
PDF


PHARMACY INDIA
PHARMACY INDIA
GET IT ON Google Play
Download
PHARMACY INDIA
App from play store

DAILY UPDATES

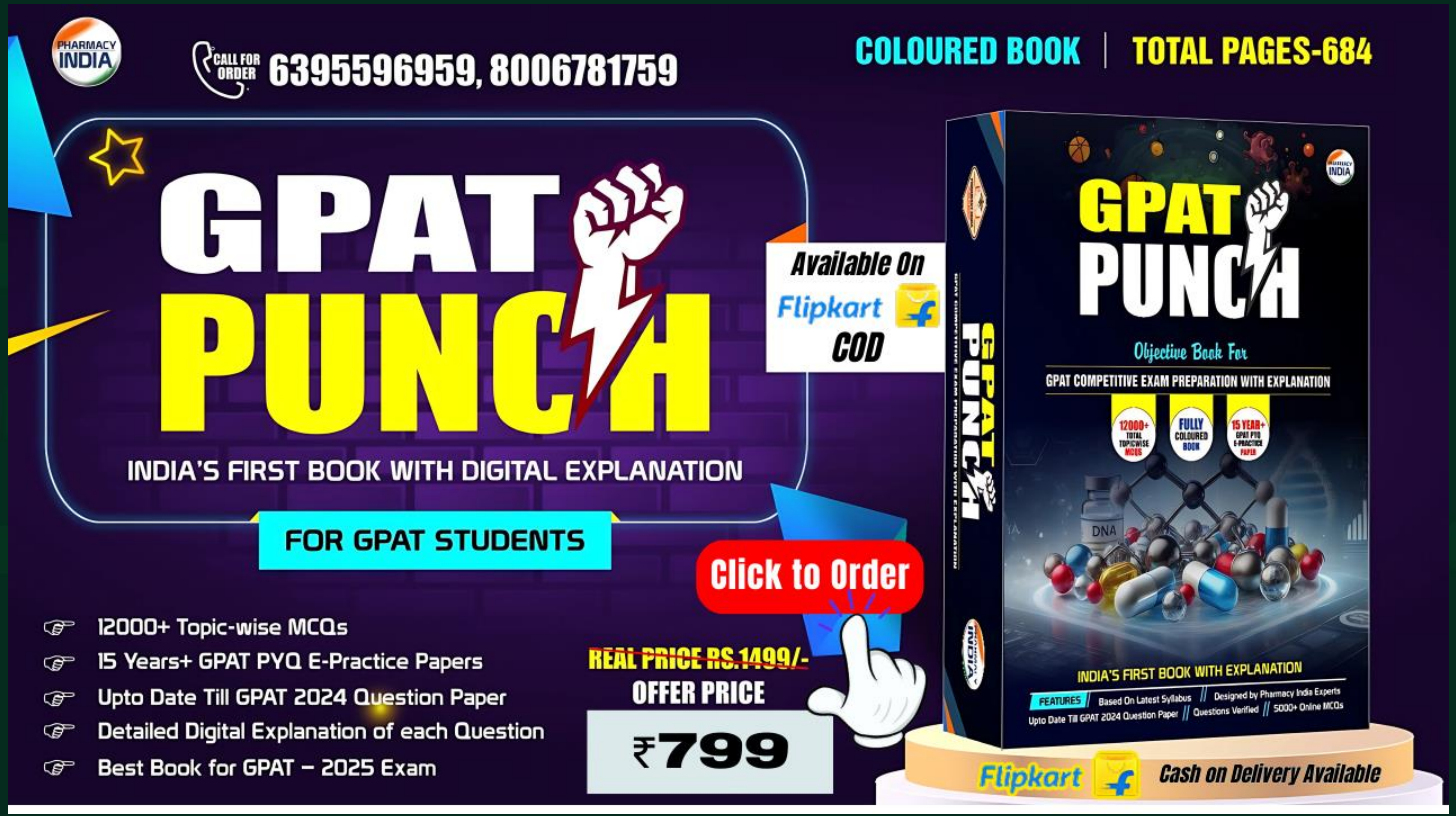
जुड़िए PHARMACY INDIA

के साथ.....

WHATSAPP & TELEGRAM SE JUDNE KE LIYE ICONS PAR CLICK KARE

WhatsApp

“GPAT Punch – Objective Book for GPAT 2025”

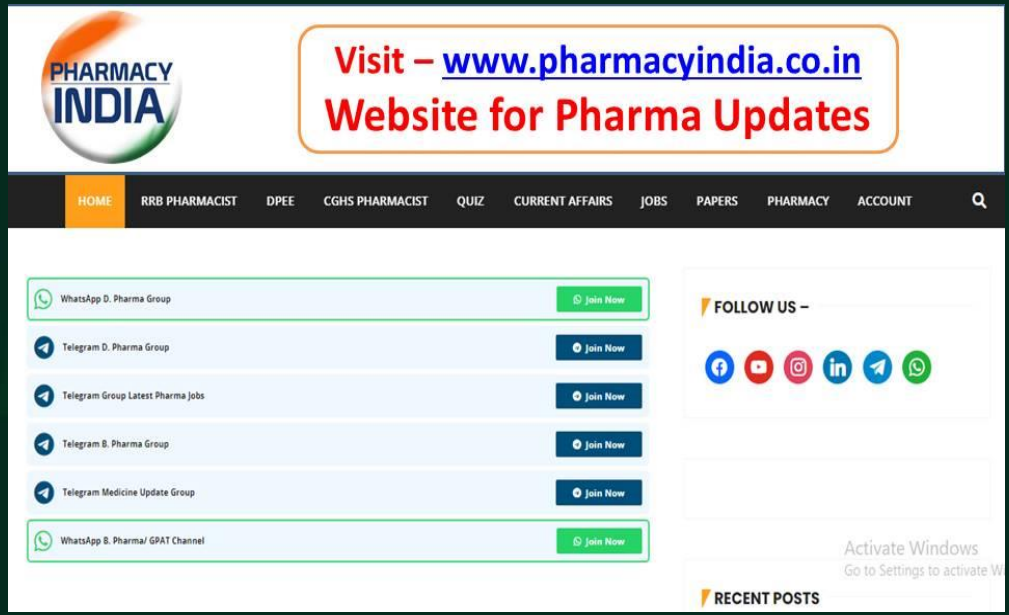


Click Here to Download Book Sample PDF

Cash on Delivery Available | Click to Order Now

Visit – pharmacyindia.co.in

- GPAT Preparation Materials
- Free GPAT Practice Quiz
- Daily Job Updates
- Previous Year GPAT Papers
- Exam Notifications & Updates
- Subject-Wise Study Notes
- College Rankings & Admissions



1. **Which of the following drug can produce mydriasis without Cycloplegia [GPAT-2023 SHIFT- I]**

(a) Atropine

(b) Tropicamide

(c) Homatropine

(d) Ephedrine

1. **Which of the following drug can produce mydriasis without Cycloplegia [GPAT-2023 SHIFT- I]**

(a) Atropine

(b) Tropicamide

(c) Homatropine

(d) Ephedrine

- **Explanation:**
- **Mydriasis** is pupil dilation, and **cycloplegia** refers to paralysis of the ciliary muscle.
- Drugs like **Ephedrine**, an indirect sympathomimetic, cause mydriasis without cycloplegia by stimulating the adrenergic receptors on the dilator pupillae muscle without affecting the ciliary muscle.

- Other drugs like **Atropine**, **Tropicamide**, and **Homatropine** block muscarinic receptors, leading to both mydriasis and cycloplegia.
- Ephedrine indirectly stimulates alpha-adrenergic receptors, causing the pupil to dilate without significant effect on accommodation.

2. **_____activates G-protein gated potassium channel resulting in membrane hyperpolarization [GPAT-2023 SHIFT-I]**

(a) α1 adrenergic receptor

(b) α2 adrenergic receptor

(c) ß1 adrenergic receptor

(d) ß2adrenergic receptor

2. **_____activates G-protein gated potassium channel resulting in membrane hyperpolarization [GPAT-2023 SHIFT-I]**

(a) α1 adrenergic receptor

(b) α2 adrenergic receptor

(c) ß1 adrenergic receptor

(d) ß2adrenergic receptor

- **Explanation:**
- α2 adrenergic receptors are Gi protein-coupled receptors.
- Upon activation, they inhibit adenylate cyclase, decreasing cAMP levels and opening G-protein gated potassium channels.
- This results in membrane hyperpolarization and reduced neuronal excitability, often contributing to a negative feedback mechanism on neurotransmitter release.

3. **The β1 and β2 adrenergic receptor subtype agonist acting at atrioventricular node produces following responses [GPAT-2021]**

(a) Increases contractility and conduction velocity

(b) Increases automaticity and conduction velocity

(c) Increases contractility and automaticity

(d) Increases conduction velocity and heart rate

3. **The β1 and β2 adrenergic receptor subtype agonist acting at atrioventricular node produces following responses [GPAT-2021]**

(a) Increases contractility and conduction velocity

(b) Increases automaticity and conduction velocity

(c) Increases contractility and automaticity

(d) Increases conduction velocity and heart rate

- **Explanation:**

- Adrenergic agonist stimulates the ß1 receptors in the heart and increases the heart rate, the force of contraction and the conduction velocity. The main effects are Increased force of contraction (Positive Inotropic effect), Increased heart rate (Positive chronotropic effect), Increased automaticity.
- Repolarisation and restoration of function following generalised cardiac depolarisation
- However it reduces the cardiac efficiency (in relation to oxygen consumption). The sympathetic and parasympathetic effects on heart is given below

Organ	Sympathetic	Adrenoceptor type	Parasympathetic	Cholinoceptor
Heart				
Sinoatrial node	Rate ↑	β_1	Rate ↓	M_2
Atrial muscle	Force ↑	β_1	Force↓	M_2
Atrioventricular node	Automaticity ↑	β_1	Conduction velocity	M_2
		β_1	Atrioventricular block	M_2
Ventricular muscle	Automaticity ↑se Force	β_1	No effect	M_2

4. **Which of the following is NOT one of the triad effects of adrenaline leading to rise in blood pressure [GPAT-2020]**

(a) A direct myocardial stimulation that increases the strength of ventricular contraction

(b) An increased heart rate (positive chronotropic action)

(c) Vasoconstriction in many vascular beds specially in precapillary resistance vessels of skin

(d) Stimulation of presynaptic alpha-2 adrenoreceptor leading to increase sympathetic tone

4. **Which of the following is NOT one of the triad effects of adrenaline leading to rise in blood pressure [GPAT-2020]**

(a) A direct myocardial stimulation that increases the strength of ventricular contraction

(b) An increased heart rate (positive chronotropic action)

(c) Vasoconstriction in many vascular beds specially in precapillary resistance vessels of skin

(d) Stimulation of presynaptic alpha-2 adrenoreceptor leading to increase sympathetic tone

- **Explanation: Adrenergic Receptors**

Alpha 1	Alpha 2	Beta 1	Beta 2
- Vasoconstriction	- Inhibits norepinephrine release	- ↑ Heart rate	- Vasodilation
- ↑ Peripheral resistance (blood flow)	- Inhibits acetylcholine release	- ↑ Lipolysis	- ↓ Peripheral resistance
- ↑ Blood pressure	- Inhibits insulin release	- ↑ Myocardial contractility	- Bronchodilation
- Mydriasis		- ↑ Renin	- ↑ Glycogenolysis (muscle, liver)

Alpha 1	Alpha 2	Beta 1	Beta 2
- ↑ Closure of bladder sphincters			- ↑ Glucagon release
			- Relaxes uterine smooth muscle

5. **Select the drug which exhibits dual alpha- and beta-adrenergic receptor agonist activities [GPAT-2018]**

(a) Terbutaline

(b) Clonidine

(c) Metaproterenol

(d) Dobutamine

5. **Select the drug which exhibits dual alpha- and beta-adrenergic receptor agonist activities [GPAT-2018]**

(a) Terbutaline

(b) Clonidine

(c) Metaproterenol

(d) Dobutamine

- **Explanation:**

Dobutamine

- A derivative of DA, but not a D1 or D2 receptor agonist.
- Though it acts on both α and β adrenergic receptors, the only prominent action of clinically employed doses (2–8 µg/kg/min i.v. infusion) is increased force of cardiac contraction and output, without significant change in heart rate, peripheral resistance and BP.

- As such, it is considered to be a relatively selective β1 agonist.
- It is used as an inotropic agent in pump failure accompanying myocardial infarction, cardiac surgery, and for short term management of severe congestive heart failure. It is less arrhythmogenic than Adr.

6. **All the dopaminergic agonists having affinity for D2 receptors are clinically used in following conditions EXCEPT [GPAT-2017]**

(a) Obsessive-compulsive disorder

(b) Hyperprolactinemia

(c) Acromegaly

(d) Parkinsonism

6. **All the dopaminergic agonists having affinity for D2 receptors are clinically used in following conditions EXCEPT [GPAT-2017]**

(a) Obsessive-compulsive disorder

(b) Hyperprolactinemia

(c) Acromegaly

(d) Parkinsonism

- **Explanation:**

VARIOUS TYPES OF DISORDERS AND THEIR TREATMENT

DISORDERS	CAUSE (MOLECULAR MECHANISM	TREATMENT
Obsessive compulsive disorder	Dopaminergic overactivity the limbic system is involved	Dopamine D_2 receptor blocking action is required.
Hyperprolactinemia	High level of prolactin is due to blocking of dopaminergic blocking action	Dopaminergic agonists such as Bromocriptine, Carbergol in will release. inhibit prolactin release.

DISORDERS	CAUSE (MOLECULAR MECHANISM	TREATMENT
Acromegaly	Excessive release of growth hormone in pituitary. Although dopamine stimulates growth hormone release in normal individuals, it inhibits growth hormone release in up to 50% of acromegalics.	Two Dopamine agonists such as Bromocriptine and Cabergoline are effective.
Parkinsonism	Is due to reduction of dopamine in the striatum	Dopaminergic agonist such as Pramipexole, Bromocriptine, Ropinirole will be effective.

7. **Match the following adrenergic drugs with their receptor affinity [GPAT-2017]**

1. Epinephrine [P] More alpha 1, no beta 1, beta 2 & dopamine
2. Noradrenaline [Q] More alpha 1 & beta 1, less beta 2, no dopamine
3. Phenylephrine [R] More beta 1 & beta 2, no alpha 1 and dopamine
4. Dobutamine [S] More alpha 1 & beta 1, no beta 2 & dopamine

(a) 1-[Q], 2-[S], 3-[P], 4-[R]

(b) 1-[P], 2-[R], 3- [S], 4-[Q]

(c) 1-[R], 2-[P], 3-[Q], 4-[S]

(d) 1-[S], 2-[Q], 3-[R], 4-[P]

7. Match the following adrenergic drugs with their receptor affinity [GPAT-2017]

1. Epinephrine [P] More alpha 1, no beta 1, beta 2 & dopamine
2. Noradrenaline [Q] More alpha 1 & beta 1, less beta 2, no dopamine
3. Phenylephrine [R] More beta 1 & beta 2, no alpha 1 and dopamine
4. Dobutamine [S] More alpha 1 & beta 1, no beta 2 & dopamine

(a) 1-[Q], 2-[S], 3-[P], 4-[R]

(b) 1-[P], 2-[R], 3- [S], 4-[Q]

(c) 1-[R], 2-[P], 3-[Q], 4-[S]

(d) 1-[S], 2-[Q], 3-[R], 4-[P]

Explanation:

DRUG ACTING ON SYMPATHETIC SYSTEM

DRUGS	OUTLINE OF STRUCTURE	AFFINITY OF RECEPTORS
Epinephrine (adrenaline)	Contain catechol with bulk group on nitrogen	α_1, α_2, β_1, less β_2, β_3, no Dopamine affinity
Noradrenaline	Contain catechol with no bulk group on nitrogen	α_1, α_2, β_1, no β_2, and dopamine affinity
Phenylephrine	Contain phenol without catechol	Selective α_1, agonist
Dobutamine	Contain catechol with bulk group on nitrogen	α_1, β_1, less β_2, no Dopamine affinity

8. **Which of the following is rate limiting step in synthesis of catecholamines [GPAT-2016]**

(a) Conversion of Dopa to dopamine by dopa decarboxylase

(b) Conversion of tyrosine to L-Dopa in presence of enzyme Tyrosine hydroxylase

(c) Conversion of dopamine to NA by dopamine ß - hydroxylase

(d) Conversion of Noradrenaline to adrenaline by N-methyltransferase

8. **Which of the following is rate limiting step in synthesis of catecholamines [GPAT-2016]**

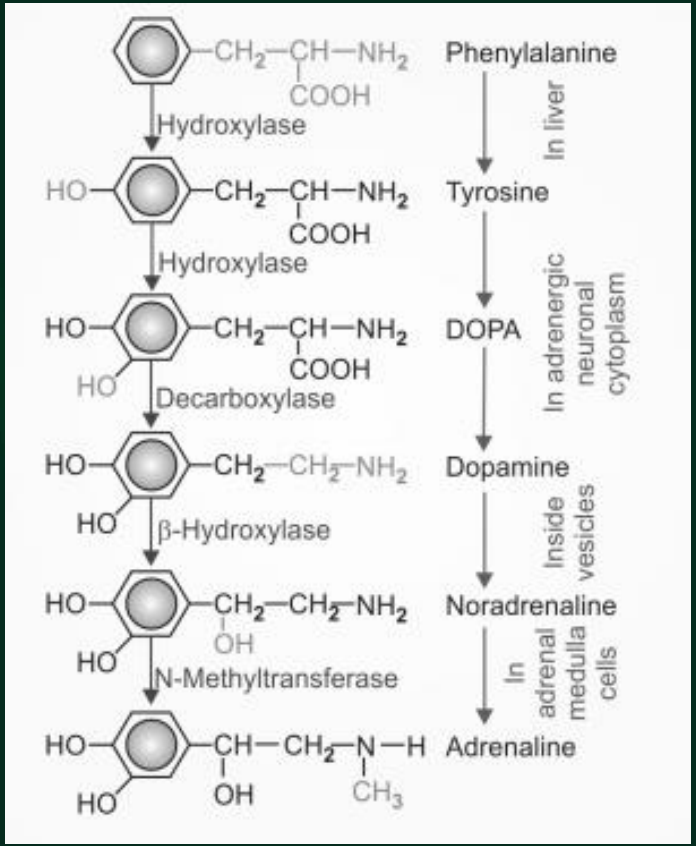
(a) Conversion of Dopa to dopamine by dopa decarboxylase

(b) Conversion of tyrosine to L-Dopa in presence of enzyme Tyrosine hydroxylase

(c) Conversion of dopamine to NA by dopamine ß - hydroxylase

(d) Conversion of Noradrenaline to adrenaline by N-methyltransferase

- **Explanation:**
- Tyrosine hydroxylase catalyzes the synthesis of L-dihydroxyphenylalanine (DOPA) from tyrosine and it is the rate-limiting step in the synthesis of the catecholamines.



9. **Which of the following drugs are often found in both prescription and over the counter nasal decongestants [GPAT-2016]**

(a) Alpha 2 agonists

(b) Alpha 1 agonists

(c) Alpha 1 antagonists

(d) Beta 2 agonists

9. **Which of the following drugs are often found in both prescription and over the counter nasal decongestants [GPAT-2016]**

(a) Alpha 2 agonists

(b) Alpha 1 agonists

(c) Alpha 1 antagonists

(d) Beta 2 agonists

- **Explanation:**

ALPHA 1 AGONISTS

- Alpha 1 agonists vasoconstrictive agents are used to reduce edema and inflammation.
- The nasal decongestants as vasoconstrictor agents is used to treat inflammation of the nasal passages or an allergy related condition, like hay fever, because inflammation can cause swelling of the mucous membrane that lines the nasal p sages and results in inordinate mucus production.

Drugs: Naphazoline, phenylephrine, Xylometolazine, Oxymetolazine

ALPHA 1 ANTAGONISTS

- Alpha 1 antagonists causes vasodilation and decreased peripheral resistance; therefore they are used in the treatment of hypertension (prazosin).

10. **Following is an analogue of Amphetamine which produces anorexia without causing stimulation**

[GPAT-2014]

(a) Fenfluramine

(b) Methyl amphetamine

(c) Methylphenidate

(d) Dextroamphetamine

10. **Following is an analogue of Amphetamine which produces anorexia without causing stimulation**

[GPAT-2014]

(a) Fenfluramine

(b) Methyl amphetamine

(c) Methylphenidate

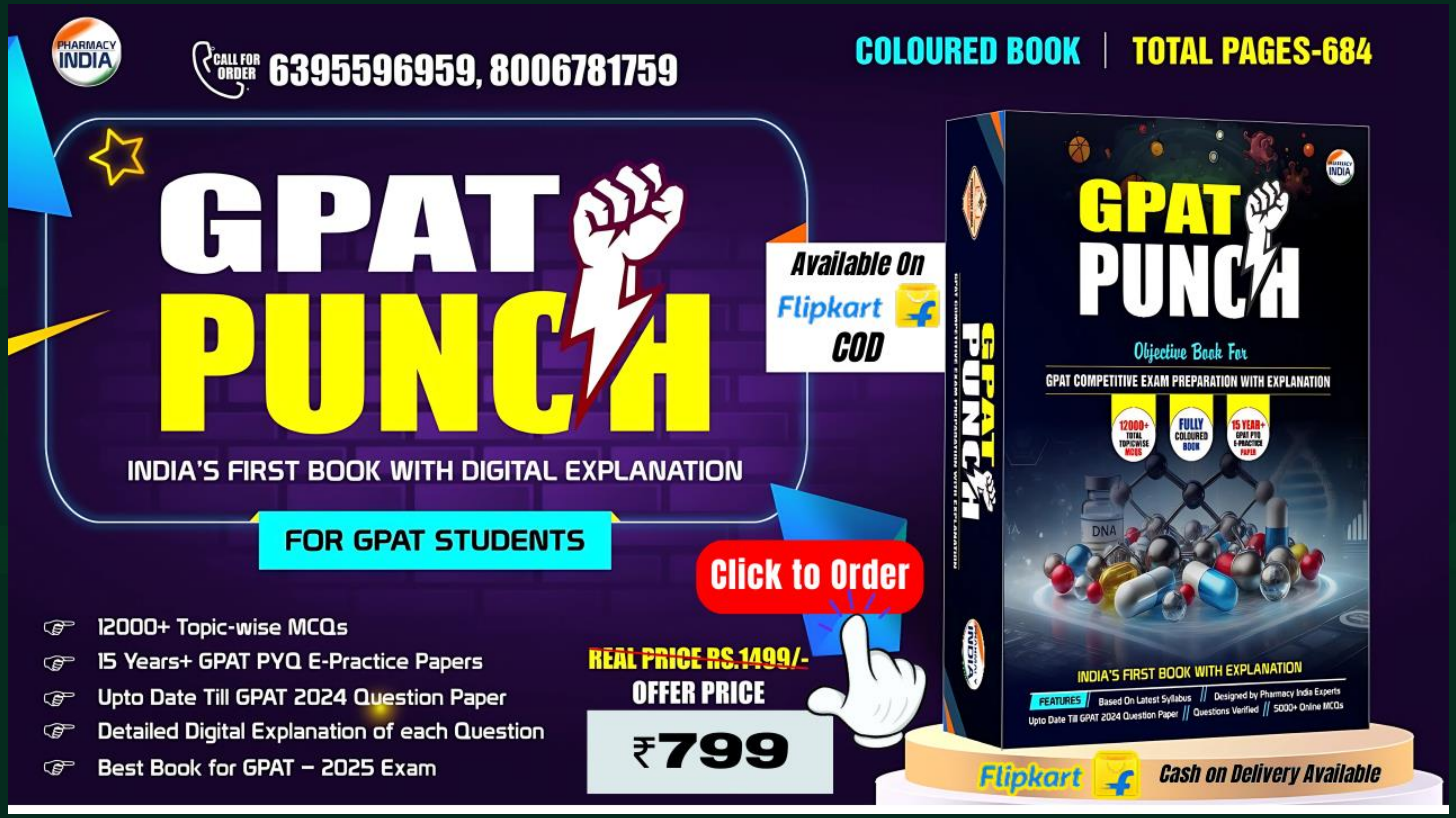
(d) Dextroamphetamine

- **Explanation:**

- Amphetamine is a synthetic substance related to natural sympathomimetic amines. However this agent is a commonly abused psychostimulant drug. Amphetamine is a chiral compound.
- The racemic mixture can be divided into its optical antipodes: levo- and dextro-amphetamine. Amphetamine is the parent compound of its own structural class, comprising a broad range of psychoactive derivatives.

- Fenfluramine is an analogue of amphetamine and are appetite suppressant and are used in the treatment of obesity. It does not cause stimulation.

“GPAT Punch – Objective Book for GPAT 2025”

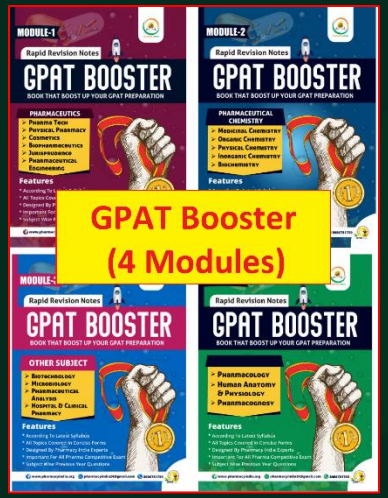


[Click Here to Download Book Sample PDF]

[Cash on Delivery Available | Click to Order Now]

Best Books For GPAT Preparation

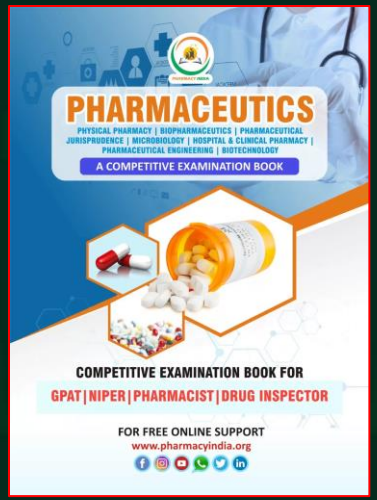
ORDER NOW



Complete GPAT Preparation
Includes All Core Subjects
COD Available

Flipkart

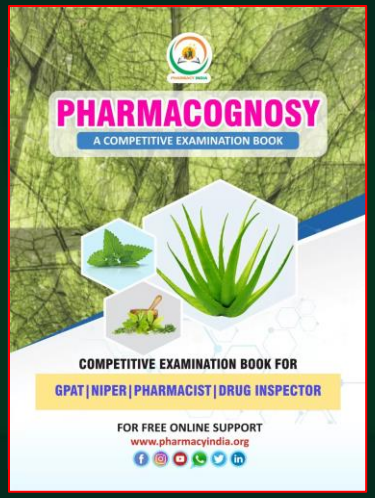
amazon



Pharmaceutics Book for
GPAT 2025 Exam
COD Available

Flipkart

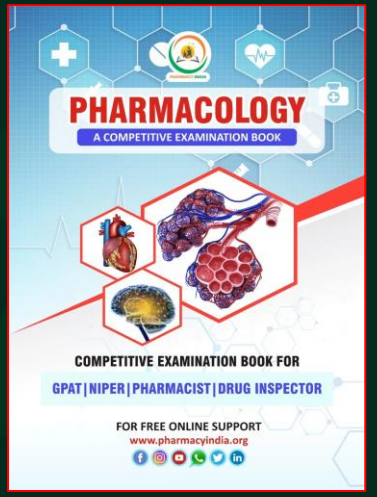
BUY NOW



Pharmacognosy Book
for GPAT 2025 Exam
COD Available

Flipkart

BUY NOW



Pharmacology Book for
GPAT 2025 Exam
COD Available

Flipkart

BUY NOW

PHARMACY INDIA

LIVE CLASSES STARTING FROM
05th FEBRUARY 2025

अभिMAAN

PREPARE TILL YOUR LAST DESTINATION

MISSION
GPAT 2025
120 DAY'S CRASH COURSE

- LIVE CLASSES
- ONLINE TEST SERIES
- E-NOTES

PHARMACY INDIA

11. **The adrenergic receptors found in fatty tissues is [GPAT-2014]**

(a) β1

(b) β2

(c) β 3

(d) $\beta 1, \beta_2$

11. **The adrenergic receptors found in fatty tissues is [GPAT-2014]**

(a) β1

(b) β2

(c) β 3

(d) $\beta 1, \beta_2$

. **Explanation:**

β RECEPTORS

RECEPTORS	LOCATION	AGONISTS	ANTAGONISTS
β_1	Heart, kidney (J .Gcell)	Dobutamine	Metaprolol, Nebivolol, Atenolol
β_2	Bronchi, urinary tract & eyes	Salbutamol, Ritordine Terbutaline, Formeterol	Butoxamine
β_3	Adipose tissue (fatty tissue)	Mirabegron	BRL37344

12. All of the given four drugs are sympathomimetics [GPAT-2012]

[P] Adrenaline

[Q] Isoprenaline

[R] Phenylephrine

[S] Noradrenaline

Choose the correct statement related to their effects on blood pressure

(a) P and Q increase systolic and diastolic blood pressure

(b) Q and R increase systolic and diastolic blood pressure

(c) R and S increase systolic blood pressure

(d) P and S increase systolic and diastolic blood pressure

12. All of the given four drugs are sympathomimetics [GPAT-2012]

[P] Adrenaline

[Q] Isoprenaline

[R] Phenylephrine

[S] Noradrenaline

Choose the correct statement related to their effects on blood pressure

(a) P and Q increase systolic and diastolic blood pressure

(b) Q and R increase systolic and diastolic blood pressure

(c) R and S increase systolic blood pressure

(d) P and S increase systolic and diastolic blood pressure

. Explanation:

DRUGS	EFFECT ON BLOOD PRESSURE
Adrenaline	Increases systolic blood pressure and decreases diastolic pressure. It increases systolic pressure due to cardiac action (B_1) and decrease in diastolic pressure due to decreased peripheral vascular resistance (B_2)
Isoprenaline	Increases systolic blood pressure due to cardiac action and decreases diastolic blood pressure due to decrease in peripheral vascular resistance
Noradrenaline	Increases systolic and diastolic pressure it does not cause vasodilation(no $ß_2$ action), peripheral vascular resistance increases due to a action
Phenylephrine	Agonist and it increase "Peripheral vascular resistance due to α_1 action, hence increases systolic and diastolic blood pressure

13. **Identify the adrenergic receptor, whose agonists can be missed used by sportsman for anabolic effects [GATE-2007]**

(a)α

(b) α2

(c) β1

(d) β_2

13. **Identify the adrenergic receptor, whose agonists can be missed used by sportsman for anabolic effects [GATE-2007]**

(a)α

(b) α2

(c) β1

(d) β_2

- **Explanation:**

- β_2 –adrenergic receptor is the main target in sport, they have bronchodilator and anabolic actions and enchance anti-inflammatory actions of corticosteroids.
- β –AR antagonists (β_2 –blockers) are used in sport that require steadiness and accuracy, such as archery and shooting, where their ability to reduce heart rate and musce tremor may improve performance.
- They have a deleterious effect in endrance sports because they reduce physical performance and maximum exercise load.
  Eg : Anadrol, Oxandrin, Nandrolone, Stanozolol, Oxymetholone

14. **The neurotransmitter is released at the end of sympathetic nerve fiber is [GATE-1991]**

(a) Epinephrine

(b) Nor-epinephrine

(c) Acetylcholine

(d) Physostigmine

14. **The neurotransmitter is released at the end of sympathetic nerve fiber is [GATE-1991]**

(a) Epinephrine

(b) Nor-epinephrine

(c) Acetylcholine

(d) Physostigmine

Explanation:

- Postganglionic neurons of the sympathetic nervous system are adrenergic and release Norepinephrine as the neurotransmitter. Approximately 50% of the sympathetic nerve fibers are afferent and 50% are efferent.

	PARASYMPATHETIC	SYMPATHETIC
Origin	Cranio-sacral	Dorso-lumbar
Distrubution	Limited to head, neck, trunk	wide
Neurotransmitter	Acetylcholine	Noradrenaline (major) Acetylcholine (minor)
Main function	Keep body cool and calm	Prepare body for fight and flight movement

15. **Repeated administration of Tyramine results in its decreasing effectiveness [GATE-1989]**

(a) Gets detoxicated easily

(b) Displaces noradrenaline from nerve ending binding site

(c) Displaces adrenaline from nerve ending binding site

(d) None of these

15. **Repeated administration of Tyramine results in its decreasing effectiveness [GATE-1989]**

(a) Gets detoxicated easily

(b) Displaces noradrenaline from nerve ending binding site

(c) Displaces adrenaline from nerve ending binding site

(d) None of these

- **Explanation:**

- **Tyramine** is an **indirect-acting sympathomimetic agent** that works by entering the nerve terminal and causing the release of **noradrenaline (norepinephrine)** from storage vesicles.
- **Repeated administration of tyramine** results in a phenomenon called **tachyphylaxis** (decreased effectiveness) because:

1. The **stores of noradrenaline in the nerve endings are gradually depleted**, reducing its availability for release upon subsequent doses of tyramine.
2. This mechanism explains why the effect of tyramine diminishes over time with repeated use.

16.

Match the followings

Receptor	Selective agonist
1.β1	(p) Clonidine
2.β2	(q) Mirabegron
3.β3	(r) Phenylephrine
4.α1	(s) Terbutaline
5.α2	(t) Dobutamine

Find the correct answer

(a) 1-(q), 2-(p), 3-(r), 4-(t), 5-(s) (b) 1-(t), 2-(s), 3-(q), 4-(p), 5-(r)

(c) 1-(t), 2-(s), 3-(q), 4-(r), 5-(p) (d) 1-(r), 2-(q), 3-(t), 4-(s), 5-(p)

16.

Match the followings

Receptor	Selective agonist
1.β1	(p) Clonidine
2.β2	(q) Mirabegron
3.β3	(r) Phenylephrine
4.α1	(s) Terbutaline
5.α2	(t) Dobutamine

Find the correct answer

(a) 1-(q), 2-(p), 3-(r), 4-(t), 5-(s) (b) 1-(t), 2-(s), 3-(q), 4-(p), 5-(r)

(c) 1-(t), 2-(s), 3-(q), 4-(r), 5-(p) (d) 1-(r), 2-(q), 3-(t), 4-(s), 5-(p)

Explanation:

1. **β1 - Dobutamine (t):**
   - **Dobutamine** is a **β1-selective agonist** that stimulates β1-adrenergic receptors in the heart, increasing cardiac contractility and heart rate. It is used in cases of acute heart failure.
2. **β2 - Terbutaline (s):**
   - **Terbutaline** is a **β2-selective agonist** that relaxes smooth muscles, particularly in the bronchi, and is used for treating asthma and bronchospasm.

3. β3 - Mirabegron (q):

- **Mirabegron** is a **β3-selective agonist** used to relax the detrusor muscle in the bladder, helping to treat overactive bladder.

4. α1 - Phenylephrine (r):

- **Phenylephrine** is an **α1-selective agonist** that causes vasoconstriction and is commonly used as a nasal decongestant or to increase blood pressure in hypotensive states.

5. α2 - Clonidine (p):

- **Clonidine** is an **α2-selective agonist** that stimulates α2 receptors in the central nervous system, reducing sympathetic outflow and lowering blood pressure.

17. **Vasopressor of choice in pregnancy is**

(a) Ephedrine

(b) Phenylephrine

(c) Methoxamine

(d) Mephentermine

17. **Vasopressor of choice in pregnancy is**

(a) Ephedrine

(b) Phenylephrine

(c) Methoxamine

(d) Mephentermine

- **Explanation:**
- **Vasopressors** are medications used to increase blood pressure, especially during conditions like **hypotension in pregnancy** (e.g., during spinal or epidural anesthesia). The choice of vasopressor depends on its safety and effectiveness for both the mother and fetus.

Why Ephedrine is the Vasopressor of Choice in Pregnancy:

1. **Mechanism of Action:**
   - Ephedrine is a **mixed-acting sympathomimetic**. It stimulates **α-adrenergic and β-adrenergic receptors**, increasing both cardiac output (via β1 stimulation) and systemic vascular resistance (via α1 stimulation).
2. **Fetal Safety:**
   - Ephedrine **does not cause significant uteroplacental vasoconstriction**, making it safer for maintaining blood flow to the fetus compared to other vasopressors like **phenylephrine**.
   - It has a long history of use with minimal adverse effects on fetal heart rate.

18. **Which of the following is not an alpha adrenoceptor agonist**

(a) Clonidine

(b) Methyldopa

(c) Guanabenz

(d) Guanfacine

18. **Which of the following is not an alpha adrenoceptor agonist**

(a) Clonidine

(b) Methyldopa

(c) Guanabenz

(d) Guanfacine

- **Explanation:**
- **Clonidine**, **Guanabenz**, **and** **Guanfacine** are **alpha-2 adrenoceptor agonists** that primarily act by stimulating **α2-receptors** in the central nervous system, leading to decreased sympathetic outflow, reduced peripheral vascular resistance, and lowered blood pressure.

Why Methyldopa is Not Classified as an Alpha Adrenoceptor Agonist:

Mechanism of Action:

- Methyldopa is a **prodrug** that is metabolized into **alpha-methyl norepinephrine**, which acts as a **false neurotransmitter**.
- While it indirectly stimulates **α2-adrenergic receptors**, its primary mechanism involves competitive inhibition of **dopa decarboxylase**, reducing norepinephrine synthesis.

- It is not a direct alpha adrenoceptor agonist like the other options.

Clinical Use:

- Methyldopa is commonly used as an **antihypertensive drug in pregnancy** because of its established safety profile.

19. **The only non-catecholamine sympathomimetic drug out of the following is**

(a) Adrenaline

(b) Ephedrine

(c) Dopamine

(d) Isoprenaline

19. **The only non-catecholamine sympathomimetic drug out of the following is**

(a) Adrenaline

(b) Ephedrine

(c) Dopamine

(d) Isoprenaline

- **Explanation:**
- **Catecholamines** are compounds that contain a **catechol nucleus** (a benzene ring with two hydroxyl groups) and an **amine group**. Examples include adrenaline, dopamine, and isoprenaline. These are derived from tyrosine and act as neurotransmitters or hormones.
- **Ephedrine** is a **non-catecholamine sympathomimetic drug**, meaning it lacks the catechol structure but still acts on adrenergic receptors to stimulate the sympathetic nervous system.

Adrenaline (Catecholamine):

- Adrenaline is a natural catecholamine with both **α and β-adrenergic agonist activity**.
- It contains a catechol nucleus and an amine group, qualifying it as a catecholamine.

Dopamine (Catecholamine):

- Dopamine is a precursor to norepinephrine and a natural catecholamine.
- It stimulates **dopaminergic receptors** at low doses and **β1-adrenergic receptors** at higher doses.

Ephedrine (Non-Catecholamine):

- Ephedrine is a **mixed-acting sympathomimetic**.
- It indirectly increases norepinephrine release and directly stimulates adrenergic receptors (**α and β**).
- It lacks the catechol nucleus, making it a **non-catecholamine**.
- It is orally active, has a longer duration of action than catecholamines, and crosses the blood-brain barrier.

Isoprenaline (Catecholamine):

- Isoprenaline is a synthetic **catecholamine** and a **β-selective agonist** (β1 and β2).
- It contains a catechol nucleus.

20. **Which of the following drugs shows the phenomenon of vasomotor reversal of Dale after administration of an β adrenergic blocker**

(a) Adrenaline

(b) Noradrenaline

(c) Isoprenaline

(d) All of the above

20. **Which of the following drugs shows the phenomenon of vasomotor reversal of Dale after administration of an β adrenergic blocker**

(a) Adrenaline

(b) Noradrenaline

(c) Isoprenaline

(d) All of the above

- **Explanation:**

The **vasomotor reversal of Dale** refers to the phenomenon in which the **pressor (vasoconstrictor)** response of **adrenaline** is converted into a **depressor (vasodilator)** response following the administration of an **α-adrenergic blocker**. This phenomenon was first observed by Sir Henry Dale.

1. Normal Adrenaline Response:

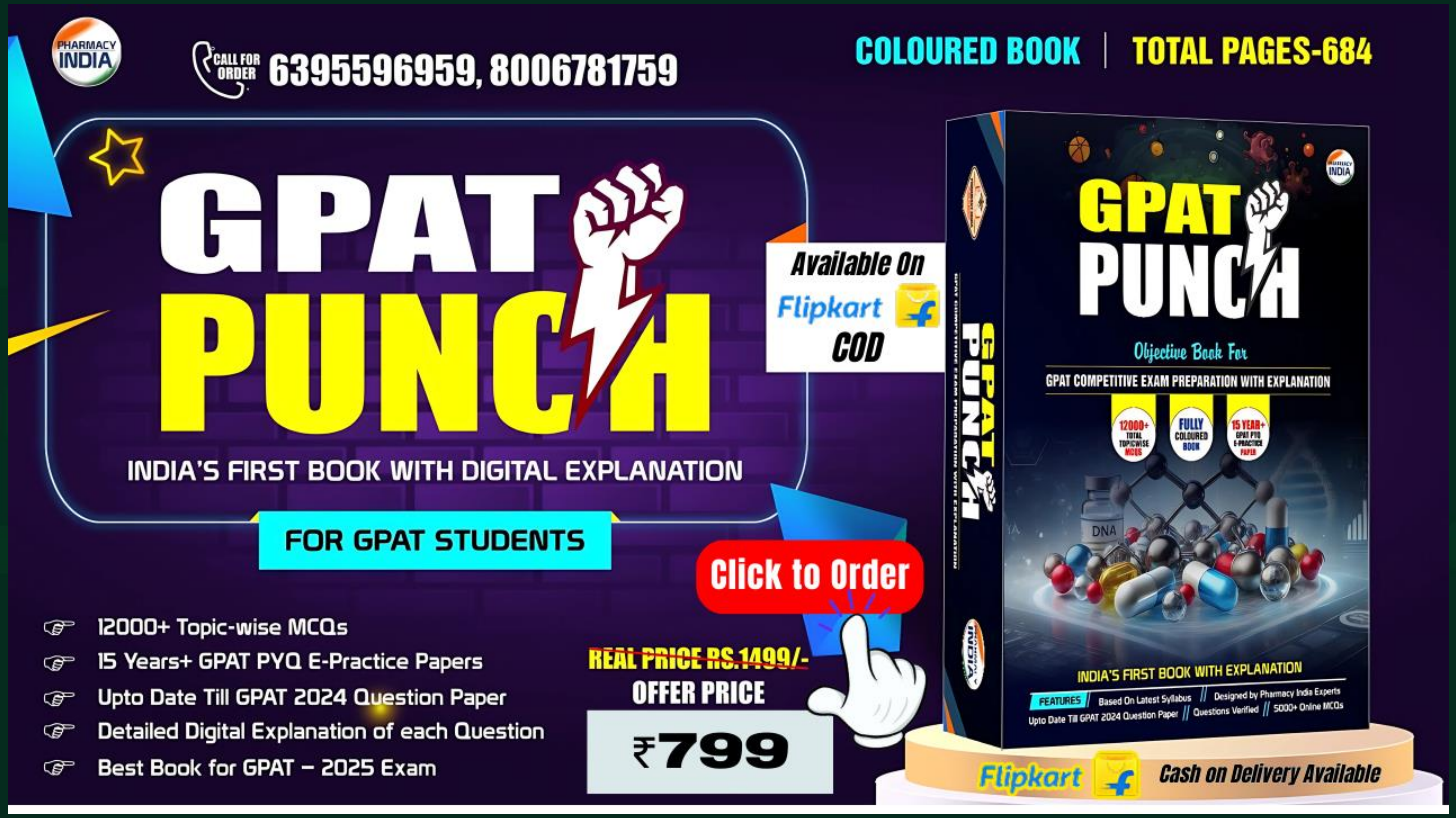
- Adrenaline stimulates both **α-adrenergic receptors** (vasoconstriction) and **β-adrenergic receptors** (vasodilation).

- The **pressor effect (vasoconstriction)** mediated by α-receptors dominates, leading to an overall increase in blood pressure.

2. After Administration of an α-Blocker (e.g., Phentolamine):

- The α-adrenergic receptors are blocked, so adrenaline's action on these receptors (vasoconstriction) is prevented.
- Adrenaline now predominantly acts on **β2-adrenergic receptors**, which mediate **vasodilation**, resulting in a **fall in blood pressure** (depressor response).

“GPAT Punch – Objective Book for GPAT 2025”

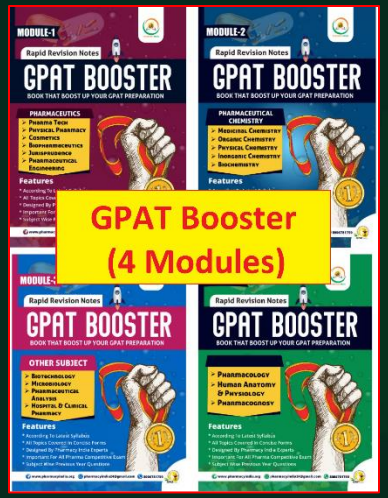


[Click Here to Download Book Sample PDF]

[Cash on Delivery Available | Click to Order Now]

Best Books For GPAT Preparation

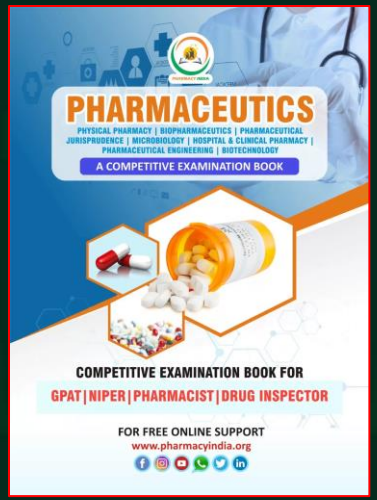
ORDER NOW



Complete GPAT Preparation
Includes All Core Subjects
COD Available

Flipkart

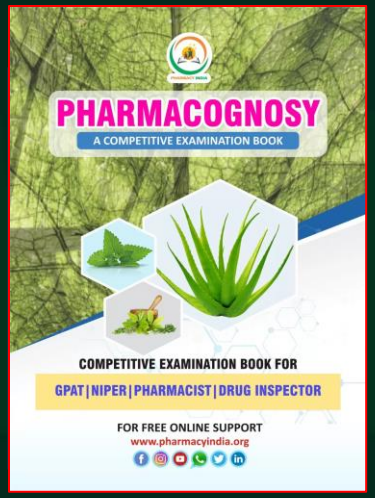
amazon



Pharmaceutics Book for
GPAT 2025 Exam
COD Available

Flipkart

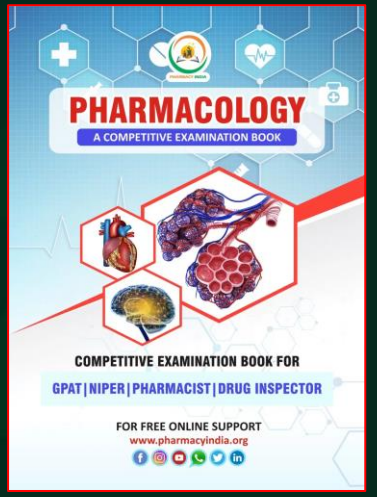
BUY NOW



Pharmacognosy Book
for GPAT 2025 Exam
COD Available

Flipkart

BUY NOW



Pharmacology Book for
GPAT 2025 Exam
COD Available

Flipkart

BUY NOW

PHARMACY INDIA

LIVE CLASSES STARTING FROM
05th FEBRUARY 2025

अभिMAAN

PREPARE TILL YOUR LAST DESTINATION

MISSION
GPAT 2025
120 DAY'S CRASH COURSE

- LIVE CLASSES
- ONLINE TEST SERIES
- E-NOTES

PHARMACY INDIA

21. **Dopamine is preferred in treatment of shock because of**

(a) Renal vasodilatory effect

(b) Increased cardiac output

(c) Peripheral vasoconstriction

(d) Prolonged action

21. **Dopamine is preferred in treatment of shock because of**

(a) Renal vasodilatory effect

(b) Increased cardiac output

(c) Peripheral vasoconstriction

(d) Prolonged action

- **Explanation:**

Dopamine is the drug of choice in the treatment of **shock** (e.g., cardiogenic or septic shock) because of its **dose-dependent effects** on adrenergic and dopaminergic receptors. It is preferred specifically because of its **renal vasodilatory effect**, which helps to preserve kidney function, an important concern during shock.

Mechanism of Action and Dose-Dependent Effects of Dopamine:

1. Low Dose (1–3 µg/kg/min):

- Stimulates **dopamine (D1) receptors** in the renal and mesenteric vasculature.
- Causes **renal vasodilation**, increasing **renal blood flow** and enhancing diuresis.
- This helps prevent or mitigate acute kidney injury (AKI), which is a common complication in shock.

2. Moderate Dose (3–10 µg/kg/min):

- Stimulates **β1-adrenergic receptors** in the heart.
- Increases **cardiac output** by enhancing heart rate and myocardial contractility.

3. High Dose (>10 µg/kg/min):

- Stimulates **α1-adrenergic receptors**.
- Causes **peripheral vasoconstriction**, which helps maintain blood pressure but may reduce renal perfusion

22. **Epinephrine is most useful in**

(a) Bronchial asthma

(b) Anaphylactic shock

(c) Peripheral vascular disease

(d) Wide angle glaucoma

22. **Epinephrine is most useful in**

(a) Bronchial asthma

(b) Anaphylactic shock

(c) Peripheral vascular disease

(d) Wide angle glaucoma

- **Explanation:**

Epinephrine is the drug of choice for **anaphylactic shock**, which is a severe, life-threatening allergic reaction characterized by respiratory distress, hypotension, and cardiovascular collapse. Its pharmacological actions make it highly effective in counteracting the symptoms of anaphylaxis.

Mechanism of Action in Anaphylactic Shock:

1. Stimulation of α1-Adrenergic Receptors:

- Causes **vasoconstriction**, which:
  - Reverses hypotension and shock.
  - Reduces mucosal edema in the upper airway, alleviating respiratory obstruction.

2. Stimulation of β1-Adrenergic Receptors:

- Increases **cardiac output** by enhancing heart rate and myocardial contractility, helping to stabilize cardiovascular function.

3. Stimulation of β2-Adrenergic Receptors:

- Causes **bronchodilation**, which:
  - Relieves bronchospasm, improving airflow in cases of respiratory distress.
- Inhibits the release of inflammatory mediators from mast cells.

23. **Which of the following drug acts as combined alpha- and beta-adrenergic receptor agonist**

(a) Dobutamine

(b) Phenylephrine

(c) Fenoldopam

(d) Noradrenaline

23. **Which of the following drug acts as combined alpha- and beta-adrenergic receptor agonist**

(a) Dobutamine

(b) Phenylephrine

(c) Fenoldopam

(d) Noradrenaline

- **Explanation:**

Noradrenaline (Norepinephrine) is a **combined α- and β-adrenergic receptor agonist**, meaning it acts on both **α-adrenergic** and **β-adrenergic receptors**. Its effects depend on its receptor affinity and their distribution in the body.

Mechanism of Action:

1. **Primary Affinity:**
    - **Strong α1 and α2 agonist activity:**
        - Causes **vasoconstriction**, leading to an increase in **systemic vascular resistance** and blood pressure.

- **Moderate β1 agonist activity:**
  - Increases **heart rate** and **myocardial contractility**, improving cardiac output.

2. Limited β2 Activity:

- Noradrenaline has minimal activity on β2 receptors, which reduces its vasodilatory effects compared to epinephrine

24. **Which of the following drug is a long acting beta2 agonist**

(a) Albuterol

(b) Salmeterol

(c) Pirbuterol

(d) Orciprenaline

24. **Which of the following drug is a long acting beta2 agonist**

(a) Albuterol

(b) Salmeterol

(c) Pirbuterol

(d) Orciprenaline

- **Explanation:**

Salmeterol is classified as a **long-acting β2-adrenergic agonist (LABA)**, meaning it provides prolonged bronchodilation by selectively stimulating **β2 receptors** in the smooth muscles of the airways. It is widely used in the management of **asthma** and **chronic obstructive pulmonary disease (COPD)**.

1. Mechanism of Action:

- Salmeterol binds selectively to **β2-adrenergic receptors** and induces relaxation of airway smooth muscles by increasing intracellular **cyclic AMP (cAMP)** levels.
- Its long duration of action (about 12 hours) is due to its **lipophilic side chain**, which anchors the molecule near the receptor.

2. Uses:

- Preventive treatment of **asthma** (in combination with inhaled corticosteroids).
- Maintenance therapy in **COPD**.
- Not suitable for **acute bronchospasm** due to its delayed onset of action.

25. **The rate limiting step for norepinephrine synthesis is**

(a) Conversion of phenylalanine to tyrosine

(b) Conversion of tyrosine to DOPA

(c) Conversion of DOPA to dopamine

(d)Conversion of dopamine to norepinephrine

25. **The rate limiting step for norepinephrine synthesis is**

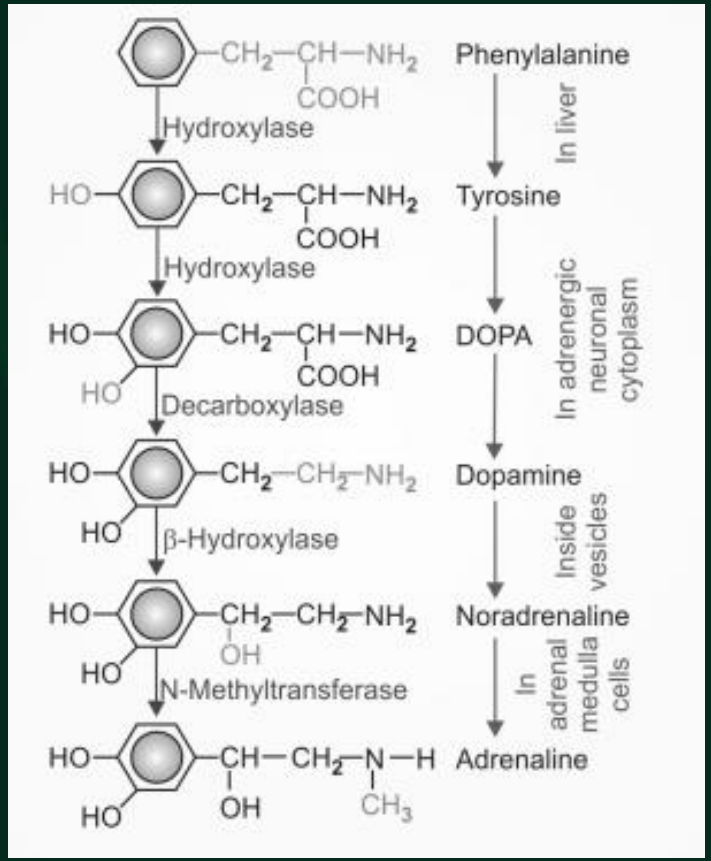
(a) Conversion of phenylalanine to tyrosine

(b) Conversion of tyrosine to DOPA

(c) Conversion of DOPA to dopamine

(d)Conversion of dopamine to norepinephrine

- **Explanation:**
- The rate-limiting step in the synthesis of norepinephrine is the conversion of tyrosine to DOPA, which is catalyzed by the enzyme tyrosine hydroxylase. This step is critical as it regulates the overall rate of norepinephrine production.



26. A patient with pheochromocytoma is undergoing surgery and has not been administered with alpha receptor blocker. If he is administered with intravenous Propranolol, then which of the following effects will be evident [GPAT-2022]

(a) There will be a rise in the blood pressure

(b) There will be a fall in the blood pressure

(c) The blood pressure will remain unchanged

(d) The patient may suffer severe bronchoconstriction

26. A patient with pheochromocytoma is undergoing surgery and has not been administered with alpha receptor blocker. If he is administered with intravenous Propranolol, then which of the following effects will be evident [GPAT-2022]

(a) There will be a rise in the blood pressure

(b) There will be a fall in the blood pressure

(c) The blood pressure will remain unchanged

(d) The patient may suffer severe bronchoconstriction

- **Explanation:**
- Pheochromocytoma i.e. tumor of adrenal gland causes increase in NA and Adrenaline level in blood.
- Increased NA and Adrenaline causes rise in BP.
- Also β-blocker is given to the patient that will act on the blood vessel and try to contract them thereby increases the BP.
- So, NA, Adrenaline and B-blocker they all causes increase in BP Therefore, it will rise in Blood Pressure.

27. **Except one of the following pairs represent drugs used in the treatment of glaucoma and their primary mechanism. Select the wrong pair from the following [GPAT-2020]**

(a) Topical prostaglandin analogues: Increase aqueous outflow

(b) Topical beta-adrenergic blockers: Decrease aqueous outflow

(c) Topical miotics: Increase aqueous outflow

(d) Topical carbonic anhydrase inhibitors: Decrease aqueous formation

27. **Except one of the following pairs represent drugs used in the treatment of glaucoma and their primary mechanism. Select the wrong pair from the following [GPAT-2020]**

(a) Topical prostaglandin analogues: Increase aqueous outflow

(b) Topical beta-adrenergic blockers: Decrease aqueous outflow

(c) Topical miotics: Increase aqueous outflow

(d) Topical carbonic anhydrase inhibitors: Decrease aqueous formation

- **Explanation:**

MODE OF ACTION OF OCULAR HYPOTENSIVE DRUG			
Drug/Class	Aqueous secretion	Trabecular	Uveoscleral outflow
1. β-blockers (Timolol)	↓	-	-
2. Adrenaline/ Dipivefrine	↓		↑
3. Brimonidine/apraclonidine	↓	-	↑
4. Prostaglandins (Latanoprost)	-	↑	↑
5. Miotics (Pilocarpine)	-	↑	-
6. Carbonic anhydrase inhibitors	↓	-	-

28. **Which of the following pair of drugs is considered as selective α_1-Blockers [GPAT-2019]**

(a) Timolol and Metoprolol

(b) Prazosin and Terazosin

(c) Formoterol and Levalbuterol

(d) Yohimbine and Corynanthine

28. **Which of the following pair of drugs is considered as selective α_1-Blockers [GPAT-2019]**

(a) Timolol and Metoprolol

(b) Prazosin and Terazosin

(c) Formoterol and Levalbuterol

(d) Yohimbine and Corynanthine

- **Explanation:**

Classification of α Blockers	Examples
Non-selective (α_1 and α_2) Blocker	
- Reversible	- Phentolamine, Tolazoline
- Irreversible	- Phenoxybenzamine
Selective α_1 Blockers	- Prazosin, Terazosin, Doxazosin, Tamsulosin, Alfuzosin
Selective α_2 Blockers	- Yohimbine

29. **Which of the following is NOT a cardio selective ß-blocker [GPAT-2019]**

(a) Bisoprolol

(b) Nebivolol

(c) Acebutolol

(d) Pindolol

29. **Which of the following is NOT a cardio selective ß-blocker [GPAT-2019]**

(a) Bisoprolol

(b) Nebivolol

(c) Acebutolol

(d) Pindolol

. Explanation:

β Adrenergic Blocking Drugs	Examples
Non-Selective (α_1 + β_2)	
- Without intrinsic activity	Sotalol, Propranolol, Timolol
- With intrinsic activity	Pindolol
- With blocking property	Carvedilol, Labetalol
Cardioselective	A - Atenolol, B - Bisoprolol, C - Celiprolol, B - Betaxolol, E - Esmolol, N - Nebivolol, A - Acebutolol, M - Metoprolol

30. **Topical application of Timolol to the eye would be expected to induce which of the following [GPAT-2017]**

(a) Decreased formation of aqueous humor

(b) Miosis

(c) Mydriasis

(d) Increased outflow of aqueous humor

30. **Topical application of Timolol to the eye would be expected to induce which of the following [GPAT-2017]**

(a) Decreased formation of aqueous humor

(b) Miosis

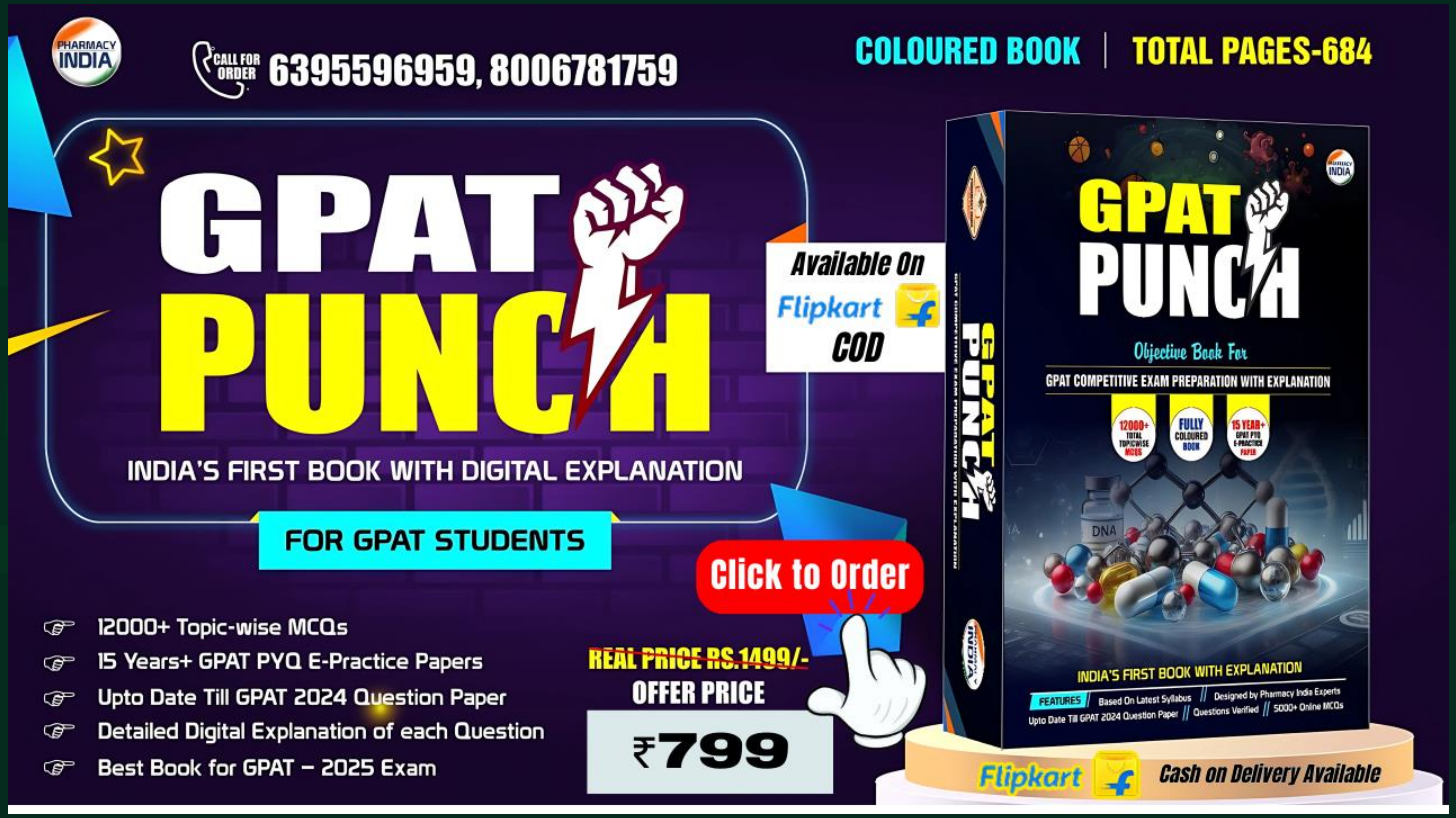
(c) Mydriasis

(d) Increased outflow of aqueous humor

. Explanation:

MODE OF ACTION OF OCULAR HYPOTENSIVE DRUG			
Drug/Class	Aqueous secretion	Trabecular	Uveoscleral outflow
1. β-blockers (Timolol)	↓	-	-
2. Adrenaline/ Dipivefrine	↓		↑
3. Brimonidine/apraclonidine	↓	-	↑
4. Prostaglandins (Latanoprost)	-	↑	↑
5. Miotics (Pilocarpine)	-	↑	-
6. Carbonic anhydrase inhibitors	↓	-	-

“GPAT Punch – Objective Book for GPAT 2025”

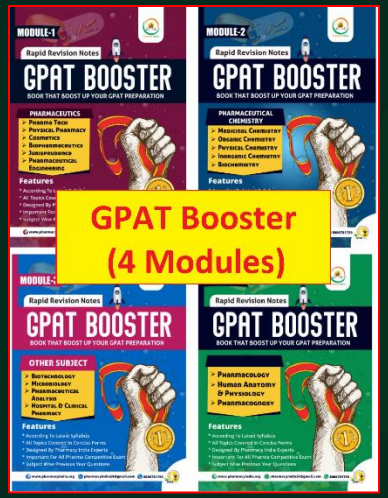


[Click Here to Download Book Sample PDF]

[Cash on Delivery Available | Click to Order Now]

Best Books For GPAT Preparation

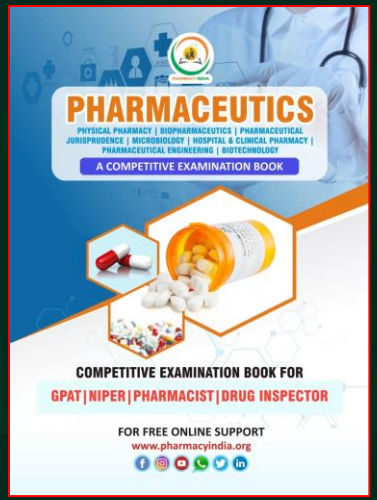
ORDER NOW



Complete GPAT Preparation
Includes All Core Subjects
COD Available

Flipkart

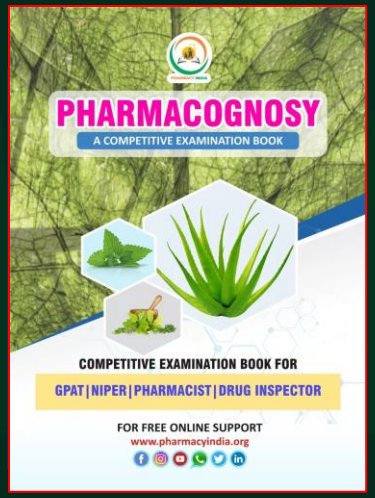
amazon



Pharmaceutics Book for
GPAT 2025 Exam
COD Available

Flipkart

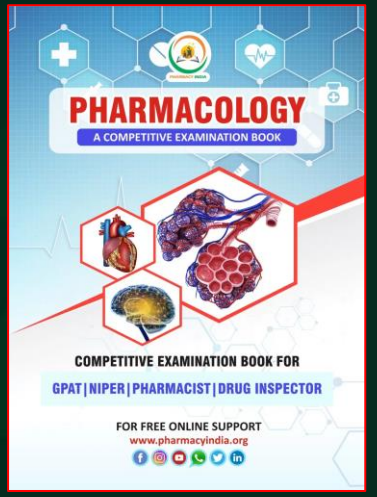
BUY NOW



Pharmacognosy Book
for GPAT 2025 Exam
COD Available

Flipkart

BUY NOW



Pharmacology Book for
GPAT 2025 Exam
COD Available

Flipkart

BUY NOW

PHARMACY INDIA

LIVE CLASSES STARTING FROM
05th FEBRUARY 2025

अभिMAAN

PREPARE TILL YOUR LAST DESTINATION

MISSION
GPAT 2025
120 DAY'S CRASH COURSE

- LIVE CLASSES
- ONLINE TEST SERIES
- E-NOTES

PHARMACY INDIA

31. Characteristics of carvedilol includes [GPAT-2016]

[P] It is a $ß_1$-selective antagonist

[Q] It has both a_1-selective and ß-blocking effects

[R] It inhibits vascular smooth muscle mitogenesis

(a) [P] true, [Q] & [R] false

(b) [P], [Q] & [R] true

(c) [Q] true, [P] & [R] false

(d) [P], [Q] & [R] false

31. Characteristics of carvedilol includes [GPAT-2016]

[P] It is a $ß_1$-selective antagonist

[Q] It has both a_1-selective and ß-blocking effects

[R] It inhibits vascular smooth muscle mitogenesis

(a) [P] true, [Q] & [R] false

(b) [P], [Q] & [R] true

(c) [Q] true, [P] & [R] false

(d) [P], [Q] & [R] false

- **Explanation:**

CARVEDILOL

- Carvedilol is a racemic mixture where the S (-) enantiomer is a beta adrenoceptor blocker and the enantiomer is both a beta and alpha-1 adrenoceptor blocker. R (+)
- Carvedilol blocks a1, B1 and B2 receptors. In addition it has antioxidant property.
- It is used in the treatment of hypertension and congestive cardiac failure.
- Carvedilol act on alpha-1 adrenergic receptors and relaxes smooth muscle in vasculature, leading to reduced peripheral vascular resistance and an overall reduction in blood pressure.

32. **Which of the following is true for a-Blocker, EXCEPT [GPAT-2015]**

(a) Blockade of vasoconstriction

(b) Cause Nasal stuffiness and miosis

(c) Increased intestinal motility

(d) Tone of smooth muscle in bladder trigone and sphincter is increased

32. **Which of the following is true for a-Blocker, EXCEPT [GPAT-2015]**

(a) Blockade of vasoconstriction

(b) Cause Nasal stuffiness and miosis

(c) Increased intestinal motility

(d) Tone of smooth muscle in bladder trigone and sphincter is increased

- **Explanation:**

MECHANISM OF ACTION OF ALPHA RECEPTOR BLOCKER

- Blockade of vasoconstriction - hypotension
- Miosis (small pupil) and nasal stuffiness
- Postural reflex is interfered - postural hypotension
- Reflex tachycardia-due to fall in BP
- Promote urinary outflow
- Failure of ejaculation

33. **Which of the following is selective α_2-selective antagonists [GPAT-2013]**

(a) Clonidine

(b) Prazosin

(c) Phentolamine

(d) Yohimbine

33. **Which of the following is selective α_2-selective antagonists [GPAT-2013]**

(a) Clonidine

(b) Prazosin

(c) Phentolamine

(d) Yohimbine

- **Explanation:**

Classification of α Blockers	Examples
Non-selective (α_1 and α_2) Blocker	
- Reversible	- Phentolamine, Tolazoline
- Irreversible	- Phenoxybenzamine
Selective α_1 Blockers	- Prazosin, Terazosin, Doxazosin, Tamsulosin, Alfuzosin
Selective α_2 Blockers	- Yohimbine

34. **The drug used during the preoperative preparation for surgical excision of pheochromocytoma is [GPAT-2013]**

(a) Atenolol

(b) Phenoxybenzamine

(c) Reserpine

(d) Clonidine

34. **The drug used during the preoperative preparation for surgical excision of pheochromocytoma is [GPAT-2013]**

(a) Atenolol

(b) Phenoxybenzamine

(c) Reserpine

(d) Clonidine

- **Explanation:**

PHEOCHROMOCYTOMA

- It is a tumour of adrenal medullary cells. Excess CAs are secreted which can cause intermittent or persistent hypertension.
- Therapeutic Surgical removal of the tumour is the first line therapeutic approach. Phenoxybenzamine can be used as definitive therapy for inoperable and malignant pheochromocytoma.

35. **Which of the following drugs does NOT induce mydriasis [GPAT-2011]**

(a) Atropine

(b) Ephedrine

(c) Phentolamine

(d) Cocaine

35. **Which of the following drugs does NOT induce mydriasis [GPAT-2011]**

(a) Atropine

(b) Ephedrine

(c) Phentolamine

(d) Cocaine

- **Explanation:**
- Mydriasis occurs with a rise in intracocular pressure due to the dilated iris blocking drainage of the intraocular fluid from the angle of the anterior chamber.
- It may precipitate angle-closure glaucoma.

DRUGS	MECHANISM FOR CAUSING MYDRIASIS
Antimuscarinic (Atropine)	Relaxation of sphincter pupillae
Alpha adrenergic (Ephedrine)	Contraction of dilator pupillae
Cocaine	By inhibiting the reuptake of noradrenaline into nerve terminals.

36. **Which of the following beta blockers has been shown clinically to reduce mortality in patients of symptomatic heart failure [GPAT-2011]**

(a) Atenolol

(b) Carvedilol

(c) Propranolol

(d) Esmolol

36. **Which of the following beta blockers has been shown clinically to reduce mortality in patients of symptomatic heart failure [GPAT-2011]**

(a) Atenolol

(b) Carvedilol

(c) Propranolol

(d) Esmolol

- **Explanation:**
- Carvediol is β_1 + β_2 + α_1 adrenoceptor blocker and also act as anti-oxidant and antiproliferative effect on vascular smooth muscle cells.
- Owing to neuroprotective effect it has the ability to offer major cardiovascular organ protection.
- In contrast to other beta blockers carvedilol causes peripheral vasoconstriction and does not alter serum lipid and blood glucose level.

- It is useful for treating the elderly hypertensive patient in whom increased peripheral vascular resistance is undesirable. It is also useful in treatment of pregnancy-induced hypertension.
- In addition, ß-blockers reduce mortality among patients with mild to moderate symptomatic heart failure.

37. **A cardio-selective beta blocker with vasodilating properties is [GATE-2008]**

(a) Pindolol

(b) Atenolol

(c) Bisoprolol

(d) Nebivolol

37. **A cardio-selective beta blocker with vasodilating properties is [GATE-2008]**

(a) Pindolol

(b) Atenolol

(c) Bisoprolol

(d) Nebivolol

- **Explanation:**

Generation	Description	Examples
First Generation	Older, nonselective	Propranolol, Timolol, Sotalol, Pindolol
Second Generation	β1-selective	Metoprolol, Atenolol, Acebutolol, Bisoprolol, Esmolol
Third Generation	With additional α-blocking and/or vasodilator properties	Labetalol, Carvedilol, Celiprolol, Nebivolol, Betaxolol

38. A 60-year-old patient presents with glaucoma. Therapy should include [GATE-2003]

[P] Topical Atropine

[Q] Topical Pilocarpine

[R] Oral Acetazolamide

[S] Oral Pilocarpine

(a) [P], [Q]

(b) [Q], [R]

(c) [R], [S]

(d) [P], [S]

38. A 60-year-old patient presents with glaucoma. Therapy should include [GATE-2003]

[P] Topical Atropine

[Q] Topical Pilocarpine

[R] Oral Acetazolamide

[S] Oral Pilocarpine

(a) [P], [Q]

(b) [Q], [R]

(c) [R], [S]

(d) [P], [S]

- **Explanation:**

- Acetazolamide: Oral treatment with Acetazolamide (0.25g 6-12 hourly) reduces aqueous formation by limiting generation of bicarbonate ion in the ciliary epithelium and its uses is Glaucoma: as adjuvant to other ocular hypotensive
- Topical Pilocarpine and/or antiChEs were the standard antiglaucoma drugs. In open angle glaucoma, they lower i.o.t. by increasing ciliary muscle tone thereby improving patency of trabeculae.

- Pilocarpine tablets are used to treat dryness of the mouth and throat caused by a decrease in the amount of saliva that may occur after radiation treatment for cancer of the head and neck or in patients with Sjogren's syndrome.
- Topical Atropine reduces myopia progression and axial elongation in children in a dose-related manner, but a rebound phenomenon occurs with higher doses. Its use has been shown to be safe, but higher doses cause pupil dilation, loss of accommodation and near vision.

39. **Metoprolol is sometimes preferred to Propranolol because [GATE-2002]**

(a) It has both a and ß adrenergic blockade

(b) It has both vasodilatory properties and ß adrenergic blocker

(c) It is a ß1 selective antagonist and it does not enter the brain

(d) It is a $ß_2$ selective antagonist

39. **Metoprolol is sometimes preferred to Propranolol because [GATE-2002]**

(a) It has both a and ß adrenergic blockade

(b) It has both vasodilatory properties and ß adrenergic blocker

(c) It is a ß1 selective antagonist and it does not enter the brain

(d) It is a $ß_2$ selective antagonist

- **Explanation:**
- The use of lipid-soluble beta blockers such as Propranolol has been associated with more CNS side effects, such as dizziness, confusion, or depression.
- However Metoprolol is β , selective antagonist and therefore act as cardioselective beta blocker. It does not enter the brain and hence the CNS effects can be avoided.

40. **Propranolol [GATE-1996]**

(a) Reduces myocardial oxygen consumption

(b) $ß_1$ receptor selective blocker

(c) Has intrinsic sympathomimetic activity

(d) Is a hypotensive agent in patients with normal blood pressure

40. **Propranolol [GATE-1996]**

(a) Reduces myocardial oxygen consumption

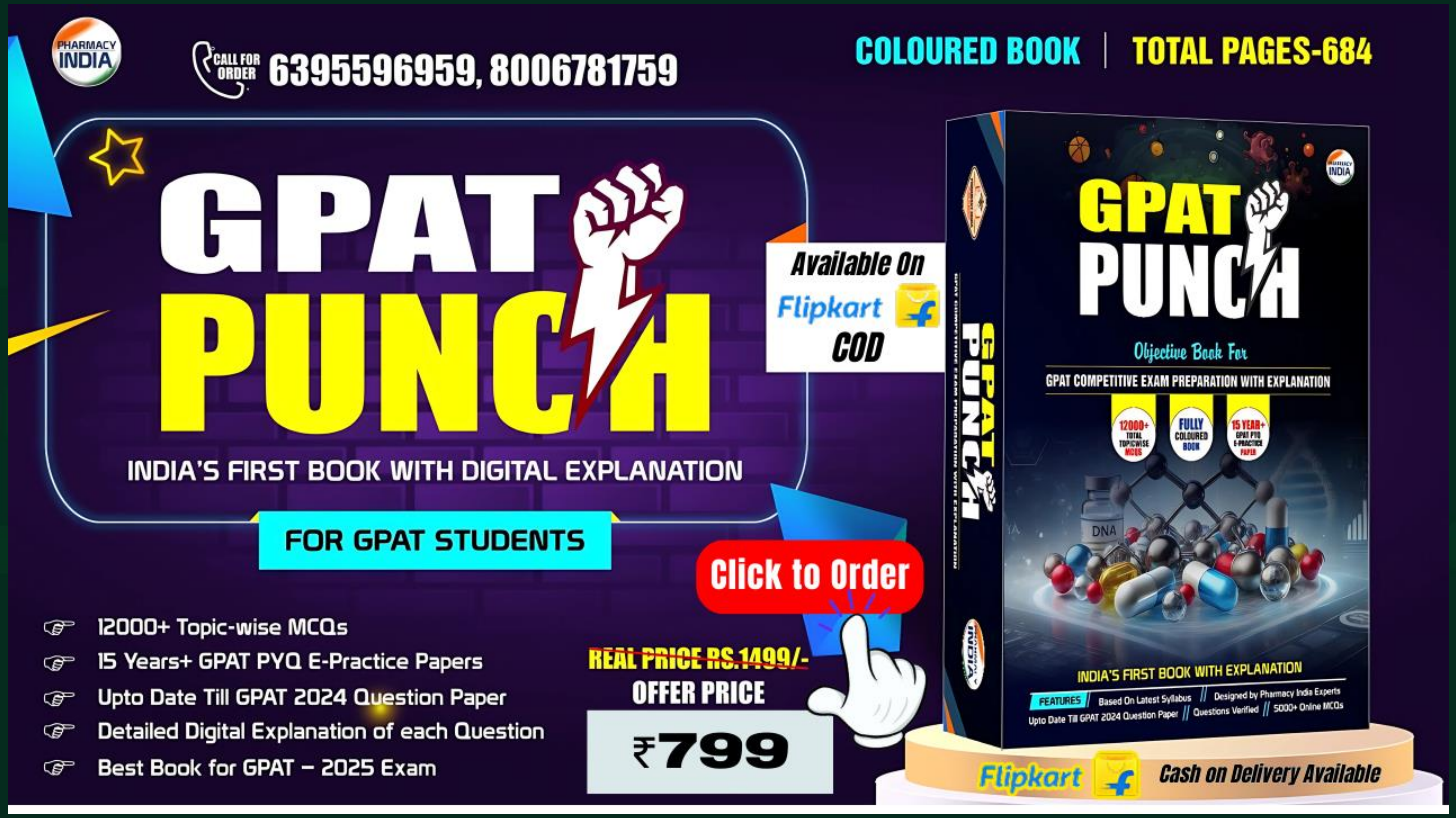
(b) $ß_1$ receptor selective blocker

(c) Has intrinsic sympathomimetic activity

(d) Is a hypotensive agent in patients with normal blood pressure

- **Explanation**:
- Propranolol reduces myocardial oxygen consumption.
- It is pure p-adrenergic antagonist and is nonselective acts as antagonist at 5, and B_2 receptors
- In a subject at rest, it does not cause change in arterial pressure, heart rate and cardiac output

“GPAT Punch – Objective Book for GPAT 2025”

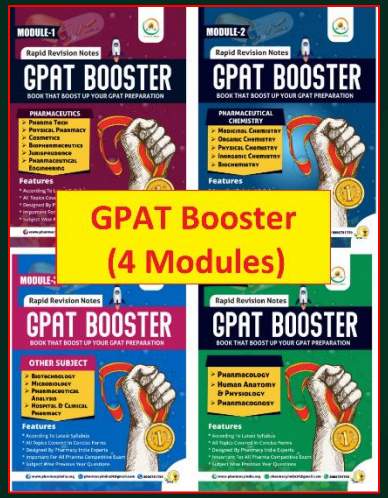


[Click Here to Download Book Sample PDF]

[Cash on Delivery Available | Click to Order Now]

Best Books For GPAT Preparation

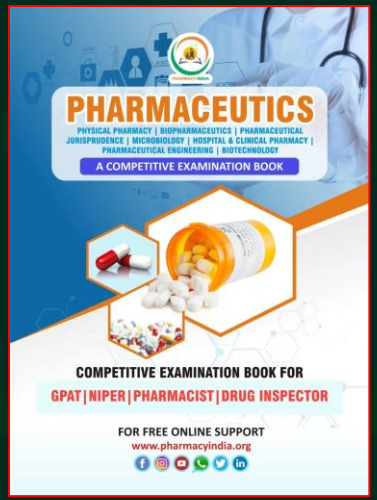
ORDER NOW



Complete GPAT Preparation
Includes All Core Subjects
COD Available

Flipkart

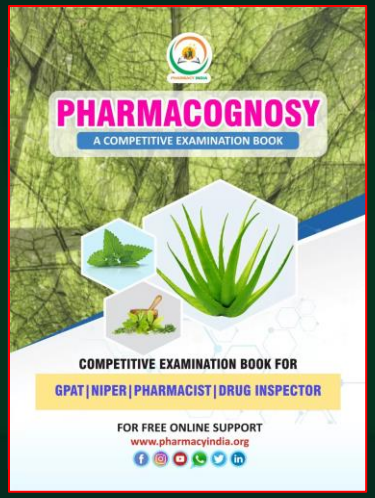
amazon



Pharmaceutics Book for
GPAT 2025 Exam
COD Available

Flipkart

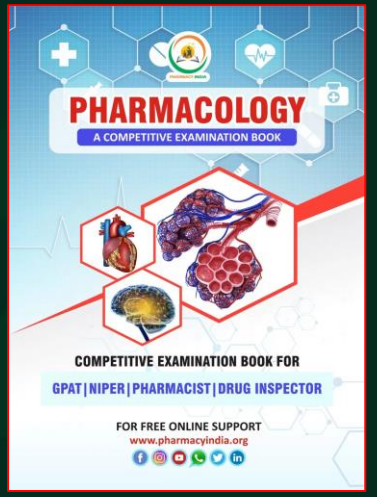
BUY NOW



Pharmacognosy Book
for GPAT 2025 Exam
COD Available

Flipkart

BUY NOW



Pharmacology Book for
GPAT 2025 Exam
COD Available

Flipkart

BUY NOW

PHARMACY INDIA

LIVE CLASSES STARTING FROM
05th FEBRUARY 2025

अभिMAAN

PREPARE TILL YOUR LAST DESTINATION

MISSION
GPAT 2025
120 DAY'S CRASH COURSE

- LIVE CLASSES
- ONLINE TEST SERIES
- E-NOTES

PHARMACY INDIA

41. **The most important action of beta blockers in glaucoma is which of the following**

(a) Membrane stabilizing effect

(b) Retinal neuron protecting effect

(c) Decrease in the production of aqueous humour

(d) Pupillary constriction

41. **The most important action of beta blockers in glaucoma is which of the following**

(a) Membrane stabilizing effect

(b) Retinal neuron protecting effect

(c) Decrease in the production of aqueous humour

(d) Pupillary constriction

- **Explanation:**

The most important action of **beta-blockers** in glaucoma is their ability to **reduce the production of aqueous humor** by the ciliary body. This action lowers intraocular pressure (IOP), which is critical in the management of **open-angle glaucoma**.

1. Site of Action:

- Beta-blockers act on **β2-adrenergic receptors** located in the **ciliary epithelium** of the eye.

1. **Reduction of Aqueous Humor Production:**
   - By blocking β2 receptors, beta-blockers reduce **cAMP levels** in the ciliary epithelium, leading to a decrease in the secretion of aqueous humor.
   - This reduction in aqueous humor production effectively lowers intraocular pressure.

2. Drugs Used:

- Common beta-blockers used in glaucoma include:
  - **Timolol** (most widely used).
  - Betaxolol (selective β1-blocker with fewer systemic side effects).
  - Levobunolol, Carteolol.

42. **All of the following are therapeutic uses of prazosin, except**

(a) Peripheral vascular disease

(b) Phaeochromocytoma

(c) Lupus Erythematous

(d) Scorpion sting

42. **All of the following are therapeutic uses of prazosin, except**

(a) Peripheral vascular disease

(b) Phaeochromocytoma

(c) Lupus Erythematous

(d) Scorpion sting

- **Explanation:**

Prazosin is an **α1-adrenergic receptor antagonist** that causes vasodilation by relaxing smooth muscles in the vasculature. It is primarily used to treat conditions related to hypertension, vascular disorders, and specific adrenergic crises. However, it is **not used for lupus erythematosus**, which is an autoimmune condition.

Therapeutic Uses of Prazosin:

1. **Peripheral Vascular Disease**
2. **Phaeochromocytoma**
3. **Scorpion Sting**

43. **All of the following are cardio selective beta blockers except**

(a) Atenolol

(b) Esmolol

(c) Bisoprolol

(d) Propranolol

43. **All of the following are cardio selective beta blockers except**

(a) Atenolol

(b) Esmolol

(c) Bisoprolol

(d) Propranolol

- **Explanation:**

Cardioselective beta blockers selectively block **β1-adrenergic receptors**, which are primarily found in the heart. This selectivity helps reduce heart rate, myocardial contractility, and oxygen demand while minimizing side effects on the lungs and peripheral vasculature that result from blocking β2 receptors.

Why Propranolol is Not Cardioselective:

1. Propranolol:

- **Non-selective beta blocker**: It blocks both **β1 and β2 receptors**.
- While it reduces heart rate and cardiac output by blocking β1 receptors, it also blocks **β2 receptors** in the bronchi and blood vessels, which can cause bronchoconstriction and vasoconstriction.
- It is unsuitable for patients with **asthma or COPD** due to its β2 blockade.

44. **Beta blocker with peripheral vasodilator action is**

(a) Carvedilol

(b) Propranolol

(c) Atenolol

(d) Acebutolol

44. Beta blocker with peripheral vasodilator action is

(a) Carvedilol

(b) Propranolol

(c) Atenolol

(d) Acebutolol

- **Explanation:**

Carvedilol is a **non-selective beta blocker** with **peripheral vasodilator action** due to its additional **α1-adrenergic receptor blocking** properties. This dual action makes it effective in conditions like hypertension and heart failure, where both beta blockade and vasodilation are beneficial.

Mechanism of Action of Carvedilol:

1. **Beta Blockade:**
   - Blocks **β1 and β2 adrenergic receptors**, reducing heart rate, myocardial contractility, and cardiac output.
2. **Alpha Blockade:**
   - Blocks **α1-adrenergic receptors** in vascular smooth muscle, causing **vasodilation**.
   - This reduces **peripheral vascular resistance**, lowering blood pressure.
3. **Antioxidant and Antiproliferative Properties:**
   - Carvedilol also exhibits antioxidant effects, which may contribute to its cardiovascular protective benefits.

45. **Combined alpha and beta blockers are**

(a) Carvedilol

(b) Prazosin

(c) Tamsulosin

(d) Milrinone

45. Combined alpha and beta blockers are

(a) Carvedilol

(b) Prazosin

(c) Tamsulosin

(d) Milrinone

- **Explanation:**

Carvedilol is a **combined alpha- and beta-adrenergic receptor blocker.** It exerts its effects by blocking both **α1-adrenergic receptors** and **β-adrenergic receptors**. This dual mechanism makes it effective in managing conditions like **hypertension** and **heart failure**.

Mechanism of Action of Carvedilol:

1. Alpha-1 Adrenergic Receptor Blockade:

- Causes **vasodilation** by relaxing vascular smooth muscle.
- Reduces **peripheral vascular resistance**, helping to lower blood pressure.

2. Beta Adrenergic Receptor Blockade:

- Blocks **β1 and β2 receptors**, reducing heart rate, myocardial contractility, and oxygen demand.
- Protects the heart in conditions like heart failure and post-myocardial infarction.

3. Additional Properties:

- **Antioxidant activity** contributes to its cardioprotective effects.
- Reduces afterload and improves cardiac function in heart failure.

46. **True statement about esmolol is/are**

(a) It is an α Blocker

(b) It has a long half-life

(c) It is not cardio selective

(d) It can cause bradycardia

46. True statement about esmolol is/are

(a) It is an α Blocker

(b) It has a long half-life

(c) It is not cardio selective

(d) It can cause bradycardia

- **Explanation:**

Esmolol is a **cardioselective β1-adrenergic blocker** known for its **short half-life** and rapid onset and offset of action. It is used in acute settings like supraventricular tachycardia and hypertensive emergencies.

47. **Contraindications of beta blockers are**

(a) Anemia

(b) Heart block

(c) Hypertension

(d) Arrhythmia

47. **Contraindications of beta blockers are**

(a) Anemia

(b) Heart block

(c) Hypertension

(d) Arrhythmia

- **Explanation:**
- **Beta blockers** are contraindicated in certain medical conditions where their pharmacological effects may worsen the condition. The most significant contraindication is **heart block**, as beta blockers reduce heart rate and AV nodal conduction, which can exacerbate bradycardia or conduction delays.
- Beta blockers decrease **AV nodal conduction** and **heart rate**, which can worsen conduction abnormalities in patients with **heart block**, particularly in second- or third-degree AV block.
- They should be avoided in these patients unless a pacemaker is in place.

48. **Which of the following is used in beta blocker overdose**

(a) Atropine

(b) Norepinephrine

(c) Insulin

(d) Thyroxin

48. **Which of the following is used in beta blocker overdose**

(a) Atropine

(b) Norepinephrine

(c) Insulin

(d) Thyroxin

- **Explanation:**

In cases of **beta blocker overdose**, the primary issue is often **bradycardia** and **hypotension** caused by excessive blockade of β1-adrenergic receptors. Management focuses on reversing these effects, and **atropine** is one of the first-line treatments for symptomatic bradycardia.

Atropine:

- **Mechanism:** Atropine is an **anticholinergic drug** that blocks parasympathetic (vagal) stimulation of the heart via muscarinic receptors.

- **Effect:** It increases heart rate and improves conduction through the AV node, counteracting the bradycardia caused by beta blocker overdose.
- **Use:** Administered as the initial treatment for severe bradycardia due to beta blocker overdose.

49. **Beta blockers with intrinsic sympathomimetic properties are**

(a) Propanolol

(b) Pindolol

(c) Esmolol

(d) Butoxamine

49. **Beta blockers with intrinsic sympathomimetic properties are**

(a) Propanolol

(b) Pindolol

(c) Esmolol

(d) Butoxamine

- **Explanation:**

Intrinsic sympathomimetic activity (ISA) refers to the ability of some beta blockers to partially activate **β-adrenergic receptors** while simultaneously blocking the stronger effects of endogenous catecholamines like epinephrine and norepinephrine. Beta blockers with ISA are less likely to cause bradycardia or a significant reduction in cardiac output compared to beta blockers without ISA.

Beta Blockers with Intrinsic Sympathomimetic Activity (ISA):

1. **Pindolol**:
   - A **non-selective beta blocker** with **strong ISA**.
   - It acts as a **partial agonist** at β1 and β2 receptors.
   - It is useful in patients requiring beta-blockade but at a lower risk of developing severe bradycardia or fatigue.
2. **Acebutolol**:
   - A **cardioselective β1 blocker** with ISA, making it suitable for patients with mild bradycardia or peripheral vascular disease.

50. **Which of the following is a selective β2 antagonist**

(a) Esmolol

(b) Betaxolol

(c) Butoxamine

(d) Celiprolol

50. **Which of the following is a selective β2 antagonist**

(a) Esmolol

(b) Betaxolol

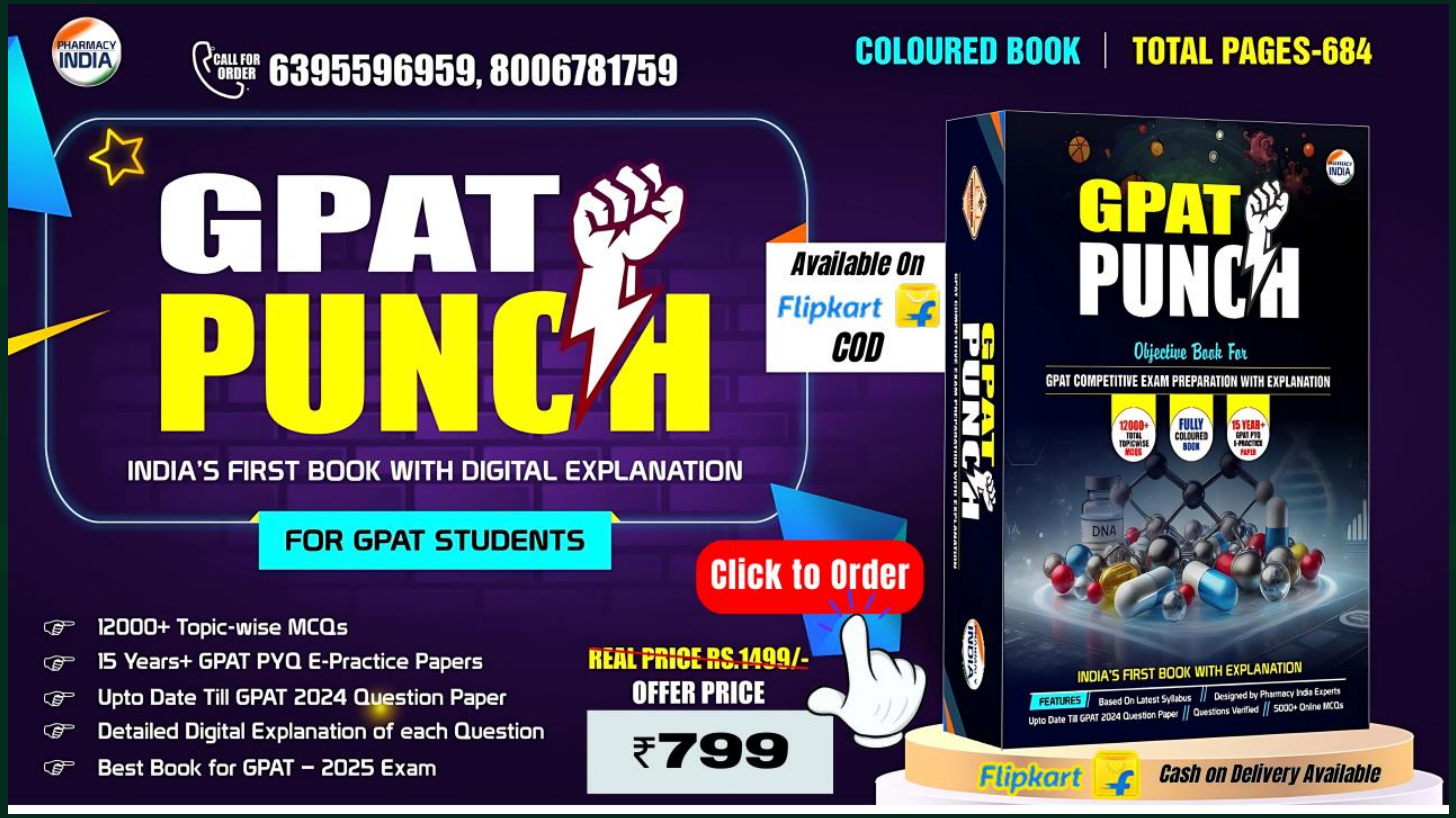
(c) Butoxamine

(d) Celiprolol

- **Explanation:**

Butoxamine is a **selective β2-adrenergic antagonist**, meaning it specifically blocks **β2 receptors** found in smooth muscle (e.g., in the bronchi, blood vessels, and uterus). It is primarily used in research settings rather than clinical practice due to its limited therapeutic applications and potential to cause bronchoconstriction.

“GPAT Punch – Objective Book for GPAT 2025”

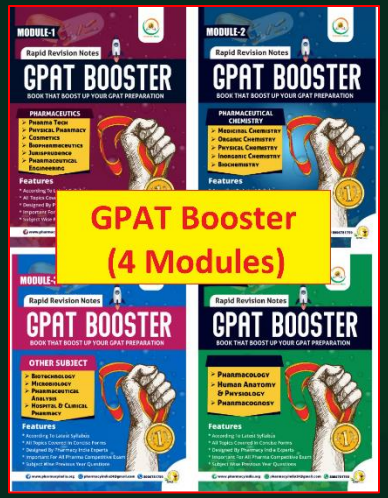


[Click Here to Download Book Sample PDF]

[Cash on Delivery Available | Click to Order Now]

Best Books For GPAT Preparation

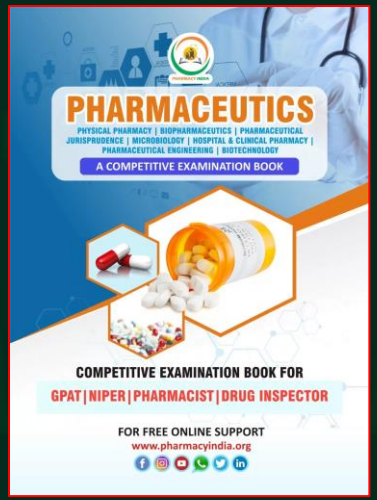
ORDER NOW



Complete GPAT Preparation
Includes All Core Subjects
COD Available

Flipkart

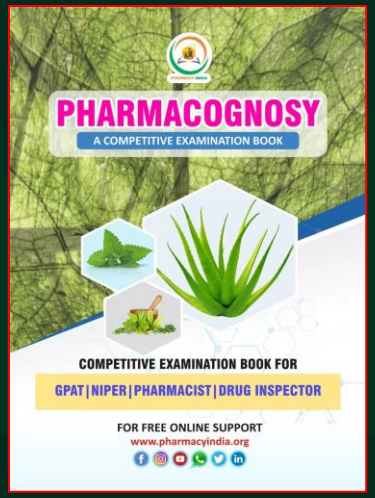
amazon



Pharmaceutics Book for
GPAT 2025 Exam
COD Available

Flipkart

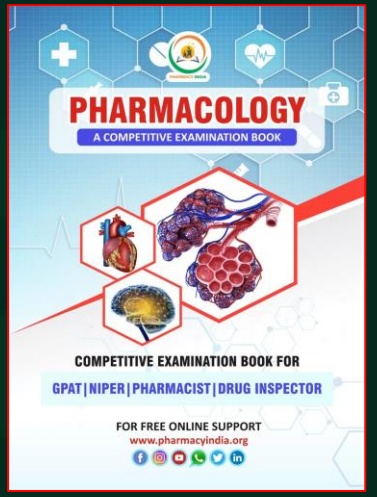
BUY NOW



Pharmacognosy Book
for GPAT 2025 Exam
COD Available

Flipkart

BUY NOW



Pharmacology Book for
GPAT 2025 Exam
COD Available

Flipkart

BUY NOW

PHARMACY INDIA

LIVE CLASSES STARTING FROM
05th FEBRUARY 2025

अभिMAAN

PREPARE TILL YOUR LAST DESTINATION

MISSION
GPAT 2025
120 DAY'S CRASH COURSE

- LIVE CLASSES
- ONLINE TEST SERIES
- E-NOTES

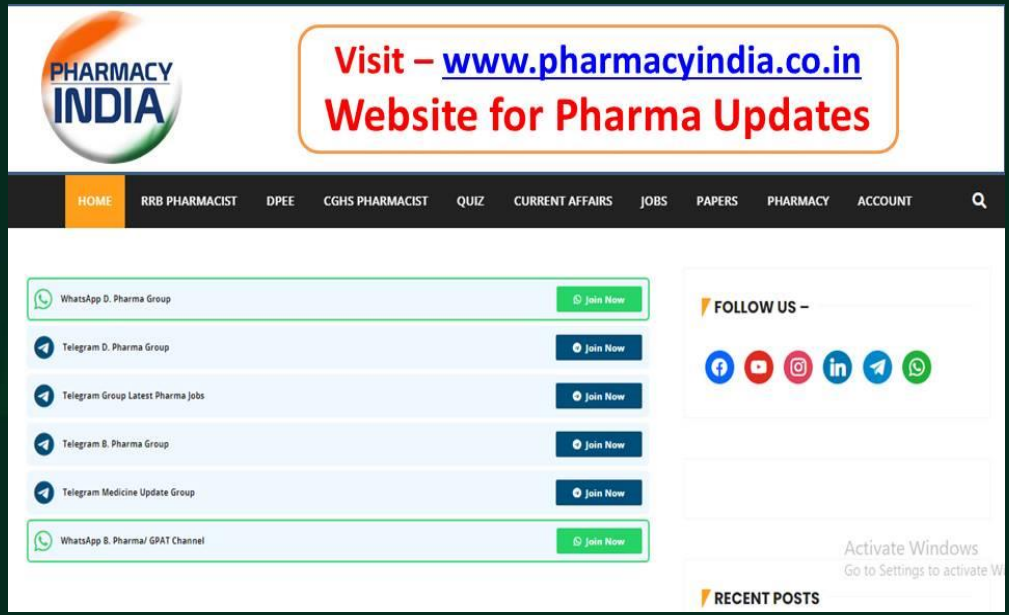
PHARMACY INDIA


PDF


PHARMACY INDIA
PHARMACY INDIA
GET IT ON Google Play
Download
PHARMACY INDIA
App from play store

Visit – pharmacyindia.co.in

- GPAT Preparation Materials
- Free GPAT Practice Quiz
- Daily Job Updates
- Previous Year GPAT Papers
- Exam Notifications & Updates
- Subject-Wise Study Notes
- College Rankings & Admissions



DOWNLOAD "PHARMACY INDIA" MOBILE APP

Mobile Phone Par Click karein

6395596959

Contact for admission related queries

DAILY UPDATES

जुड़िए PHARMACY INDIA

के साथ.....

WHATSAPP & TELEGRAM SE JUDNE KE LIYE ICONS PAR CLICK KARE

WhatsApp

THANK YOU