takeaway: **Scopolamine** is a tertiary antimuscarinic that ***crosses the BBB***, blocks ***vestibular inputs*** and ***vomiting center***, thereby preventing motion sickness while causing typical ***anticholinergic effects.***

Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

1 DEFINITION

- Physostigmine is a reversible anticholinesterase drug.
- It inhibits acetylcholinesterase and increases acetylcholine at synapses.
- It is a tertiary amine, so it crosses the blood-brain barrier.

2 CLASSIFICATION

- **Class:** Indirect-acting cholinomimetic.
- **Type:** Reversible acetylcholinesterase inhibitor.
- **Chemical nature:** Natural carbamate alkaloid.
- Tertiary amine → CNS active.

3 MECHANISM OF ACTION

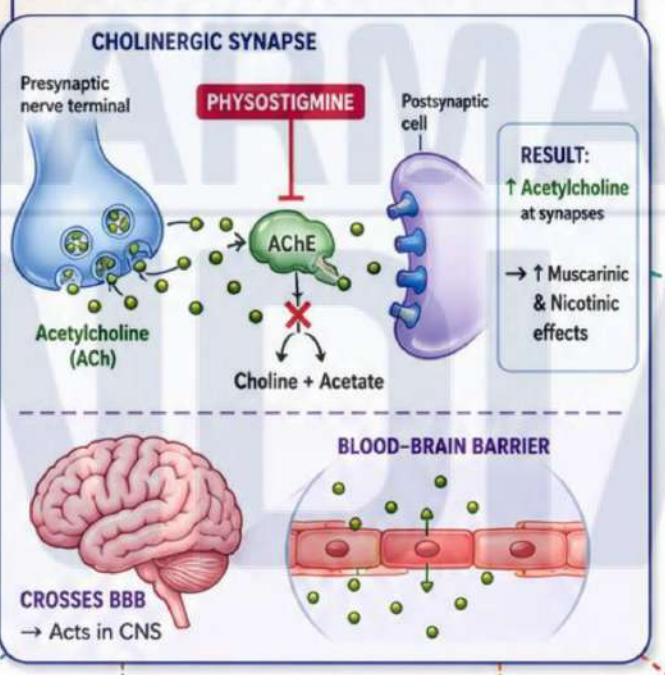
- Reversibly inhibits acetylcholinesterase.
- Prevents breakdown of acetylcholine.
- Increases ACh at muscarinic and nicotinic receptors.
- Enhances cholinergic transmission in peripheral and central nervous systems.
- Crosses BBB and reverses central anticholinergic toxicity.

4 PHARMACOLOGICAL EFFECTS

- **Eye:** miosis, spasm of accommodation, reduced intraocular pressure.
- **GIT:** increased motility and secretions.
- **Glands:** increased salivation, sweating, lacrimation.
- **CVS:** bradycardia.
- **Skeletal muscle:** improved neuromuscular transmission.
- **CNS:** enhances central cholinergic activity.

PHYSOSTIGMINE*

Reversible AChE inhibitor; crosses BBB



5 THERAPEUTIC USES

- Antidote in atropine poisoning / anticholinergic toxicity.
- Reversal of central and peripheral antimuscarinic effects.
- Occasionally used in glaucoma.
- Historical / less common use in myasthenia gravis.

6 ADVERSE EFFECTS

- Salivation, sweating, lacrimation.
- Nausea, vomiting, diarrhea, abdominal cramps.
- Bradycardia and hypotension.
- Bronchospasm.
- Muscle cramps or fasciculations.
- CNS effects: seizures at high dose.

7 CONTRAINDICATIONS

- Asthma or severe COPD.
- Peptic ulcer disease.
- Bradycardia or heart block.
- GI or urinary obstruction.
- Epilepsy / seizure disorders: caution.

8 DRUG INTERACTIONS

- Antimuscarinics (e.g., atropine) antagonize many muscarinic effects.
- Other cholinomimetics or AChE inhibitors increase toxicity.
- Beta-blockers may enhance bradycardia.
- Drugs lowering seizure threshold may worsen CNS toxicity risk.

9 IMPORTANT EXAMPLES

- Physostigmine salicylate.
- Compare related reversible AChE inhibitors:
  - Neostigmine
  - Pyridostigmine
  - Edrophonium
- ★ Highlight: Physostigmine is the one that crosses BBB.

10 MNEMONICS

"PHYSO = PHYSically enters the brain"

- ☑ Physostigmine = tertiary amine → crosses BBB.
- ☑ Use in atropine poisoning.

11 EXAM POINTS

- ★ Reversible AChE inhibitor.
- ★ Tertiary amine; crosses BBB.
- ★ Drug of choice for atropine toxicity / central anticholinergic syndrome.
- ★ Increases both muscarinic and nicotinic actions indirectly.
- ★ Different from neostigmine: neostigmine does **NOT** cross BBB.

12 IMPORTANT QUESTIONS

1. Why is physostigmine used in atropine poisoning?
2. How does physostigmine differ from neostigmine?
3. Why can physostigmine produce CNS effects?
4. Mention therapeutic uses and adverse effects of physostigmine.
5. Explain its mechanism as a reversible AChE inhibitor.

EXAM BOOSTER ★

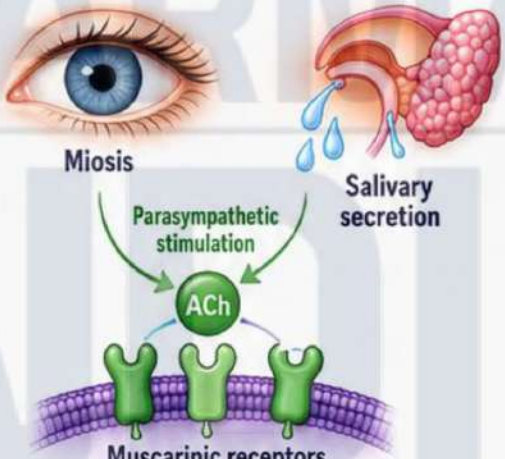
High-Yield Fact:

Physostigmine is the classic reversible AChE inhibitor that crosses the BBB and is used in anticholinergic toxicity.

Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

PILOCARPINE*

Muscarinic agonist; increases salivation and miosis



1. DEFINITION

- Pilocarpine is a direct-acting **parasympathomimetic** drug.
- Natural alkaloid and selective **muscarinic** receptor agonist.
- Increases exocrine secretions and causes **miosis**.

2. CLASSIFICATION

- **Class:** Cholinergic agonist.
- **Type:** Direct-acting muscarinic agonist.
- **Source:** Natural alkaloid from *Pilocarpus*.

3. MECHANISM OF ACTION

- Stimulates muscarinic receptors, mainly M3.
- Contracts iris sphincter → **miosis**.
- Contracts ciliary muscle → opens trabecular meshwork.
- Increases aqueous humor outflow → **lowers intraocular pressure**.
- Increases salivary, lacrimal and sweat secretion.

4. PHARMACOLOGICAL EFFECTS

- Marked salivation and sweating.
- Miosis and spasm of accommodation.
- Decreased intraocular pressure.
- Increased bronchial and lacrimal secretions.
- Increased GI motility and mild bradycardia.

5. THERAPEUTIC USES

- Xerostomia (dry mouth).
- Sjögren syndrome.
- Glaucoma, especially to reduce intraocular pressure.
- Produce miosis in ophthalmic practice.

6. ADVERSE EFFECTS

- Excess sweating.
- Excess salivation.
- Nausea, abdominal cramps, diarrhea.
- Bronchospasm.
- Bradycardia and hypotension.
- Blurred vision or brow ache.

7. CONTRAINDICATIONS

- Asthma or severe COPD.
- Peptic ulcer disease.
- Severe bradycardia or hypotension.
- GI or urinary obstruction.
- Caution in iritis/uveitis.

8. DRUG INTERACTIONS

- Antimuscarinics (e.g. atropine) block its action.
- Other cholinomimetics or anticholinesterases increase cholinergic effects.
- Additive bradycardia with other rate-lowering drugs.

9. IMPORTANT EXAMPLES

- ✓ Pilocarpine eye drops.
- ✓ Oral pilocarpine tablets.
- ✓ Related muscarinic agonists: bethanechol, methacholine, cevimeline.

10. MNEMONICS

"PILO = Pupil In, Liquids Out"

Pupil in = miosis;
Liquids out = salivation, sweating, tears.

11. EXAM POINTS

- Direct muscarinic agonist; no significant nicotinic action.
- Natural alkaloid.
- Used in xerostomia and glaucoma.
- Causes miosis and ciliary muscle contraction.
- Lowers IOP by increasing aqueous outflow.

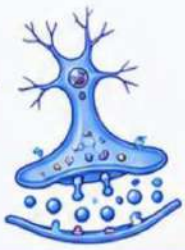
12. IMPORTANT QUESTIONS

1. Write the mechanism of action of pilocarpine.
2. Why is pilocarpine used in xerostomia?
3. Mention therapeutic uses of pilocarpine.
4. List adverse effects of muscarinic agonists.
5. Differentiate pilocarpine and atropine.

Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

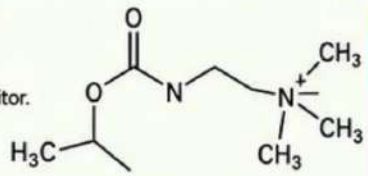
1. DEFINITION

- Reversible anticholinesterase drug.
- Inhibits acetylcholinesterase and increases acetylcholine at cholinergic synapses.



2. CLASSIFICATION

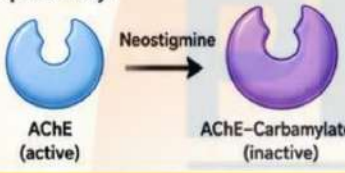
- Indirect-acting cholinomimetic.
- Reversible acetylcholinesterase inhibitor.
- **Carbamate ester.**
- Quaternary ammonium compound.
- Mainly peripheral acting.

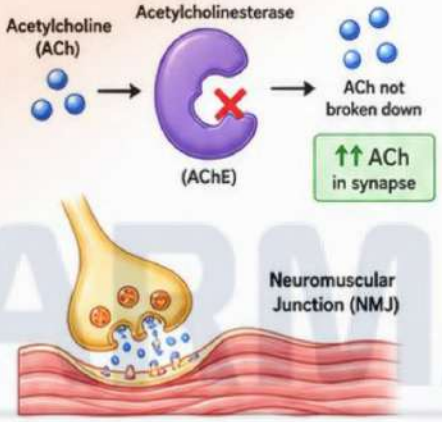


Chemical Class:
Carbamate ester

3. MECHANISM OF ACTION

- Carbamylates acetylcholinesterase reversibly.
- Prevents breakdown of acetylcholine.
- Increases both muscarinic and nicotinic activity.
- Enhances transmission at neuromuscular junction.
- Poor CNS entry because it is quaternary.





NEOSTIGMINE*

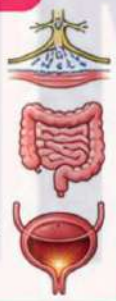
Reversible acetylcholinesterase inhibitor.

4. PHARMACOLOGICAL EFFECTS

- Miosis.
- Bradycardia.
- Increased salivation and secretions.
- Bronchoconstriction.
- Increased GI motility.
- Bladder contraction.
- Improves skeletal muscle strength in myasthenia gravis.

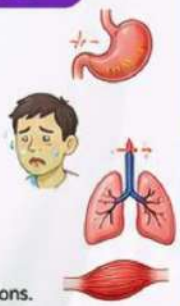
5. THERAPEUTIC USES

- Myasthenia gravis.
- Reversal of non-depolarizing neuromuscular block.
- Postoperative paralytic ileus.
- Postoperative/non-obstructive urinary retention.



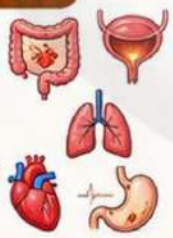
ADVERSE EFFECTS

- Abdominal cramps.
- Diarrhea.
- Nausea and vomiting.
- Excess salivation.
- Sweating.
- Bradycardia.
- Bronchospasm.
- Muscle cramps or fasciculations.



7. CONTRAINDICATIONS

- Intestinal obstruction.
- Urinary obstruction.
- Asthma or severe COPD.
- Bradycardia or heart block.
- Peptic ulcer.



9. IMPORTANT EXAMPLES

- Neostigmine methylsulfate (injection).
- Neostigmine bromide (oral).

Related reversible inhibitors:
- Pyridostigmine.
- Physostigmine.

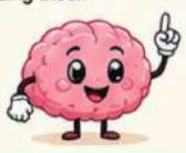
DRUG INTERACTIONS

- Atropine antagonizes muscarinic effects.
- Glycopyrrolate often co-used during reversal.
- Aminoglycosides may worsen neuromuscular weakness.
- May prolong effect of succinylcholine.
- Additive effect with other cholinergic drugs.

10. MNEMONICS

"NEO-STIM"

- **N** – NMJ strength ↑
- **E** – Empty bladder
- **O** – Opposes non-depolarizing block
- **S** – Secretions ↑
- **T** – Tears / sweating
- **I** – Ileus relief
- **M** – Myasthenia gravis



11. EXAM POINTS

- Reversible AChE inhibitor.
- Quaternary ammonium → poor BBB penetration.
- Acts on both muscarinic and nicotinic sites indirectly.
- Used to reverse non-depolarizing muscle relaxants.
- Often given with atropine or glycopyrrolate.

EXAM BOOSTER

IMPORTANT QUESTIONS

1. **What type of drug is neostigmine?**
   → Reversible AChE inhibitor.
2. **Does it cross BBB well?**
   → No, poor CNS entry.
3. **Main use in muscle disease?**
   → Myasthenia gravis.
4. **Reverses which block?**
   → Non-depolarizing neuromuscular block.
5. **Muscarinic adverse effects are treated with?**
   → Atropine.

Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

1. DEFINITION

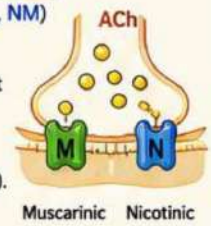
- Acetylcholine (ACh) is an endogenous neurotransmitter that acts as an **agonist** at both **muscarinic** and **nicotinic** receptors.

2. CLASSIFICATION

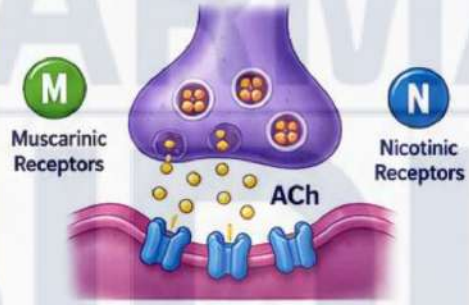
- **By Source:** Endogenous
- **By Receptor:**
  - Muscarinic Receptor Agonist
  - Nicotinic Receptor Agonist
- **Chemical Class:** Choline Ester

3. MECHANISM OF ACTION

- Binds to and activates muscarinic (M1–M5) and nicotinic (N_N, NM) receptors.
- Mimics the action of ACh at cholinergic synapses.
- Rapidly hydrolyzed by acetylcholinesterase (AChE).



ACETYLCHOLINE* – MUSCARINIC AND NICOTINIC RECEPTOR AGONIST



Endogenous neurotransmitter at cholinergic synapses

4. PHARMACOLOGICAL EFFECTS

Muscarinic (M)

- Miosis
- Bronchoconstriction
- ↓ Heart rate
- ↑ Salivation, lacrimation
- ↑ GI motility & secretion
- Urination (detrusor contraction)

Nicotinic (N)

- N_N: Ganglionic stimulation (↑ autonomic activity)
- N_M: Skeletal muscle contraction

5. THERAPEUTIC USES

- Post-operative ileus & urinary retention
- Myasthenia gravis
- Reversal of non-depolarizing neuromuscular blockers
- Glaucoma (topical; intraocular pressure reduction)
- Diagnostic use in autonomic function tests

6. ADVERSE EFFECTS

- Bradycardia
- Hypotension
- Bronchospasm
- Excess salivation, sweating & lacrimation
- Nausea, vomiting, diarrhea
- Muscle cramps, fasciculations (N_M stimulation)

7. CONTRAINDICATIONS

- Asthma or severe COPD
- Peptic ulcer disease
- Mechanical obstruction (GI or urinary)
- Bradycardia or heart block
- Hypersensitivity to drug

8. DRUG INTERACTIONS

- **Anticholinesterases** (e.g., Neostigmine) ↑↑ prolongs ACh action
- **Anticholinergics** (e.g., Atropine) ↓↓ block effects
- **β-Blockers** ↑ risk of bradycardia
- **Depolarizing NM blockers** (e.g., **Succinylcholine**) ↑ prolonged neuromuscular block

9. IMPORTANT EXAMPLES

- ✓ **Acetylcholine chloride (ACh)** – Natural neurotransmitter
- ✓ **Carbachol** – More stable cholinergic agonist
- ✓ **Methacholine** – Selective muscarinic agonist
- ✓ **Nicotine** – Nicotinic receptor agonist (non-selective)

10. MNEMONICS

"MUSCLES & GLANDS GO, NERVE NODES GO TOO!"

M = Muscarinic – Glands, Gut, Heart, Eye
N = Nicotinic – Nerve ganglia, Muscles

Think: ACh Activates All!

11. EXAM POINTS

- ACh is rapidly inactivated by acetylcholinesterase.
- Produces brief and short-lived effects.
- Stimulates both muscarinic and **nicotinic** receptors.
- Basis for parasympathomimetic actions.
- Key neurotransmitter of both autonomic & somatic systems.

12. IMPORTANT QUESTIONS

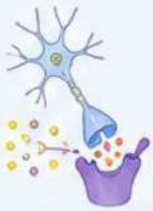
1. Differentiate between muscarinic and nicotinic actions of acetylcholine.
2. Why is acetylcholine not used therapeutically systemically?
3. How does acetylcholine help in myasthenia gravis?
4. List the adverse effects of acetylcholine.
5. Mention drugs that potentiate and block the action of acetylcholine.

High-Yield Fact: ACh is the first discovered neurotransmitter and the master key of cholinergic transmission!

Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

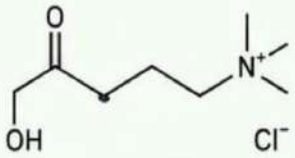
DEFINITION

Bethanechol is a direct-acting cholinergic (muscarinic) agonist which stimulates muscarinic receptors.



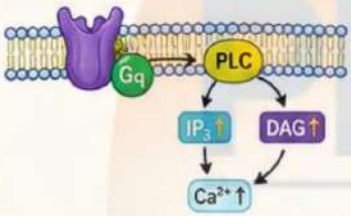
CLASSIFICATION

- **Class:** Cholinergic agonist
- **Subclass:** Muscarinic agonist
- **Chemical Class:** Choline ester
- **Mechanism:** Direct-acting
- **Source:** Synthetic



MECHANISM OF ACTION

- Directly stimulates muscarinic receptors (M_2, M_3).
- Activates G_q protein → Phospholipase C activation → ↑ IP_3 & DAG → ↑ intracellular Ca^{2+} → Smooth muscle contraction & glandular secretion.



BETHANECHOL*

Direct muscarinic agonist; stimulates bladder contraction.

PHARMACOLOGICAL EFFECTS

- Stimulates bladder contraction (↑ detrusor tone)
- Increases GI motility
- Increases salivary, lacrimal, bronchial & gastric secretions
- Causes miosis (pupil constriction)
- May cause bradycardia
- Facilitates nasal drainage

THERAPEUTIC USES

- Postoperative or postpartum urinary retention
- Neurogenic (atonic) bladder
- Non-obstructive urinary retention
- To assist in bladder emptying (e.g., after surgery or spinal injury)

ADVERSE EFFECTS

- Abdominal cramps
- Diarrhea
- Nausea & vomiting
- Increased salivation
- Urinary urgency
- Bradycardia
- Sweating
- Bronchospasm (in susceptible)

CONTRAINDICATIONS

- Mechanical obstruction in GI or urinary tract
- Asthma or severe COPD
- Bradycardia or heart block
- Peptic ulcer
- Peritonitis
- Hypersensitivity

IMPORTANT EXAMPLES

- Bethanechol chloride (Most commonly used)

DRUG INTERACTIONS

- Anticholinergics (e.g., atropine): Antagonize effect
- Cholinesterase inhibitors: Prolong & enhance effect
- Beta-blockers: ↑ risk of bradycardia
- Succinylcholine: Prolongs apnea

MNEMONICS

"BETHA – Bladder Tightens"

- B – Bladder contraction
- E – Enhances GI motility
- T – Tears & Saliva ↑
- H – Heart rate ↓
- A – Assists urination

EXAM POINTS

- ★ Direct muscarinic agonist
- ★ Not degraded by AChE easily
- ★ Poor CNS penetration
- ★ Used in atonic bladder
- ★ Causes miosis & bradycardia

EXAM BOOSTER

IMPORTANT QUESTIONS

1. **What is bethanechol?**
   → Direct muscarinic receptor agonist.
2. **Main use of bethanechol?**
   → Atonic (neurogenic) bladder.
3. **Does it cross BBB?**
   → Poorly.
4. **Major adverse effect?**
   → Bradycardia.

Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

1. DEFINITION

- Selective α_1-adrenergic receptor blocker
- Peripheral vasodilator
- Antihypertensive and anti-BPH drug

2. CLASSIFICATION

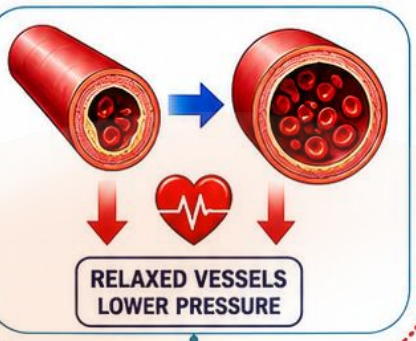
- Sympatholytic drug
- Selective α_1 blocker
- Antihypertensive agent
- Uroselective benefit in BPH

3. MECHANISM OF ACTION

- Blocks postsynaptic α_1 receptors in arterioles and veins
- Prevents IP3 / Ca^{2+} mediated smooth-muscle contraction
- Causes vasodilation
- Decreases peripheral vascular resistance
- Relaxes smooth muscle in bladder neck and prostate
- Minimal effect on presynaptic α_2 receptors

4. PHARMACOLOGICAL EFFECTS

- Decreases blood pressure
- Reduces preload and afterload
- Improves urine flow in BPH
- Causes less reflex tachycardia than non-selective α blockers
- Improves symptoms of Raynaud-like vasospasm in some patients



Prazosin

α_1 blocker causing vasodilation

5. THERAPEUTIC USES

- Hypertension
- Benign prostatic hyperplasia (BPH)
- Adjunct in heart failure (historical / limited role)
- Raynaud phenomenon
- PTSD-related nightmares / sleep disturbance (exam note: more class-related use, mention cautiously)

6. ADVERSE EFFECTS

- First-dose postural hypotension / syncope
- Dizziness
- Headache
- Palpitations / mild tachycardia
- Weakness / fatigue
- Nasal congestion
- Edema

7. CONTRANIDICATIONS

- Severe hypotension
- History of syncope with α blockers
- Use cautiously in elderly patients
- Use cautiously with volume depletion
- Caution with other antihypertensives and PDE5 inhibitors

11. EXAM POINTS

- Prototype selective α_1 blocker
- Major danger: first-dose phenomenon
- Lowers BP mainly by vasodilation, not by β blockade
- Useful in BPH because it relaxes bladder neck and prostate
- Compared with non-selective α blockers, causes less reflex tachycardia
- Common adverse effects: postural hypotension, dizziness, palpitations

12. IMPORTANT QUESTIONS

1. What is the mechanism of action of prazosin?
2. Why does prazosin cause first-dose syncope?
3. Mention four therapeutic uses of prazosin.
4. Write common adverse effects of prazosin.
5. How does prazosin differ from phenoxybenzamine and tamsulosin?

8. DRUG INTERACTIONS

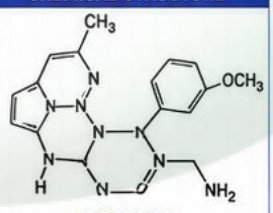
- Other antihypertensives enhance hypotension
- Diuretics may increase first-dose hypotension
- PDE5 inhibitors increase risk of severe postural hypotension
- Alcohol may worsen dizziness
- NSAIDs may reduce antihypertensive response slightly

9. IMPORTANT EXAMPLES

- Prazosin
- Terazosin
- Doxazosin
- Tamsulosin
- Alfuzosin

Prazosin, terazosin, doxazosin = α_1 blockers; tamsulosin is more uroselective.

CHEMICAL STRUCTURE



PRAZOSIN

10. MNEMONICS

PRAZO = Pressure Relieved And Zone Open

- P = Pressure falls
- R = Relaxes vessels
- A = α_1 blocked
- Z = Zone of prostate relaxes
- O = Orthostatic hypotension

ROUTE	MAIN ACTION	RESULT	SPECIAL NOTE
Oral	↓ peripheral resistance	↓ BP, ↑ urine flow	first-dose effect

★ HIGH-YIELD TAKEAWAY ★

Prazosin is a selective α_1 blocker that causes arteriolar and venous vasodilation, lowering blood pressure and improving urinary outflow in BPH.

Important exam point: first-dose postural hypotension / syncope.

Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

SLOWER HEART
BETTER CONTROL

Metoprolol

β_1 blocker reducing heart rate

1 DEFINITION

- Selective β_1-adrenergic receptor blocker
- Cardioselective beta blocker
- Reduces heart rate and myocardial contractility
- Lowers blood pressure and cardiac workload

2 CLASSIFICATION

- Sympatholytic drug
- Selective β_1 blocker
- Class II antiarrhythmic
- Antihypertensive / antianginal agent

3 MECHANISM OF ACTION

- Blocks β_1 receptors in heart and juxtaglomerular cells
- Decreases G_s-mediated cAMP formation
- Slows SA node firing and AV conduction
- Decreases contractility and cardiac output
- Reduces renin release from kidney
- Overall lowers sympathetic cardiac stimulation

4 PHARMACOLOGICAL EFFECTS

- Decreases heart rate
- Decreases blood pressure
- Decreases myocardial oxygen demand
- Antianginal effect
- Suppresses some supraventricular arrhythmias
- Reduces renin-mediated effects

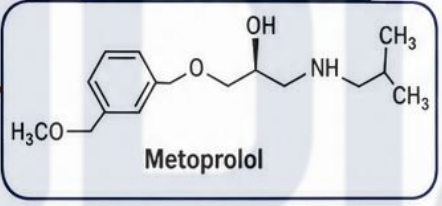
5 THERAPEUTIC USES

- Hypertension
- Chronic stable angina
- Rate control in tachyarthythmias
- Post-myocardial infarction therapy
- Chronic heart failure (metoprolol succinate)
- Symptomatic relief in hyperthyroidism
- Migraine prophylaxis

6 ADVERSE EFFECTS

- Bradycardia
- Fatigue / weakness
- Hypotension
- Dizziness
- Cold extremities
- Sleep disturbance or depression
- Bronchospasm at high dose / in susceptible patients
- Sexual dysfunction

CHEMICAL STRUCTURE



Metoprolol

7 CONTRAINDICATIONS

- Severe bradycardia
- Second- or third-degree AV block
- Cardiogenic shock
- Acute decompensated heart failure
- Severe hypotension
- Use cautiously in asthma / COPD and diabetes

8 DRUG INTERACTIONS

- Verapamil or diltiazem may increase bradycardia
- Digoxin may further slow AV conduction
- Other antihypertensives increase hypotension
- Insulin / antidiabetics: may mask hypoglycemia symptoms
- CYP2D6 inhibitors may increase metoprolol effect
- Clonidine withdrawal with beta blocker may worsen rebound hypertension

ROUTE: Oral / IV

FORM: Tartrate (IR), Succinate (ER)

MAIN ACTION: ↓ HR, ↓ contractility

RESULT: ↓ BP, ↓ O_2 demand

9 IMPORTANT EXAMPLES

- Metoprolol tartrate
- Metoprolol succinate
- Atenolol
- Bisoprolol
- Nebivolol
- Esmolol

★ Metoprolol tartrate = immediate release; metoprolol succinate = extended release, commonly used in heart failure.

10 MNEMONICS

METO = Make Every Tachycardia Outcome slower

- **M** = Myocardial rate slows
- **E** = Eases angina
- **T** = Treats hypertension / tachycardia
- **O** = Output and oxygen demand fall

EXAM BOOSTER — EXAM POINTS

- Prototype cardioselective β_1 blocker
- Lowers HR more than non-cardiac effects at usual dose
- Post-MI and heart failure benefits are important exam facts
- Metoprolol succinate is used in chronic HFrEF
- Common adverse effects: bradycardia, fatigue, hypotension
- Cardioselective does not mean absolutely safe in asthma

12 IMPORTANT QUESTIONS

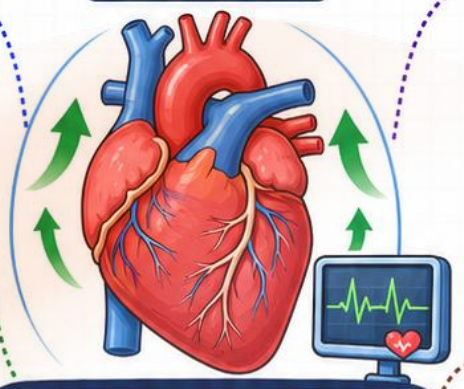
1. What is the mechanism of action of metoprolol?
2. Why is metoprolol called a cardioselective beta blocker?
3. Mention four therapeutic uses of metoprolol.
4. Write common adverse effects and contraindications.
5. Differentiate metoprolol tartrate and metoprolol succinate.

★ HIGH-YIELD TAKEAWAY ★

Metoprolol is a **cardioselective β_1 blocker** that **slows heart rate, reduces cardiac workload,** and **lowers blood pressure.** It is widely used in **hypertension, angina, post-MI care,** and **chronic heart failure,** but may cause **bradycardia** and **hypotension.**

Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

MORE PUMP BETTER OUTPUT



Dobutamine

β1 agonist increasing cardiac output

1 DEFINITION

- Selective β_1-adrenergic agonist
- Direct-acting sympathomimetic inotrope
- Increases myocardial contractility and cardiac output
- Given mainly by IV infusion

2 CLASSIFICATION

- Sympathomimetic drug
- Direct-acting adrenergic agonist
- Predominantly β_1 agonist
- Cardiac stimulant / positive inotrope

3 MECHANISM OF ACTION

- Stimulates β_1 receptors in the heart
- Activates G_s protein → adenylyl cyclase
- Increases cAMP and activates PKA
- Increases Ca^{2+} influx and Ca^{2+} availability
- Produces strong positive inotropic effect
- Mild increase in heart rate compared with contractility

4 PHARMACOLOGICAL EFFECTS

- Increases force of cardiac contraction
- Increases stroke volume and cardiac output
- Mild increase in heart rate
- Improves tissue perfusion in low-output states
- May slightly reduce peripheral vascular resistance
- Little change or mild increase in blood pressure

5 THERAPEUTIC USES

- Acute heart failure
- Cardiogenic shock with *low cardiac output*
- Low-output state *after cardiac surgery*
- Acute decompensated heart failure
- Stress echocardiography
- Septic shock *with myocardial dysfunction* (adjunct)

6 ADVERSE EFFECTS

- Tachycardia
- Palpitations
- Arrhythmias
- Angina / chest pain
- Hypertension or hypotension
- Headache
- Nausea

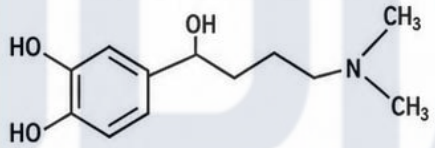
7 CONTRAINDICATIONS

- Hypertrophic obstructive cardiomyopathy
- Significant tachyarrhythmias
- Atrial fibrillation with rapid ventricular response
- Severe ischemic heart disease (use cautiously)
- Hypersensitivity to the drug

8 DRUG INTERACTIONS

- β blockers antagonize effect
- MAO inhibitors may potentiate response
- Tricyclic antidepressants may enhance effects
- Halogenated anesthetics increase arrhythmia risk
- Other sympathomimetics increase cardiovascular toxicity

CHEMICAL STRUCTURE



Catecholamine derivative

ROUTE:	ONSET:	DURATION:	USE SETTING:
IV infusion	1–2 min	Very short	ICU / emergency / monitored care

9 IMPORTANT EXAMPLES

- Dobutamine
- Dopamine (β_1 effect at moderate dose)
- Isoproterenol (non-selective β agonist)
- Dopexamine

Dobutamine is the main selective cardiac inotrope among these.

10 MNEMONICS

DOBUTA = Drive Output Better Under Tired heart Activity

- **D** = Direct β_1 agonist
- **O** = Output increases
- **B** = Beats stronger
- **U** = Used by IV infusion
- **T** = Tachycardia risk
- **A** = Acute heart failure use

11 EXAM POINTS

- Prototype selective β_1 agonist and positive inotrope
- Major action: increases contractility more than heart rate
- Used only by IV because of very short action
- Important use: acute heart failure / cardiogenic shock
- Not a vasopressor; severe hypotension may need another drug too
- Common adverse effects: tachycardia, arrhythmias, angina

12 IMPORTANT QUESTIONS

1. What is the mechanism of action of dobutamine?
2. Why is dobutamine called a positive inotrope?
3. Mention four therapeutic uses of dobutamine.
4. Write common adverse effects of dobutamine.
5. How does dobutamine differ from dopamine?

HIGH-YIELD TAKEAWAY

Dobutamine is a selective **β_1 agonist** that increases cardiac contractility and cardiac output rapidly. It is mainly used in **acute heart failure** and **cardiogenic shock**, but may cause ***tachycardia*** and ***arrhythmias***.

Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

1 DEFINITION

- Selective β_2-adrenergic agonist
- Short-acting bronchodilator
- Relieves acute bronchospasm quickly

2 CLASSIFICATION

- Sympathomimetic drug
- Selective β_2 agonist
- SABA (short-acting β_2 agonist)
- Antiasthmatic bronchodilator

3 MECHANISM OF ACTION

- Stimulates β_2 receptors in bronchial smooth muscle
- Activates G_s protein → adenylyl cyclase
- Increases cAMP and activates PKA
- Decreases intracellular Ca2+ and MLCK activity
- Produces smooth muscle relaxation
- Causes bronchodilation and less mediator release

4 PHARMACOLOGICAL EFFECTS

- Bronchodilation
- Decreases airway resistance
- Increases mucociliary clearance
- Mild uterine relaxation
- Drives K+ into cells
- May cause slight tachycardia

5 THERAPEUTIC USES

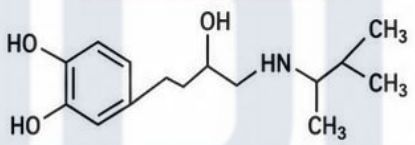
- Acute bronchial asthma attack
- Rescue inhaler in asthma
- COPD with reversible bronchospasm
- Prevention of exercise-induced bronchospasm
- Adjunct in hyperkalemia
- Occasionally as tocolytic / uterine relaxant

OPEN AIRWAYS
EASY BREATHING

Salbutamol

β_2 agonist causing bronchodilation

CHEMICAL STRUCTURE



IUPAC: 4-[2-(tert-Butylamino)-1-hydroxyethyl] benzene-1,2-diol

ROUTE:	ONSET:	DURATION:	FORM:
Inhalation (Preferred)	2–5 min	4–6 hours	MDI, DPI, Nebulizer, Tablets

6 ADVERSE EFFECTS

- Fine tremor
- Tachycardia / palpitations
- Nervousness or restlessness
- Headache
- Hypokalemia
- Muscle cramps
- Hyperglycemia in some patients

7 CONTRAINDICATIONS

- Tachyarrhythmias
- Severe uncontrolled cardiac disease
- Hypersensitivity to the drug
- Use cautiously in hyperthyroidism
- Use cautiously in diabetes and hypertension

8 DRUG INTERACTIONS

- Non-selective β blockers antagonize effect
- MAO inhibitors and TCAs may increase cardiovascular effects
- Diuretics and corticosteroids increase hypokalemia risk
- Other sympathomimetics increase adverse effects
- Digoxin effect may be altered

9 IMPORTANT EXAMPLES

- Salbutamol
- Terbutaline
- Bambuterol
- Salmeterol
- Formoterol

Salbutamol and terbutaline = short-acting; salmeterol and formoterol = long-acting.

10 MNEMONICS

B2 = Breathe Better

- **B** = Bronchodilation
- **R** = Rescue in asthma
- **E** = Exercise-induced asthma prevention
- **A** = Activates cAMP
- **T** = Tremor / tachycardia
- **H** = Helps shift K+ into cells

11 EXAM POINTS

- Prototype short-acting selective β_2 agonist
- Best known as a **rescue** bronchodilator
- Works by β_2 → G_s → **cAMP** pathway
- Not a steroid and not anti-inflammatory by itself
- Common adverse effects: **tremor, tachycardia, hypokalemia**
- Preferred route in asthma: **inhalation**

12 IMPORTANT QUESTIONS

1. What is the mechanism of action of salbutamol?
2. Why is salbutamol called a rescue drug?
3. Mention four therapeutic uses of salbutamol.
4. Write common adverse effects of salbutamol.
5. How does salbutamol differ from non-selective β agonists?

HIGH-YIELD TAKEAWAY

Salbutamol is a SABA that stimulates β_2 receptors → ↑ cAMP → bronchodilation. It is used mainly for rapid relief of bronchospasm, but may cause **tremor**, **tachycardia**, and **hypokalemia**.

Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

Clonidine

α_2 agonist reducing sympathetic outflow

1 DEFINITION

- Centrally acting antihypertensive drug
- Selective α_2-adrenergic receptor agonist
- Reduces sympathetic outflow from CNS

2 CLASSIFICATION

- Sympatholytic drug
- Centrally acting α_2 agonist
- Antihypertensive agent

3 MECHANISM OF ACTION

- Stimulates α_2 receptors in brainstem
- Decreases norepinephrine release
- Lowers sympathetic tone
- Reduces heart rate and peripheral vascular resistance
- Also decreases renin release

4 PHARMACOLOGICAL EFFECTS

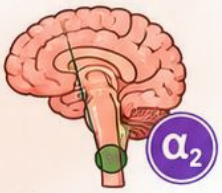
- Decreases blood pressure
- Causes bradycardia
- Produces sedation
- Reduces cardiac output slightly
- Decreases plasma catecholamines

5 THERAPEUTIC USES

- Hypertension
- Hypertensive urgency adjunct
- ADHD
- Migraine prophylaxis adjunct
- Opioid withdrawal symptoms
- Menopausal hot flushes
- Neuropathic pain adjunct / epidural analgesia adjunct

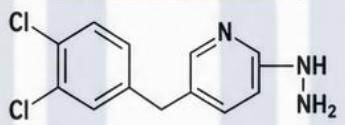
BRAINSTEM (Medulla)

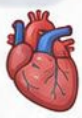
α_2 Receptors in Nucleus Tractus Solitarius



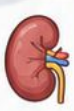
↓ Sympathetic Outflow

CLONIDINE STRUCTURE




↓ Heart Rate (Bradycardia)


↓ Peripheral Vascular Resistance


↓ Renin Release


↓ Norepinephrine Release

RESULT: DECREASED BLOOD PRESSURE

6 ADVERSE EFFECTS

- Dry mouth
- Sedation / drowsiness
- Bradycardia
- Constipation
- Dizziness
- Postural hypotension
- Rebound hypertension on sudden withdrawal

7 CONTRAINDICATIONS

- Severe bradycardia
- Heart block
- Hypotension
- Use cautiously in depression
- Caution in renal impairment

8 DRUG INTERACTIONS

- Sedatives and alcohol increase CNS depression
- Other antihypertensives increase hypotension
- Beta blockers may worsen withdrawal hypertension if clonidine is stopped abruptly
- Tricyclic antidepressants may reduce clonidine effect

9 IMPORTANT EXAMPLES

- Clonidine
- Methyldopa
- Guanfacine
- Dexmedetomidine

α_2 AGONISTS

Note: Clonidine and guanfacine are α_2 agonists; dexmedetomidine is used mainly for sedation.

10 MNEMONICS

"Clonidine CALMS the Sympathetic System"

- **C** = CNS action
- **A** = α_2 agonist
- **L** = Lowers BP
- **M** = Makes mouth dry
- **S** = Sedation

EXAM BOOSTER

11 EXAM POINTS

- Prototype centrally acting α_2 agonist
- Major danger: rebound hypertension if stopped suddenly
- Common side effects: dry mouth and sedation
- Acts by reducing sympathetic outflow, not by direct vasodilation
- Also used in ADHD and withdrawal states

12 IMPORTANT QUESTIONS

1. What is the mechanism of clonidine?
2. Why does sudden withdrawal cause rebound hypertension?
3. Name two clinical uses other than hypertension.
4. Mention two common adverse effects.
5. How is clonidine different from peripheral adrenergic blockers?

HIGH-YIELD TAKEAWAY: Clonidine reduces sympathetic outflow via central α_2 receptors → ↓ HR, ↓ BP, ↓ renin. Use carefully and never stop abruptly!

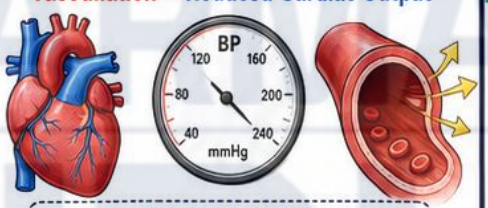
Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

Labetalol

(α and β Receptor Blocker)

Combined α_1 Adrenergic Antagonist + Non-selective β-Blocker

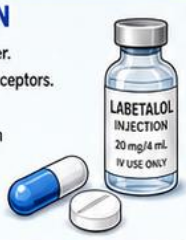
Lowers Blood Pressure by Vasodilation + Reduced Cardiac Output



RECEPTORS BLOCKED

- α_1 Receptor BLOCKED
- β_1 Receptor BLOCKED
- β_2 Receptor BLOCKED

1 DEFINITION



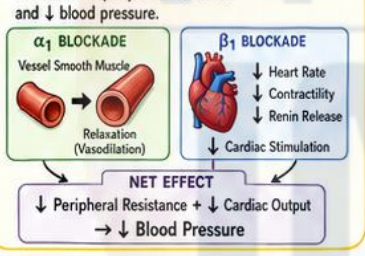
- Mixed adrenergic blocker.
- Blocks α_1, β_1, and β_2 receptors.
- Antihypertensive drug.
- Causes vasodilation with less reflex tachycardia.
- Used orally and IV.

2 CLASSIFICATION

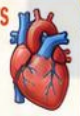
- Combined α_1 blocker + non-selective β-blocker.
- Adrenergic antagonist.
- Prototype mixed blocker.
- Used mainly in cardiovascular disorders.
- Useful in hypertensive emergencies and pregnancy hypertension.

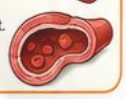
3 MECHANISM OF ACTION

- α_1 blockade → vasodilation.
- β_1 blockade → ↓ heart rate and ↓ contractility.
- β_1 blockade also ↓ renin release.
- β_2 blockade present due to non-selective β blockade.
- Net effect: ↓ peripheral resistance and ↓ blood pressure.



4 PHARMACOLOGICAL EFFECTS





- Decreases blood pressure.
- Reduces peripheral vascular resistance.
- Mild decrease in heart rate.
- Decreases cardiac output.
- Antianginal and antihypertensive effect.
- Little reflex tachycardia due to β blockade.

5 THERAPEUTIC USES

- Hypertension.
- Hypertensive emergency.
- Pregnancy-induced hypertension / preeclampsia.
- Chronic hypertension in pregnancy.
- Aortic dissection or severe BP control settings.
- Sometimes used perioperatively.

6 ADVERSE EFFECTS

- Postural hypotension.
- Dizziness.
- Bradycardia.
- Fatigue.
- Bronchospasm.
- Scalp tingling / paresthesia.
- Nausea.

7 CONTRAINDICATIONS / CAUTIONS

- Bronchial asthma or severe COPD.
- Severe bradycardia.
- AV block.
- Cardiogenic shock.
- Decompensated heart failure.
- Use cautiously in liver disease.
- Monitor in diabetes mellitus.

8 DRUG INTERACTIONS

- Other antihypertensives may increase hypotension.
- Verapamil or diltiazem may increase bradycardia risk.
- Anesthetics may enhance BP fall.
- Insulin / antidiabetics: may mask hypoglycemia.
- NSAIDs may reduce antihypertensive effect.
- Use cautiously with clonidine.

EXAM BOOSTER

9 IMPORTANT EXAMPLES

- Labetalol tablets.
- Labetalol injection.
- Common mixed α + β blocker example.
- Oral and IV formulations.

10 MNEMONICS

- "LA-BETA-LOW" = lowers BP by β block.
- "LABE = alpha + beta together.
- Remember: vessel relaxation + heart slowing.
- Key use: pregnancy hypertension.

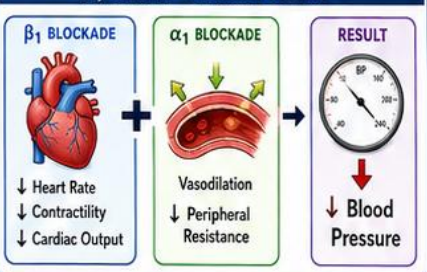
11 EXAM POINTS

- Mixed α_1 + β blocker.
- Non-selective β blockade with α_1 blockade.
- Important drug for hypertensive emergency.
- Preferred and important in pregnancy hypertension.
- Causes postural hypotension.
- Avoid in asthma.
- IV form is high-yield.

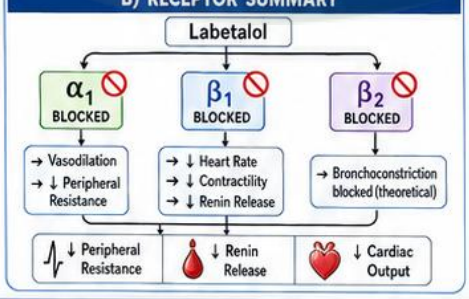
12 IMPORTANT QUESTIONS

- Why does labetalol cause less reflex tachycardia?
- Why is it useful in hypertensive emergency?
- Why is it preferred in pregnancy hypertension?
- Why should it be avoided in asthma?
- Which receptors are blocked by labetalol?

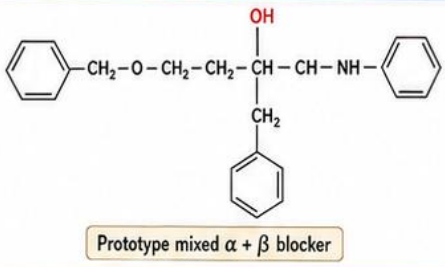
A) HEART + VESSEL EFFECT



B) RECEPTOR SUMMARY



C) CHEMICAL STRUCTURE OF LABETALOL



Prototype mixed α + β blocker

α_1 + β BLOCKADE → VASODILATION + ↓ HEART WORK → ↓ BP

Mixed Blocker • Hypertensive Emergency • Pregnancy Hypertension

Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

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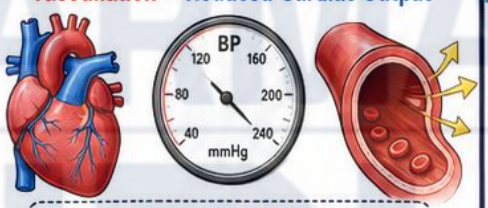

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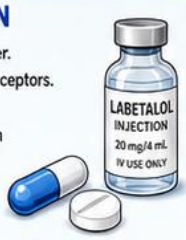

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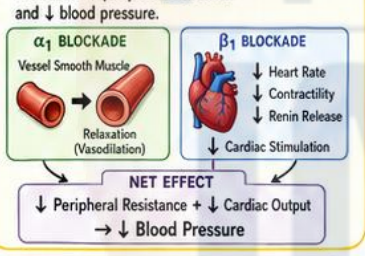
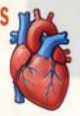
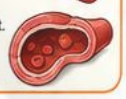


4 PHARMACOLOGICAL EFFECTS





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EXAM BOOSTER

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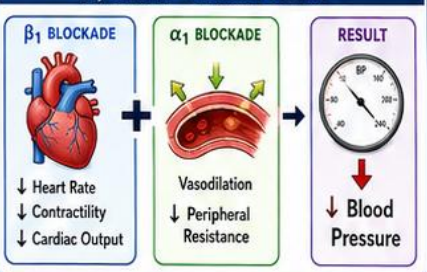

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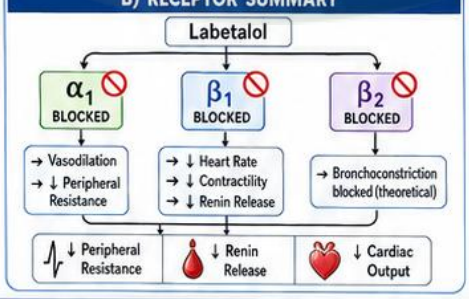

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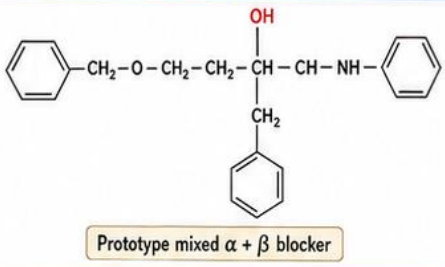


Prototype mixed α + β blocker

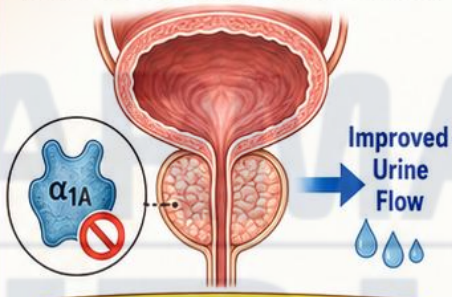
α_1 + β BLOCKADE → VASODILATION + ↓ HEART WORK → ↓ BP

Mixed Blocker • Hypertensive Emergency • Pregnancy Hypertension

Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

Tamsulosin
(Selective α_{1A} Blocker)

Uroselective α_{1A} Adrenergic Antagonist
Relaxes Prostate and Bladder Neck in BPH



★ Exam Booster ★

1 Definition

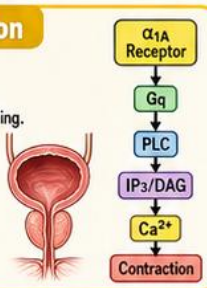
- Selective α_{1A}-adrenergic blocker.
- Uroselective drug for BPH.
- Relaxes smooth muscle in prostate and bladder neck.
- Improves urinary flow.
- Less effect on blood pressure than non-selective α_1 blockers.

2 Classification

- α_1-adrenergic antagonist.
- Selective α_{1A} blocker.
- Uroselective sympatholytic drug.
- Used mainly for lower urinary tract symptoms.
- Safer than non-selective α_1 blockers for BP effects.

3 Mechanism of Action

- Blocks α_{1A} receptors in prostate, prostatic urethra, and bladder neck.
- Prevents $G_q \rightarrow PLC \rightarrow IP_3/DAG$ signaling.
- Decreases intracellular Ca^{2+}.
- Causes smooth muscle relaxation.
- Reduces urinary outflow resistance.
- Improves urine passage in BPH.

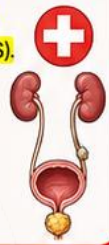


4 Pharmacological Effects

- Relaxes prostate and bladder neck.
- Increases urinary flow rate.
- Reduces hesitancy and weak stream.
- Decreases incomplete bladder emptying.
- Reduces frequency and nocturia.
- Minimal BP lowering at usual dose.

5 Therapeutic Uses

- Benign prostatic hyperplasia (BPH).
- Lower urinary tract symptoms (LUTS).
- Helpful in urinary hesitancy.
- Improves obstructive urinary symptoms.
- Sometimes used in medical expulsive therapy for ureteric stones.



6 Adverse Effects

- Dizziness.
- Postural hypotension (less common).
- Headache.
- Abnormal ejaculation / retrograde ejaculation.
- Rhinitis or nasal congestion.
- Fatigue.

7 Contraindications / Cautions

- Use cautiously in hypotension.
- Caution with severe hepatic impairment.
- Caution in patients using antihypertensives.
- Inform ophthalmologist before cataract surgery.
- Risk of intraoperative floppy iris syndrome.
- Avoid sudden position changes.

8 Drug Interactions

- CYP3A4 inhibitors increase levels.
- CYP2D6 inhibitors may increase exposure.
- PDE-5 inhibitors may increase hypotension.
- Other antihypertensives enhance BP fall.
- Cimetidine may increase plasma concentration.
- Use carefully with alcohol or vasodilators.

9 Important Examples

- Tamsulosin capsules.
- Tamsulosin modified-release formulations.
- Tamsulosin + dutasteride combination.
- Common uroselective α_1 blocker example.

10 Mnemonics

- "TAM = Tract And Male urine flow."
- "Tamsulosin helps the stream."
- "α_{1A} = A for urinary outlet area."
- Remember: prostate relaxation with less BP drop.

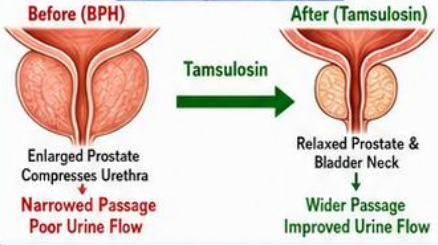
11 Exam Points

- Selective α_{1A} blocker.
- Drug of choice for symptomatic BPH.
- Relaxes prostate and bladder neck.
- Less first-dose hypotension than prazosin-type drugs.
- May cause ejaculation problems.
- Important adverse effect: floppy iris syndrome.
- Minimal effect on blood pressure.

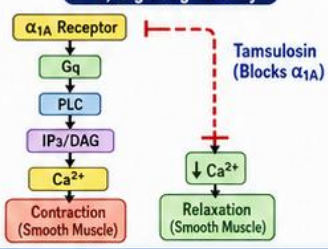
12 Important Questions

- Why is tamsulosin preferred in BPH?
- Why does it cause less hypotension?
- Why can ejaculation be affected?
- Why must cataract surgery history be asked?
- How does α_{1A} blockade improve urine flow?

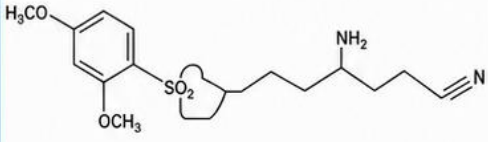
A) Urinary Outflow in BPH



B) Signaling Pathway



C) Chemical Structure of Tamsulosin



Prototype uroselective α_{1A} blocker

α_{1A} BLOCKADE → PROSTATE RELAXATION → BETTER URINE FLOW

Uroselective • BPH Relief • Less BP Effect

Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

1 DEFINITION

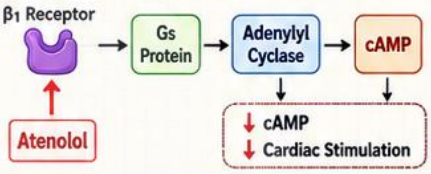
- Cardioselective β-blocker.
- Selectively blocks β_1 receptors.
- Sympatholytic, direct receptor antagonist.
- Mainly acts on the heart.
- Lowers heart rate and blood pressure.
- Less bronchoconstriction than non-selective β-blockers.

2 CLASSIFICATION

- Second-generation β-blocker.
- β_1-selective (cardioselective) agent.
- No intrinsic sympathomimetic activity.
- Relatively hydrophilic drug.
- Used mainly in cardiovascular disorders.

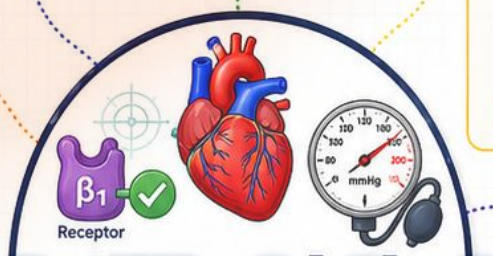
3 MECHANISM OF ACTION

- Competitively blocks β_1 adrenergic receptors.
- Reduces effects of adrenaline and noradrenaline on the heart.
- Decreases heart rate (negative chronotropic effect).
- Decreases contractility (negative inotropic effect).
- Slows AV conduction.
- Decreases renin release from kidney.
- Net effect: ↓ cardiac output and ↓ blood pressure.



4 PHARMACOLOGICAL EFFECTS

- Decreases heart rate.
- Decreases myocardial contractility.
- Lowers cardiac output.
- Decreases blood pressure.
- Reduces myocardial oxygen demand.
- Antianginal and antiarrhythmic effect.
- Minimal effect on β_2 receptors at usual doses.



Atenolol

(Selective β_1 Blocker)

Cardioselective β-blocker

Reduces Heart Rate, Contractility and Blood Pressure

★ EXAM BOOSTER ★

5 THERAPEUTIC USES

- Hypertension.
- Angina pectoris.
- Supraventricular tachyarrhythmias.
- Post-myocardial infarction prophylaxis.
- Chronic stable angina.
- Rate control in some arrhythmias.
- Sometimes used in hyperthyroid symptoms.

6 ADVERSE EFFECTS

- Bradycardia.
- Hypotension.
- Fatigue and weakness.
- Dizziness.
- Cold extremities.
- Sleep disturbance (less CNS effect than lipophilic β-blockers).
- May mask hypoglycemia symptoms.

7 CONTRAINDICATIONS / CAUTIONS

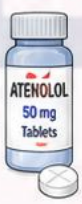
- Severe bradycardia.
- AV block.
- Cardiogenic shock.
- Decompensated heart failure.
- Use cautiously in diabetes mellitus.
- Use cautiously in peripheral vascular disease.
- Caution in asthma/COPD, though safer than non-selective β-blockers.
- Do not stop abruptly.

8 DRUG INTERACTIONS

- Verapamil or diltiazem may increase bradycardia risk.
- Digoxin may enhance AV nodal suppression.
- Insulin and antidiabetics: may mask hypoglycemia.
- NSAIDs may reduce antihypertensive effect.
- Other antihypertensives may increase hypotension.
- Clonidine withdrawal may worsen rebound hypertension.

9 IMPORTANT EXAMPLES

- Atenolol tablets.
- Atenolol oral formulations.
- Common prototype of cardioselective β_1 blockers.
- Compare with metoprolol and bisoprolol.



10 MNEMONICS

- "ATENO-LOW" = lowers heart rate and BP.
- "A = Acts on β_1".
- Remember: cardioselective β-blocker.
- Think: heart protection with less lung effect.

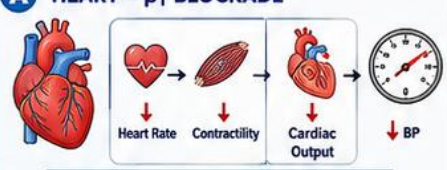
11 EXAM POINTS

- Selective β_1 blocker.
- Cardioselective β-blocker.
- Prototype β_1-selective blocker.
- Less bronchospasm than propranolol.
- Decreases renin release.
- Useful in hypertensioes and angina.
- Can mask hypoglycemia.
- Do not withdraw suddenly.

12 IMPORTANT QUESTIONS

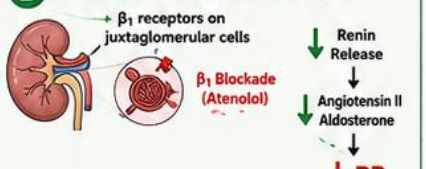
- Why is atenolol called cardioselective?
- Why is atenolol safer than propranolol in asthma?
- How does β_1 blockade reduce blood pressure?
- Why can atenolol mask hypoglycemia?
- Why should β-blockers not be stopped abruptly?

A HEART – β_1 BLOCKADE



Less workload, less oxygen demand

B KIDNEY – RENIN REDUCTION



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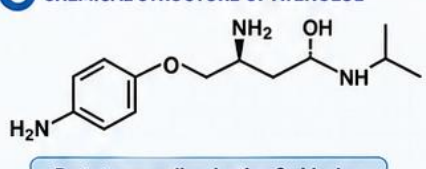


Prototype cardioselective β_1 blocker

β_1 BLOCKADE → ↓ HEART RATE → ↓ BP

Cardioselective • Antihypertensive • Heart Protection

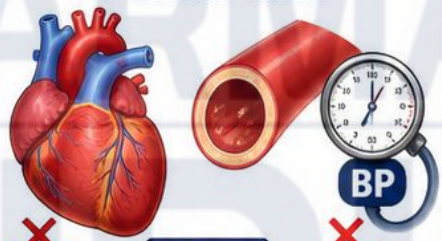
Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

Propranolol

(Non-selective β-Blocker)

Blocks β_1 and β_2 Adrenergic Receptors

Reduces Heart Rate, Contractility and Blood Pressure



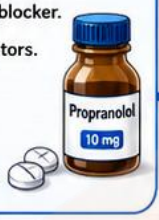
BLOCKADE
↓ cAMP
↓ PKA activity
↓ Cellular Response

β_1 β_2

Exam Booster

1 Definition

- Sympatholytic drug.
- Non-selective β-adrenergic blocker.
- Blocks both β_1 and β_2 receptors.
- Lipophilic agent with CNS penetration.
- Used in cardiovascular and non-cardiovascular disorders.

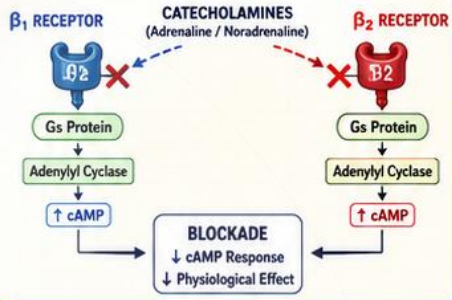


2 Classification

- β-blocker.
- First-generation non-selective β-blocker.
- Direct receptor antagonist.
- No intrinsic sympathomimetic activity.
- Membrane-stabilizing effect at high doses.

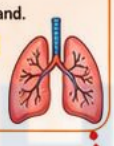
3 Mechanism of Action

- Competitively blocks β_1 and β_2 receptors.
- Inhibits catecholamine action of adrenaline and noradrenaline.
- β_1 blockade → ↓ heart rate and ↓ contractility.
- β_1 blockade → ↓ renin release from kidney.
- β_2 blockade → bronchoconstriction and reduced glycogenolysis.
- Net effect: ↓ cardiac output and ↓ blood pressure.



4 Pharmacological Effects

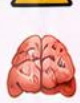
- Decreases heart rate.
- Decreases myocardial contractility.
- Slows AV conduction.
- Lowers blood pressure.
- Decreases myocardial oxygen demand.
- May cause bronchoconstriction.
- Reduces tremor and somatic anxiety symptoms.



5 Therapeutic Uses

- Hypertension.
- Angina pectoris.
- Supraventricular arrhythmias.
- Post-myocardial infarction prophylaxis.
- Migraine prophylaxis.
- Essential tremor.
- Hyperthyroidism / thyrotoxicosis symptom control.
- Portal hypertension.
- Adjunct in pheochromocytoma after α-blockade.

6 Adverse Effects



- Bradycardia.
- Hypotension.
- Fatigue and weakness.
- Bronchospasm.
- Cold extremities.
- Depression, sleep disturbances or nightmares.
- Sexual dysfunction.
- Masks hypoglycemia.

7 Contraindications / Cautions

- Bronchial asthma or severe COPD.
- Severe bradycardia.
- AV block.
- Cardiogenic shock.
- Decompensated heart failure.
- Use cautiously in diabetes mellitus.
- Use cautiously in peripheral vascular disease.
- Do not stop abruptly.

8 Drug Interactions

- Verapamil or diltiazem may increase bradycardia/heart block risk.
- Digoxin may enhance AV nodal suppression.
- Insulin and oral antidiabetics: masks hypoglycemia symptoms.
- NSAIDs may reduce antihypertensive effect.
- Other antihypertensives can increase hypotension.
- Clonidine withdrawal with β-blocker may worsen rebound hypertension.

9 Important Examples

- Propranolol tablets.
- Propranolol sustained-release capsules.
- Propranolol injection.
- Common prototype of non-selective β-blockers.

10 Mnemonics

- ★ "PROPRANOLOL = PROtects the heart by slowing it."
- ★ "β_1 + β_2 both blocked = non-selective blocker."
- ★ "P for Pulse down, Pressure down."
- ★ Remember: can cause bronchospasm, so avoid in asthma."

11 Exam Points

- ☑ Prototype non-selective β-blocker.
- ☑ Blocks both β_1 and β_2 receptors.
- ☑ Lipophilic → crosses BBB.
- ☑ Useful in migraine prophylaxis and essential tremor.
- ☑ Can mask hypoglycemia.
- ☑ Contraindicated in asthma.
- ☑ Do not withdraw suddenly.

12 Important Questions

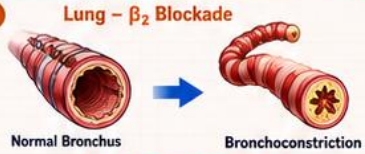
1. Why is propranolol contraindicated in asthma?
2. Why can propranolol mask hypoglycemia?
3. Why must propranolol not be stopped abruptly?
4. Why is propranolol useful in migraine prophylaxis?
5. Which receptors are blocked by propranolol?

A Heart – β_1 Blockade

- ↓ Heart Rate (HR)
- ↓ Contractility
- ↓ Cardiac Output

→ ↓ Blood Pressure

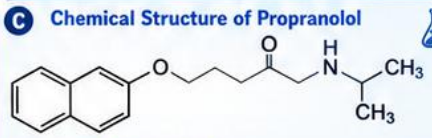
B Lung – β_2 Blockade



Normal Bronchus → Bronchoconstriction

Risk of Bronchospasm

C Chemical Structure of Propranolol



- Prototype non-selective β-blocker.

β_1 + β_2 BLOCKADE → ↓ HEART RATE → ↓ BP

"Less Cardiac Workload • Lower Blood Pressure • Protects the Heart"

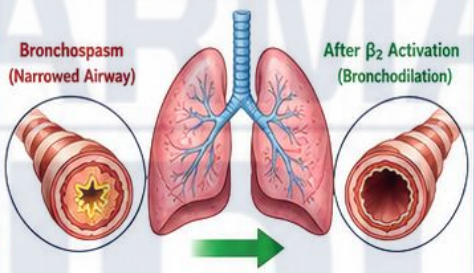
CAUTION
Bronchospasm Risk!

Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

Terbutaline
(β2 Agonist)

Selective β2 Adrenergic Receptor Agonist

Relaxes Bronchial Smooth Muscle



Exam Booster

1 Definition

- Sympathomimetic bronchodilator.
- Selective β2 receptor agonist.
- Relaxes bronchial smooth muscle.
- Used in asthma and bronchospasm.
- Also acts as a tocolytic.

2 Classification

- Adrenergic agonist.
- Direct-acting sympathomimetic.
- Selective β2 agonist.
- Short-acting β2 agonist (SABA).
- Non-catecholamine compound.

3 Mechanism of Action

- Stimulates β2 receptors on bronchial smooth muscle.
- Activates Gs → adenylyl cyclase → ↑ cAMP.
- cAMP activates protein kinase A.
- Reduces intracellular Ca^{2+}.
- Causes smooth muscle relaxation.
- Produces bronchodilation.
- Also relaxes uterine smooth muscle.

4 Pharmacological Effects

- Bronchodilation and ↓ airway resistance.
- Relieves wheeze and dyspnea.
- Improves airflow in obstructive airway disease.
- Inhibits mediator release from mast cells.
- Relaxes uterine smooth muscle.
- May cause mild tachycardia and tremor.

5 Therapeutic Uses

- Bronchial asthma.
- Acute bronchospasm relief.
- Prevention of exercise-induced bronchospasm.
- COPD with reversible bronchospasm.
- Tocolytic use in premature labor.

6 Adverse Effects

- Tremor.
- Tachycardia and palpitations.
- Nervousness or restlessness.
- Headache.
- Hypokalemia.
- Hyperglycemia.
- Muscle cramps.

7 Contraindications / Cautions

- Use cautiously in cardiac arrhythmias.
- Caution in ischemic heart disease.
- Caution in hyperthyroidism.
- Use carefully in diabetes mellitus.
- Caution in severe hypertension.
- Avoid overuse.

8 Drug Interactions

- β-blockers antagonize bronchodilator action.
- MAO inhibitors may enhance cardiovascular effects.
- Tricyclic antidepressants may increase sympathomimetic effects.
- Other sympathomimetics increase toxicity.
- Diuretics may worsen hypokalemia.
- Corticosteroids may add to hypokalemia risk.

9 Important Examples

- Terbutaline sulfate inhaler.
- Terbutaline tablets.
- Terbutaline injection.
- Nebulizer solution preparations.
- Common exam example of a β2 agonist.

10 Mnemonics

- "TERBUTA-LUNG" = opens the lungs.
- "β2 = 2 lungs" for bronchodilation.
- Terbutaline = bronchodilator + tocolytic.
- Remember: tremor is a common β2 effect.

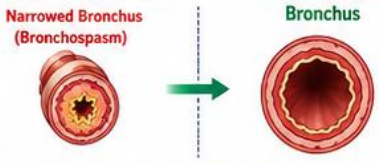
11 Exam Points

- Selective β2 agonist.
- Short-acting bronchodilator.
- Causes bronchodilation by ↑ cAMP.
- Useful in asthma and bronchospasm.
- Can relax uterus in preterm labor.
- Common adverse effect: tremor.
- Cardiac effects may occur at higher doses.

12 Important Questions

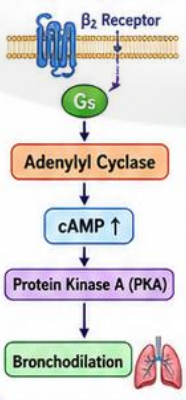
- Why does terbutaline cause tremor?
- Why can tachycardia occur with a β2 agonist?
- Why is terbutaline useful in asthma?
- Why can it be used as a tocolytic?
- How does ↑ cAMP produce bronchodilation?

A) Airway / Bronchus

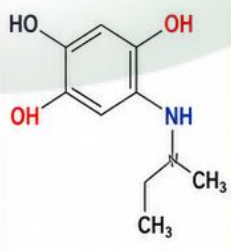


β2 Activation → Smooth Muscle Relaxation

B) Signaling Pathway



C) Chemical Structure of Terbutaline



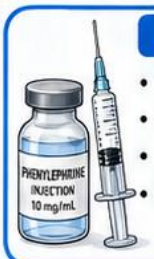
Molecular Formula: $C_{12}H_{19}NO_3$

Molecular Weight: 225.28 g/mol

β2 STIMULATION → BRONCHODILATION → EASIER BREATHING

Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

1) Definition

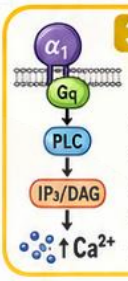


- Sympathomimetic drug.
- Direct-acting selective α_1 agonist.
- Produces vasoconstriction.
- Used as pressor, decongestant, and mydriatic.

2) Classification

- Adrenergic agonist.
- Direct-acting sympathomimetic.
- Selective α_1 receptor agonist.
- Non-catecholamine.
- Mainly peripheral action.

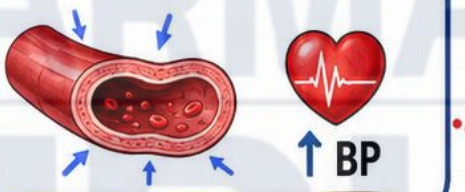
3) Mechanism of Action



- Stimulates α_1 receptors on vascular smooth muscle.
- Activates Gq → PLC → IP3/DAG pathway.
- Increases intracellular Ca^{2+}.
- Causes smooth muscle contraction.
- Leads to vasoconstriction and raised BP.
- In iris radial muscle causes mydriasis.

Phenylephrine (α_1 Agonist)

Selective α_1 Adrenergic Receptor Agonist
Produces Vasoconstriction



★ Exam Booster ★

4) Pharmacological Effects

- Vasoconstriction of arterioles and veins.
- Increases systolic and diastolic BP.
- May cause reflex bradycardia.
- Decreases nasal mucosal edema.
- Produces mydriasis without cycloplegia.

5) Therapeutic Uses

- Hypotension during anesthesia or shock.
- Nasal decongestant.
- Mydriatic in eye examination.
- Support in supraventricular tachycardia vagal maneuver setting.
- Topical relief in hemorrhoids.

6) Adverse Effects

- Hypertension.
- Reflex bradycardia.
- Headache and anxiety.
- Palpitations.
- Ischemia with excessive vasoconstriction.
- Rebound nasal congestion on prolonged topical use.

7) Contraindications / Cautions

- Severe hypertension.
- Ischemic heart disease.
- Hyperthyroidism.
- Narrow-angle glaucoma.
- Use cautiously in elderly patients.
- Avoid excessive topical overuse.

8) Drug Interactions

- MAO inhibitors potentiate action.
- Tricyclic antidepressants increase pressor response.
- Other sympathomimetics may increase toxicity.
- Use carefully with antihypertensive therapy.
- β-blockers may enhance reflex bradycardia.

9) Important Examples

- Phenylephrine injection.
- Phenylephrine nasal drops / spray.
- Phenylephrine ophthalmic drops.
- Common α_1 agonist exam example.

10) Mnemonics

- "PHE = PRESSURE" → raises BP.
- "PhenylEYEphrine" → dilates pupil.
- "α_1 = one vessel tightens".
- Remember: vasoconstrictor + decongestant.

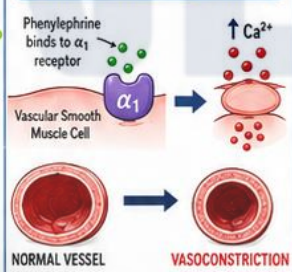
11) Exam Points

- Selective α_1 agonist.
- Causes vasoconstriction and increases BP.
- Can produce reflex bradycardia.
- Mydriasis occurs without cycloplegia.
- Used in hypotension and nasal congestion.
- Important non-catecholamine sympathomimetic.

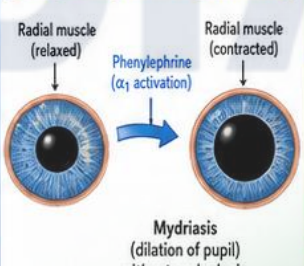
12) Important Questions

- Why does phenylephrine cause reflex bradycardia?
- Why does it produce mydriasis without cycloplegia?
- Why are MAOIs important with phenylephrine?
- Why is it useful as a nasal decongestant?

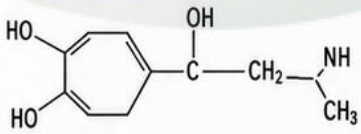
A) Blood Vessel – α_1 Activation



B) Eye – Mydriasis (Pupil Dilation)



Chemical Structure of Phenylephrine



Molecular Formula: $C_9H_{13}NO_3$
Molecular Weight: 183.20 g/mol

α_1 STIMULATION → VASOCONSTRICTION → ↑ BP

Pharmacology Classification + Specific Mechanism of Action (MOA) – 200 High-Yield One-Liners

Pralidoxime (2-PAM)

Acetylcholinesterase Reactivator in Organophosphate Poisoning

1) Definition

- Oxime antidote.
- Reactivates phosphorylated acetylcholinesterase.
- Used in organophosphate (OP) poisoning.
- Best given **early** with atropine.

2) Classification

- Cholinesterase reactivator.
- Specific antidote for OP poisoning.
- Oxime compound.
- Peripheral action > CNS due to poor BBB entry.

3) Mechanism of Action

- Binds to phosphorus attached to acetylcholinesterase.
- Removes phosphate from inhibited enzyme.
- Restores acetylcholine breakdown.
- Reverses nicotinic effects, especially at **NMJ.**
- Most effective **before "aging"** of enzyme occurs.
- Does **NOT** directly antagonize muscarinic receptors.

4) Pharmacological Effects

- Improves skeletal muscle weakness.
- Relieves fasciculations and paralysis.
- Improves **respiratory** muscle function.
- Helps reduce excess acetylcholine at synapses.
- Limited effect on CNS symptoms.

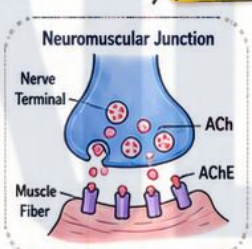
5) Therapeutic Uses

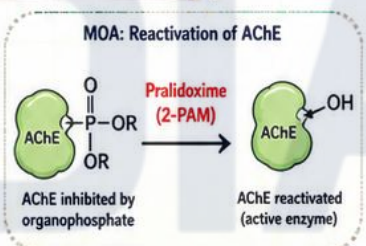
- Organophosphate insecticide poisoning.
- **Nerve** agent poisoning.
- Given along with atropine and supportive care.
- Useful for **respiratory** muscle weakness.
- **Not useful** for carbamate poisoning in many cases.

6) Adverse Effects

- Nausea, dizziness.
- Tachycardia.
- Hypertension.
- Blurred vision or diplopia.
- Injection-site pain.
- Neuromuscular stiffness if excessive dose.

Exam Booster

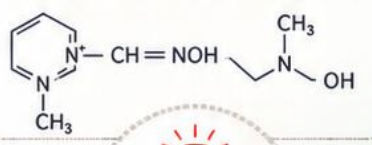




7) Contraindications / Cautions

- Use with caution in renal impairment.
- Benefit is greatest when given **early.**
- Reduced usefulness after enzyme **aging.**
- Avoid delay in severe poisoning.
- Use carefully if diagnosis is uncertain.

Chemical Structure of Pralidoxime (2-PAM)



8) Drug Interactions

- Osed synergistically with **atropine.**
- Supportive role with diazepam in seizures.
- **Does not** replace atropine.
- Response may vary with type of OP compound.

9) Important Examples

- Pralidoxime.
- Obidoxime.
- Other oxime reactivators.
- Key exam example: 2-PAM = pralidoxime.

10) Mnemonics

- "PAM = **Pulls AChE free** from phosphate".
- "Pralidoxime = OP **antidote** partner of **atropine**".
- Highlight the words "**Before aging**".

Before aging!

11) Exam Points

- Antidote for organophosphate poisoning.
- Must be given **early.**
- Reactivates AChE **before aging.**
- Better for nicotinic than muscarinic symptoms.
- Always remember: **combine with atropine.**
- Poor CNS penetration.

12) Important Questions

- Why is pralidoxime ineffective after aging?
- Why is it given with atropine?
- Why is it more useful in OP than carbamate poisoning?
- What symptoms improve most with pralidoxime?

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