

GPAT

2027-28

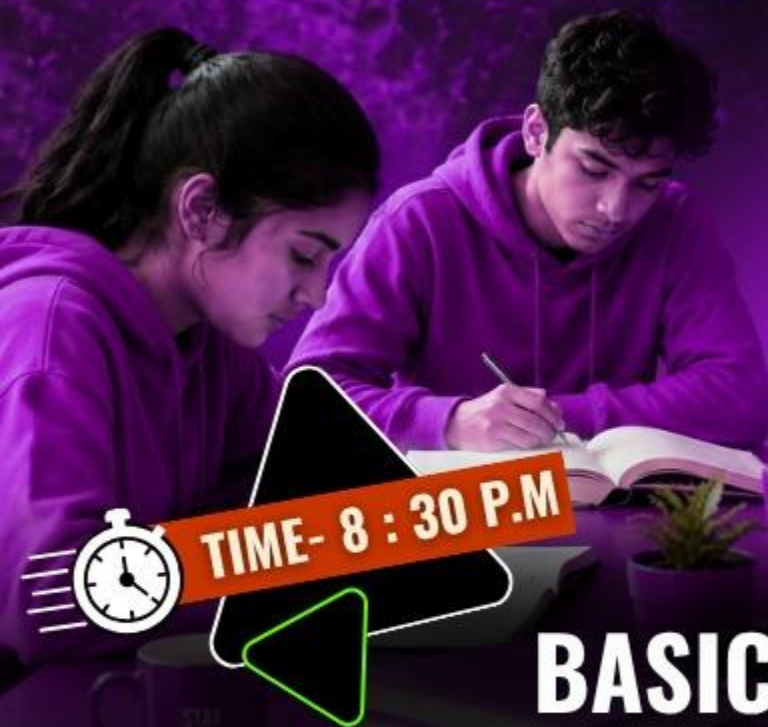


PATHOPHYSIOLOGY

CLASS- 6

BASIC PRINCIPLES & CARDIOVASCULAR SYSTEM PATHOPHYSIOLOGY

UPDATED QUESTIONS TILL GPAT 2026



TIME- 8 : 30 P.M

GPAT



VIDEO
LECTURE



PDF



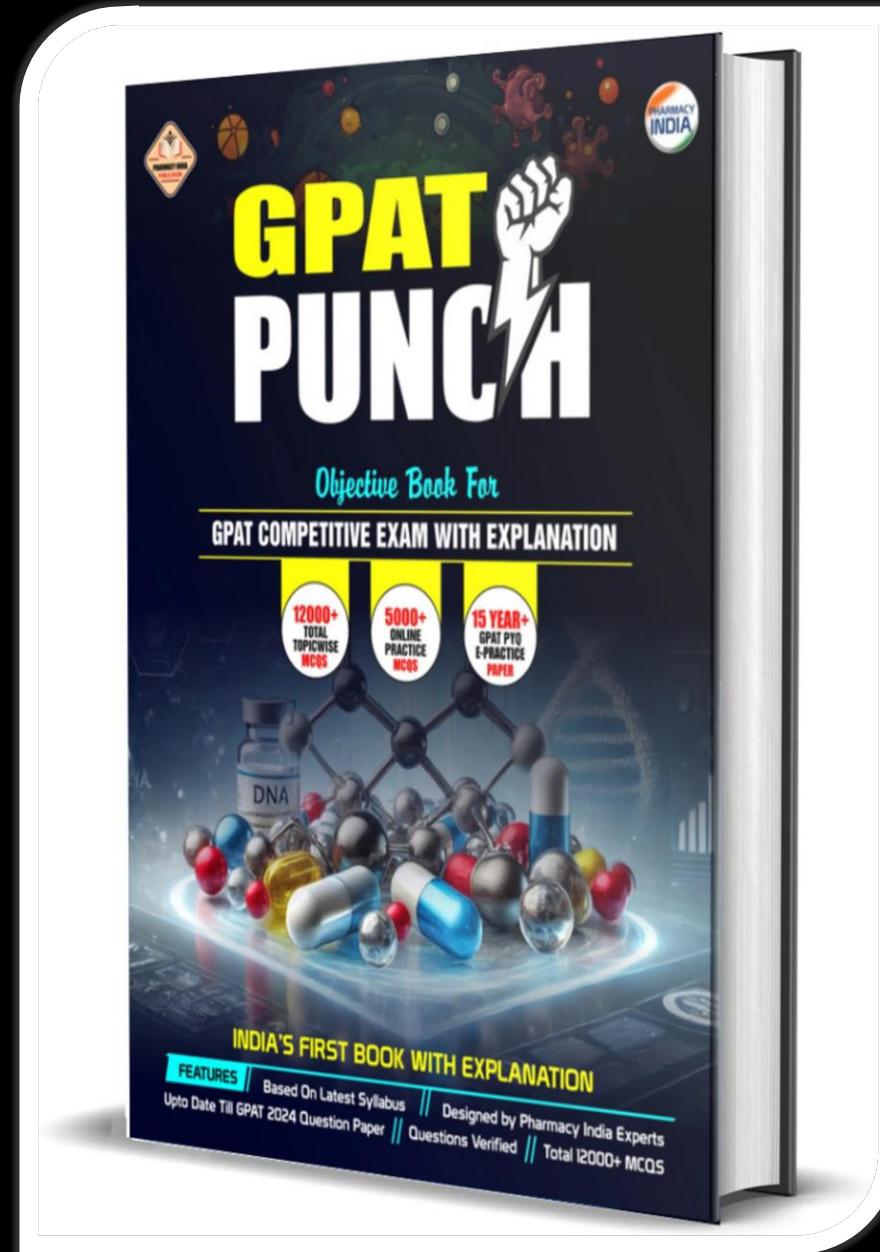
**STEP 1 – DOWNLOAD PHARMACY INDIA
MOBILE APP FROM PLAY STORE**

**STEP 2 – VISIT STORE OPTION – SEARCH
GPAT – ACCESS PDF + VIDEO**

Best Book for GPAT Exam

AVAILABLE ON

Flipkart  amazon



GPAT PUNCH OBJECTIVE BOOK

- Based on Latest Syllabus
- Up to Date Till GPAT 2026 Question Paper
- 5000+ Online Practice MCQs
- Total 12000+ MCQs
- 15 Year+ GPAT PYQ E-Practice Paper

DAILY UPDATES
जुड़िए **PHARMACY INDIA**
के साथ.....

**WHATSAPP & TELEGRAM SE JUDNE KE LIYE
ICONS PAR CLICK KARE**

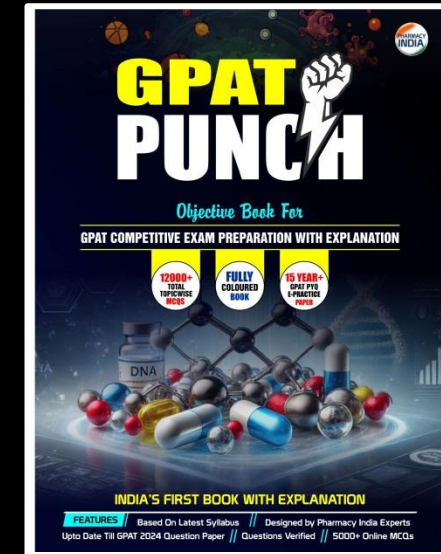




@PharmacyIndia

01. The earliest manifestation of almost all forms of cellular injury is: (GPAT shift – I 2023)

- (a) Cellular swelling
- (b) Fatty change
- (c) Calcification
- (d) Necrosis



Download Pharmacy India Mobile App from Playstore



01. The earliest manifestation of almost all forms of cellular injury is:
(GPAT Shift – I 2023)

(a) Cellular swelling

(b) Fatty change_ Abnormal accumulation of fat (triglycerides) inside cells, especially in the liver.

(c) Calcification_ Abnormal deposition of calcium salts in tissues.

(d) Necrosis



Download Pharmacy India Mobile App from Playstore

Earliest manifestation of almost all forms of cellular injury

CELLULAR SWELLING

(Reversible change)

Why does it happen?

- ↓ ATP
- Failure of Na^+/K^+ pump
- Influx of Na^+ and water
- Cellular **distension**



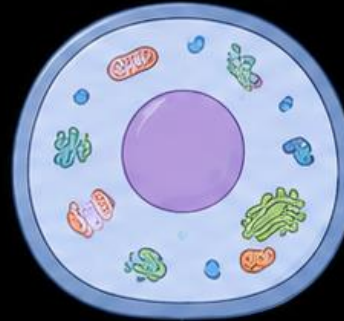
Key Point

It is the **earliest detectable** change seen in **almost all** forms of injury.

NORMAL CELL



EARLY INJURY (Cell Swelling)



Cell enlarges
Organelles appear paler & distended

PROGRESSIVE INJURY



Membrane blebs
↓ Organelles function
May lead to death

OUTCOMES

If injury is mild
→ **Reversible**
(Cell recovers)

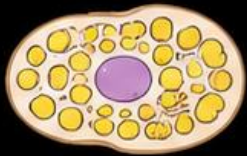


If injury is severe
→ **Irreversible**
(Leads to necrosis or apoptosis)



Other changes occur later

Fatty change



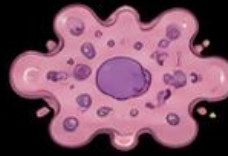
Due to abnormal fat accumulation

Calcification



Deposition of calcium salts in tissues

Necrosis



Irreversible cell death

Take Home ★

Cellular swelling is the **earliest** manifestation of **almost all** forms of **cellular injury**.

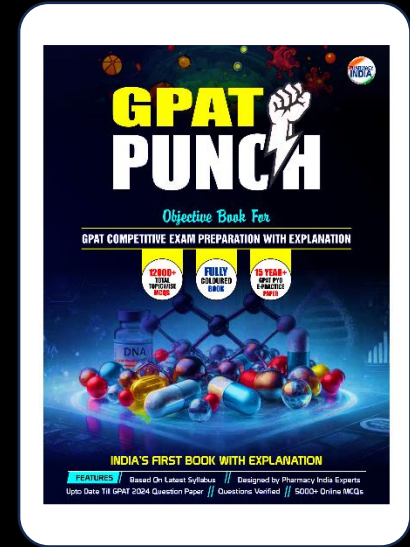




@PharmacyIndia

02. **Reversible cell injury is primarily characterized by:**

- (a) Nuclear pyknosis
- (b) Cellular swelling
- (c) Karyolysis
- (d) Karyorrhexis



Download Pharmacy India Mobile App from Playstore



@PharmacyIndia

02. Reversible cell injury is primarily characterized by:

(a) Nuclear pyknosis

→ Nucleus shrinks

(b) Cellular swelling

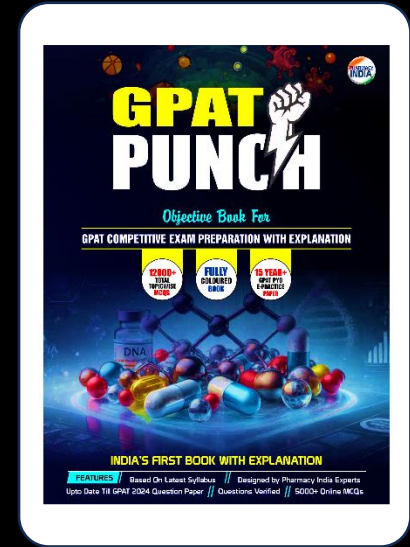
→ Cell becomes enlarged

(c) Karyolysis

→ Nucleus dissolves

(d) Karyorrhexis

→ Nucleus breaks into fragments

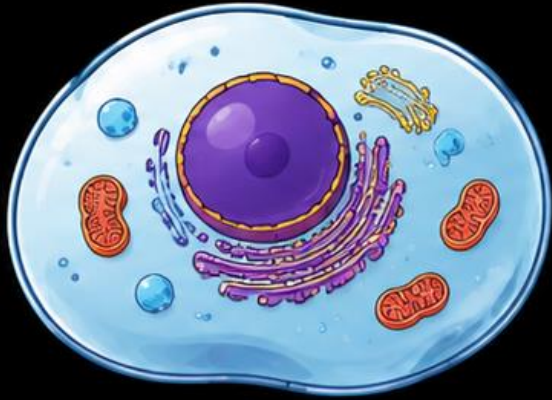


Download Pharmacy India Mobile App from Playstore

Reversible Cell Injury is primarily characterized by Cellular Swelling

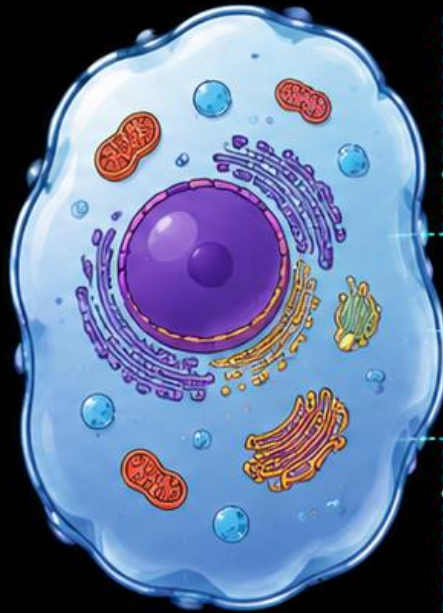
REVERSIBLE CELL INJURY

Early, potentially reversible response to stress



Main Feature:
CELLULAR SWELLING
(Hydropic change)

CELLULAR SWELLING



Water influx
(↓ ATP, Na⁺ pump failure)



Cell enlargement
(swelling)



Pale cytoplasm
(↓ protein synthesis)



Mild mitochondrial swelling



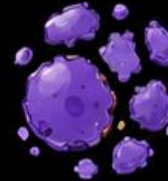
Plasma membrane intact

If stress is removed → Cell returns to normal

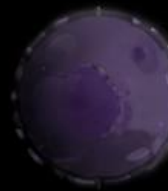
NUCLEAR CHANGES IN IRREVERSIBLE INJURY



Pyknosis
Nucleus shrinks, chromatin condenses



Karyorrhexis
Nucleus breaks into fragments



Karyolysis
Nucleus fades away, complete dissolution



Reversible = Swelling
Irreversible = Nuclear changes



@PharmacyIndia



03.

Free radical-mediated cell injury is mainly caused by:

- (a) Protein deficiency
- (b) Reactive oxygen species
- (c) Calcium deficiency
- (d) Decreased blood urea



Download Pharmacy India Mobile App from Playstore



@PharmacyIndia



03. Free radical-mediated cell injury is mainly caused by:

- (a) Protein deficiency
- (b) Reactive oxygen species**
- (c) Calcium deficiency
- (d) Decreased blood urea



Download Pharmacy India Mobile App from Playstore



Free Radical–Mediated Cell Injury



Mainly due to Reactive Oxygen Species (ROS)

ROS = superoxide,
hydroxyl radical,
hydrogen peroxide



Superoxide



Hydroxyl
radical



Hydrogen
peroxide

COMMON SOURCES OF ROS



Inflammation



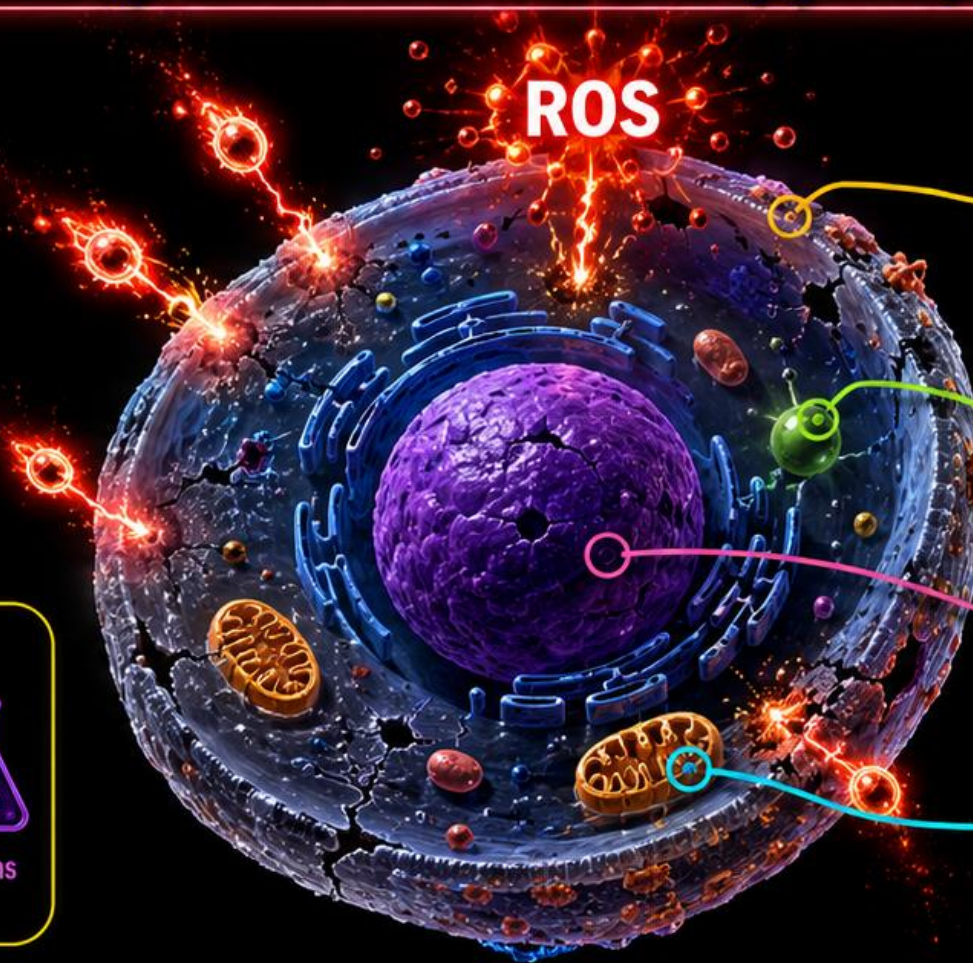
Ischemia-
reperfusion



Radiation

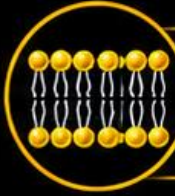


Toxins



ROS

They attack **lipids**, **proteins** & **DNA**



Lipid peroxidation
→ membrane damage



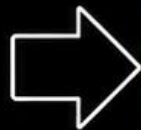
Protein oxidation
→ enzyme dysfunction



DNA damage
→ mutation /
cell death



Mitochondrial injury
→ energy failure



RESULT:



Cell
dysfunction



Necrosis



Apoptosis



Excess ROS overwhelms
antioxidant defenses
→ **cell injury**



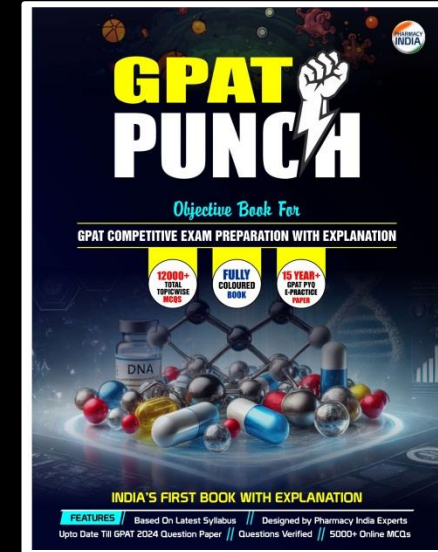
@PharmacyIndia



04.

A hallmark feature of apoptosis is:

- (a) Cell swelling
- (b) Loss of membrane integrity
- (c) Caspase activation
- (d) Leakage of lysosomal enzymes



Download Pharmacy India Mobile App from Playstore



04.

A hallmark feature of apoptosis is:

- (a) Cell swelling
- (b) Loss of membrane integrity
- (c) Caspase activation**
- (d) Leakage of lysosomal enzymes

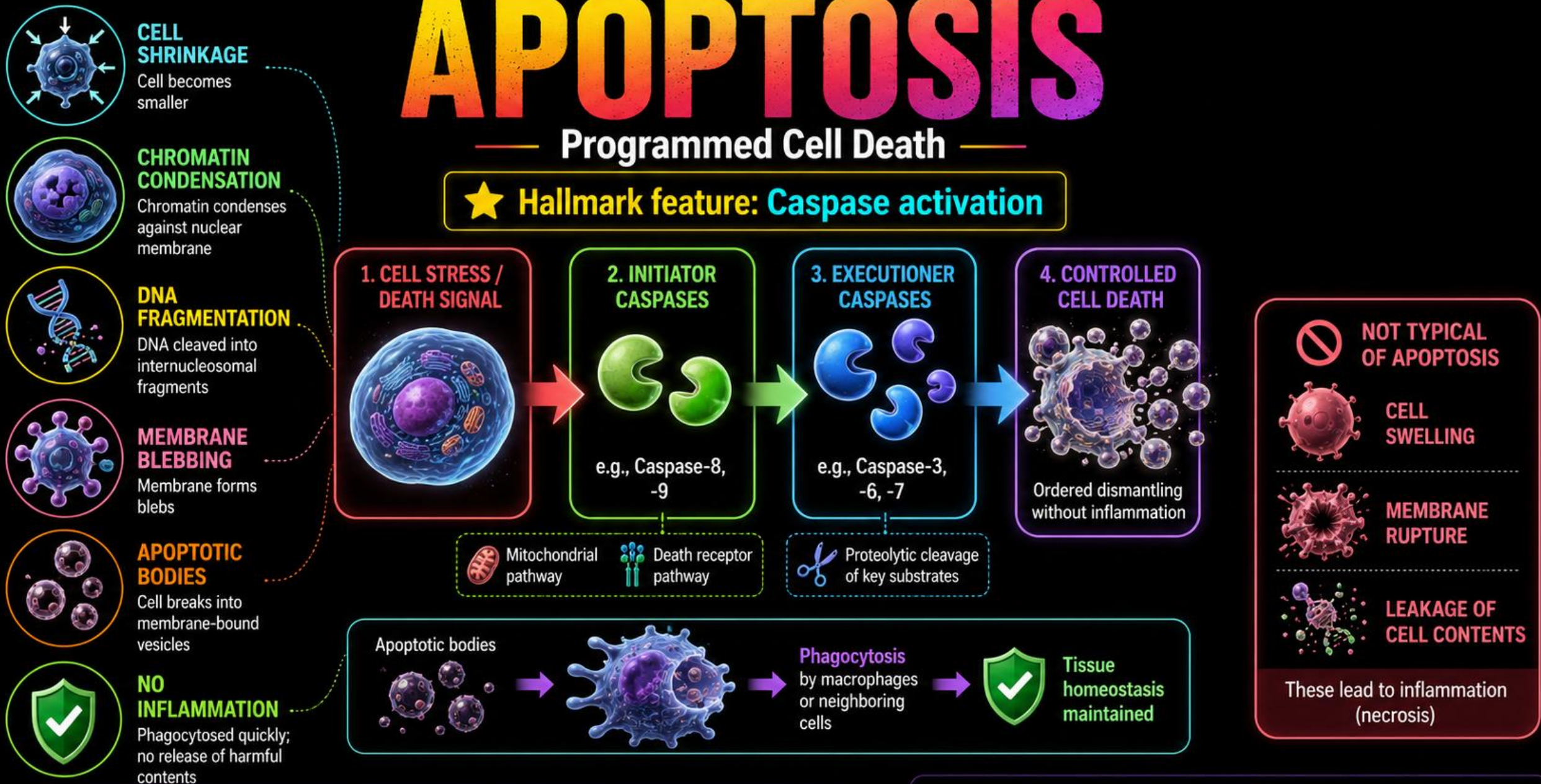


Download Pharmacy India Mobile App from Playstore

APOPTOSIS

Programmed Cell Death

★ **Hallmark feature: Caspase activation**



KEY TAKEAWAY: Apoptosis is a clean, controlled process driven by **caspase activation**, ensuring cells die without harming surrounding tissue.



Physiological roles



Development



Immune regulation



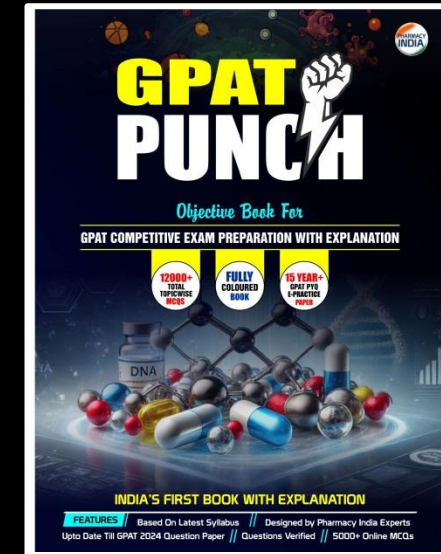
Removal of damaged cells



@PharmacyIndia

05. Chronic inflammation is characterized by all EXCEPT:

- (a) Macrophages
- (b) Lymphocytes
- (c) Fibroblast proliferation
- (d) Neutrophil predominance



Download Pharmacy India Mobile App from Playstore



05. Chronic inflammation is characterized by all EXCEPT:

- (a) Macrophages
- (b) Lymphocytes
- (c) Fibroblast proliferation
- (d) Neutrophil predominance**



Download Pharmacy India Mobile App from Playstore

CHRONIC INFLAMMATION

Key characteristics



Long-standing
inflammatory
response



Dominant cells:
macrophages &
lymphocytes



Fibroblast
proliferation



Tissue destruction
+ **repair** occur
together



Fibrosis / scar
formation may
develop



Macrophage

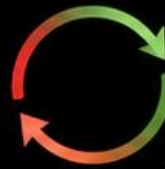
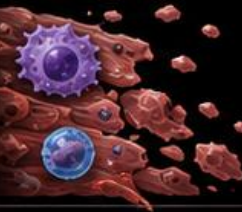


Lymphocyte

Often accompanied
by **plasma cells**



DESTRUCTION
Enzymes, ROS,
cytokines



REPAIR
Fibroblasts,
new matrix,
angiogenesis



Normal tissue



Chronic inflammation



Fibrosis / scar

Fibroblast
Collagen fibers
Blood vessel



CONTRAST



Neutrophil
predominance
is typical of
ACUTE
inflammation,
not **chronic**
inflammation.



Neutrophil



PERSISTENT
STIMULUS



IMMUNE RESPONSE
THAT ENDURES



DESTRUCTION + REPAIR
SIDE BY SIDE



STRUCTURAL CHANGE
OVER TIME



LEADS TO DYSFUNCTION
OF THE AFFECTED ORGAN

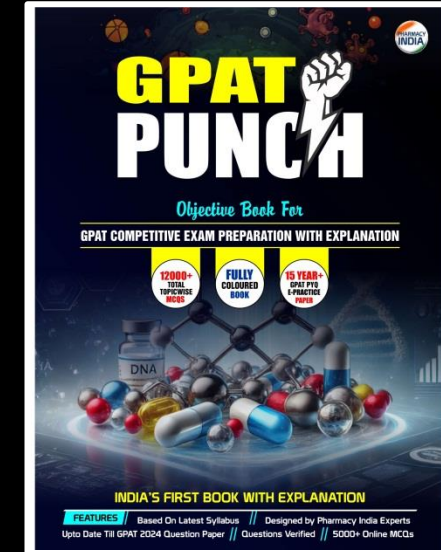


@PharmacyIndia

06.

Which cell is the first to appear at site of acute inflammation?

- (a) Basophils
- (b) Eosinophils
- (c) Neutrophils
- (d) Macrophages



Download Pharmacy India Mobile App from Playstore



06.

Which cell is the first to appear at site of acute inflammation?

(a) Basophils

(b) Eosinophils

(c) Neutrophils

(d) Macrophages



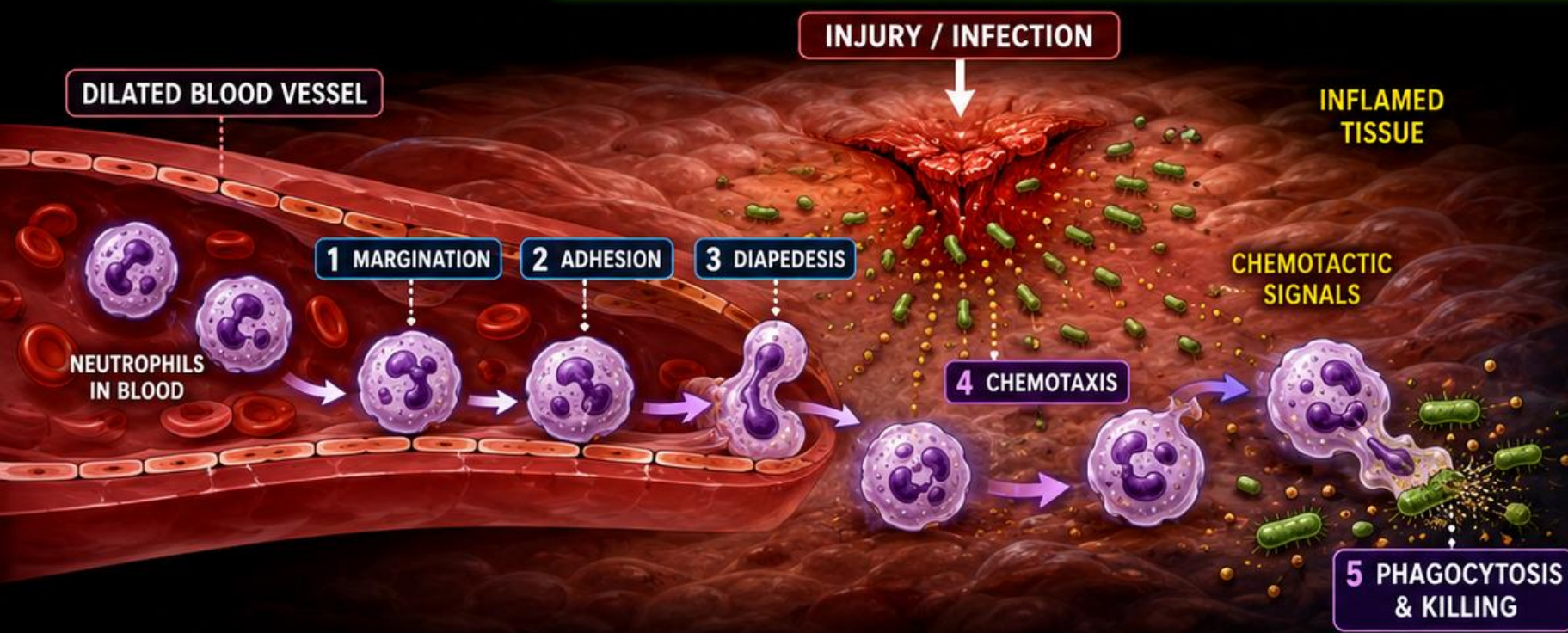
Download Pharmacy India Mobile App from Playstore



ACUTE INFLAMMATION: FIRST RESPONDER



NEUTROPHILS ARRIVE FIRST



-  **1** EARLIEST LEUKOCYTES AT THE INJURY SITE
-  **2** DOMINATE IN THE FIRST **6-24 HOURS**
-  **3** MOVE BY **CHEMOTAXIS**
-  **4** STRONG **PHAGOCYTIC ACTION**
-  **5** KILL **MICROBES** QUICKLY
-  **6** **MACROPHAGES** APPEAR LATER



ACUTE PHASE
→ **NEUTROPHILS FIRST**



0-24 HOURS

LATER PHASE
→ **MACROPHAGES**



CLEAR DEBRIS & PROMOTE HEALING



NEUTROPHILS = BODY'S RAPID RESPONSE TEAM

FIRST IN – FAST ACTION – PROTECTS THE BODY

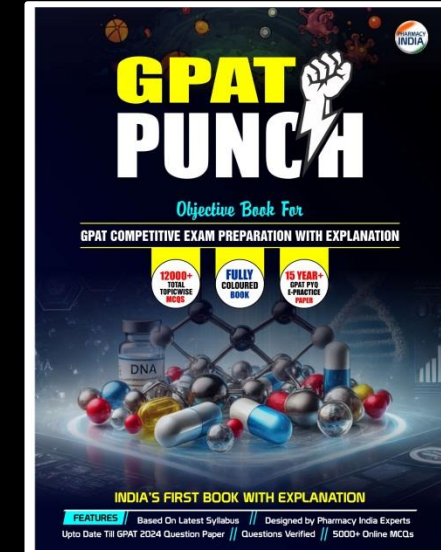


@PharmacyIndia



07. Cardinal sign “dolor” in inflammation is caused by:

- (a) Serotonin
- (b) Prostaglandin I₂
- (c) Histamine
- (d) Bradykinin



Download Pharmacy India Mobile App from Playstore



07. Cardinal sign “dolor(pain)” in inflammation is caused by:

(a) Serotonin

(b) Prostaglandin I₂

(c) Histamine

(d) Bradykinin

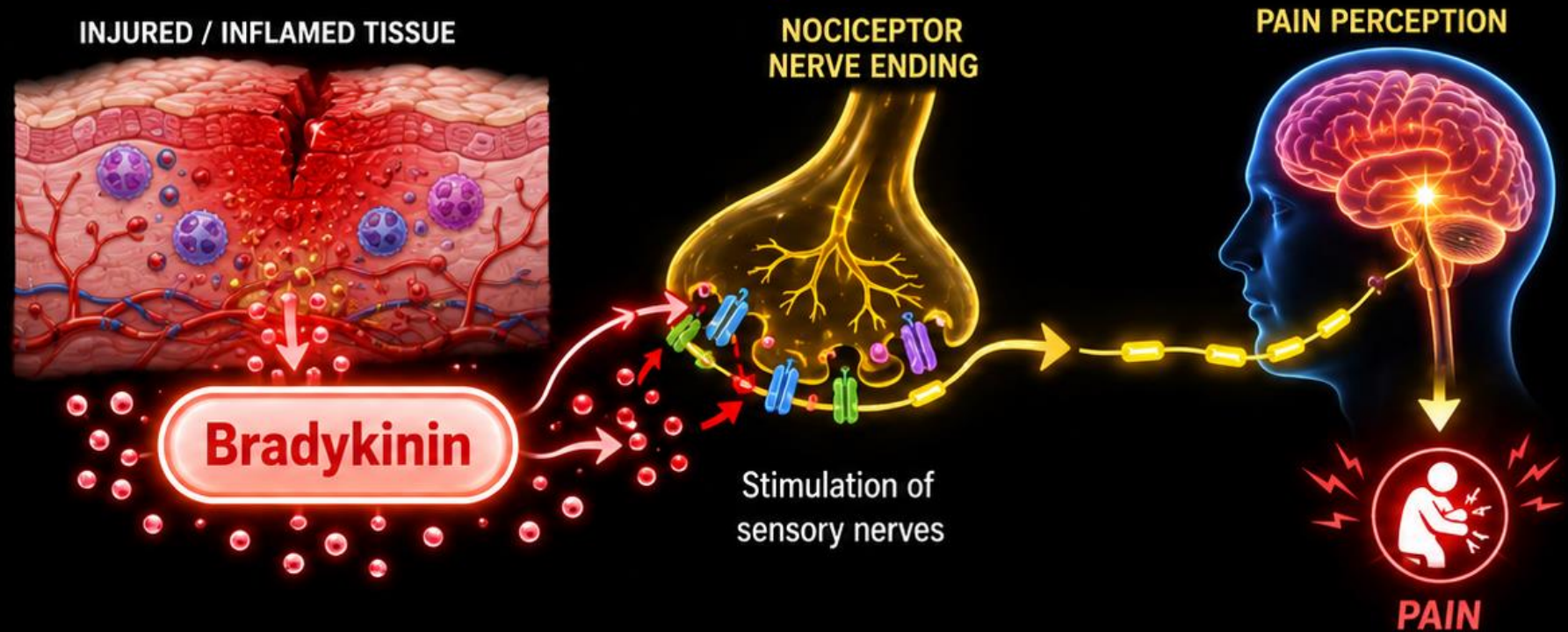


Download Pharmacy India Mobile App from Playstore

Dolor in Inflammation

Pain occurs mainly due to **bradykinin**

-  **Dolor** = pain
-  **Bradykinin** strongly stimulates pain receptors
-  It activates **nociceptor** nerve endings
-  **Prostaglandins** can enhance pain sensitivity
-  Result: **tenderness** + pain at inflamed site



★ **KEY MEDIATOR FOR PAIN**

BRADYKININ



Primary mediator of pain

HISTAMINE



Mainly causes vasodilation & itching

SEROTONIN



Contributes to vasodilation

PGI₂ (PROSTACYCLIN)



Vasodilation, inhibits platelet aggregation



Secondary / Less role in pain



Remember: Dolor → Bradykinin





@PharmacyIndia

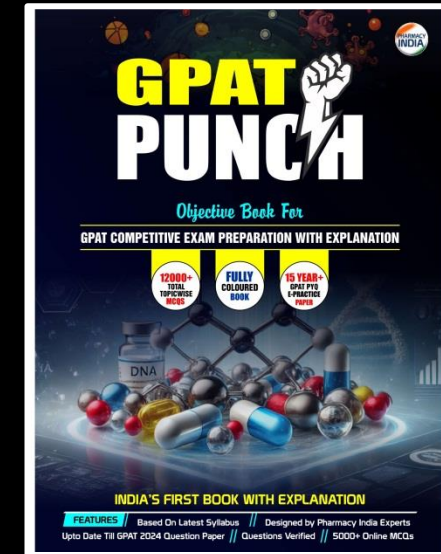


Granulomas are **small groups of immune cells** formed when the body tries to **trap and isolate** something it cannot remove (like TB germs or foreign material).

08.

Granuloma formation is a hallmark of:

- (a) Acute pyogenic infection
- (b) Tuberculosis
- (c) Viral hepatitis
- (d) Anaphylaxis



Download Pharmacy India Mobile App from Playstore



@PharmacyIndia



08.

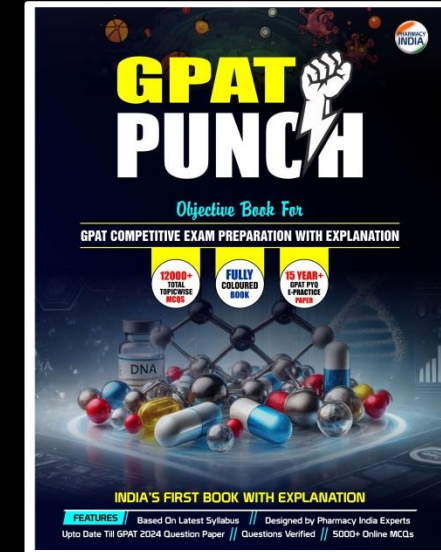
Granuloma formation is a hallmark of:

(a) Acute pyogenic infection

(b) Tuberculosis

(c) Viral hepatitis

(d) Anaphylaxis



Download Pharmacy India Mobile App from Playstore

GRANULOMA FORMATION

Classic hallmark of *tuberculosis*



Chronic
inflammatory
response



Body walls off
persistent
infection



Central caseation
is characteristic



Seen classically
in tuberculosis

Epithelioid
Macrophages

Langhans
Giant Cell

CASEOUS
NECROSIS

Lymphocytes

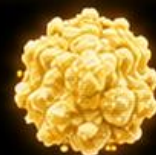


*Mycobacterium
tuberculosis*

TB



Granuloma



Caseation



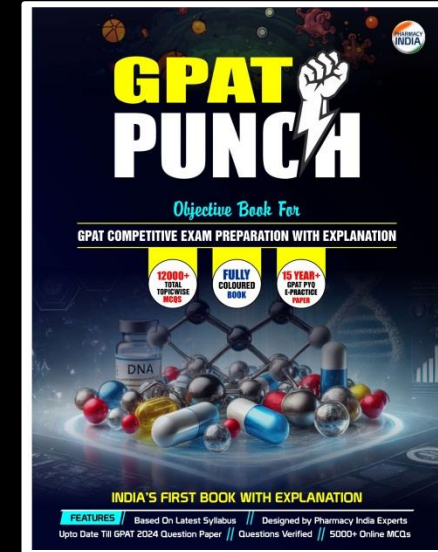
@PharmacyIndia



09.

Atherosclerosis begins with:

- (a) Fatty streak formation
- (b) Endothelial injury
- (c) Thrombosis
- (d) Fibrous cap formation



Download Pharmacy India Mobile App from Playstore



09.

Atherosclerosis begins with:

(a) Fatty streak formation

(b) Endothelial injury

(c) Thrombosis

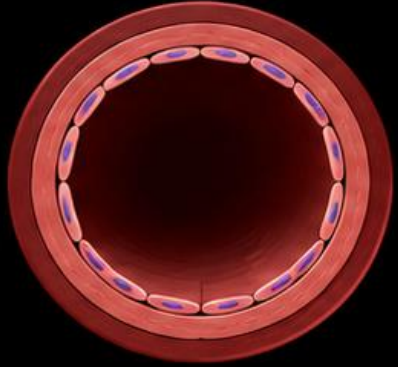
(d) Fibrous cap formation



Download Pharmacy India Mobile App from Playstore

Atherosclerosis Begins With Endothelial Injury

1. NORMAL ENDOTHELIUM



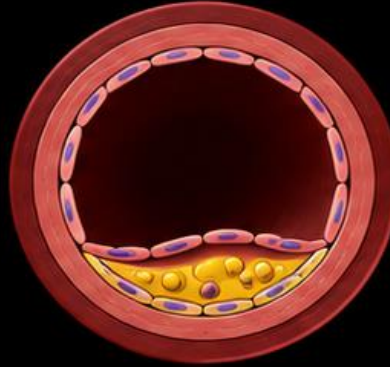
- ✓ Intact endothelium
- ✓ Anti-thrombotic
- ✓ Anti-inflammatory

2. ENDOTHELIAL INJURY



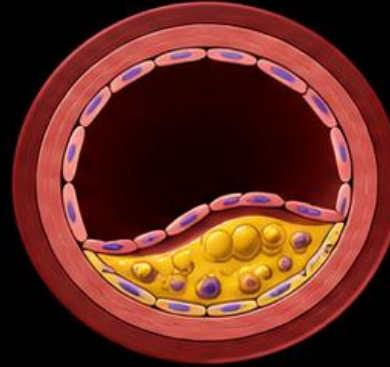
- Risk factors: ↑LDL, HTN, Smoking, DM, Oxidative stress
- ↑ Permeability & LDL entry

3. FATTY STREAK FORMATION



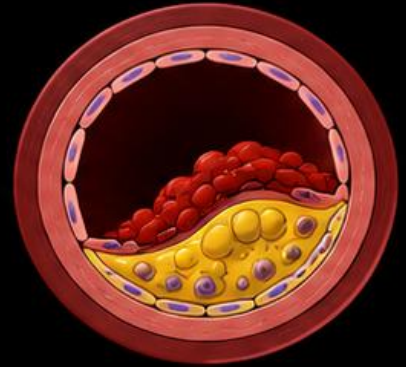
LDL oxidation → Monocyte adhesion → Foam cells
→ Fatty streak

4. PLAQUE DEVELOPMENT



Smooth muscle migration, ECM deposition, Fibrous cap forms

5. COMPLICATIONS



- Plaque rupture
- Thrombosis
- Ischemic events

★ Endothelial injury is the **initiating event** that triggers the entire atherosclerotic process.

CAUSES OF ENDOTHELIAL INJURY



High LDL



Hypertension



Smoking



Diabetes



Oxidative Stress



Inflammation



Genetic Factors



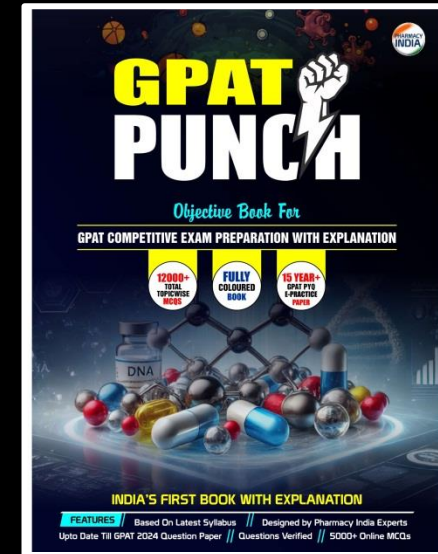
@PharmacyIndia



10.

Foam cells in atherosclerosis are formed due to uptake of: (GPAT - 2022)

- (a) Oxidized LDL by macrophages
- (b) HDL by smooth muscle cells
- (c) Triglycerides by neutrophils
- (d) VLDL by fibroblasts



Download Pharmacy India Mobile App from Playstore



10. Foam cells in atherosclerosis are formed due to uptake of: (GPAT - 2022)

(a) Oxidized LDL by macrophages

(b) HDL by smooth muscle cells

(c) Triglycerides by neutrophils

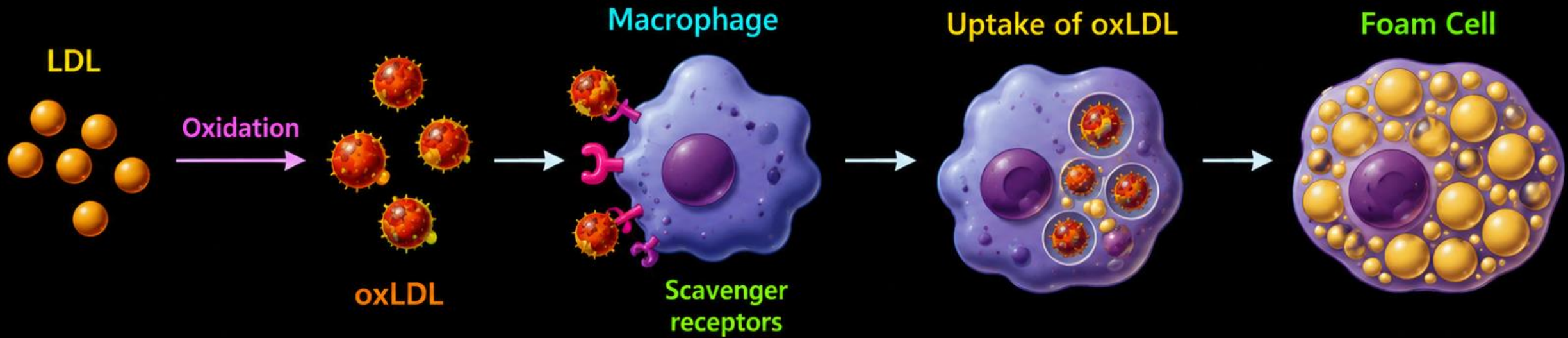
(d) VLDL by fibroblasts



Download Pharmacy India Mobile App from Playstore

★ Foam Cells in Atherosclerosis ★

Foam cells are formed due to uptake of oxidized LDL (oxLDL) by macrophages.



Key Point:

Macrophages take up oxLDL via scavenger receptors → accumulate lipid → become foam cells.

Why oxLDL?

- ✓ Not recognized by normal LDL receptors
- ✓ Taken up in excess via scavenger receptors
- ✓ Leads to cholesterol accumulation
- ✓ Forms foam cells → key in plaque formation

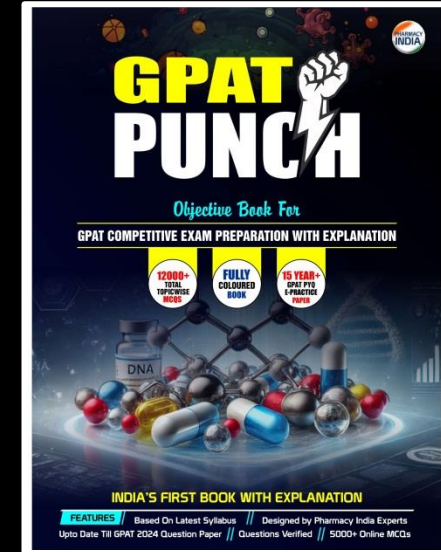


@PharmacyIndia

11.

The primary determinant of systolic blood pressure is:

- (a) Total peripheral resistance
- (b) Venous tone
- (c) Blood viscosity
- (d) Stroke volume



Download Pharmacy India Mobile App from Playstore



- 11.** **The primary determinant of systolic blood pressure is:**
- (a) Total peripheral resistance**
 - (b) Venous tone**
 - (c) Blood viscosity**
 - (d) Stroke volume _ Normal value $\approx$ 70 mL per beat**



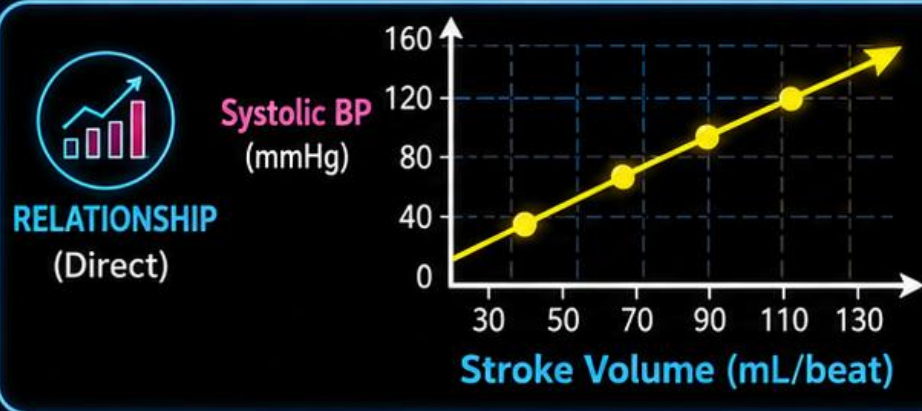
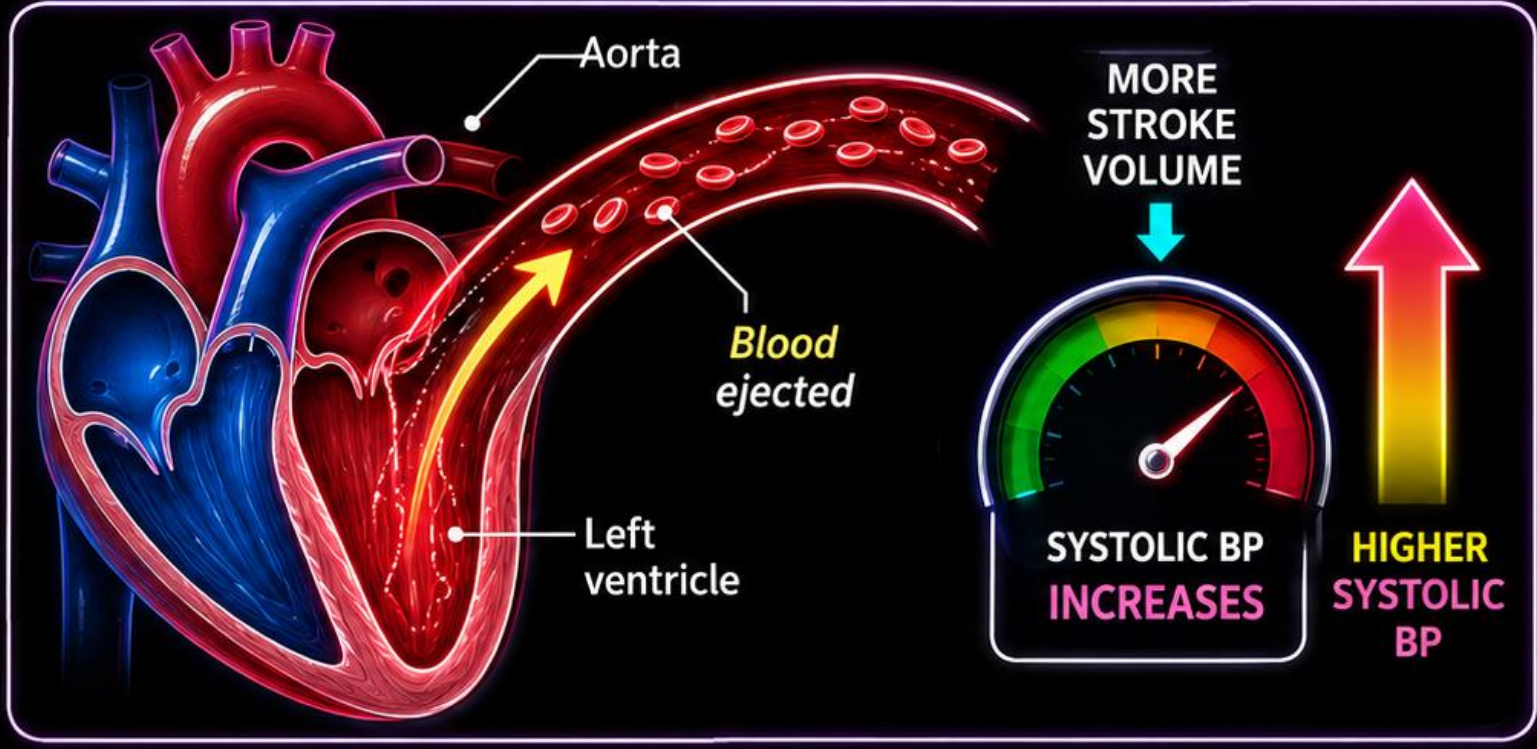
♥ **Systolic BP** – Key Explanation **💡**

1  **Systolic BP** reflects force during **ventricular contraction**

2  Key determinant = **Stroke volume**

3  **↑ Stroke volume**
→ **↑ blood** ejected
→ **↑ systolic BP**

4  Normal stroke volume **≈ 70 mL/beat**



 As stroke volume increases, **systolic BP rises.**

 **Quick clue:**
More blood pumped per beat
=
higher systolic pressure

 **CONTRAST** 
Total Peripheral Resistance (TPR) mainly influences **diastolic BP**, not systolic BP.

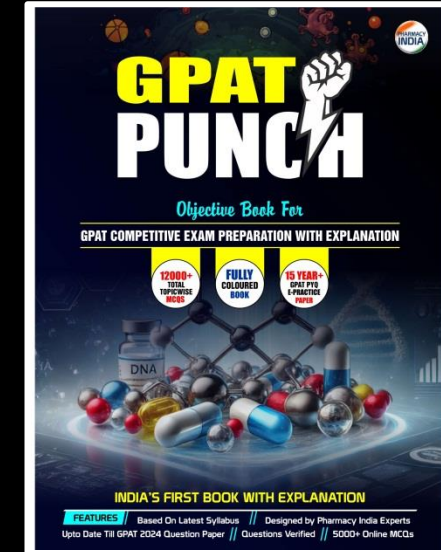




@PharmacyIndia



12. **Diastolic blood pressure is primarily maintained by:**
- (a) Stroke volume
 - (b) Total peripheral resistance
 - (c) Venous return
 - (d) Heart rate



Download Pharmacy India Mobile App from Playstore



12. Diastolic blood pressure is primarily maintained by:

(a) Stroke volume

(b) Total peripheral resistance The resistance that blood faces while flowing through all the blood vessels in the body.

(c) Venous return

(d) Heart rate



Download Pharmacy India Mobile App from Playstore

DIASTOLIC BLOOD PRESSURE

• Pressure during cardiac relaxation



- Occurs when the heart **relaxes**.



- Pressure is sustained by **arteriolar tone**.



- Peripheral **resistance** prevents a rapid pressure fall.



- Vasoconstriction **↑ diastolic pressure**.



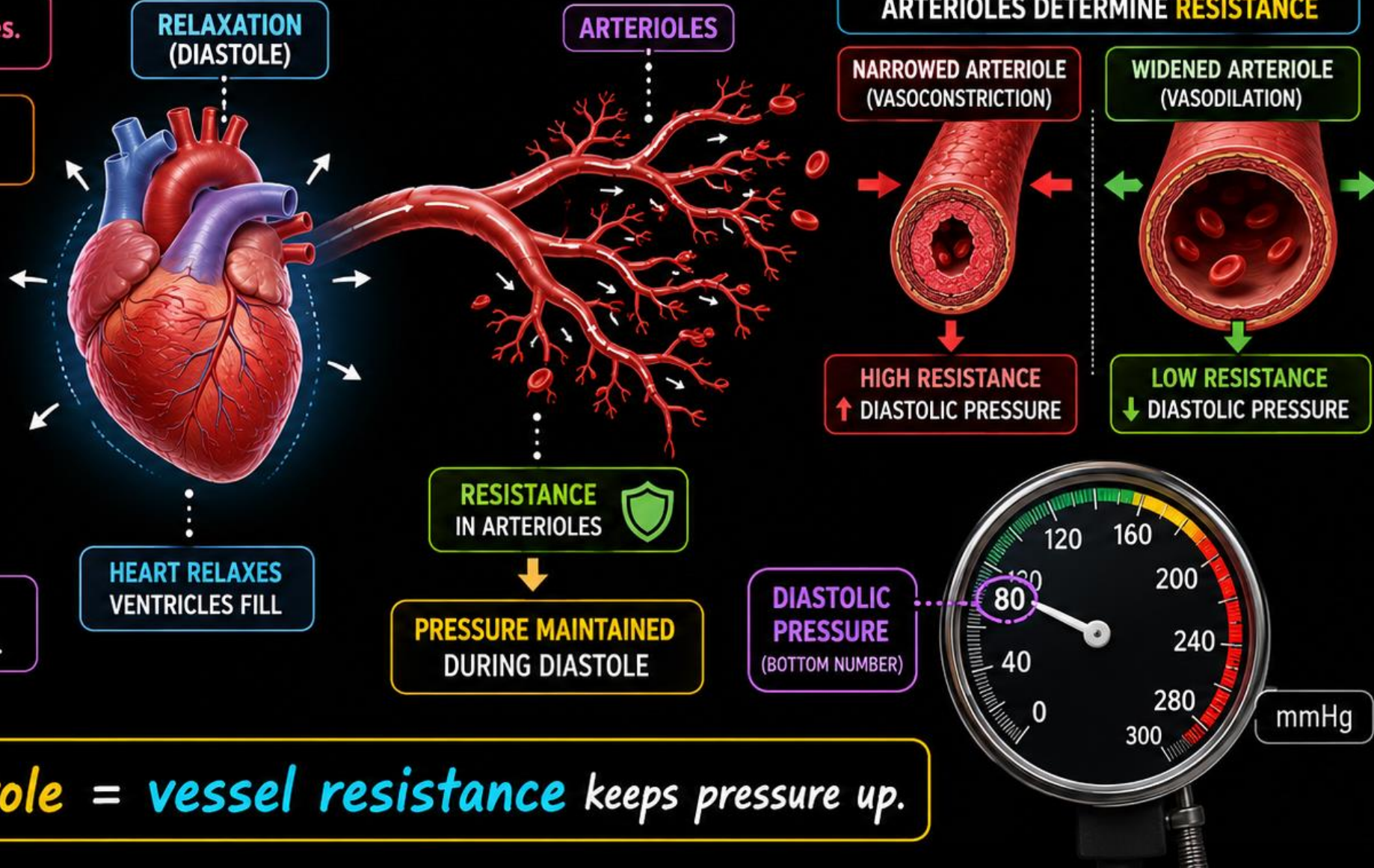
- Vasodilation **↓ diastolic pressure**.



- Best reflects **resistance** in small arteries / arterioles.



Think: **Diastole** = **vessel resistance** keeps pressure up.





@PharmacyIndia

13.

Essential hypertension is mainly associated with:

Essential (primary) HTN has no identifiable cause.

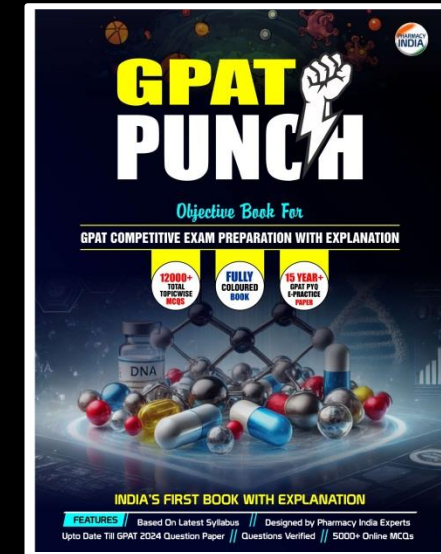
(a) Identifiable secondary causes

(b) Renal artery stenosis

Narrowing of the artery that supplies blood to the kidney.

(c) Genetic and environmental factors

(d) Pheochromocytoma



Download Pharmacy India Mobile App from Playstore



13. Essential hypertension is mainly associated with:

- (a) Identifiable secondary causes
- (b) Renal artery stenosis
- (c) Genetic and environmental factors**
- (d) Pheochromocytoma



Download Pharmacy India Mobile App from Playstore

Essential Hypertension = Primary (95%)

Mainly associated with **Genetic** & **Environmental** factors

Genetic Factors



- Family history
- Polygenic inheritance
- Gene variants affecting BP regulation

Environmental Factors



High salt



Unhealthy diet



Sedentary lifestyle



Stress



Obesity

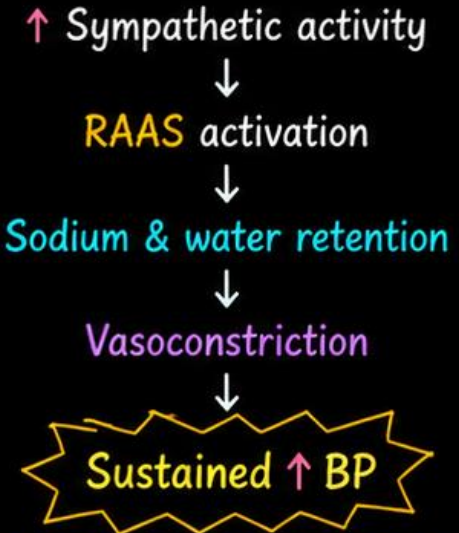


Smoking



Alcohol

How it leads to ↑ BP



How it is DIFFERENT from Secondary Hypertension



No identifiable secondary cause



Not due to renal artery stenosis



Not due to pheochromocytoma



Key Takeaway

Essential hypertension is mainly due to **genetic** predisposition + **lifestyle** & **environmental factors**, not due to any identifiable secondary cause.





@PharmacyIndia



14.

Left ventricular hypertrophy occurs mainly due to:

- (a) Decreased preload
- (b) Increased afterload
- (c) Reduced venous return
- (d) Mitral stenosis



Download Pharmacy India Mobile App from Playstore



14. Left ventricular hypertrophy occurs mainly due to:

- (a) Decreased preload
- (b) Increased afterload**
- (c) Reduced venous return
- (d) Mitral stenosis



Left Ventricular Hypertrophy occurs mainly due to Increased Afterload

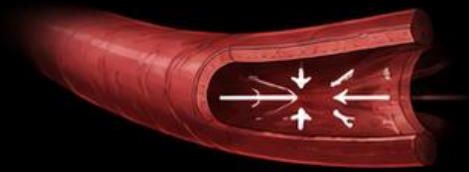
CAUSE

Increased Afterload

(↑ Resistance the LV must pump against)

Common causes:

- Systemic Hypertension
- Aortic Stenosis
- Coarctation of Aorta



High resistance

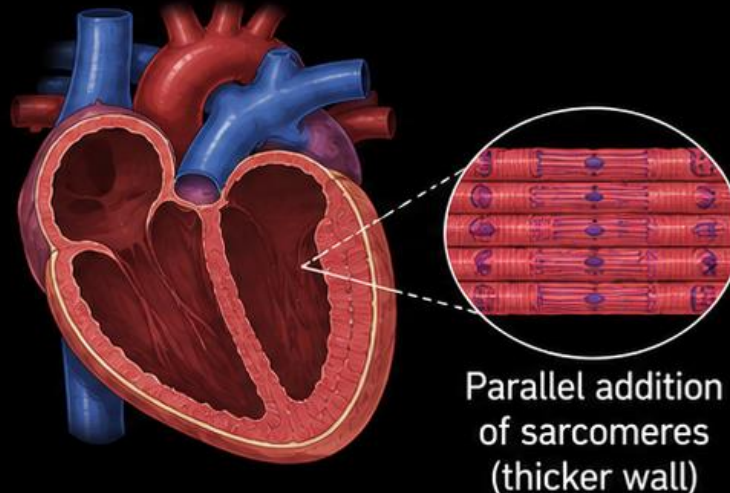
MECHANISM

Pressure Overload on LV

LV must generate more force to eject blood

Myocardial adaptation:

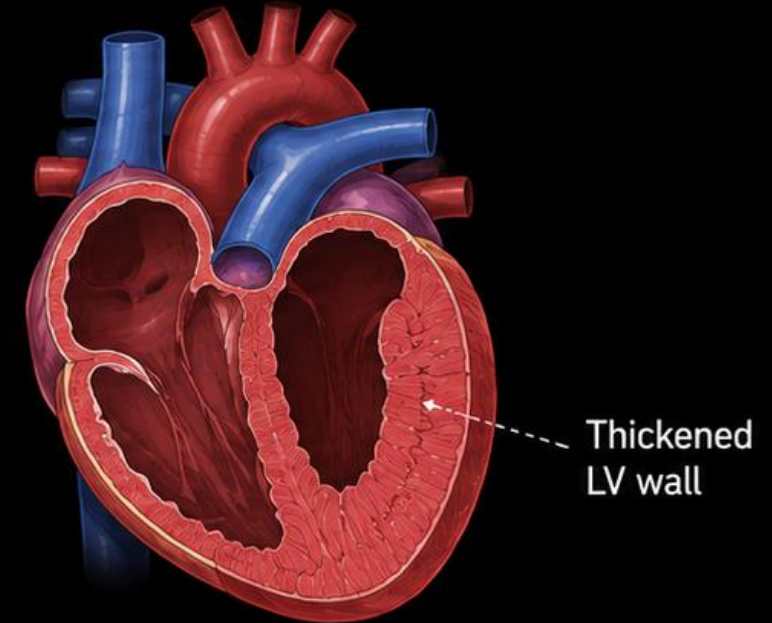
Sarcomeres added in parallel



RESULT

Left Ventricular Hypertrophy

Thickening of LV wall (Concentric Hypertrophy)



KEY POINT:

Afterload ↑ → Pressure overload ↑ → LV wall thickens → Left Ventricular Hypertrophy (Concentric Hypertrophy)

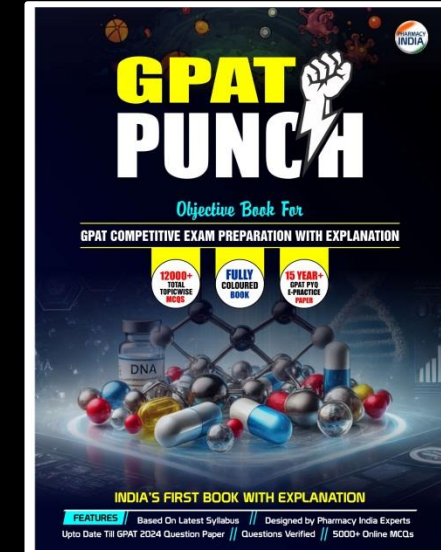


@PharmacyIndia



15. The earliest symptom of left-sided heart failure is:

- (a) Pedal edema
- (b) Nocturnal dyspnea
- (c) Exertional dyspnea
- (d) Jugular venous distension



Download Pharmacy India Mobile App from Playstore



15. The earliest symptom of left-sided heart failure is:

- (a) Pedal edema
- (b) Nocturnal dyspnea
- (c) Exertional dyspnea**
- (d) Jugular venous distension



LEFT-SIDED HEART FAILURE

— EARLIEST SYMPTOM: **EXERTIONAL DYSPNEA** ★

Left ventricle fails to pump effectively



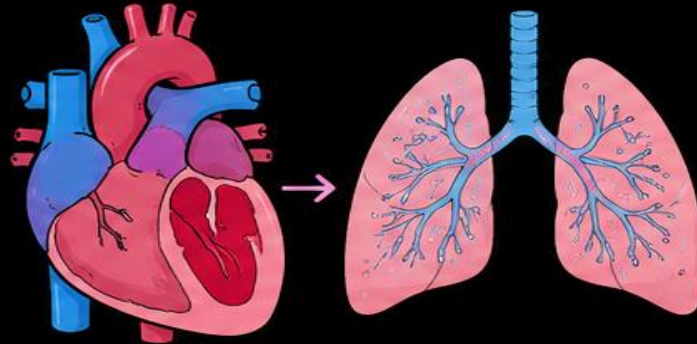
Blood backs up into left atrium & pulmonary veins



↑ Pulmonary capillary pressure



Pulmonary congestion



↑ Pressure → Fluid leaks into interstitium



Impaired gas exchange



Breathlessness

PROGRESSION OF DYSPNEA



1. EXERTIONAL DYSPNEA

Breathlessness on physical activity or exertion

EARLIEST SYMPTOM



2. ORTHOPNEA

Breathlessness when lying flat; relief on sitting up



3. NOCTURNAL DYSPNEA

Breathlessness that wakes from sleep



4. LATE SIGNS

Pedal edema, JVD, fatigue, reduced exercise tolerance

MECHANISM: ROSENOW'S CHAIN

LV Failure → ↑ LA Pressure → ↑ Pulmonary Pressure → Pulmonary Congestion → Dyspnea (Earliest on Exertion)



KEY POINT

Dyspnea on exertion is the **earliest symptom** in left-sided heart failure due to pulmonary congestion occurring first during increased activity.



@PharmacyIndia



16.

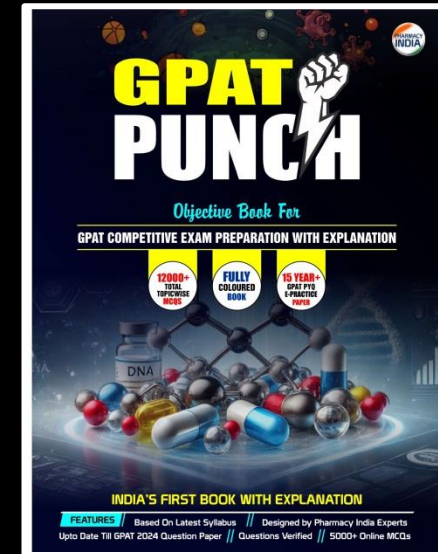
Right-sided heart failure primarily causes:

- (a) Pulmonary edema
- (b) Ascites and peripheral edema
- (c) Pink frothy sputum
- (d) Orthopnea

Ascites : Abnormal fluid accumulation in the abdomen, making the belly swollen.

Peripheral edema :Swelling in legs, feet, or hands due to fluid buildup in tissues.

Orthopnea: Difficulty in breathing while lying flat,



Download Pharmacy India Mobile App from Playstore



16. Right-sided heart failure primarily causes:

(a) Pulmonary edema

(b) Ascites and peripheral edema

(c) Pink frothy sputum

(d) Orthopnea

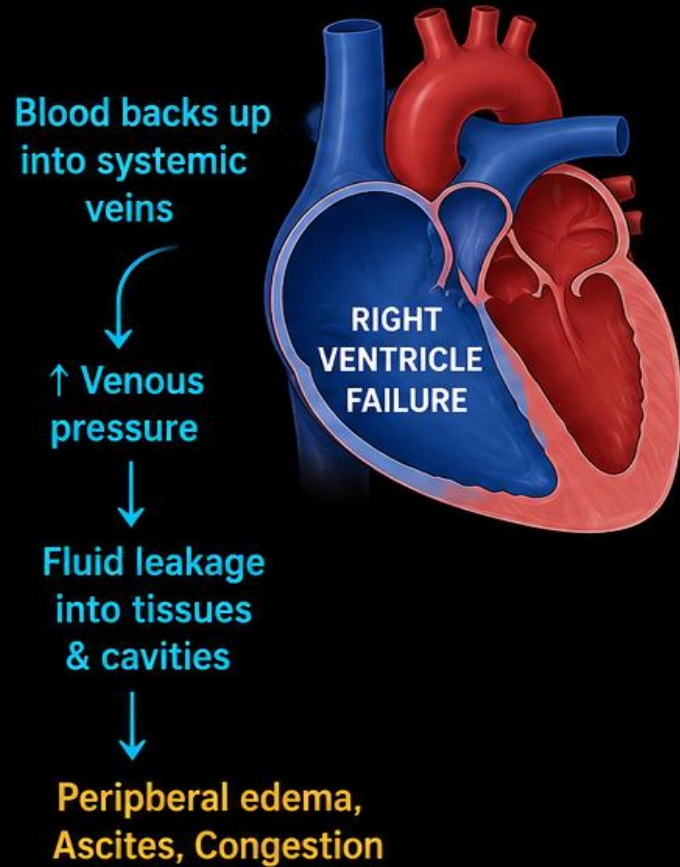


Download Pharmacy India Mobile App from Playstore

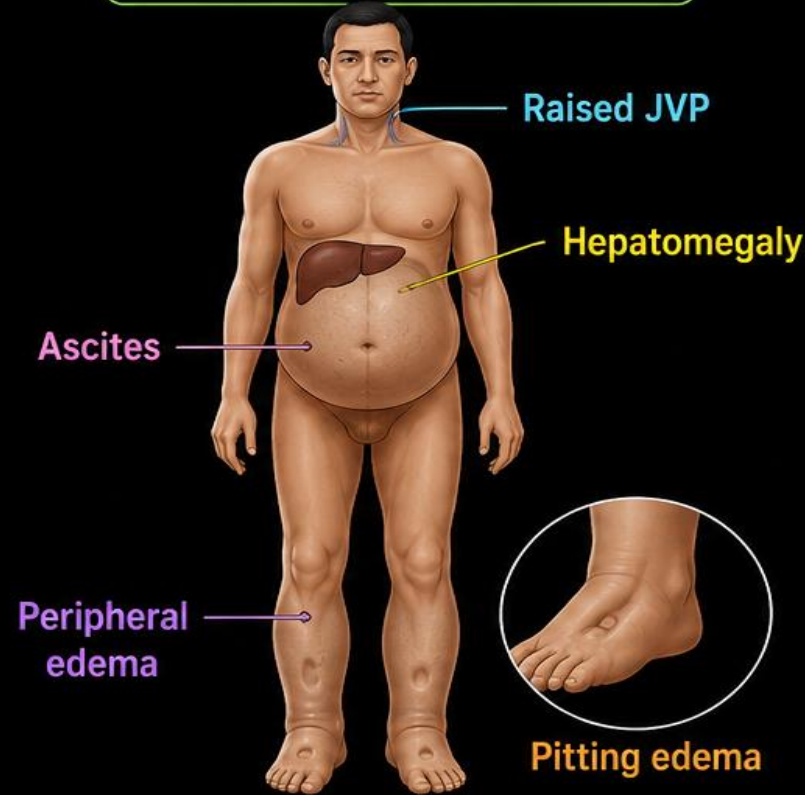
RIGHT-SIDED HEART FAILURE

Primary Effect → Systemic Venous Congestion

PATHOPHYSIOLOGY



RIGHT HEART FAILURE CLINICAL FEATURES



IN CONTRAST: LEFT HEART FAILURE (PULMONARY CONGESTION)



Pulmonary edema
Fluid in alveoli



Pink frothy sputum
Alveolar edema



Orthopnea
Breathlessness when lying flat



Dyspnea
Due to pulmonary congestion

KEY TAKEAWAY



RIGHT-SIDED HEART FAILURE → SYSTEMIC CONGESTION

Mainly causes: **ASCITES** and **PERIPHERAL EDEMA**



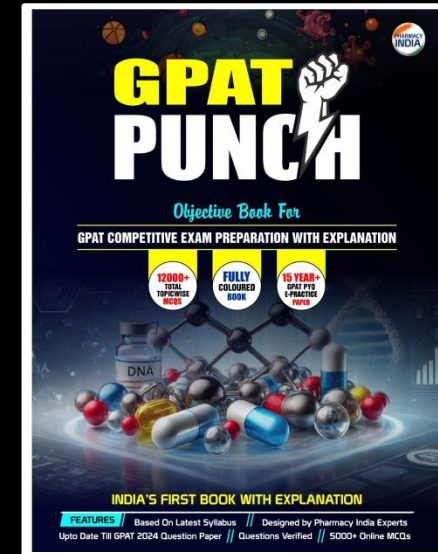
@PharmacyIndia

17.

Angina pectoris occurs primarily due to:

- (a) Increased myocardial oxygen supply
- (b) Reduced oxygen demand
- (c) Transient myocardial ischemia
- (d) Myocardial necrosis

Transient myocardial ischemia = Temporary decrease in blood flow to the heart muscle, causing reduced oxygen supply.



Download Pharmacy India Mobile App from Playstore



17. Angina pectoris occurs primarily due to:

(a) Increased myocardial oxygen supply

(b) Reduced oxygen demand

(c) Transient myocardial ischemia

(d) Myocardial necrosis



Download Pharmacy India Mobile App from Playstore

ANGINA PECTORIS

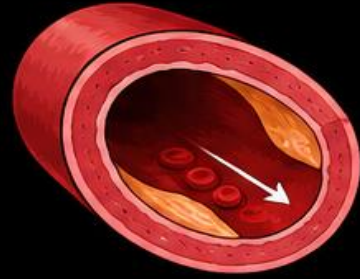
Occurs primarily due to: **TRANSIENT MYOCARDIAL ISCHEMIA**

1. CORONARY ATHEROSCLEROSIS



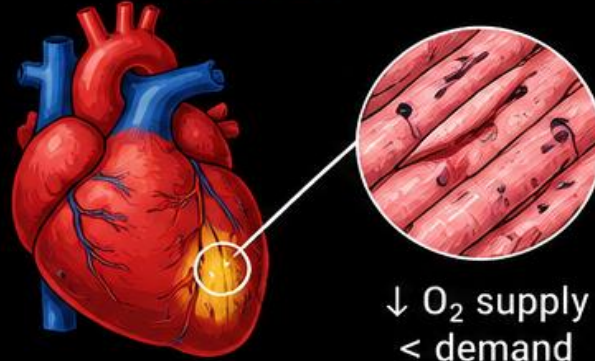
Plaque →
Narrowed artery

2. TRANSIENT REDUCTION IN BLOOD FLOW



↓ Coronary blood flow
(e.g., during exertion,
stress, cold)

3. TRANSIENT MYOCARDIAL ISCHEMIA



↓ O₂ supply
< demand

4. ANGINA (CHEST PAIN)



Substernal chest pain/pressure
(reversible on rest or GTN)

KEY POINTS

- ★ Angina pectoris = **Transient myocardial ischemia**
- ★ Caused by imbalance: **O₂ supply < O₂ demand**
- ★ **Reversible**; no myocardial necrosis
- ★ Triggered by exertion, stress, heavy meals, cold, smoking
- ★ Relieved by rest or **GTN** (vasodilation)

Ischemia is **temporary** → Pain is **temporary** → No **necrosis**

ANGINA vs MYOCARDIAL INFARCTION

ANGINA PECTORIS

- ✓ Transient ischemia
- ✓ Reversible
- ✓ No necrosis
- ✓ Pain relieved by rest/GTN

MYOCARDIAL INFARCTION

- ✗ Prolonged ischemia
- ✗ Irreversible
- ✗ Myocardial necrosis
- ✗ Pain not relieved by rest/GTN



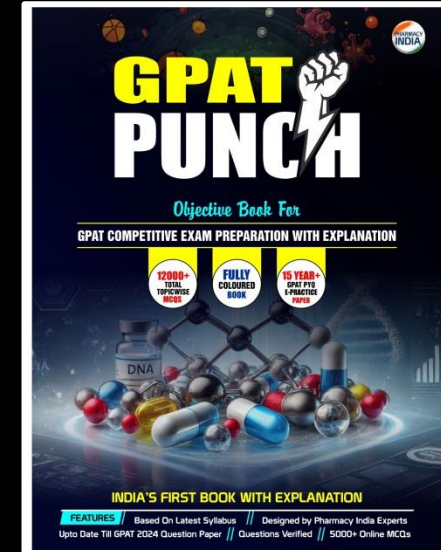
@PharmacyIndia



18.

Unstable angina is characterized by:

- (a) Predictable chest pain on exertion
- (b) Pain relieved by rest
- (c) Increasing frequency/duration of pain
- (d) Transient ST elevation



Download Pharmacy India Mobile App from Playstore



18.

Unstable angina is characterized by:

- (a) Predictable chest pain on exertion
- (b) Pain relieved by rest
- (c) Increasing frequency/duration of pain**
- (d) Transient ST elevation



Download Pharmacy India Mobile App from Playstore

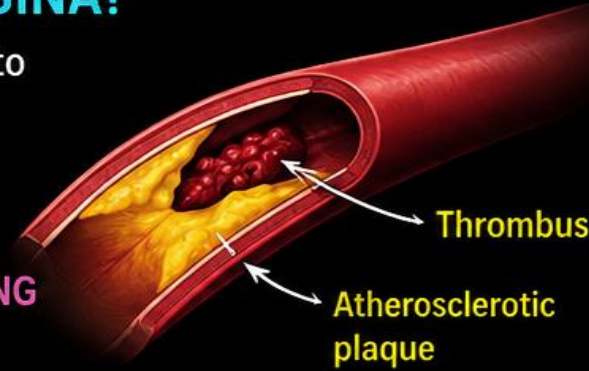
UNSTABLE ANGINA

CHARACTERIZED BY

INCREASING FREQUENCY / DURATION OF PAIN

WHAT IS UNSTABLE ANGINA?

- Acute coronary syndrome due to **partial & transient occlusion**
- **Plaque rupture** / erosion with superimposed thrombus
- Pain pattern is **NEW, WORSENING** or occurs at **REST**



UNSTABLE ANGINA – PAIN PATTERN

INCREASING FREQUENCY



More episodes than before

INCREASING DURATION



Each episode lasts longer

INCREASING SEVERITY



More severe pain

AT REST



Occurs even without exertion

UNSTABLE vs STABLE ANGINA

Feature	Stable Angina	Unstable Angina
Pattern	Predictable on exertion 	Increasing frequency / duration or at rest 
Duration	Usually < 10–15 min 	Longer, may be prolonged 
Relief	Relieved by rest or GTN 	May not be relieved by rest or GTN 
ECG	Normal or ST depression 	ST depression ± T inversion 
Risk	Lower	High – may progress to MI

KEY POINT



Unstable angina = **CHANGE** in pattern
Warns of **HIGH RISK** of Myocardial Infarction

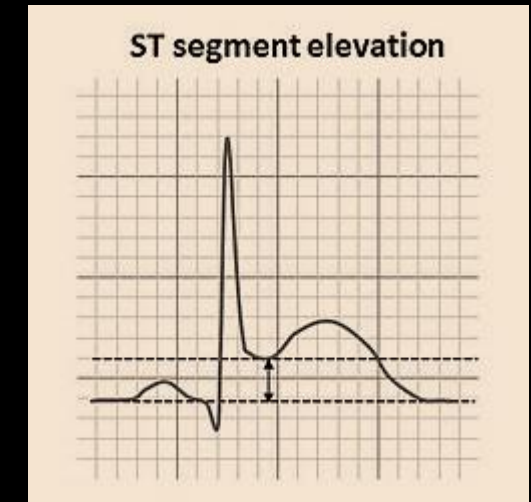


UNSTABLE ANGINA = WORSENING / NEW PAIN PATTERN = MEDICAL EMERGENCY



19. When the ECG shows ST-segment elevation, it usually means:

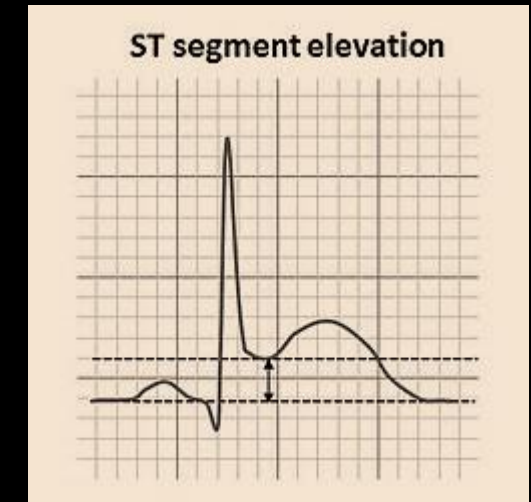
- (a) Only the inner layer of the heart is damaged**
- (b) Full-thickness damage of the heart muscle (transmural MI)**
- (c) A temporary spasm of a coronary artery**
- (d) Nothing abnormal**





19. When the ECG shows ST-segment elevation, it usually means:

- (a) Only the inner layer of the heart is damaged**
- (b) Full-thickness damage of the heart muscle (transmural MI)**
- (c) A temporary spasm of a coronary artery**
- (d) Nothing abnormal**



Transmural myocardial infarction (damage extends through the entire thickness of the heart wall).



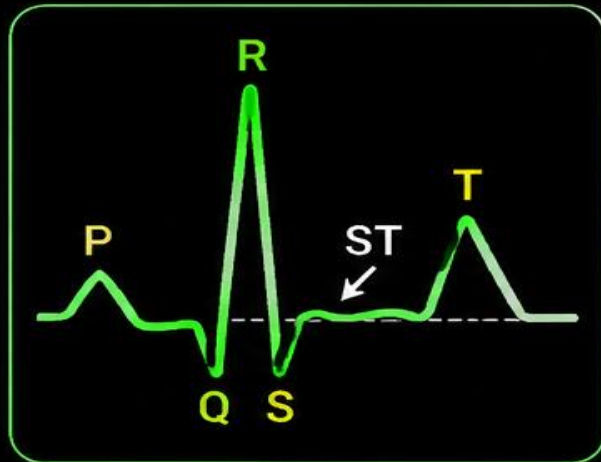
Download Pharmacy India Mobile App from Playstore

ST-SEGMENT ELEVATION

= TRANSMURAL MYOCARDIAL INFARCTION (STEMI)

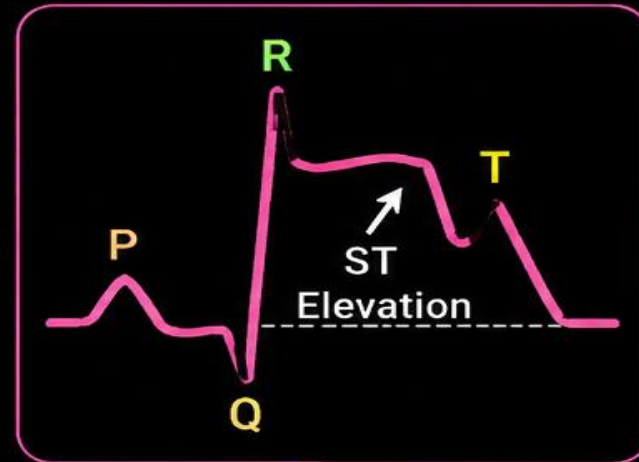
→ **ST-segment elevation** on ECG usually means **full-thickness (transmural)** damage of the heart muscle due to **acute coronary occlusion**.

NORMAL ECG



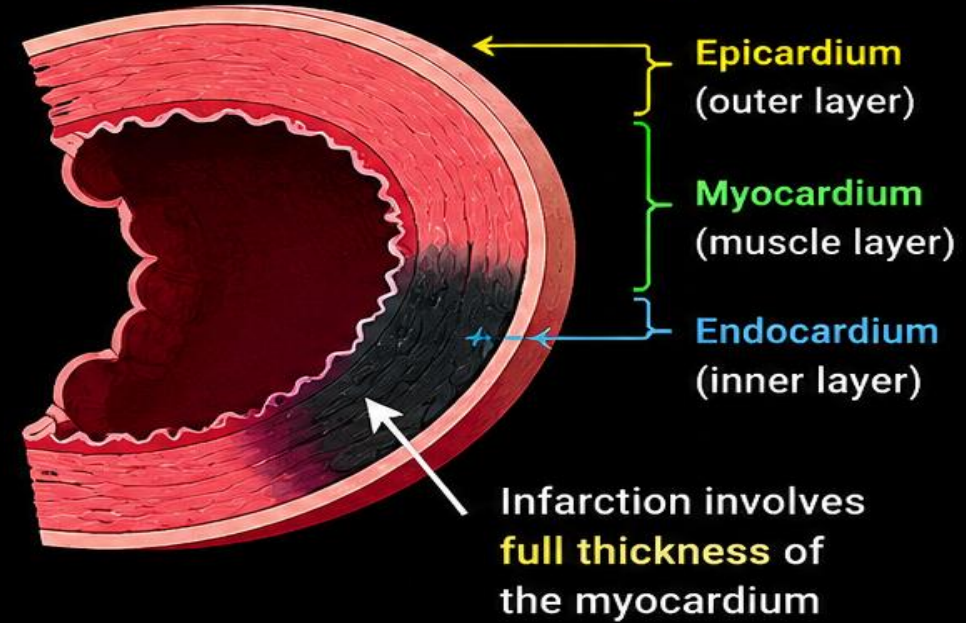
ST segment is on the **isoelectric line**

ST ELEVATION (STEMI)



ST segment is **elevated** above baseline

TRANSMURAL (FULL-THICKNESS) INFARCTION



WHY ST ELEVATION?



MEMORY:

ST Elevation = **STEMI**
= **Transmural MI**

★ Think: **Full Thickness Damage**

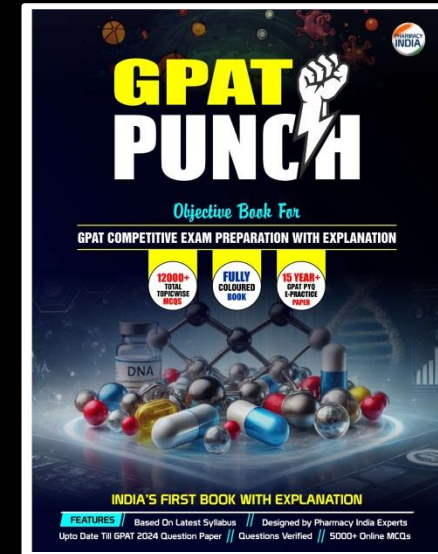


@PharmacyIndia



20. The earliest and most sensitive marker of myocardial infarction is:

- A. Creatine Kinase - Myocardial Band
- B. Aspartate Aminotransferase
- C. Troponin I
- D. Lactate Dehydrogenase



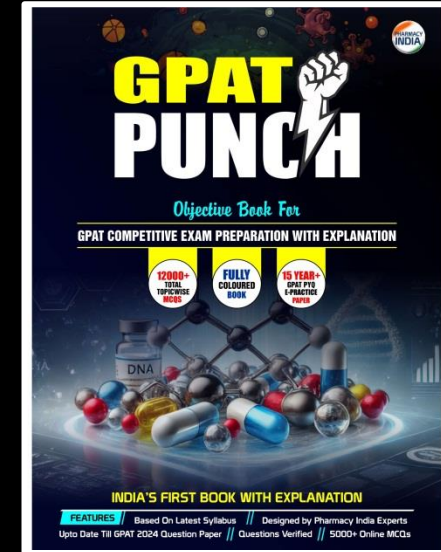
Download Pharmacy India Mobile App from Playstore



@PharmacyIndia

20. The earliest and most sensitive marker of myocardial infarction is:

- A. Creatine Kinase - Myocardial Band
- B. Aspartate Aminotransferase
- C. Troponin I
- D. Lactate Dehydrogenase



Download Pharmacy India Mobile App from Playstore

EARLIEST & MOST SENSITIVE MARKER OF MYOCARDIAL INFARCTION



TROPONIN I is the earliest and most sensitive marker for myocardial infarction.



Rises
3–6 hrs

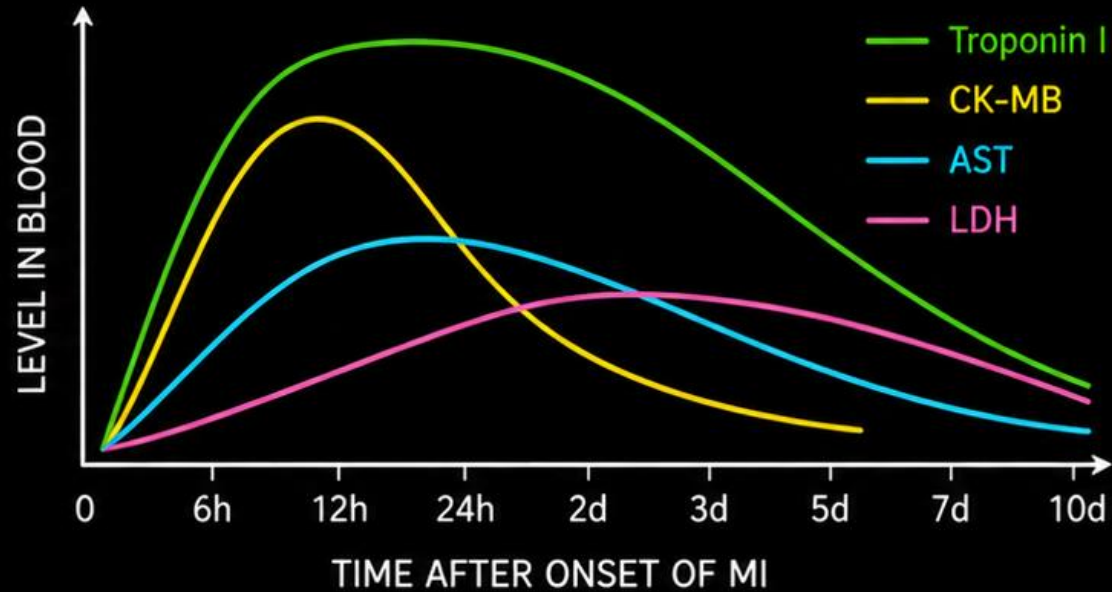


Highest
sensitivity



Remains
elevated
7–10 days

RELEASE KINETICS IN MI



COMPARISON OF MARKERS

 MARKER	 RISES	 PEAK	 RETURNS TO NORMAL
Troponin I	3–6 hrs	12–24 hrs	7–10 days
CK-MB	3–6 hrs	18–24 hrs	2–3 days
AST	6–12 hrs	24–36 hrs	3–5 days
LDH	12–24 hrs	2–3 days	7–10 days



Troponin I is specific to cardiac muscle and detects even **minimal** myocardial injury earlier than other markers.



KEY POINT

Troponin I = Earliest rise + Highest sensitivity
= Best marker for MI diagnosis



@PharmacyIndia



21.

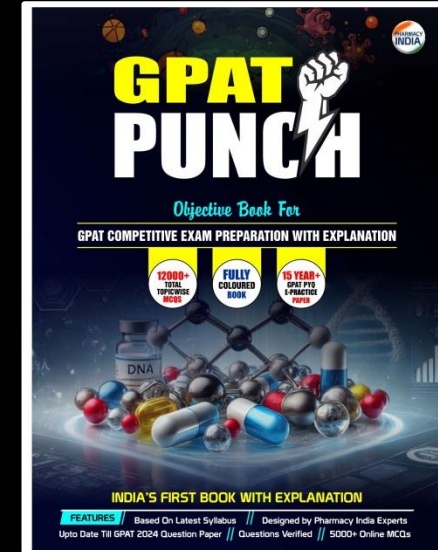
Cardiac output is determined by:

(a) Stroke volume $\times$ Heart rate

(b) Preload $\times$ Afterload

(c) Blood pressure $\times$ Heart rate

(d) Contractility $\times$ TPR



Download Pharmacy India Mobile App from Playstore



21. Cardiac output is determined by:

(a) Stroke volume $\times$ Heart rate

(b) Preload $\times$ Afterload

(c) Blood pressure $\times$ Heart rate

(d) Contractility $\times$ TPR



Download Pharmacy India Mobile App from Playstore

CARDIAC OUTPUT

• is determined by •

$$\text{CARDIAC OUTPUT (CO)} = \text{STROKE VOLUME (SV)} \times \text{HEART RATE (HR)}$$

1. STROKE VOLUME (SV)

Amount of blood pumped by each ventricle per beat

Determined by:

Preload



(EDV)

Afterload



(Arterial resistance)

Contractility



(Inotropy)



Normal Range

4 – 8 L/min



2. HEART RATE (HR)

Number of heartbeats per minute

Influenced by:



Parasympathetic
(↓ HR)



Sympathetic
(↑ HR)



Exercise,
stress, hormones

• KEY POINT •



Cardiac Output = Stroke Volume × Heart Rate



Reflects the ability of the heart to pump blood to the body.



Increase in either SV or HR increases CO.



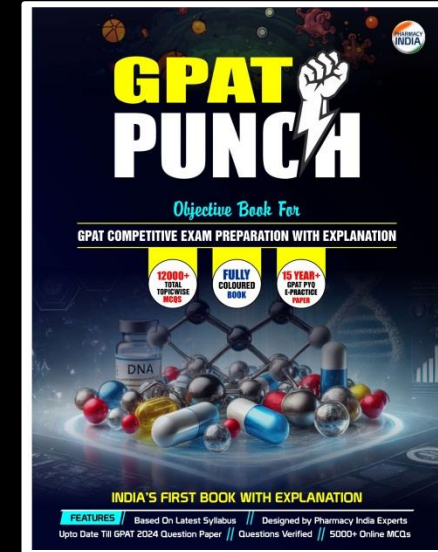
@PharmacyIndia



22.

Increased preload primarily leads to:

- (a) Decreased stroke volume
- (b) Reduced contractility
- (c) Decreased ejection fraction
- (d) Increased stroke volume



Download Pharmacy India Mobile App from Playstore



22. Increased preload primarily leads to:

- (a) Decreased stroke volume
- (b) Reduced contractility
- (c) Decreased ejection fraction
- (d) Increased stroke volume



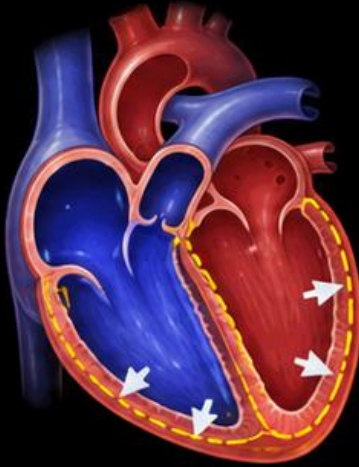
Increased Preload → Increased Stroke Volume

★ **Preload** = Initial stretching of **ventricular fibers** at **end-diastole**
→ Determined by **venous return** / **EDV**

↑ **Preload**
↑ Venous return



↑ **EDV**
(End-diastolic volume)



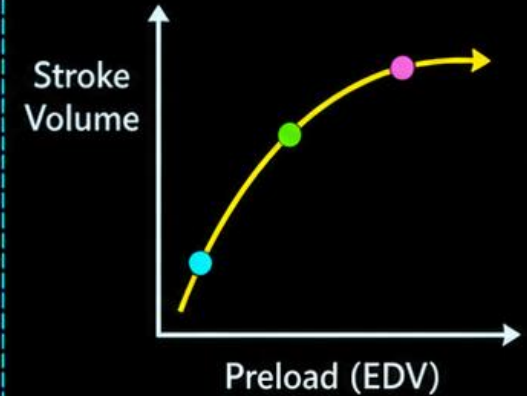
↑ **Myocardial stretch**
(Frank–Starling)



↑ **Stroke Volume**
(More blood ejected)



Frank–Starling Law



Key Point:

↑ **Preload** → ↑ **EDV** → ↑ **Myocardial stretch** → ↑ **Stroke Volume**
(Via Frank–Starling Mechanism)

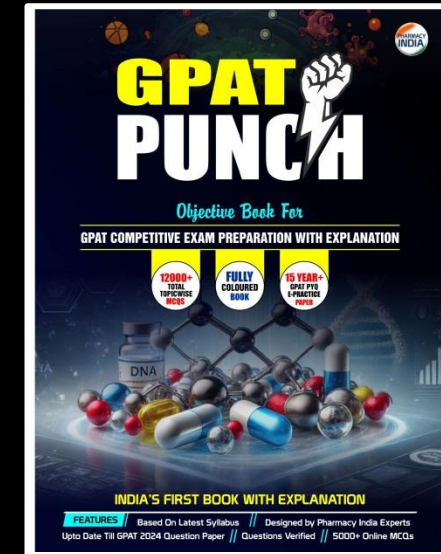


@PharmacyIndia



23. Atrial fibrillation is characterized by: (GPAT - 2024)

- (a) Regular rhythm
- (b) Absence of P waves
- (c) Tall peaked P waves
- (d) Prolonged PR interval



Download Pharmacy India Mobile App from Playstore



23. Atrial fibrillation is characterized by: (GPAT - 2024)

(a) Regular rhythm

(b) Absence of P waves

(c) Tall peaked P waves

(d) Prolonged PR interval



Download Pharmacy India Mobile App from Playstore



Atrial Fibrillation – Key Explanation



1



No distinct
P waves

2



Irregularly
irregular rhythm

3



Chaotic
atrial activity

4



Fibrillatory baseline
replaces normal
P waves

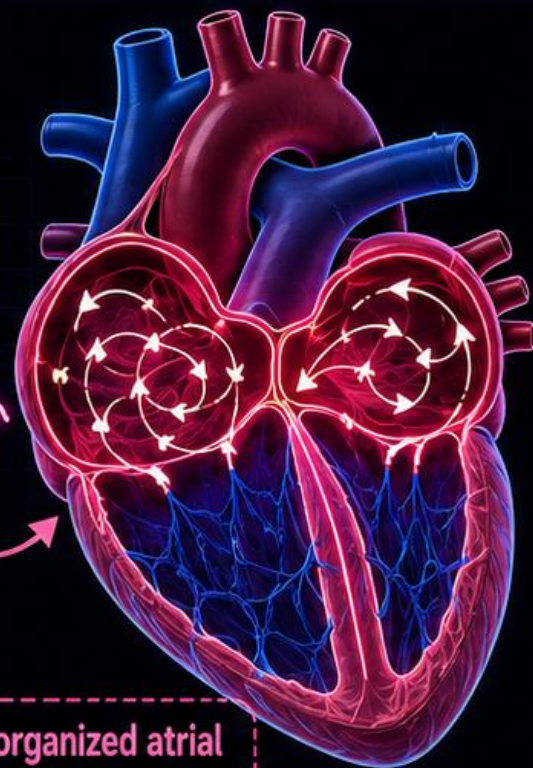
ECG FINDINGS

Irregular R-R intervals

No P waves

f waves / fibrillatory activity

Disorganized atrial
electrical impulses



Quick clue:

AF = absent P waves + irregular rhythm



Not **regular** rhythm



Not prolonged **PR interval**

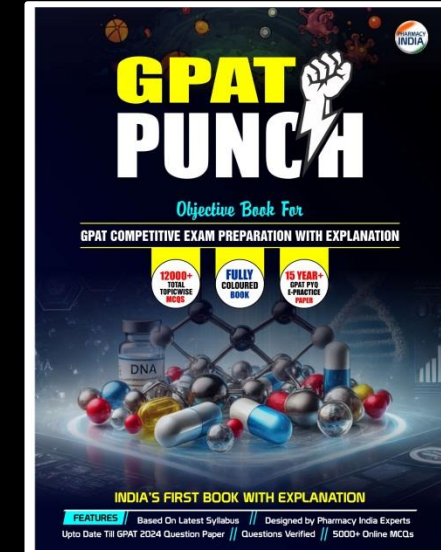


@PharmacyIndia



24. Ventricular tachycardia is dangerous because:

- (a) It reduces ventricular filling time
- (b) It increases cardiac output
- (c) It improves coronary perfusion
- (d) It decreases myocardial oxygen demand



Download Pharmacy India Mobile App from Playstore



24. Ventricular tachycardia is dangerous because:

- (a) It reduces ventricular filling time**
- (b) It increases cardiac output**
- (c) It improves coronary perfusion**
- (d) It decreases myocardial oxygen demand**



Download Pharmacy India Mobile App from Playstore

Ventricular Tachycardia is dangerous because:



What is Ventricular Tachycardia (VT)?

- Rapid abnormal rhythm from ventricles (~120–250 bpm)



Why it's dangerous?

↓ Very short diastole → ↓ ventricular filling time (↓ preload)



↓ Stroke volume → ↓ Cardiac output (hypotension, shock)

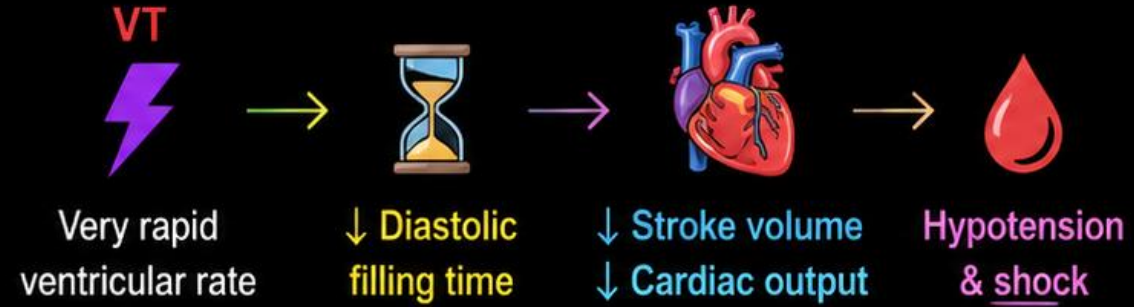


↓ Coronary perfusion (coronary flow occurs in diastole)



↑ Myocardial oxygen demand (tachycardia) + ↓ Supply → Ischemia

Hemodynamic Impact



Normal vs VT (at same overall time)

Normal
(60–80 bpm)



VT
(120–250 bpm)



Very short diastole



Result: ↓ Cardiac output + ↓ Coronary perfusion + ↑ Oxygen demand = Ischemia, Hypotension, Heart failure, Sudden death

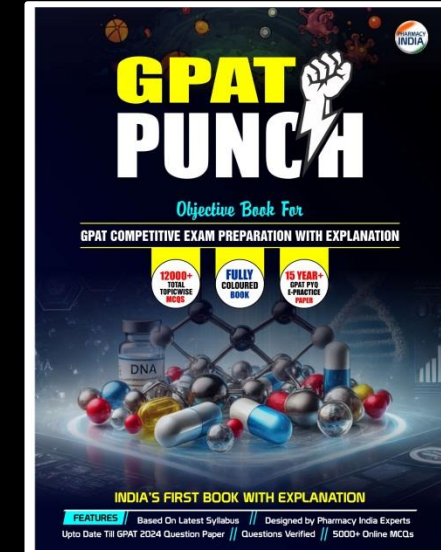


@PharmacyIndia



25. The most common cause of left-sided heart failure is:

- (a) Pulmonary hypertension
- (b) Systemic hypertension
- (c) COPD
- (d) Pulmonary embolism



Download Pharmacy India Mobile App from Playstore



25. The most common cause of left-sided heart failure is:

(a) Pulmonary hypertension

(b) Systemic hypertension

(c) COPD

(d) Pulmonary embolism



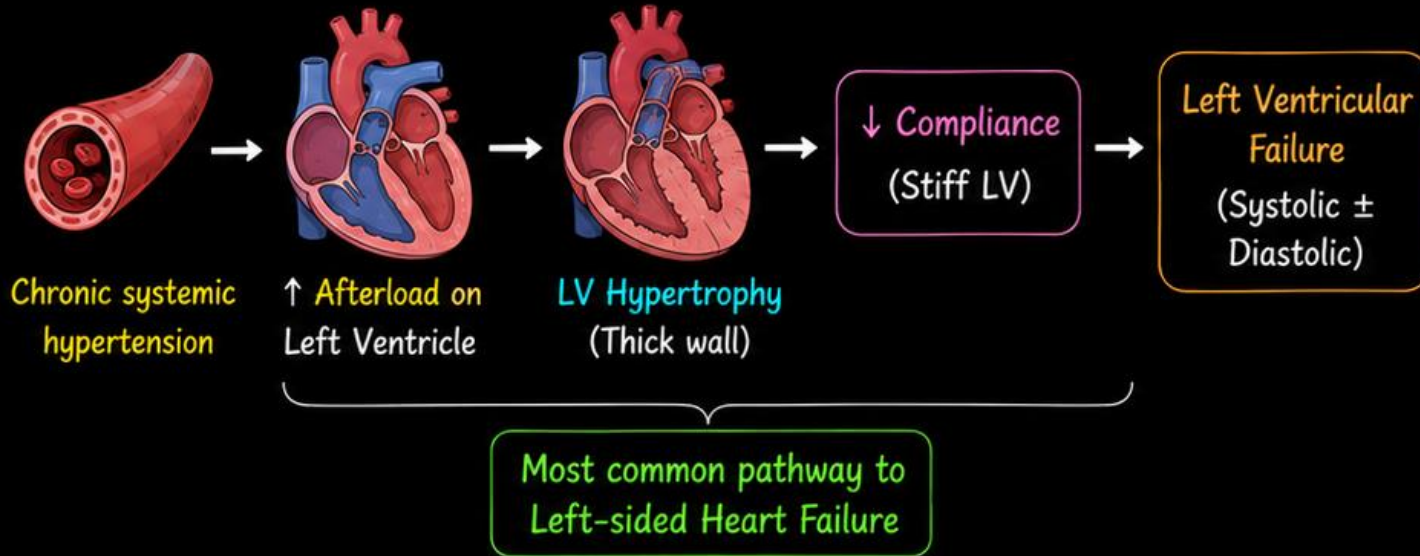
Download Pharmacy India Mobile App from Playstore

Most common cause of Left-Sided Heart Failure

SYSTEMIC HYPERTENSION

Why?

Chronic ↑ systemic BP → Increased **afterload** on **Left Ventricle**
→ **LV hypertrophy** → ↓ **compliance** → **LV failure**



Why others are less common causes?



Pulmonary Hypertension

→ Mainly causes **Right-sided** heart failure



COPD

→ Leads to **Right-sided** heart failure (cor pulmonale)



Pulmonary Embolism

→ Acute **Right** ventricular failure (not left-sided)



Systemic Hypertension

→ Directly increases **afterload** on LV → **Left-sided** failure



Key Takeaway

Systemic hypertension is the most common cause of **left-sided** heart failure due to chronic ↑ **afterload** → **LV hypertrophy** → ↓ **compliance** → **LV failure**.





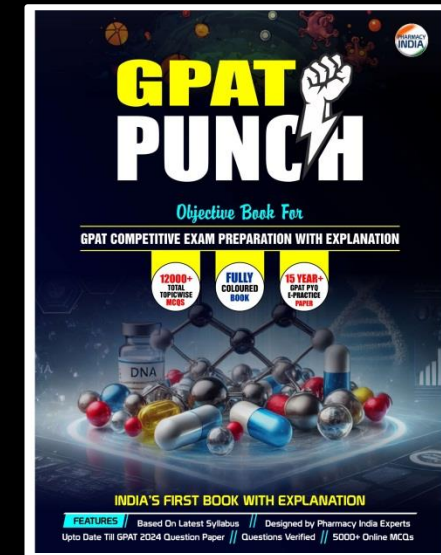
@PharmacyIndia

26.

Fluid accumulation in lungs in left-sided heart failure is due to:

- (a) decreased oncotic pressure
- (b) increased vascular resistance
- (c) lymphatic obstruction
- (d) increased pulmonary capillary hydrostatic pressure

- **Oncotic pressure** is the **pulling force** created by **plasma proteins (mainly albumin)** in the blood, which **draws water into the bloodstream** and prevents fluid from leaking into tissues.
- **Lymphatic obstruction** : **Blockage of the lymphatic vessels**



Download Pharmacy India Mobile App from Playstore



- 26. Fluid accumulation in lungs in left-sided heart failure is due to:**
- (a) decreased oncotic pressure**
 - (b) increased vascular resistance**
 - (c) lymphatic obstruction**
 - (d) increased pulmonary capillary hydrostatic pressure**



LEFT-SIDED HEART FAILURE: WHY FLUID ACCUMULATES IN LUNGS

↑ Pulmonary capillary hydrostatic pressure

Mechanism



LV pump failure



Pulmonary venous congestion



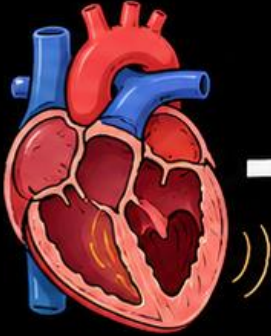
↑ Hydrostatic pressure



Transudation of fluid into alveoli

①

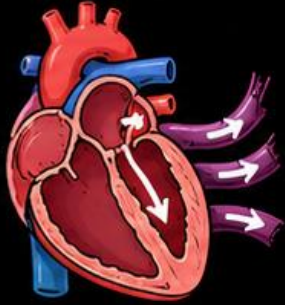
Left ventricle failure (weak left heart)



Reduced LV pumping ability

②

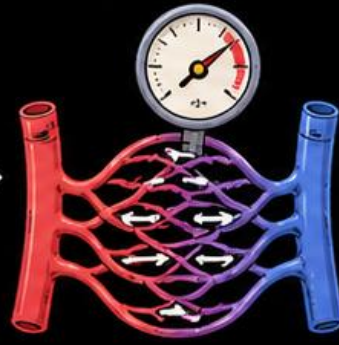
Blood backs up into left atrium and pulmonary veins



Pulmonary venous congestion

③

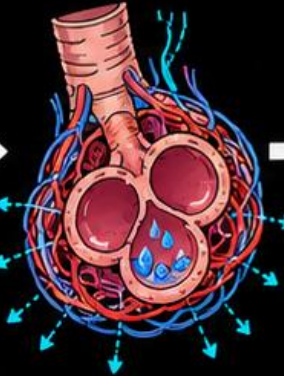
Pulmonary capillary pressure rises



↑ Hydrostatic pressure in pulmonary capillaries

④

Fluid leaks from capillaries into interstitium and alveoli



Fluid moves out due to ↑ pressure

⑤

Pulmonary edema / breathlessness



Fluid in alveoli
Impaired gas exchange
→ Breathlessness

Not mainly due to



Decreased oncotic pressure



Lymphatic obstruction



General vascular resistance



KEY TAKEAWAY

In left-sided heart failure, lung fluid buildup occurs because blood backs up into pulmonary circulation, raising pulmonary capillary hydrostatic pressure and pushing fluid into alveoli.





@PharmacyIndia

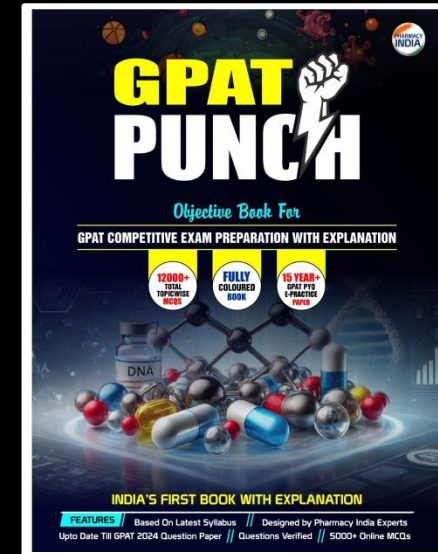


27.

Cardiogenic shock is caused by:

- (a) Severe blood loss
- (b) Myocardial infarction
- (c) Allergic reaction
- (d) Sepsis

Cardiogenic shock is a life-threatening condition where the **heart fails to pump enough blood**, leading to **severe drop in blood pressure** and **inadequate supply of oxygen** to organs.



Download Pharmacy India Mobile App from Playstore



27.

Cardiogenic shock is caused by:

(a) Severe blood loss

(b) Myocardial infarction

(c) Allergic reaction

(d) Sepsis



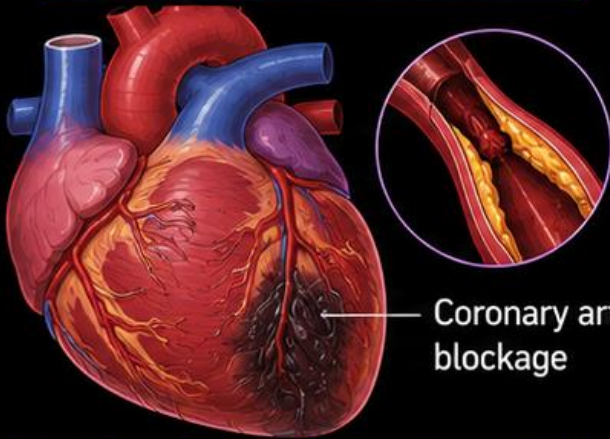
Download Pharmacy India Mobile App from Playstore

Cardiogenic Shock is caused by Myocardial Infarction

CAUSE

Myocardial Infarction

Acute severe damage to heart muscle



Ischemia → Infarction → Pump Failure

MECHANISM



Severe ↓ in Myocardial Contractility



↓ Cardiac Output



↓ Tissue Perfusion



Organ Dysfunction



Shock

RESULT

Clinical Features

- Hypotension
- Cold, clammy skin
- Weak pulse
- Altered mental status
- Low urine output



Cardiogenic shock = Pump failure
due to heart muscle failure (most commonly due to **Myocardial Infarction**)

WHY OTHER OPTIONS ARE INCORRECT?



Severe blood loss

Causes hypovolemic shock, not cardiogenic shock



Allergic reaction

Causes anaphylactic shock



Sepsis

Causes distributive (septic) shock



Key Point

Cardiogenic shock is due to failure of the heart to pump effectively, most commonly due to **Myocardial Infarction**.

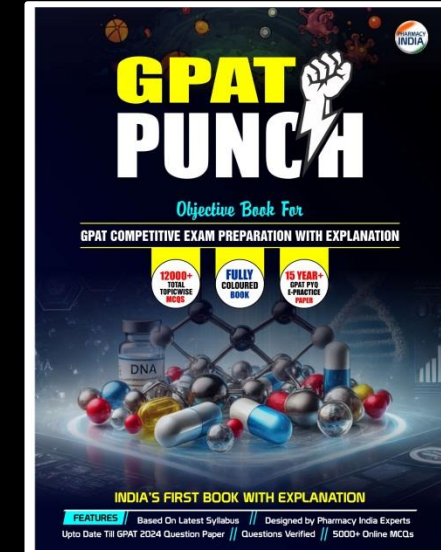


@PharmacyIndia



28. A key ECG change in ischemia (not infarction) is:

- (a) ST elevation
- (b) Pathological Q waves
- (c) ST depression
- (d) T wave inversion only



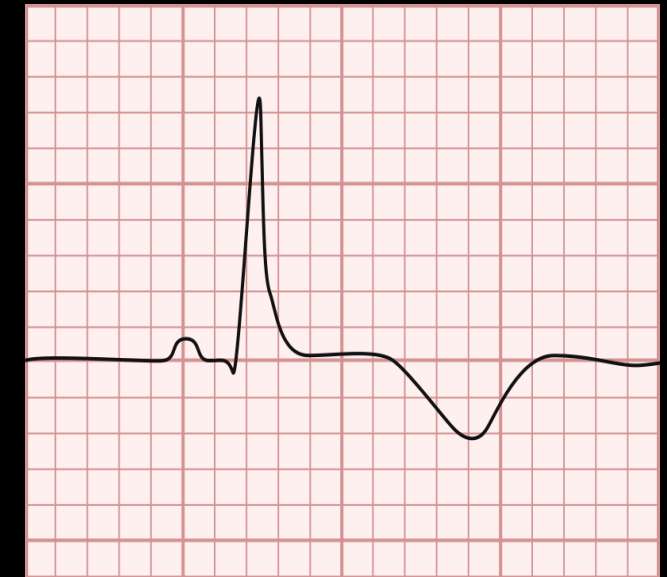
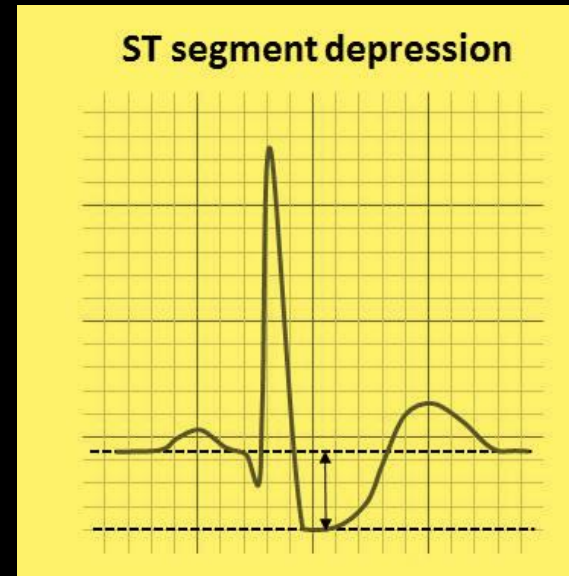
Download Pharmacy India Mobile App from Playstore



28. A key ECG change in ischemia (not infarction) is:

- (a) ST elevation
- (b) Pathological Q waves
- (c) ST depression**
- (d) T wave inversion only

Pathological Q waves are abnormally deep and wide Q waves seen on ECG,



T wave inversion



Download Pharmacy India Mobile App from Playstore



ECG IN ISCHEMIA

KEY FINDING: ST DEPRESSION

★ Typical in Angina / Subendocardial Ischemia

ISCHEMIA ≠ INFARCTION

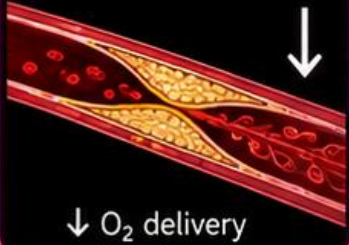
Ischemia = ↓ O₂ supply
(reversible)

Infarction = cell death
(usually irreversible)

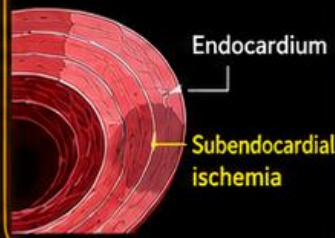


WHY DOES ST DEPRESSION OCCUR?

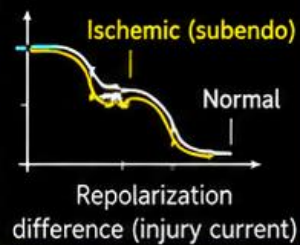
1. REDUCED CORONARY BLOOD FLOW



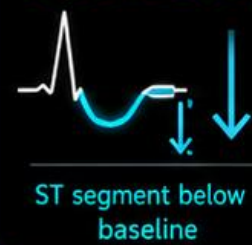
2. SUBENDOCARDIAL ISCHEMIA



3. ALTERED REPOLARIZATION



4. ECG CHANGE ST DEPRESSION



ISCHEMIA vs INFARCTION

	ISCHEMIA	INFARCTION
ST SEGMENT	 DEPRESSION (classic)	 ELEVATION (typical)
Q WAVES	 ABSENT	 PATHOLOGIC Q WAVES (necrosis clue)
T WAVES	 T-WAVE INVERSION May occur (nonspecific)	 Often present (evolves over time)



BOTTOM LINE:

ST DEPRESSION = Ischemia

ST ELEVATION & PATHOLOGIC Q WAVES = Infarction

CLINICAL CONTEXT



Exertional chest pain (angina)



Pain relieved by rest/GTN



Demand ischemia



ECG may normalize when ischemia resolves



HIGH-YIELD PEARL: ST DEPRESSION = Classic sign of ischemia (especially subendocardial). Think: Demand ischemia, stable/unstable angina, NSTEMI.

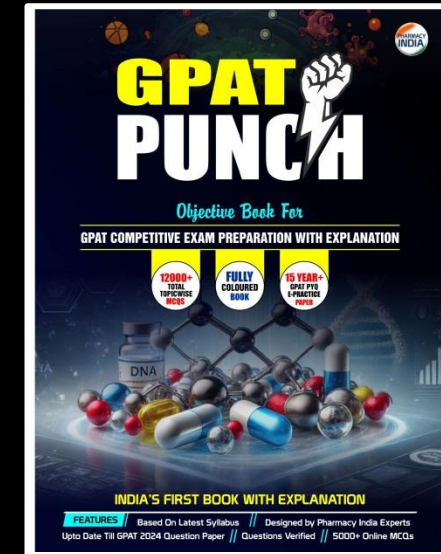


@PharmacyIndia



29. The zone of necrosis in MI occurs due to:

- (a) Increased oxygen supply
- (b) Prolonged ischemia
- (c) Vasodilation
- (d) Increased contractility



Download Pharmacy India Mobile App from Playstore



29. The zone of necrosis in MI occurs due to:

(a) Increased oxygen supply

(b) Prolonged ischemia

(c) Vasodilation

(d) Increased contractility



Download Pharmacy India Mobile App from Playstore

ZONE OF NECROSIS IN MI

Develops due to prolonged ischemia

1



Coronary artery **blockage** reduces blood flow

2



Prolonged **ischemia** = severe **O₂** deprivation

3



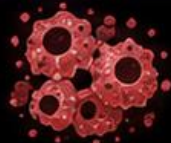
ATP falls, cells fail, membrane **damage** occurs

4



Irreversible injury leads to myocyte **necrosis**

5

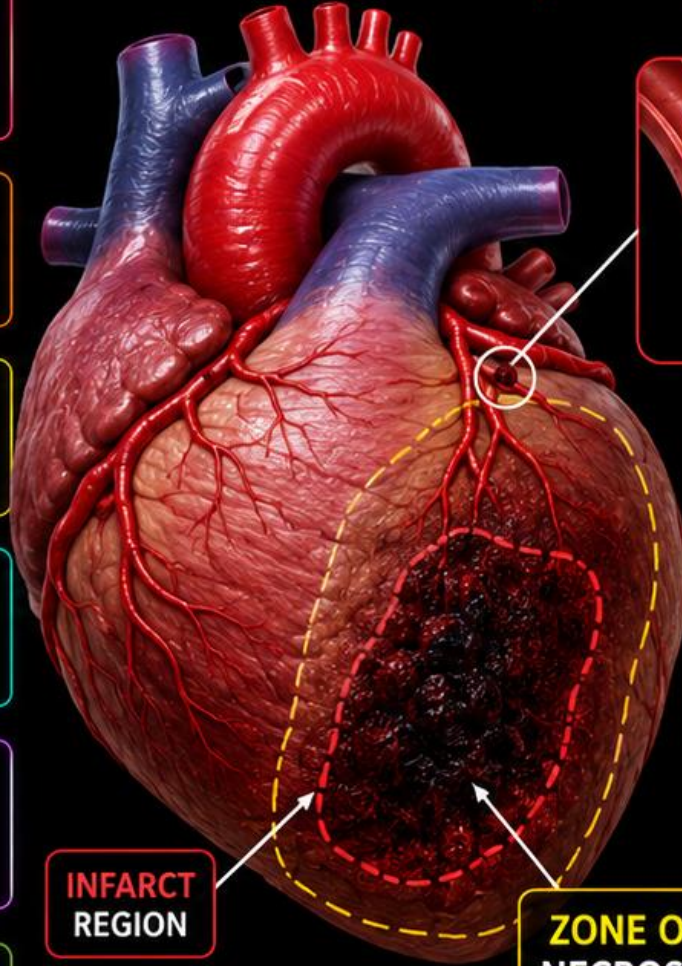


Necrotic core forms in the infarcted area

6

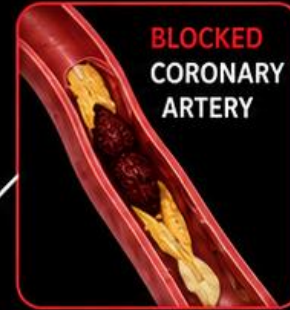


If blood flow is **restored early**, surrounding tissue may **survive**



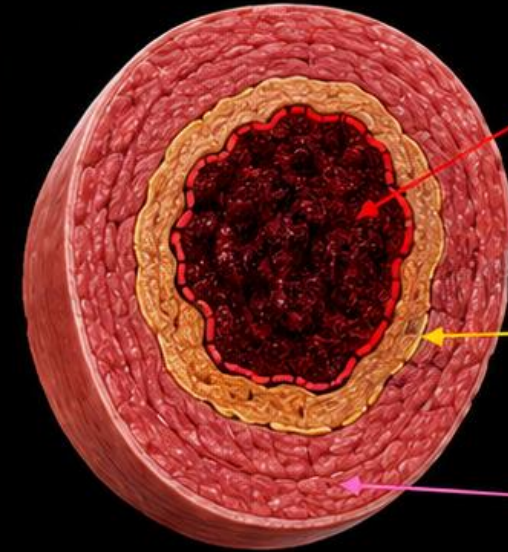
INFARCT REGION

ZONE OF NECROSIS



BLOCKED CORONARY ARTERY

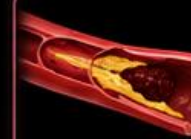
CROSS-SECTION OF MYOCARDIUM



NECROTIC CORE
Irreversibly damaged tissue (cell death)

ISCHEMIC BUT POTENTIALLY SALVAGEABLE AREA (PENUMBRA)
At risk, may survive if reperfused early

NORMAL VIABLE MYOCARDIUM
Adequate blood supply



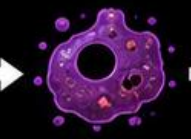
BLOCKAGE



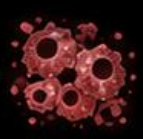
ISCHEMIA



ATP DEPLETION



IRREVERSIBLE INJURY



NECROSIS



KEY IDEA: Necrosis in MI appears when ischemia is **prolonged** enough to cause **irreversible cell death**.



@PharmacyIndia



30.

The “pressure overload” heart failure results in:

- (a) Concentric hypertrophy
- (b) Eccentric hypertrophy
- (c) Dilated cardiomyopathy
- (d) Restrictive cardiomyopathy



Download Pharmacy India Mobile App from Playstore



30. The “pressure overload” heart failure results in:

(a) Concentric hypertrophy Ventricular walls thicken, chamber becomes small.

(b) Eccentric hypertrophy

(c) Dilated cardiomyopathy

(d) Restrictive cardiomyopathy



Download Pharmacy India Mobile App from Playstore

PRESSURE OVERLOAD → CONCENTRIC HYPERTROPHY



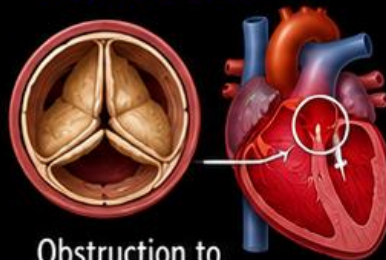
PRESSURE OVERLOAD = HEART PUMPS AGAINST **HIGH RESISTANCE**

COMMON CAUSES

HYPERTENSION (Systemic)

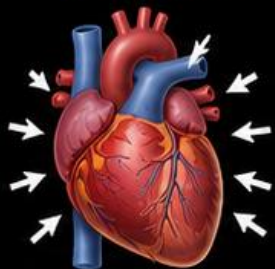


AORTIC STENOSIS



Obstruction to LV outflow

1. PRESSURE OVERLOAD



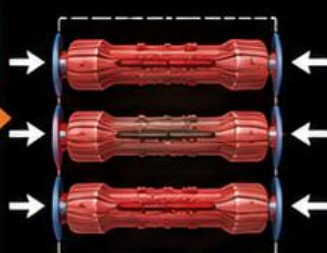
Heart pumps against **high resistance**

2. INCREASED AFTERLOAD



LV must generate more pressure

3. SARCOMERES ADDED IN PARALLEL



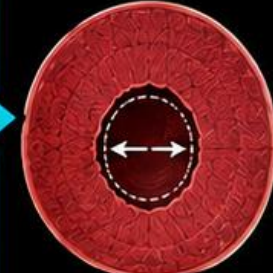
More sarcomeres **side-by-side** (not end-to-end)

4. WALL THICKENING



LV wall becomes **thick**

5. SMALLER CHAMBER LUMEN



Chamber size **decreases**

6. CONCENTRIC HYPERTROPHY



Pattern = **concentric hypertrophy**

LATER MAY IMPAIR FILLING

- ↓ Compliance (stiff ventricle)
- Impaired diastolic relaxation
- May lead to **DIASTOLIC DYSFUNCTION**

LEFT VENTRICLE: CROSS-SECTION COMPARISON

NORMAL

Normal wall thickness

Large chamber lumen

CONCENTRIC HYPERTROPHY

Thickened wall

Smaller chamber lumen



Common in **HYPERTENSION** & **AORTIC STENOSIS**



KEY IDEA: HIGH PRESSURE LOAD MAKES THE VENTRICLE **THICKER**, **NOT WIDER**.

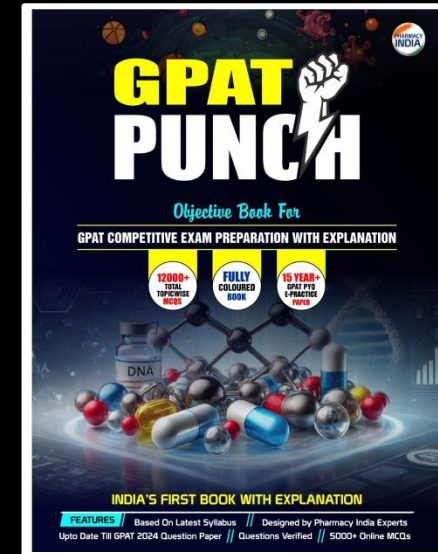


@PharmacyIndia

31.

Prinzmetal (variant) angina occurs due to:

- (a) Atherosclerotic plaque rupture
- (b) Coronary artery vasospasm
- (c) Increased oxygen demand
- (d) Thrombus formation



Download Pharmacy India Mobile App from Playstore



31. Prinzmetal (variant) angina occurs due to:

(a) Atherosclerotic plaque rupture

(b) Coronary artery vasospasm

(c) Increased oxygen demand

(d) Thrombus formation



Download Pharmacy India Mobile App from Playstore



PRINZMETAL (VARIANT) ANGINA



TEMPORARY CORONARY VASOSPASM



TRANSIENT SPASM
OF A CORONARY
ARTERY



TEMPORARY
↓ BLOOD FLOW
TO MYOCARDIUM



PAIN OFTEN
OCCURS AT REST,
USUALLY AT NIGHT



CAN CAUSE
TRANSIENT
ST ELEVATION



NOT PRIMARILY
DUE TO PLAQUE
RUPTURE



VASOSPASM



↓ CORONARY FLOW



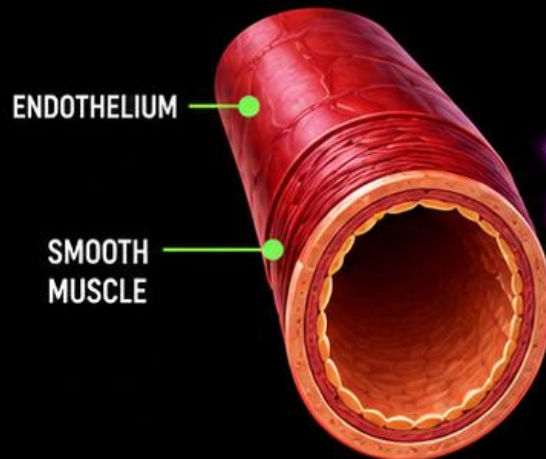
MYOCARDIAL ISCHEMIA



CHEST PAIN



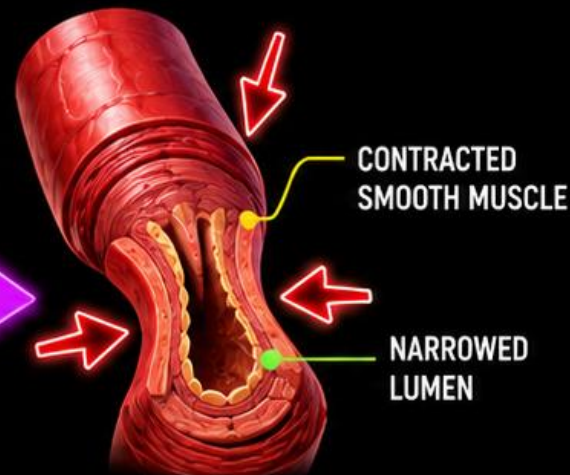
NORMAL CORONARY ARTERY



WIDE LUMEN
NORMAL BLOOD FLOW

**TRANSIENT
VASOSPASM**

CORONARY ARTERY IN SPASM



REDUCED BLOOD FLOW
MYOCARDIAL ISCHEMIA



**TEMPORARY
MYOCARDIAL
ISCHEMIA**

ECG DURING SPASM



ST ELEVATION

RESOLUTION



NORMAL ST SEGMENT

TRIGGERS



SMOKING

R_x

VASOCONSTRICTORS
(e.g., ERGOT, COCAINE)



COLD EXPOSURE



**EMOTIONAL
STRESS**



**AUTONOMIC
HYPERACTIVITY**



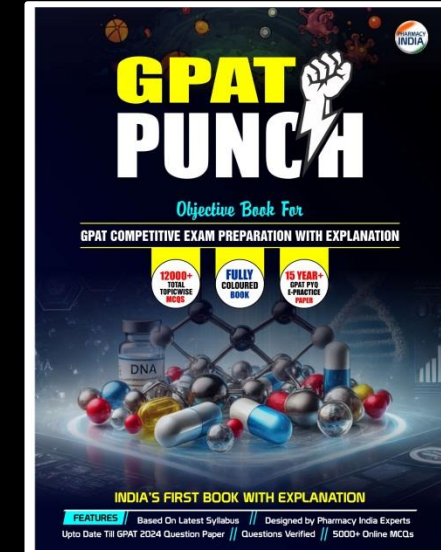
LOW Mg²⁺



@PharmacyIndia

32. Aortic stenosis leads to which type of ventricular hypertrophy?

- (a) Eccentric
- (b) Concentric
- (c) Dilated
- (d) Restrictive



Download Pharmacy India Mobile App from Playstore



@PharmacyIndia



32.

Aortic stenosis leads to which type of ventricular hypertrophy?

(a) Eccentric

(b) Concentric

(c) Dilated

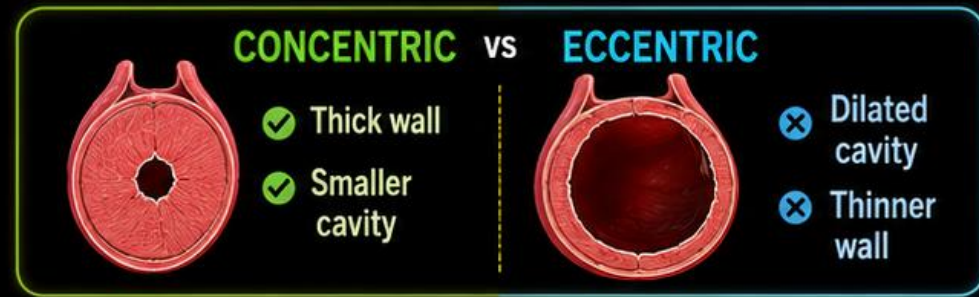
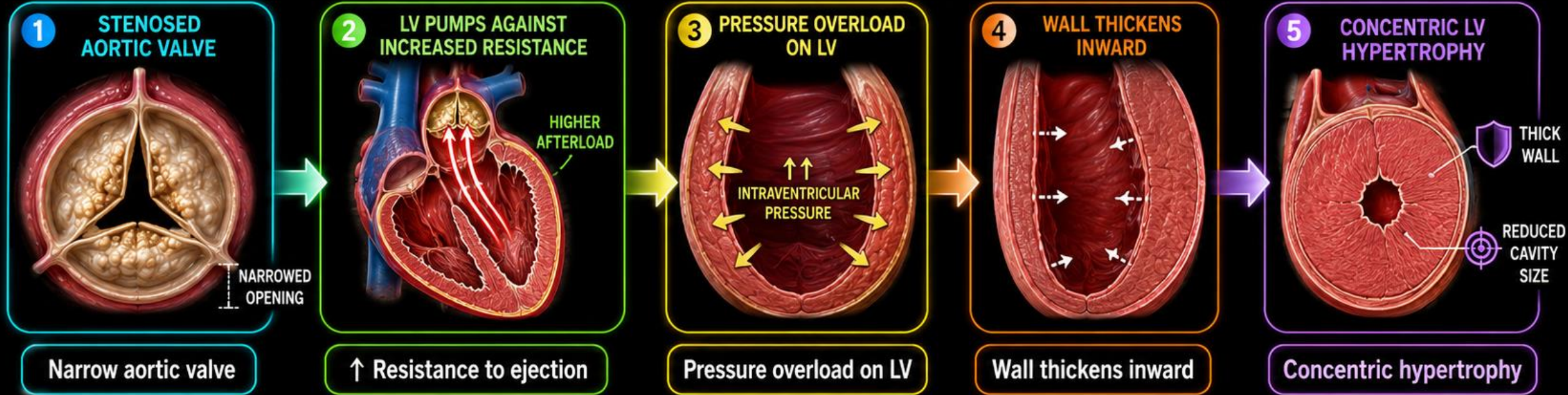
(d) Restrictive



Download Pharmacy India Mobile App from Playstore

Aortic Stenosis

Pressure overload → Concentric LV Hypertrophy



Remember: Pressure overload = Concentric hypertrophy





@PharmacyIndia

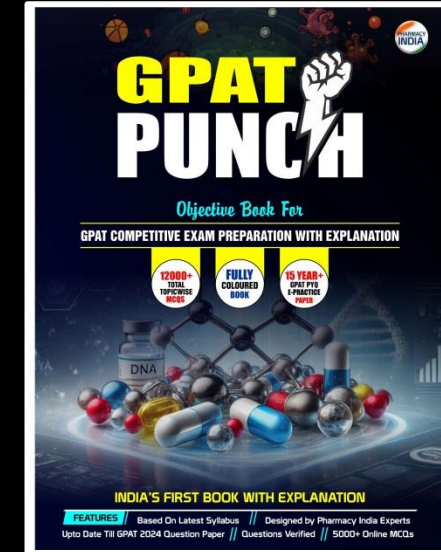


33.

Rapid ventricular filling sound

S3 heart sound is most commonly associated with:

- (a) Hypertension
- (b) Aortic regurgitation
- (c) Congestive heart failure
- (d) Pericarditis



Download Pharmacy India Mobile App from Playstore



33. S3 heart sound is most commonly associated with:

- (a) Hypertension
- (b) Aortic regurgitation
- (c) Congestive heart failure**
- (d) Pericarditis



Download Pharmacy India Mobile App from Playstore

S3 HEART SOUND

Classically associated with congestive heart failure



1

Occurs in
early diastole



2

Caused by
rapid filling of an
overloaded ventricle



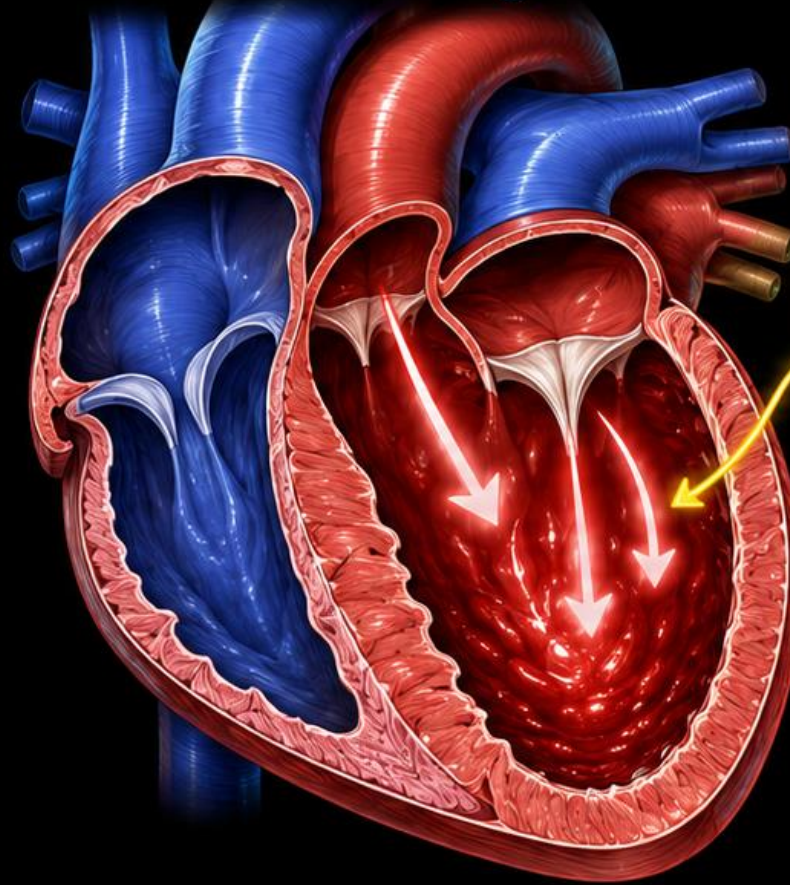
3

Suggests
**volume overload /
dilated ventricle**



4

Seen commonly in
**congestive
heart failure**



**Rapid
ventricular
filling**



Can be normal in
**children &
young adults**



S3



S3 = Ventricular gallop



Memory: **Kentucky rhythm**

S1
Lub

S2
Dub

S3
Lub

S4
Dub





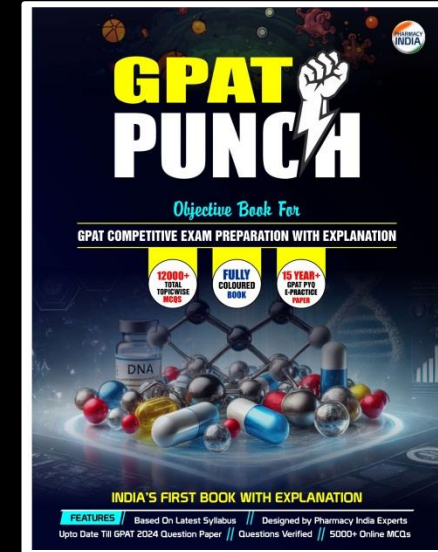
@PharmacyIndia



34.

S4 heart sound is associated with:

- (a) Atrial fibrillation
- (b) ischemic heart disease
- (c) Atrial contraction against stiff ventricle
- (d) Increased preload



Download Pharmacy India Mobile App from Playstore



34.

S4 heart sound is associated with:

- (a) Atrial fibrillation
- (b) ischemic heart disease
- (c) Atrial contraction against stiff ventricle**
- (d) Increased preload



Download Pharmacy India Mobile App from Playstore

S4



S4 HEART SOUND



Late diastolic sound

Produced by **atrial contraction** forcing blood into a **stiff, non-compliant ventricle**.

MECHANISM

1 ATRIAL CONTRACTION
Atrial kick

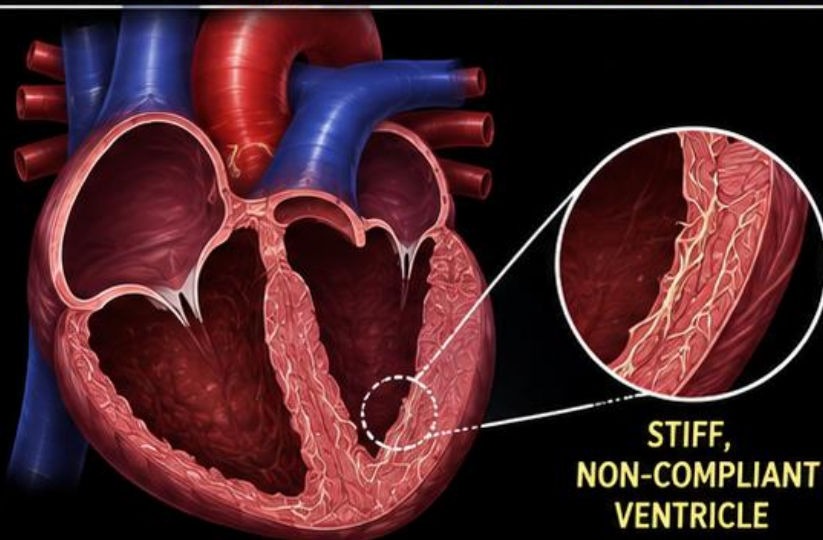
2 FORCES BLOOD INTO **STIFF, NON-COMPLIANT VENTRICLE**

3 RAPID DECELERATION OF BLOOD + VENTRICULAR WALL VIBRATION = **S4 SOUND**

atrial kick + stiff ventricle = vibration (S4)

MEMORY CLUE

S4 = stiff ventricle



STIFF, NON-COMPLIANT VENTRICLE

TIMING: OCCURS JUST BEFORE S1



CLINICAL ASSOCIATIONS

Seen with ventricular stiffness



LV
HYPERTROPHY



HYPERTENSION



AORTIC
STENOSIS



ISCHEMIC
HEART DISEASE



IMPORTANT CONTRAST

Usually absent in

ATRIAL FIBRILLATION

because effective atrial contraction is lost.



NO EFFECTIVE ATRIAL KICK

KEY POINTS



Late diastolic (presystolic) sound



Low pitch, best at the apex



Indicator of ventricular stiffness



ATRIAL CONTRACTION AGAINST A STIFF VENTRICLE PRODUCES S4.





@PharmacyIndia



Dilated cardiomyopathy

Large, weak heart with poor pumping.

35.

Dilated cardiomyopathy is primarily due to:

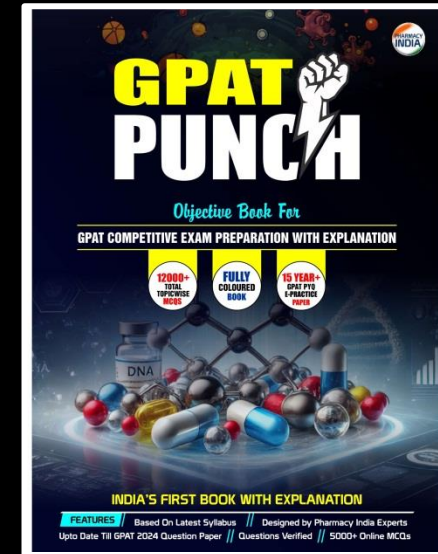
(a) Hypertension

(b) Chronic alcohol use

(c) Aortic stenosis

(d) Amyloidosis

abnormal proteins (amyloid) get deposited in organs causing them to become stiff and weak.



Download Pharmacy India Mobile App from Playstore



35. Dilated cardiomyopathy is primarily due to:

(a) Hypertension

(b) Chronic alcohol use

(c) Aortic stenosis

(d) Amyloidosis



Download Pharmacy India Mobile App from Playstore

Dilated Cardiomyopathy is primarily due to Chronic Alcohol Use

★ Dilated Cardiomyopathy (DCM)

Dilatation of ventricles with **impaired systolic function** (↓ **contractility**).

Features

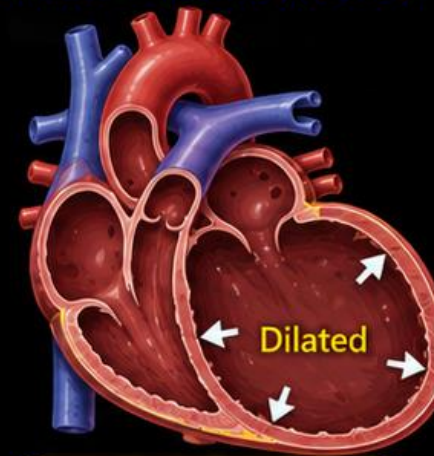
- ✓ Ventricular dilatation
- ✓ Thin, stretched walls
- ✓ ↓ Contractility
- ✓ ↓ Ejection fraction
- ✓ Heart failure

Normal Heart



Normal size ventricle
Normal contractility

Dilated Cardiomyopathy



Dilated ventricle
Thin wall
Poor contractility
↓ Ejection fraction



Key Point

DCM = Dilated,
Weak, Poor Pump

Think:

Alcohol → Toxic to
myocardium →
DCM

Causes of Dilated Cardiomyopathy

Hypertension



Causes concentric hypertrophy, **not DCM**

Chronic Alcohol Use



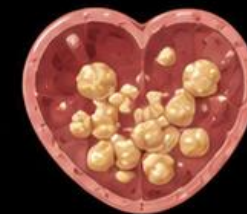
Alcohol is directly
toxic to myocardium →
leads to DCM

Aortic Stenosis



Causes pressure overload
→ concentric hypertrophy,
not DCM

Amyloidosis



Causes restrictive
cardiomyopathy,
not DCM



Take-Home

★ Dilated cardiomyopathy
= dilated + weak heart.

★ Most common cause:
Chronic Alcohol Use

★ Others cause different
types of cardiomyopathy.



@PharmacyIndia

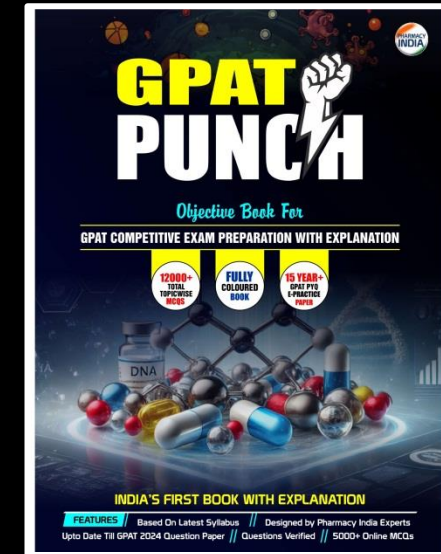


36.

(Creatine Kinase—Myocardial Band

In myocardial infarction, CK-MB is particularly useful to: (GPAT - 2020)

- (a) Detect very early MI
- (b) Diagnose Prinzmetal angina
- (c) Detect stable angina
- (d) Detect reinfarction



Download Pharmacy India Mobile App from Playstore



36. In myocardial infarction, CK-MB is particularly useful to:
(GPAT - 2020)

- (a) Detect very early MI
- (b) Diagnose Prinzmetal angina
- (c) Detect stable angina
- (d) Detect reinfarction**



Download Pharmacy India Mobile App from Playstore

CK-MB in MI – Key Explanation

1



CK-MB rises after MI

2



Rises in about 4–6 hours

3



Peaks at 18–24 hours

4



Returns to normal in 48–72 hours

5

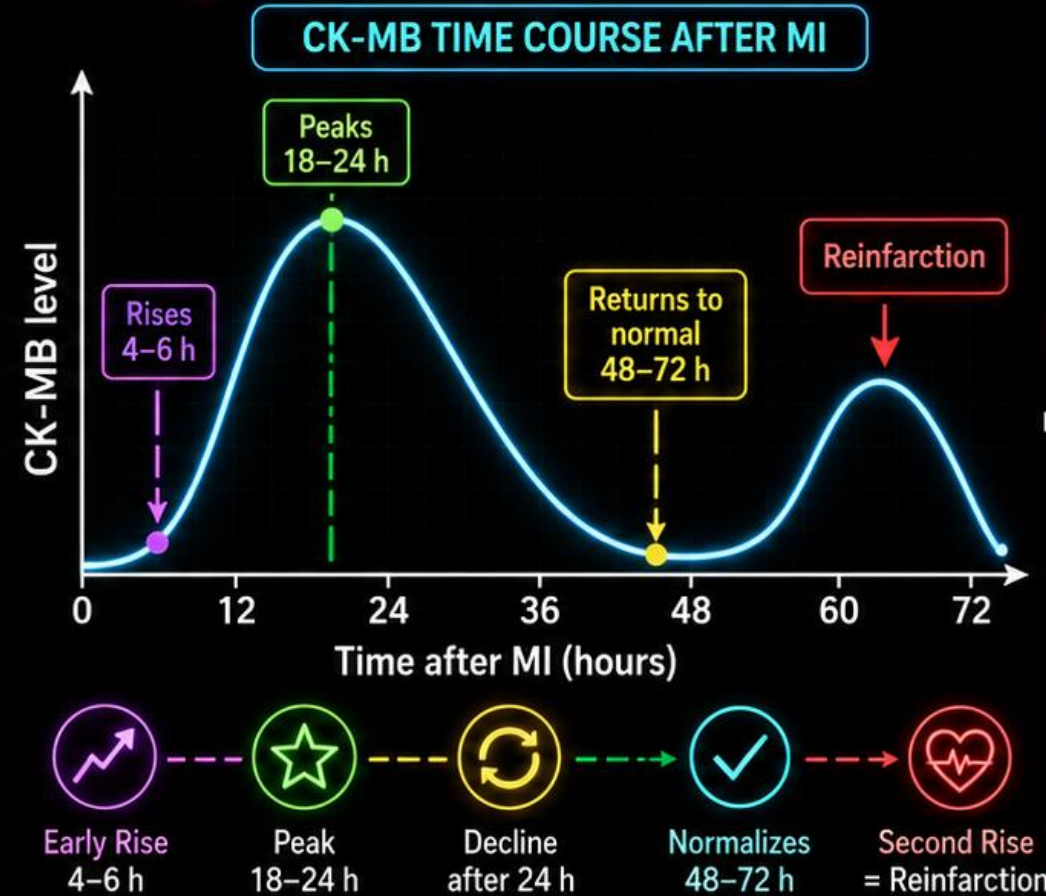


A second rise after normalization suggests reinfarction

6



Useful for detecting reinfarction



ECG



MYOCARDIUM



LAB TEST



BIOMARKER



Quick clue:

CK-MB normalizes early,
so repeat elevation = **reinfarction clue**



CONTRAST / REMINDER

- Troponin stays elevated longer
- CK-MB is less useful than troponin for late persistent elevation





@PharmacyIndia

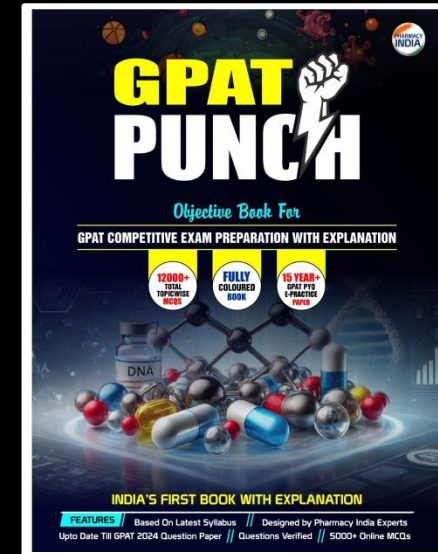


Hypertrophic cardiomyopathy (HCM) is a condition where the heart muscle (**especially the left ventricle**) becomes abnormally thick, making it hard for the heart to relax and fill with blood.

37.

Hypertrophic cardiomyopathy most commonly leads to:

- (a) Dilated ventricles
- (b) Sudden cardiac death in young athletes
- (c) Low ejection fraction
- (d) Concentric thickening due to hypertension



Download Pharmacy India Mobile App from Playstore



- 37. Hypertrophic cardiomyopathy most commonly leads to:**
- (a) Dilated ventricles**
 - (b) Sudden cardiac death in young athletes**
 - (c) Low ejection fraction**
 - (d) Concentric thickening due to hypertension**



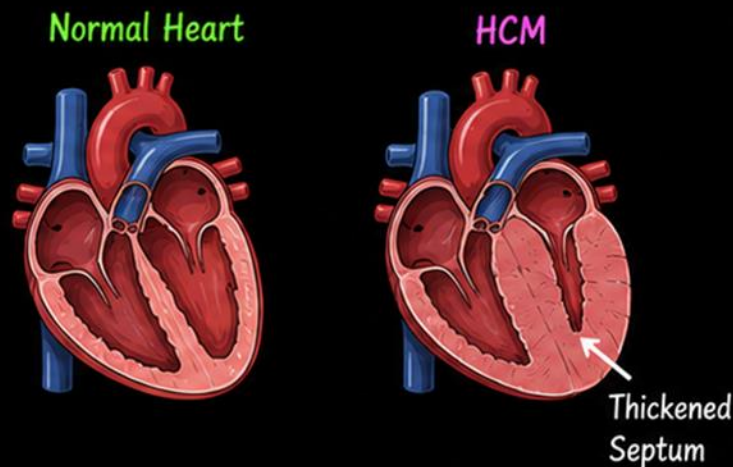
HYPERTROPHIC CARDIOMYOPATHY (HCM)

Most commonly leads to: **SUDDEN CARDIAC DEATH IN YOUNG ATHLETES**

What is HCM?

Genetic disorder (usually **autosomal dominant**) causing **marked thickening of myocardium** (especially **interventricular septum**)

Normal Heart vs HCM



Why Sudden Cardiac Death?



Myocyte disarray & fibrosis
→ Ventricular arrhythmias



Dynamic LV outflow obstruction
→ Ischemia, arrhythmias



High adrenergic state in athletes (exercise/competition)
→ Triggers fatal arrhythmias

Who is at Risk?



Young athletes (10–30 yrs)



Family history of HCM or sudden death



Unexplained syncope

Why other options are incorrect?

(a) Dilated ventricles

✗ HCM has **small/normal ventricular cavity**, not dilated.



(c) Low ejection fraction

✗ EF is usually **normal or increased** in HCM until late stages.



(d) Concentric thickening due to hypertension

✗ HTN causes concentric LVH, but not the typical **septal hypertrophy + disarray** seen in HCM.



Key Takeaway

HCM → **Most commonly** causes **sudden cardiac death** in **young athletes** due to **ventricular arrhythmias**.





@PharmacyIndia



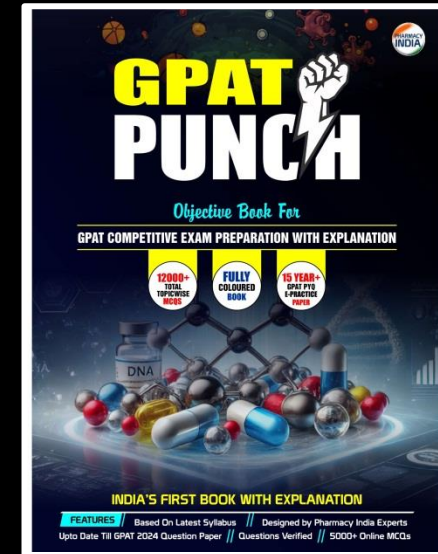
38.

Mitral stenosis most commonly occurs due to:

- (a) Atherosclerosis
- (b) Rheumatic fever
- (c) Infective endocarditis
- (d) Myxomatous degeneration

Rheumatic fever is an inflammatory disease that occurs after **untreated streptococcal throat infection**, and it can damage the **heart valves**, joints, skin, and brain.

Myxomatous degeneration : weakening and degeneration of connective tissue



Download Pharmacy India Mobile App from Playstore



38. Mitral stenosis most commonly occurs due to:

(a) Atherosclerosis

(b) Rheumatic fever

(c) Infective endocarditis

(d) Myxomatous degeneration



Download Pharmacy India Mobile App from Playstore

Mitral Stenosis most commonly occurs due to Rheumatic Fever

CAUSE

Rheumatic Fever

Autoimmune reaction after
Group A streptococcal
pharyngitis



Chronic inflammation
and scarring of
mitral valve



Fusion of commissures,
thickened leaflets,
calcification

Leads to narrowing of
mitral valve orifice
= Mitral Stenosis

PATHOLOGY

Normal Mitral Valve



Open mitral orifice

Mitral Stenosis



Narrowed mitral orifice

HEMODYNAMIC CONSEQUENCE



↑ Pressure in
left atrium



Pulmonary venous
congestion



Right heart
strain



Systemic
congestion

WHY OTHER OPTIONS ARE LESS LIKELY



Atherosclerosis

Affects large and medium arteries,
not heart valves



Infective Endocarditis

Causes acute valve destruction
and regurgitation, not isolated
mitral stenosis



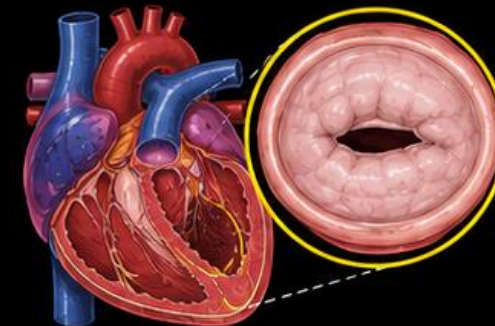
Myxomatous Degeneration

Leads to mitral valve prolapse
and regurgitation, not stenosis



KEY POINT

Rheumatic fever is the most common cause of mitral stenosis,
especially in developing countries.





@PharmacyIndia

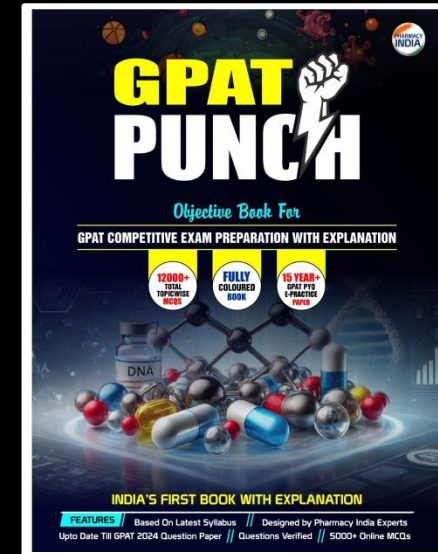


39.

The main mechanism of orthopnea in left-sided heart failure is:

Lying down

- (a) Increased venous pooling
- (b) Increased venous return in supine position
- (c) Reduced preload
- (d) Decreased sympathetic tone



Download Pharmacy India Mobile App from Playstore



39. The main mechanism of orthopnea in left-sided heart failure is:

- (a) Increased venous pooling
- (b) Increased venous return in supine position**
- (c) Reduced preload
- (d) Decreased sympathetic tone





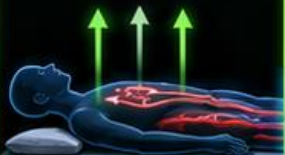
ORTHOPNEA IN LEFT-SIDED HEART FAILURE



• Breathlessness worsens on lying flat •

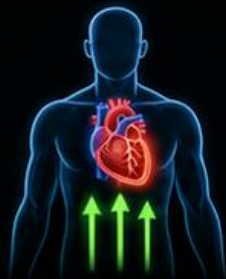
1

In supine position, blood from legs and splanchnic circulation returns to the chest.



2

This causes increased venous return / increased preload.



3

The weak left ventricle cannot handle the extra volume.



4

Pulmonary venous pressure rises.



5

Pulmonary congestion / interstitial edema develops.



6

Result: **dyspnea** when lying flat (**orthopnea**).



7

Sitting up relieves symptoms by reducing venous return.



LYING FLAT (SUPINE)



Gravity eliminated
→ more blood from legs & abdomen returns to the chest



Weak left ventricle overloaded



Pulmonary congestion & interstitial edema



Chest tightness



Rapid, shallow breathing



Worse at night, hard to sleep (needs to sit up)



EXAM PEARL

Orthopnea occurs because the supine position **increases venous return** and **worsens pulmonary congestion**.

SITTING UP (RELIEF)



Why it helps:

- ✓ Gravity pulls blood to the lower body
- ↓
- ✓ Decreased venous return (preload)
- ↓
- ✓ Lower pulmonary venous pressure
- ↓
- ✓ Less pulmonary congestion
- ↓
- ✓ Easier breathing



@PharmacyIndia

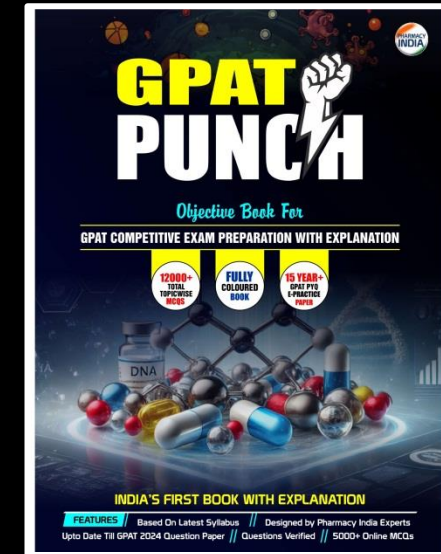


Pericardial tamponade is a medical emergency where fluid rapidly accumulates around the heart (in the pericardial sac) and compresses the heart, preventing it from filling and pumping properly.

40.

Pericardial tamponade classically shows:

- (a) Widened pulse pressure
- (b) Pulsus paradoxus
- (c) Bounding pulses
- (d) Hypertension



Download Pharmacy India Mobile App from Playstore



@PharmacyIndia

Pericardial tamponade is a medical emergency where fluid rapidly accumulates around the heart (in the pericardial sac) and compresses the heart, preventing it from filling and pumping properly.

40.

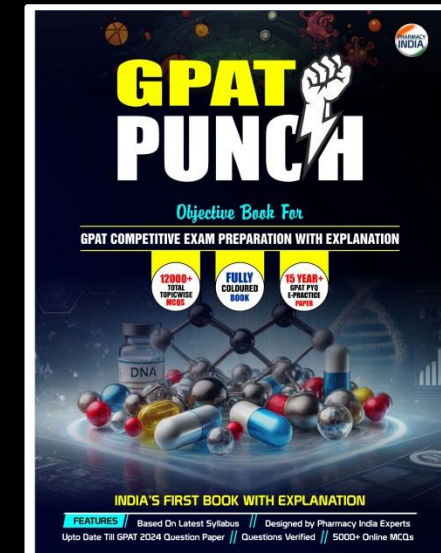
Pericardial tamponade classically shows:

(a) **Widened pulse pressure:** Systolic–diastolic BP difference is large.

(b) **Pulsus paradoxus:** Systolic BP falls >10 mmHg on inspiration.

(c) **Bounding pulses:** Pulse is very strong and forceful.

(d) **Hypertension:** High blood pressure.

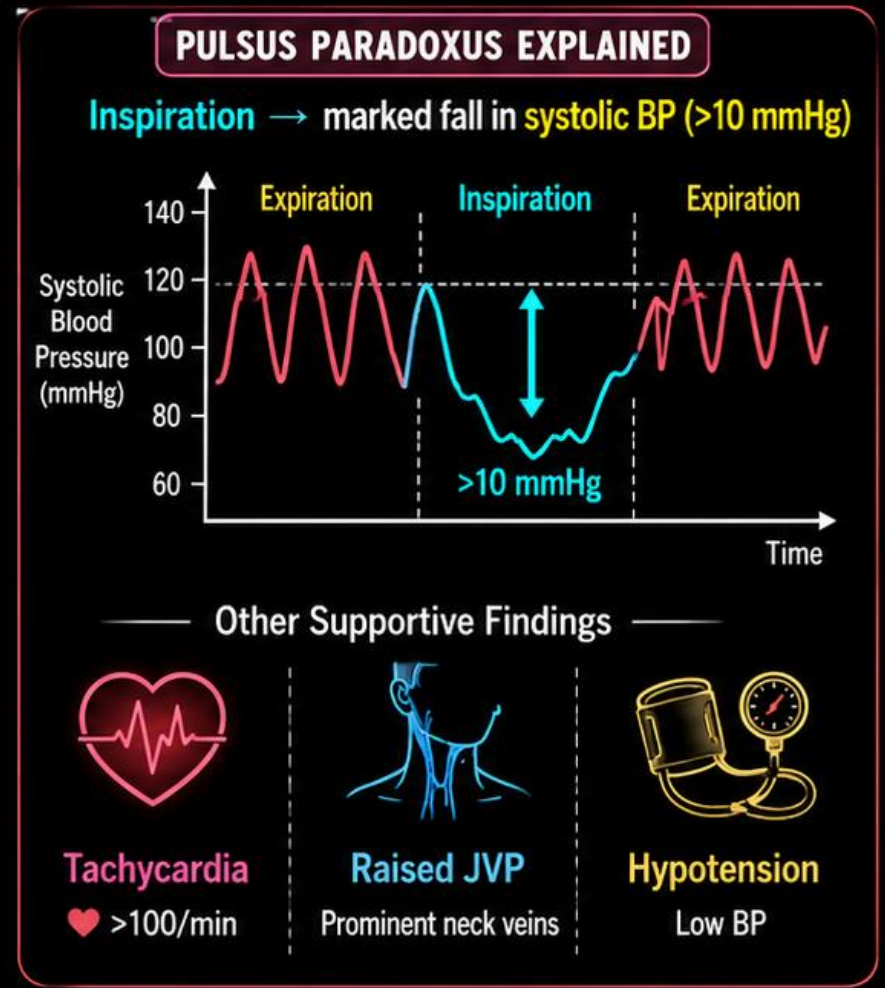
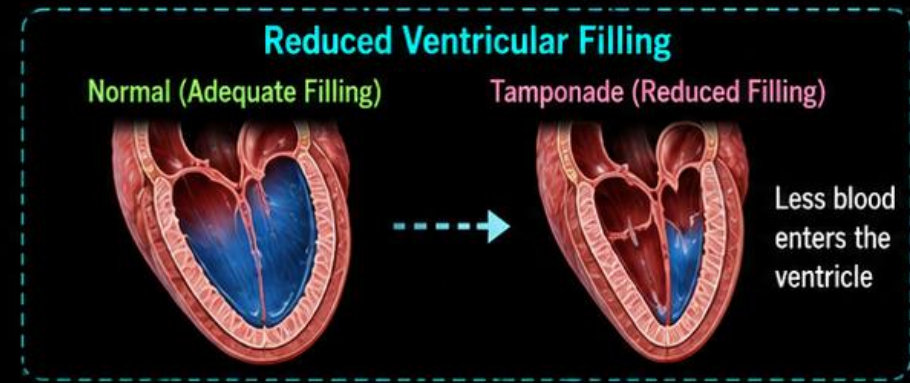
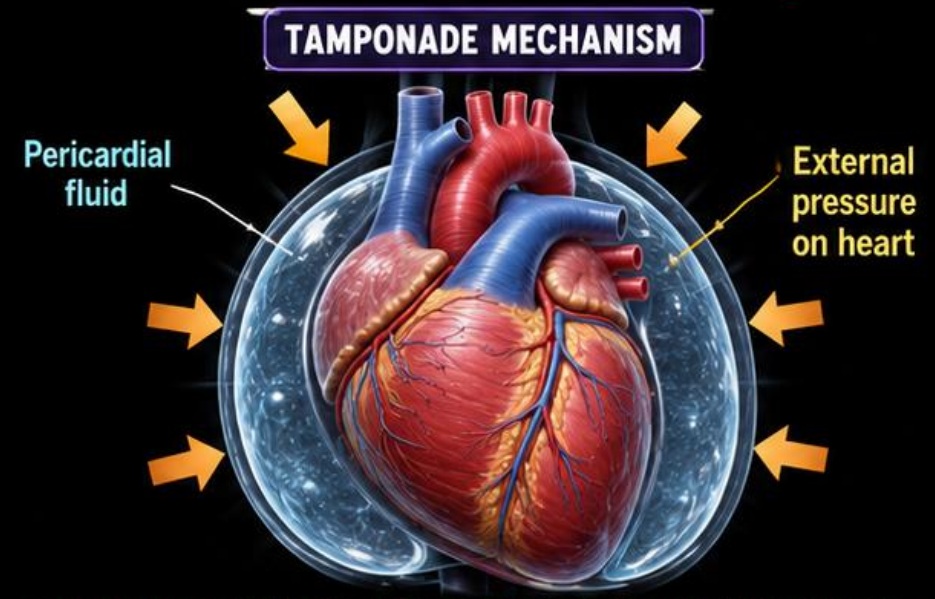


Download Pharmacy India Mobile App from Playstore

PERICARDIAL TAMPONADE

• Classical sign: **Pulsus paradoxus** •

-  Fluid collects in pericardial sac
-  Heart gets compressed
-  Ventricular filling falls
-  Cardiac output drops
-  During inspiration, systolic BP falls more than normal
-  This exaggerated fall is called **pulsus paradoxus**



KEY IDEA: tamponade restricts cardiac filling; the classic bedside clue is **pulsus paradoxus**.



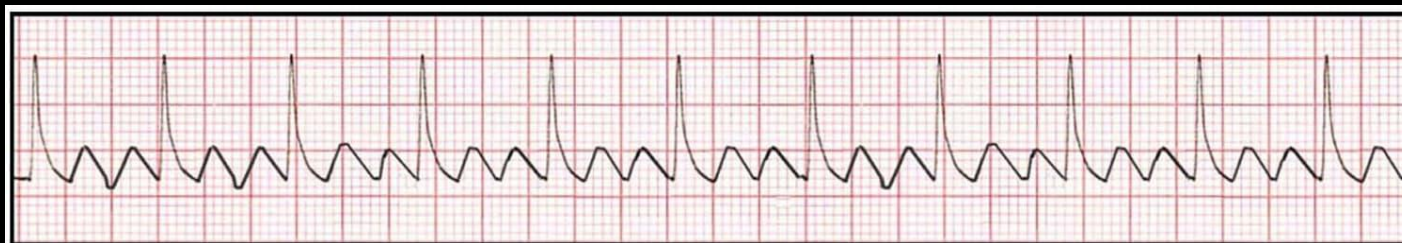


@PharmacyIndia

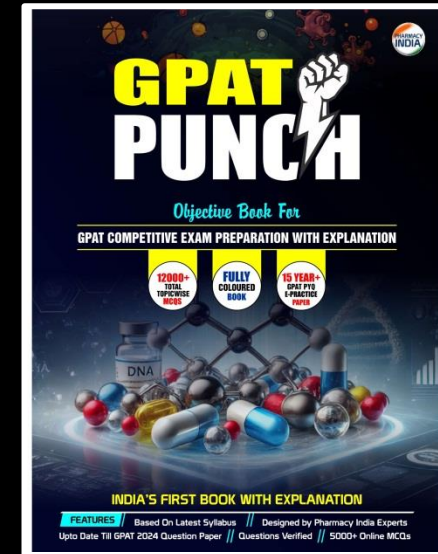
41.

Which arrhythmia is characterized by “saw-tooth” flutter waves on ECG?

- (a) Atrial fibrillation
- (b) Atrial flutter
- (c) Ventricular fibrillation
- (d) Ventricular tachycardia



Download Pharmacy India Mobile App from Playstore





41.

Which arrhythmia is characterized by “saw-tooth” flutter waves on ECG?

- (a) Atrial fibrillation
- (b) Atrial flutter**
- (c) Ventricular fibrillation
- (d) Ventricular tachycardia



Download Pharmacy India Mobile App from Playstore

ATRIAL FLUTTER

Saw-tooth flutter waves on ECG

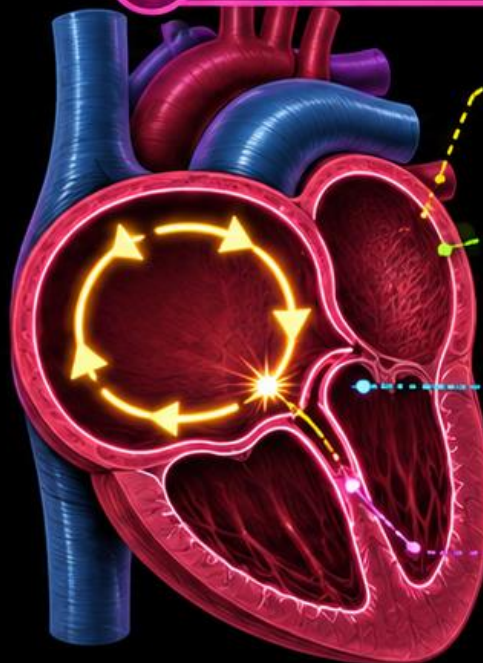
ECG FINDING

Regular saw-tooth F waves



F waves = atrial depolarizations seen between QRS complexes

MECHANISM



Rapid atrial rate

Organized atrial rhythm

Usually 250–350/min

Re-entry circuit in atrium

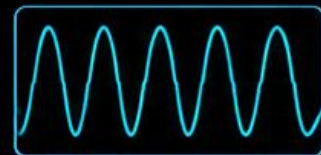
NOT THIS PATTERN



Atrial fibrillation: irregular, no saw-tooth



Ventricular tachycardia: wide fast QRS



Ventricular fibrillation: chaotic tracing



1

Saw-tooth flutter waves are classic



2

Most often seen in atrial flutter



3

Atrial activity is **fast** and **regular**



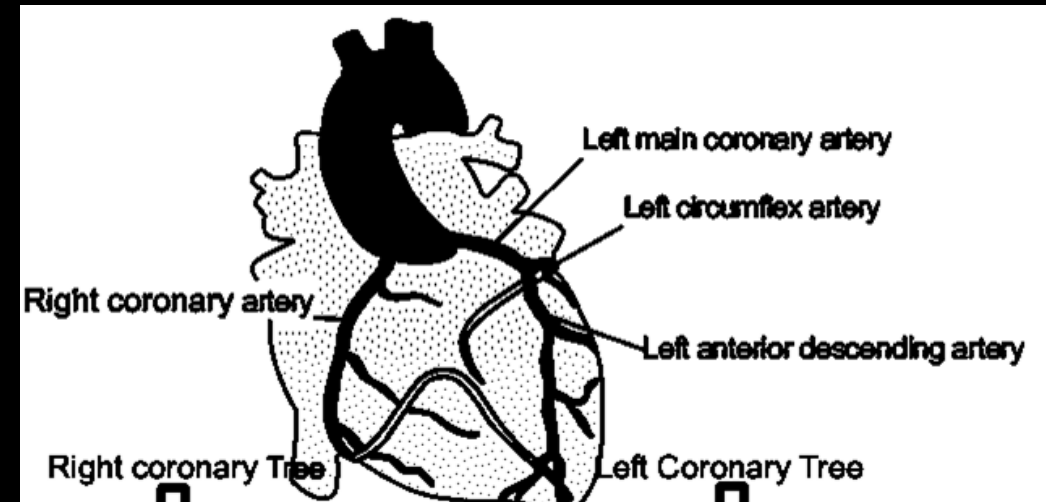
4

ECG clue: **flutter waves** between QRS complexes



42. The “widow-maker” artery refers to critical occlusion of:

- (a) Right coronary artery
- (b) Left circumflex artery
- (c) Left anterior descending artery
- (d) Posterior descending artery





42. The “widow-maker” artery refers to critical occlusion of:

- (a) Right coronary artery
- (b) Left circumflex artery
- (c) Left anterior descending artery**
- (d) Posterior descending artery



Download Pharmacy India Mobile App from Playstore

Widow-Maker Artery



Refers to **critical blockage** of the **Left Anterior Descending (LAD)** artery.



- LAD supplies a **major part** of the **left ventricle**.



- Sudden occlusion may cause a **massive anterior wall** heart attack.



- Can **sharply reduce** blood flow to the **main pumping chamber**.



- High risk of **collapse**, **arrhythmia**, and **sudden death**.

ECG: Anterior Wall MI Pattern



ST
ELEVATION
V1-V4

LAD

(Left Anterior
Descending
Artery)

RIGHT
CORONARY
ARTERY

CIRCUMFLEX
ARTERY

AORTA

LEFT
CORONARY
ARTERY

**BLOCKAGE
(CLOT)**

BLOOD FLOW



**HIGH
RISK**



COLLAPSE



ARRHYTHMIA



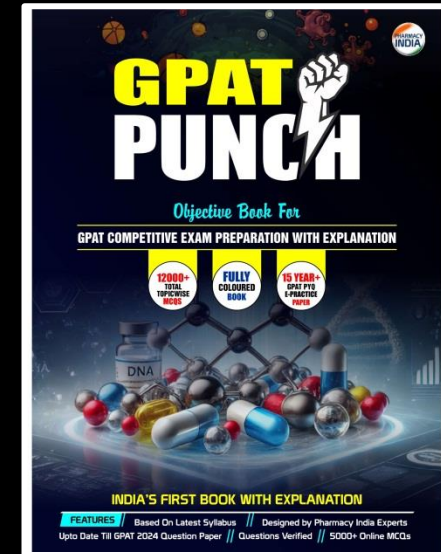
**SUDDEN
DEATH**



@PharmacyIndia

43. Which electrolyte abnormality most commonly precipitates ventricular fibrillation?

- (a) Hypocalcemia
- (b) Hyperkalemia
- (c) Hypokalemia
- (d) Hypermagnesemia



Download Pharmacy India Mobile App from Playstore



43.

Which electrolyte abnormality most commonly precipitates ventricular fibrillation?

(a) Hypocalcemia

(b) Hyperkalemia

(c) Hypokalemia

(d) Hypermagnesemia



Download Pharmacy India Mobile App from Playstore

HYPOKALEMIA & VENTRICULAR FIBRILLATION

Low K^+ makes the heart electrically unstable

K^+ ↓ K^+ increases ventricular irritability

⌚ Delays repolarization → ectopic beats

$\frac{QT}{QU}$ QT / QU prolongation may appear

⚡ Can trigger VT, then VF

ECG clues: flat T waves, prominent U waves

🐚 Clinical pearl: potassium imbalance can be life-threatening



LOW K^+
=
HIGH RISK
OF VF

NORMAL SINUS RHYTHM



VENTRICULAR FIBRILLATION (VF)

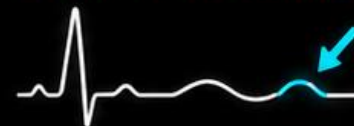


ECG CLUES
IN HYPOKALEMIA

FLAT T WAVES



PROMINENT U WAVES



PROLONGED QT / QU



MONITOR **POTASSIUM** IN CARDIAC PATIENTS





@PharmacyIndia



44.

The primary biochemical cause of chest pain during ischemia is:

- (a) Calcium influx
- (b) Lactate accumulation
- (c) Troponin release
- (d) Oxidized LDL



Download Pharmacy India Mobile App from Playstore



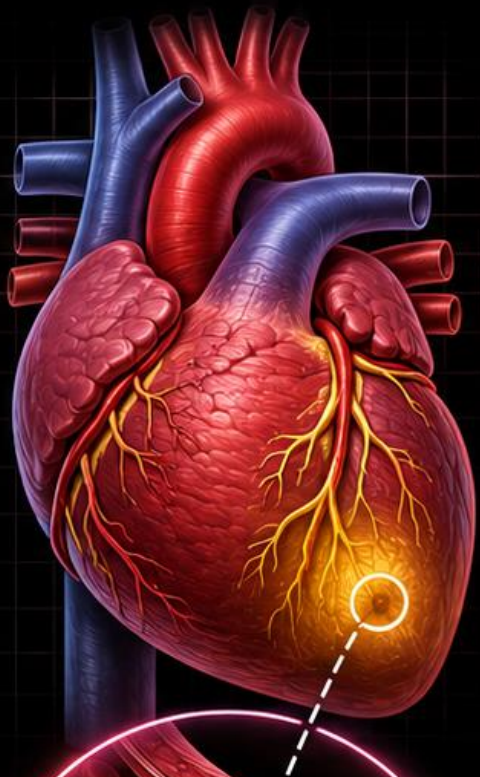
44.

The primary biochemical cause of chest pain during ischemia is:

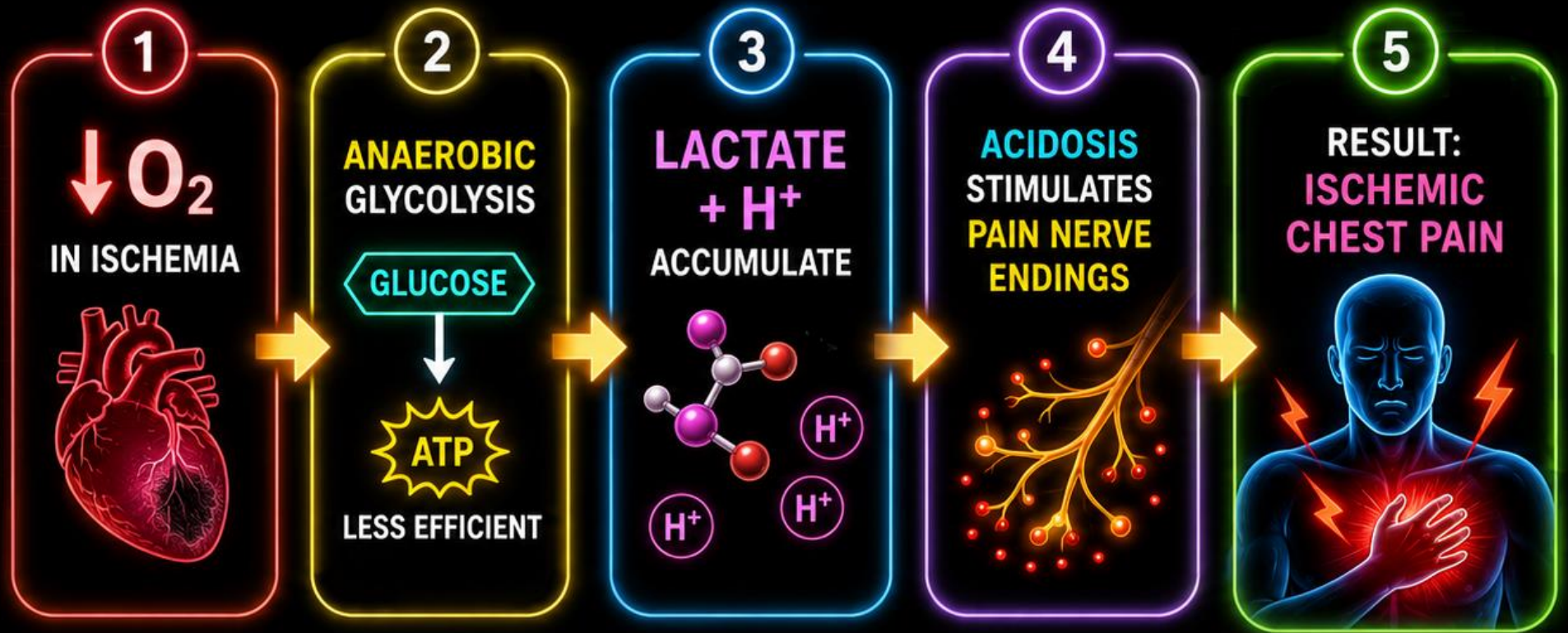
- (a) Calcium influx
- (b) Lactate accumulation**
- (c) Troponin release
- (d) Oxidized LDL



Download Pharmacy India Mobile App from Playstore



**CORONARY
NARROWING**



HIGH-YIELD:
PAIN IS MAINLY DUE TO
METABOLITE BUILDUP,
NOT TROPONIN RELEASE



LACTATE



ACIDITY





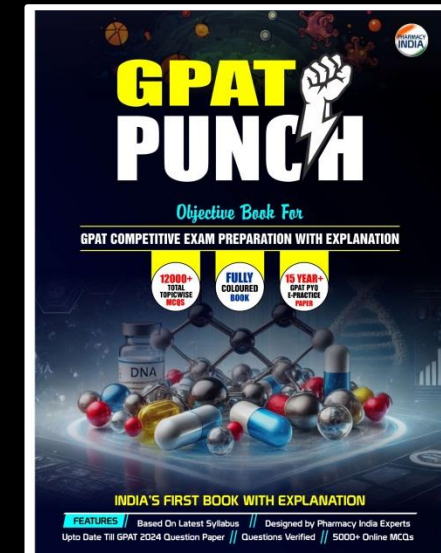
@PharmacyIndia

45.

Which hemodynamic change is seen in early septic shock?

Septic shock is a severe, life-threatening condition where a serious infection causes dangerously low blood pressure and organ failure due to widespread inflammation and blood vessel dilation.

- (a) Cold skin
- (b) Decreased cardiac output
- (c) Increased cardiac output
- (d) Increased systemic vascular resistance



Download Pharmacy India Mobile App from Playstore



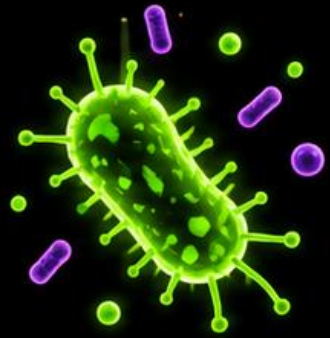
45. Which hemodynamic change is seen in early septic shock?

- (a) Cold skin
- (b) Decreased cardiac output
- (c) Increased cardiac output**
- (d) Increased systemic vascular resistance

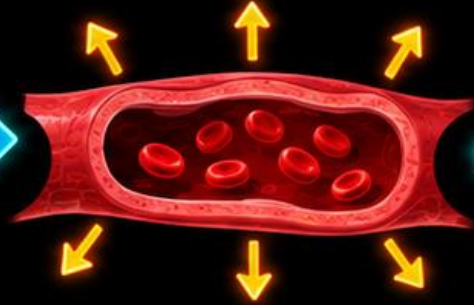


EARLY SEPTIC SHOCK

Hyperdynamic / Warm shock



Infection →
cytokines +
nitric oxide



Peripheral
vasodilation



↓ Systemic
vascular
resistance
(SVR)



↑ Compensatory
Cardiac output



Warm
flushed skin



Bounding pulse • Tachycardia



Perfusion may still be
poor at tissue level



Memory: Early sepsis = warm, vasodilated, high-output state





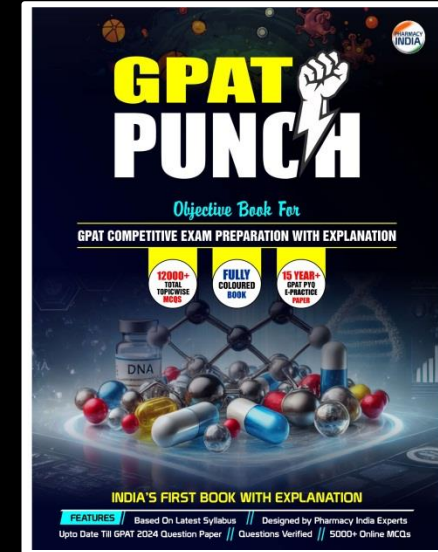
@PharmacyIndia



46.

Hypertensive emergency is defined as:

- (a) BP >160/100 mmHg
- (b) BP >200/130 mmHg without organ damage
- (c) Severe hypertension with acute organ damage
- (d) Hypertension with ankle edema



Download Pharmacy India Mobile App from Playstore



- 46. Hypertensive emergency is defined as:**
- (a) BP >160/100 mmHg**
 - (b) BP >200/130 mmHg without organ damage**
 - (c) Severe hypertension with acute organ damage**
 - (d) Hypertension with ankle edema**



Hypertensive Emergency

Severe hypertension + acute target-organ damage

★ Defined by **organ damage**,
not **BP number** alone



SEVERE HYPERTENSION
(Usually $\geq 180/120$ mmHg)



BRAIN:
encephalopathy,
stroke



HEART:
MI, acute LV failure,
pulmonary edema



KIDNEY:
acute renal injury



EYE:
retinopathy /
papilledema



**ACUTE
TARGET-
ORGAN
DAMAGE**



**Needs
immediate
BP control**



**Usually
IV antihypertensives
+ monitoring**



**ALTERED
MENTAL STATUS**



**CHEST PAIN /
DYSPNEA**



**↓ URINE
OUTPUT**



**SEVERE
HEADACHE**



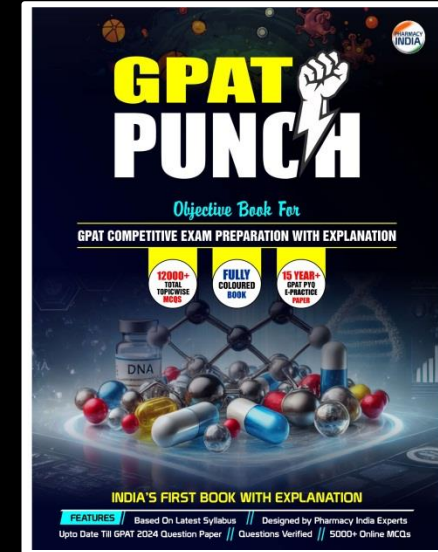
@PharmacyIndia



47.

The most common cause of secondary hypertension is:

- (a) Pheochromocytoma
- (b) Renal artery stenosis
- (c) Primary aldosteronism
- (d) Cushing's syndrome



Download Pharmacy India Mobile App from Playstore



47.

The most common cause of secondary hypertension is:

(a) Pheochromocytoma

Secondary hypertension is high blood pressure caused by an underlying disease or condition,

(b) Renal artery stenosis

(c) Primary aldosteronism

(d) Cushing's syndrome



Download Pharmacy India Mobile App from Playstore

Secondary Hypertension

Common reversible causes matter

Primary aldosteronism
= common listed cause



ALDOSTERONE

A

Aldosterone
holds salt + water



Excess aldosterone → Na^+ & water retention



Blood volume rises → BP increases



Often causes hypokalemia K^+ ↓



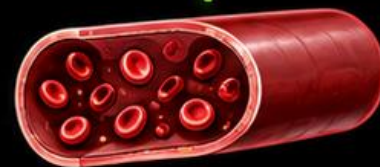
Think of resistant /
difficult-to-control hypertension



Cause is identifiable and
potentially treatable



Na^+ & H_2O
RETENTION



BLOOD VOLUME
RISES

Hypokalemia
clue

K^+ ↓



BP
UP



@PharmacyIndia



48.

In heart failure, activation of RAAS leads to:

- (a) Reduced preload
- (b) Reduced afterload
- (c) Sodium & water retention
- (d) Decreased sympathetic activity



Download Pharmacy India Mobile App from Playstore



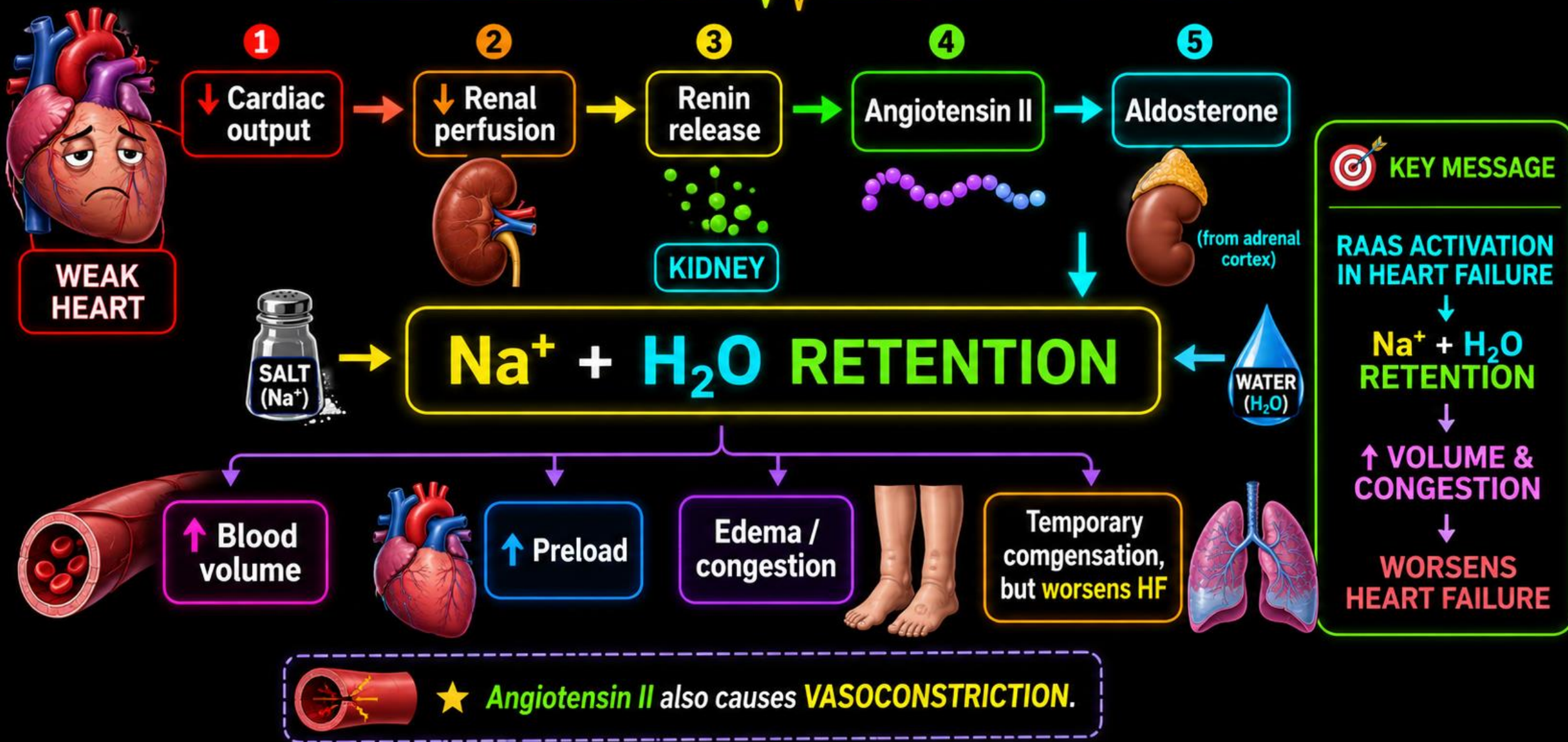
48. In heart failure, activation of RAAS leads to:

- (a) Reduced preload
- (b) Reduced afterload
- (c) Sodium & water retention**
- (d) Decreased sympathetic activity



Download Pharmacy India Mobile App from Playstore

RAAS in Heart Failure





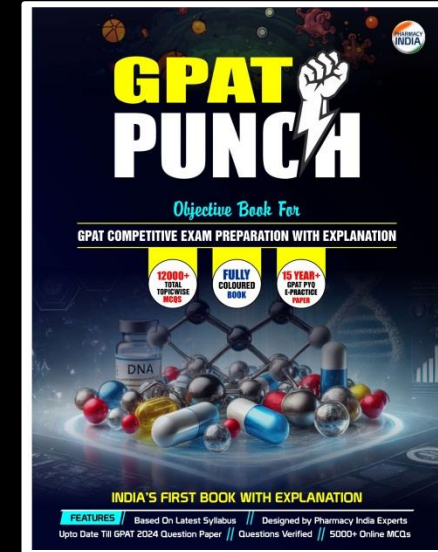
@PharmacyIndia



49.

Cor pulmonale refers to:

- (a) Right heart failure due to left HF
- (b) Right ventricular hypertrophy due to lung disease
- (c) Cardiac arrest due to embolism
- (d) Pulmonary edema due to LV failure



Download Pharmacy India Mobile App from Playstore



49.

Cor pulmonale refers to:

- (a) Right heart failure due to left HF
- (b) Right ventricular hypertrophy due to lung disease**
- (c) Cardiac arrest due to embolism
- (d) Pulmonary edema due to LV failure



Download Pharmacy India Mobile App from Playstore

COR PULMONALE

RIGHT HEART STRAIN FROM LUNG DISEASE



Cor pulmonale =
right-sided heart change
due to lung disease



Main feature:
Right ventricular
hypertrophy



Cause:
Pulmonary hypertension
secondary to chronic
lung disease



Common in COPD
and other chronic
pulmonary disorders



Not caused by
left ventricular failure



Key idea:
Lung pathology ->
pressure load on RV



RIGHT VENTRICLE (RV)
Highlighted

PATHOPHYSIOLOGY: THE CHAIN OF EVENTS

1

CHRONIC LUNG DISEASE



- COPD
- Pulmonary fibrosis
- Bronchiectasis
- Sleep apnea
- Other chronic lung disorders

2

PULMONARY HYPERTENSION



↑ Pulmonary
vascular resistance
=
↑ Pulmonary
artery pressure

3

RIGHT VENTRICULAR OVERLOAD



RV faces increased
pressure load to
pump against high
pulmonary pressure

4

RIGHT VENTRICULAR HYPERTROPHY



RV muscle thickens
(hypertrophy) to
generate more
force

5

POSSIBLE RIGHT HEART FAILURE



- Systemic venous congestion
- Peripheral edema
- Ascites
- Hepatomegaly
- Fatigue, exertional intolerance

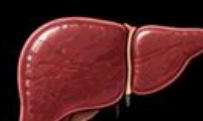
CONSEQUENCES OF RIGHT HEART FAILURE



↑ JVP
(Raised venous
pressure)



Peripheral
edema



Hepatomegaly



Ascites



Fatigue,
exertional
intolerance



ESSENCE: Chronic lung disease causes pulmonary hypertension, leading to RV hypertrophy and possible right heart failure.



@PharmacyIndia



50.

The most common cause of sudden cardiac death post-MI is:

- (a) Ventricular fibrillation
- (b) Atrial flutter
- (c) Complete heart block
- (d) Dilated cardiomyopathy



Download Pharmacy India Mobile App from Playstore



50.

The most common cause of sudden cardiac death post-MI is:

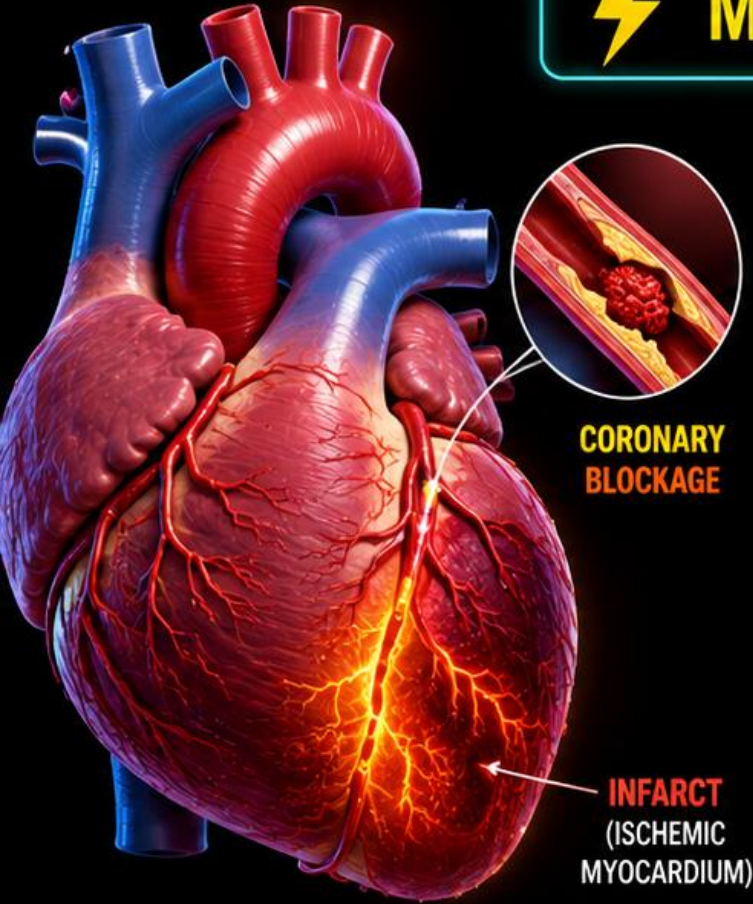
- (a) Ventricular fibrillation**
- (b) Atrial flutter**
- (c) Complete heart block**
- (d) Dilated cardiomyopathy**



Download Pharmacy India Mobile App from Playstore

Sudden Cardiac Death Post-MI

⚡ Most common trigger: **Ventricular fibrillation**



DEFIBRILLATION



SHOCK

- 1 ⚡ Ischemic myocardium becomes **electrically unstable**
- 2 🧠 Chaotic ventricular **impulses** develop
- 3 🌊 Ventricles **quiver** instead of pumping
- 4 📉 Cardiac output **falls** to **near zero**
- 5 🏃 Rapid **collapse** without urgent **defibrillation**

NO EFFECTIVE PUMP



SUDDEN DEATH



HIGH-YIELD:

VF is the common early **fatal arrhythmia** after **MI**



GPAT PLUS

38 PREVIOUS YEAR QUESTION PAPERS
WITH DIGITAL EXPLANATION

←..... FEATURES→



Based on Latest
GPAT Syllabus



Subjectwise
Division



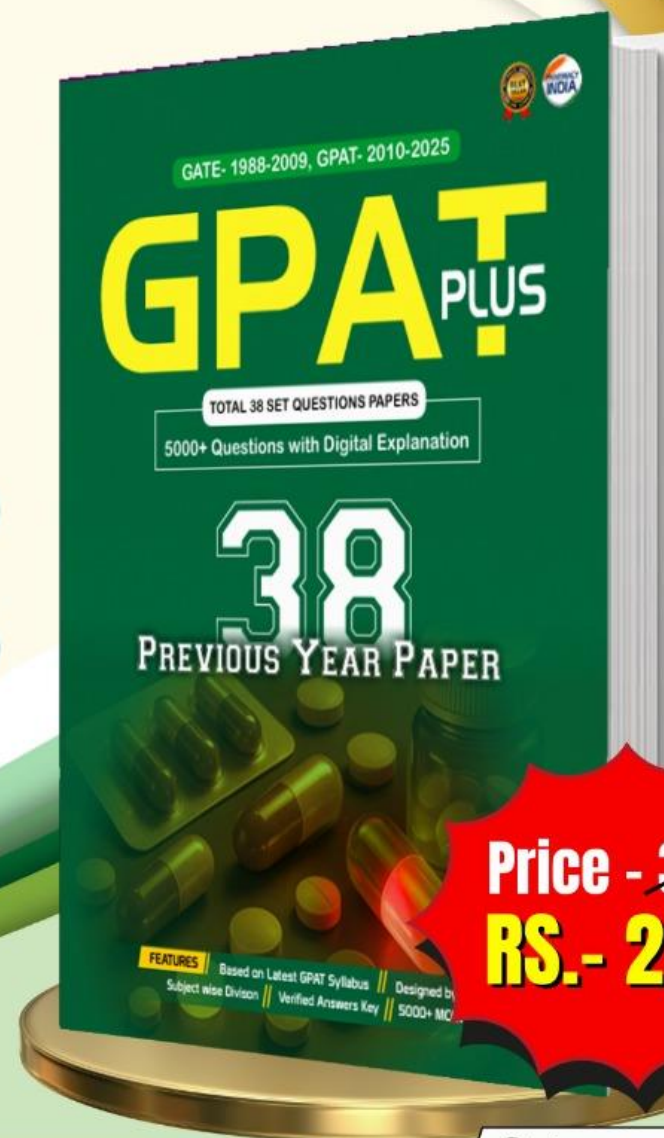
Verified
Questions



Designed
by Experts



Digital
Explanation



Price - ~~399~~
RS.- 299/-



DOWNLOAD PHARMACY INDIA APP FROM PLAYSTORE

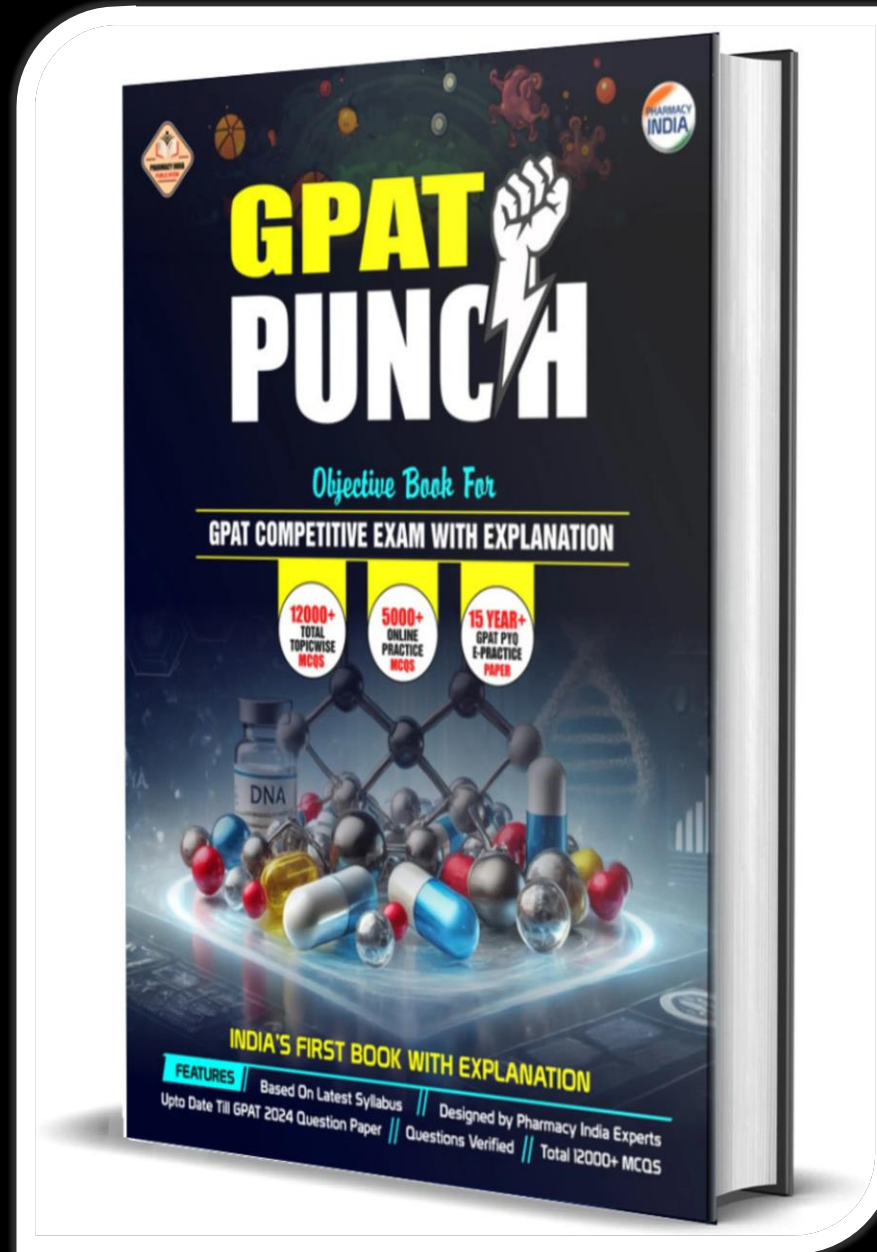


6395596959
8006781759

Best Book for GPAT Exam

AVAILABLE ON

Flipkart  amazon



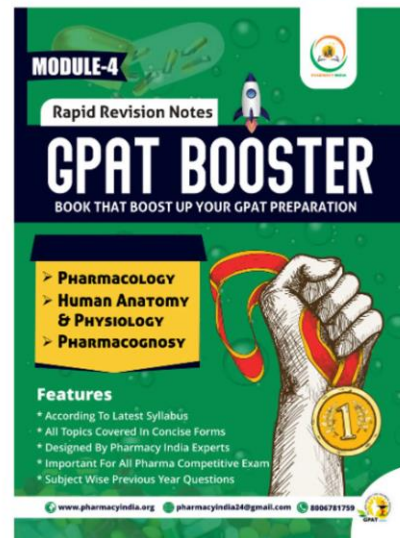
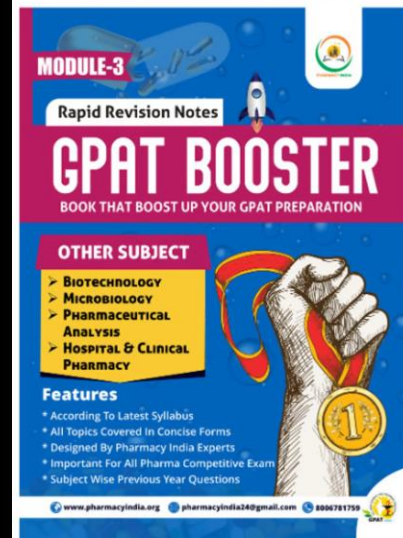
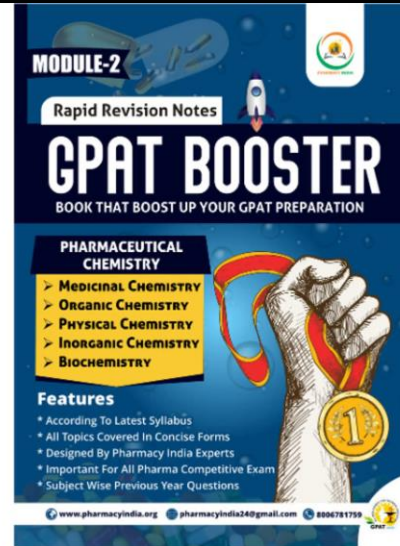
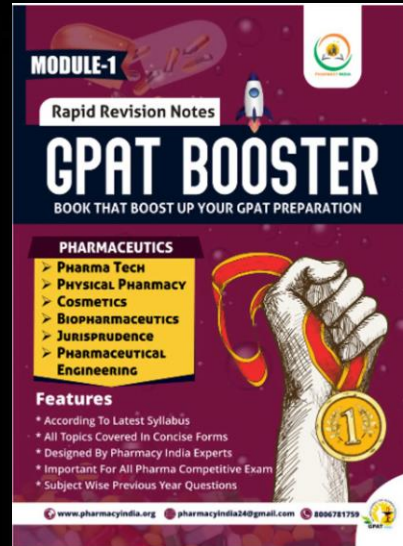
GPAT PUNCH OBJECTIVE BOOK

- Based on Latest Syllabus
- Upto Date Till GPAT 2026 Question Paper
- 5000+ Online Practice MCQs
- Total 12000+ MCQs
- 15 Year+ GPAT PYQ E-Practice Paper

Best Books for GPAT Exam

AVAILABLE ON

Flipkart  amazon



G
P
A
T

GPAT BOOSTER BOOKS

- According to Latest Syllabus
- All Topics Covered in Concise Forms
- Important for GPAT Exam
- Subject-wise Previous Year Questions included
- Includes short notes for rapid last-minute revision

DAILY UPDATES
जुड़िए PHARMACY INDIA
के साथ.....

WHATSAPP & TELEGRAM SE JUDNE KE LIYE
ICONS PAR CLICK KARE

