❤️ Cardiovascular Physiology

🫀 Cardiac Output Basics

📌 Key Formula

- Cardiac Output (CO) = Heart Rate (HR) × Stroke Volume (SV)

💥 Stroke Volume (SV)

- Stroke volume (SV) is the amount of blood pumped out of the heart's left ventricle with each single heartbeat.

📌 Determinants

SV depends on:

- Contractility
- Preload
- Afterload

💡 Memory Trick: "SV CAP"

➡️ Contractility, Afterload, Preload

🔄 Effects on Stroke Volume

Factor	Effect on SV
↑ Contractility (exercise, anxiety)	↑ SV
↑ Preload (early pregnancy)	↑ SV
↓ Afterload	↑ SV

⚙️ Stroke Work (SW)

- Work done by ventricle to eject blood

📌 Relationship:

- SW $\propto$ SV × Mean Arterial Pressure (MAP)

💡 Clinical:

- Heart failure → ↓ SV → ↓ stroke work

💪 Contractility

📌 Definition

- Intrinsic ability of myocardium to contract (independent of preload)

🔼 Factors Increasing Contractility

1. β1 receptor stimulation 💪

β1 receptor stimulation (catecholamines) → ↑ cAMP → Activation of protein kinase A (PKA) →

i. SR effect (storage): Phosphorylation of phospholamban → ↑ SERCA activity → ↑ Ca^{2+} storage in SR

ii. Membrane effect (entry): Phosphorylation of Ca^{2+} channels → ↑ Ca^{2+} influx → ↑ Ca^{2+}-induced Ca^{2+} release from SR

Result: ↑ intracellular Ca^{2+} during systole → ↑ contractility (positive inotropy)

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2. Drugs Increasing Contractility 💊

- Digoxin

- Blocks Na^+/K^+ ATPase → ↑ intracellular Na^+ → ↓ Na^+/Ca^{2+} exchanger → ↑ intracellular Ca^{2+} → ↑ contractility

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🔽 Factors Decreasing Contractility

- β1-blockers
- Systolic heart failure
- Acidosis
- Hypoxia / hypercapnia
- Non-dihydropyridine Ca^{2+} channel blockers (e.g., verapamil)

🫀 Preload

📌 Definition

- Degree of ventricular stretch at end diastole
- Approximated by End-Diastolic Volume (EDV)

🔄 Determinants

- Venous tone
- Blood volume

💊 Clinical

- Venous vasodilators (e.g., nitroglycerin) → ↓ venous return → ↓ preload

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🫀 Afterload

📌 Definition

- Resistance against which ventricle pumps
- Approximated by Mean Arterial Pressure (MAP)

🔄 Mechanism (Laplace Relationship)

Increased pressure in LV (hypertension, aortic stenosis)

→ ↑ wall tension → ↑ afterload

💊 Clinical Effects

- Arterial vasodilators (e.g., hydralazine) → ↓ afterload
- ACE inhibitors / ARBs → ↓ preload + ↓ afterload

🧠 Adaptive Response

Chronic ↑ afterload (e.g., hypertension) → LV hypertrophy → ↓ wall stress (compensation)

❤️ Cardiac Oxygen Demand

📌 Increased by:

- ↑ Contractility
- ↑ Afterload (↑ arterial pressure)
- ↑ Heart rate
- ↑ Ventricular radius (dilation)🫀

📐 Laplace's Law

$T = P \times r$

Where:

- T = wall tension
- P = pressure
- r = radius

📌 Wall Stress

$$\sigma = \frac{pressure \times radius}{2 \times h}$$

Where:

- σ = wall stress
- h = wall thickness

🧠 Key Insights

- ↑ Pressure or ↑ radius → ↑ wall tension
- ↑ Wall thickness (hypertrophy) → ↓ wall stress

💡 This is why LV hypertrophy is compensatory

🩸 Coronary Oxygen Extraction

- Coronary sinus contains most deoxygenated blood in the body 😮
- Because myocardium extracts maximum O_2 at baseline 💡

💡 Takeaways

- SV = CAP (Contractility, Afterload, Preload)
- $\beta 1$ stimulation → ↑ Ca^{2+} → ↑ contractility
- Digoxin → ↑ intracellular Ca^{2+} → ↑ contractility
- Preload = EDV; Afterload = MAP
- Hypertension → LV hypertrophy (compensation)

- O_2 demand depends on pressure, HR, size, contractility
- Laplace law: T = P x r

❤️ Cardiac Output Equations

🫀 Stroke Volume (SV)

- Stroke volume (SV) is the amount of blood pumped out of the heart's left ventricle with each single heartbeat.

📌 Core Equation

SV = EDV - ESV

- EDV (End-Diastolic Volume): volume before contraction

- ESV (End-Systolic Volume): volume after contraction

💡 Interpretation:

- ↑ EDV (preload) → ↑ SV
- ↑ ESV (poor contractility) → ↓ SV → that's why ESV has a "minus" sign in the equation

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💥 Ejection Fraction (EF)

- Ejection fraction (EF) is the percentage of blood pumped out of the left ventricle with each heartbeat

📌 Formula

EF = SV / EDV

🧠 Key Concepts

- Normal EF: 50–70% ✅

- Marker of ventricular contractility

⚠️ Clinical Correlation

Condition	EF
Systolic heart failure	↓ EF 🚨
Diastolic heart failure	Normal EF

💡 Exam trick:

- If EF is normal but patient has HF → think diastolic dysfunction

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❤️ Cardiac Output (CO)

- Cardiac output (CO) is the total volume of blood pumped by the heart's left ventricle per minute

📌 Formula

$CO = SV \times HR$

🧪 Fick Principle

📌 Equation

$$CO = \frac{VO_2}{Ca - Cv}$$

Where:

- VO_2 = oxygen consumption
- $CaO_2 - CvO_2$ = arterial − venous O_2 difference

💡 Used in cardiac catheterization questions

🏃 Exercise Physiology

🔄 CO Changes with Exercise

Early exercise → ↑ HR + ↑ SV → ↑ CO

Later stages → SV plateaus → CO maintained by ↑ HR only

⚠️ High HR Effect

Very high HR (e.g., ventricular tachycardia) → ↓ diastolic filling time → ↓ EDV → ↓ SV → ↓ CO 🚨

💉 Pulse Pressure (PP)

- It represents the force of blood ejected by the heart into the arterial system

📌 Formula

PP = SBP − DBP

🧠 Key Relationships

- PP ∝ Stroke Volume
- PP ∝ 1 / Arterial compliance

🔼 Increased Pulse Pressure

Seen in:

- Aortic regurgitation 💥 (systolic BP increases and diastolic BP decreases → wide pulse pressure)
- Aortic stiffness (aging)
- High-output states (anemia, hyperthyroidism)
- Exercise
- Obstructive sleep apnea

🔽 Decreased Pulse Pressure

Seen in:

- Aortic stenosis
- Cardiogenic shock
- Cardiac tamponade 🚨
- Advanced heart failure

🩺 Mean Arterial Pressure (MAP)

- It reflects the perfusion pressure of organs
- It tells you how well blood is being delivered to tissues

📌 Formula

$MAP = CO \times TPR$

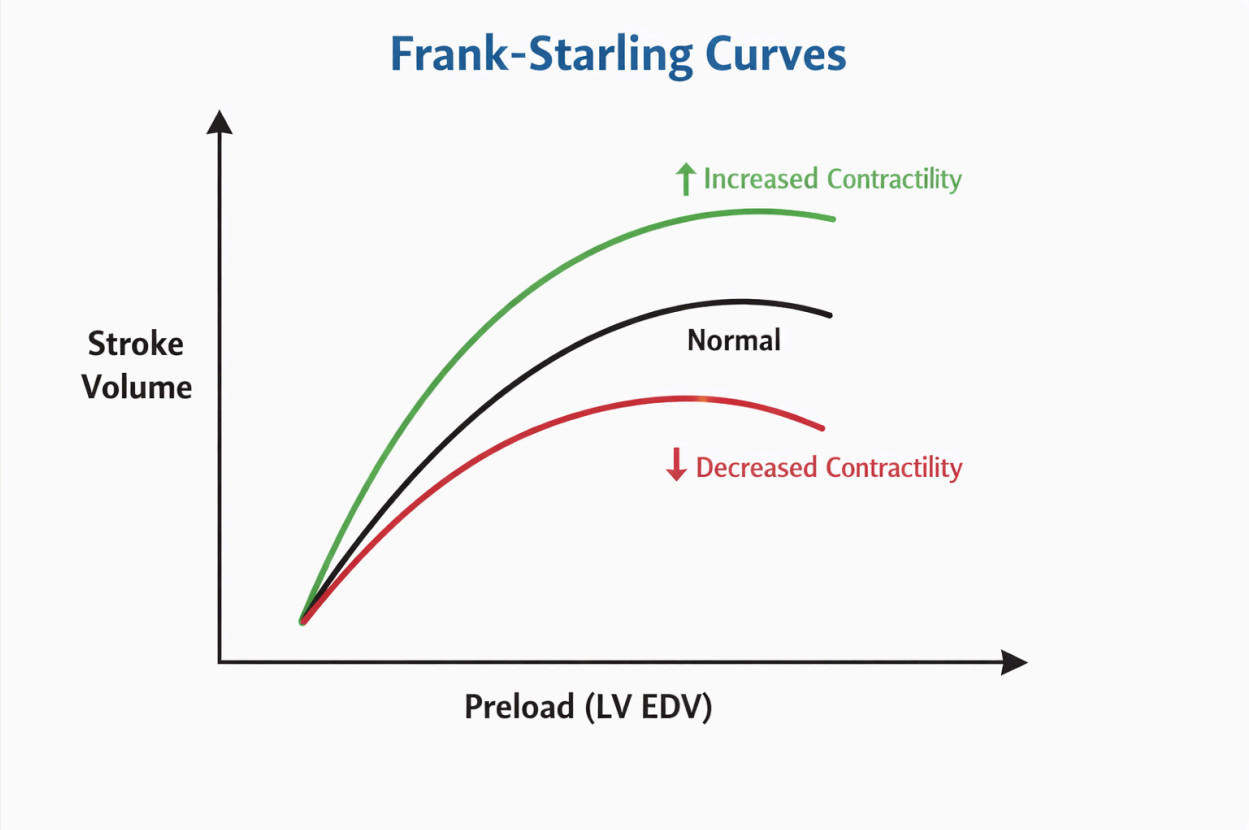
📌 Clinical Approximation

$$MAP = DBP + \frac{1}{3}(SBP - DBP)$$

💡 Interpretation:

- Heavily influenced by diastolic pressure (since diastole lasts longer)

📈 Frank-Starling Relationship



📌 Core Concept

- Force of contraction ∝ preload (EDV)

🔄 Flowchart (Conceptual)

↑ Venous return → ↑ EDV (preload) → ↑ myocardial fiber stretch → ↑ force of contraction → ↑ SV ❤️

📊 Curve Interpretation

Curve Shift	Meaning
Upward shift	↑ Contractility (exercise, inotropes)
Downward shift	↓ Contractility (heart failure)

🧠 Clinical States

- Exercise: shifts curve upward
- Heart failure: downward shift
- Inotropes (dobutamine, digoxin): restore curve upward

💪 Myocardial Contractility

📌 Increased by:

- Catecholamines ($\beta 1$ stimulation)
- Positive inotropes:
  - Dobutamine
  - Milrinone
  - Digoxin

📌 Decreased by:

- Myocardial infarction (loss of muscle)
- β-blockers (acute effect)
- Non-DHP Ca^{2+} channel blockers
- Heart failure

💡 Takeaways

- SV = EDV − ESV
- EF = contractility indicator
- CO = HR × SV
- Fick = gold standard CO measurement
- High HR → ↓ filling → ↓ CO
- PP reflects SV & arterial compliance
- MAP depends on CO + TPR
- Frank-Starling = preload → output relationship

❤️ Resistance, Pressure & Flow

🌊 Flow Fundamentals

📌 Volumetric Flow Rate

$$Q = v \times A$$

Where:

- Q = flow rate
- v = velocity
- A = cross-sectional area

🧠 Key Insight

- Capillaries → highest total cross-sectional area + lowest velocity 🐢
- Allows time for gas & nutrient exchange 💡

🧱 Resistance (Poiseuille's Law)

📌 Core Equation

$$R = \frac{8\eta L}{\pi r 4}$$

🧠 Key Relationships

- R ∝ viscosity (η)
- R ∝ vessel length (L)
- R ∝ 1 / r^4 🚨

💡 Takeaway: Small ↓ in radius → HUGE ↑ in resistance

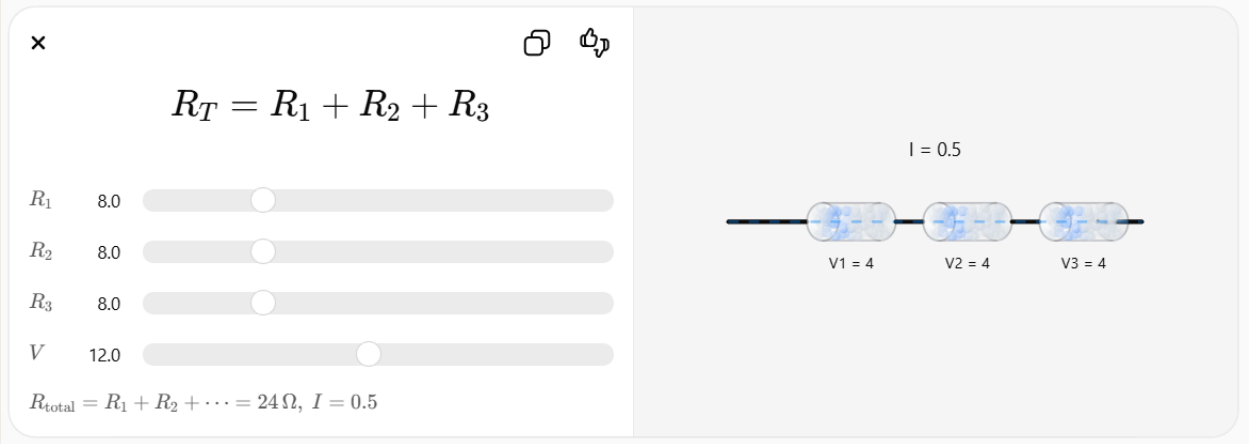
🔥 Radius–Flow Relationship: Q ∝ r

💡 Clinical relevance:

- Vasoconstriction → ↓ radius → ↓ flow dramatically
- Vasodilation → ↑ radius → ↑ flow massively

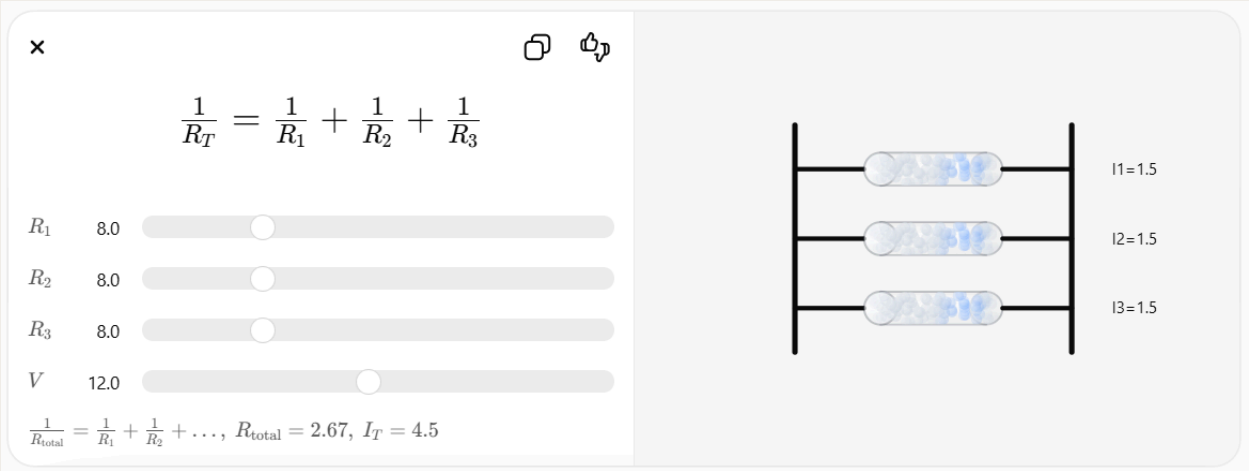
🧩 Resistance in Vessel Networks

📌 Series Arrangement



- Resistance adds up ➕
- Seen in systemic circulation segments (aorta → arteries → arterioles → capillaries → veins)

📌 Parallel Arrangement



- Total resistance decreases
- Adding vessels → ↓ total resistance
- Seen in capillary beds (ensures tissues get adequate perfusion) 💡

🩸 Pressure & Flow

📌 Driving Force

Flow occurs due to pressure gradient:

High pressure → Blood flow → Low pressure

🧠 Vessel Function

Vessel Type	Function
Arterioles	Major contributors to Total Peripheral Resistance (TPR) 🚨
Veins	Major blood reservoir (high capacitance)

🧪 Blood Viscosity

- The thickness or "stickiness" of blood, reflecting its resistance to flow.

📌 Determinants

- Primarily depends on hematocrit

🔼 Increased Viscosity

- Polycythemia
- Hyperproteinemia (e.g., multiple myeloma)

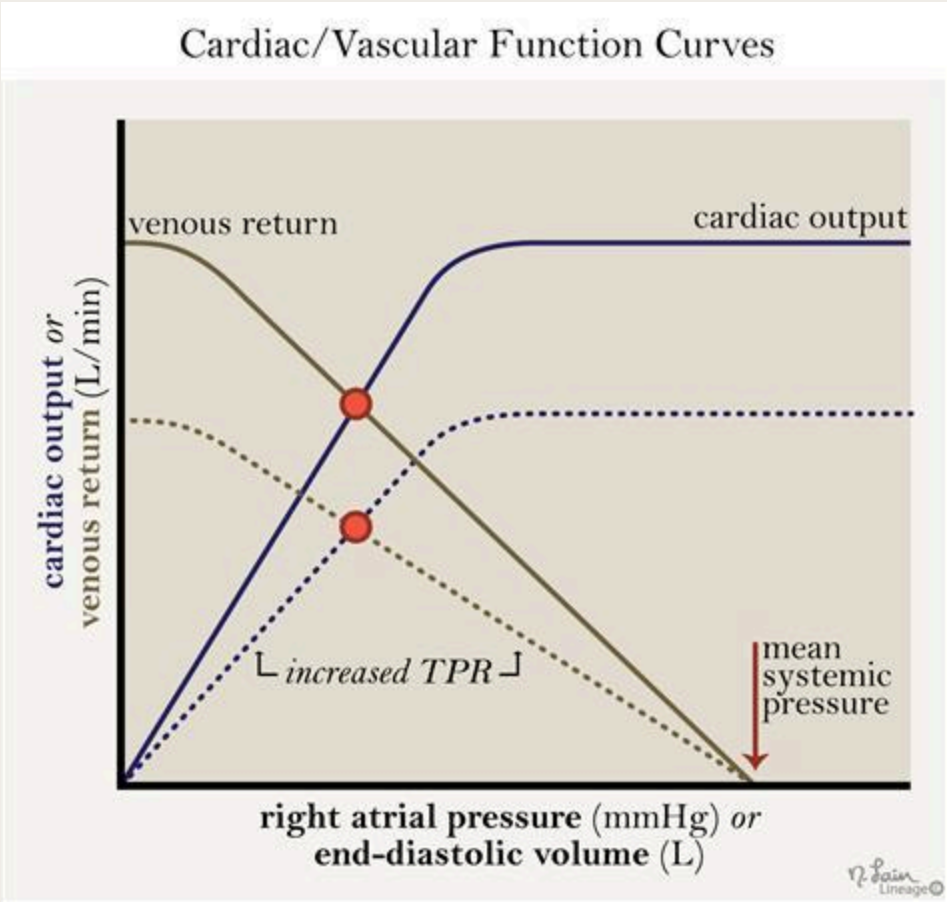
🔽 Decreased Viscosity

- Anemia

💡 Clinical:

- ↑ Viscosity → ↑ resistance → ↓ flow

📈 Cardiac & Vascular Function Curves



Source: step1.medbullets.com

🎯 Key Concept

- Intersection point = operating point of heart
  - Cardiac Output (CO) = Venous Return (VR)
  - System is closed loop

🔄 Understanding the Axes

- X-axis: Right atrial pressure (RAP)
- Y-axis: CO / Venous return

💪 Changes in Inotropy (Contractility)

🔄 Flowchart

↑ Contractility (catecholamines, exercise, digoxin) → ↑ SV → ↑ CO + ↓ RAP (right atrium empties more efficiently)

📊 Effects

Change	Effect

↑ Inotropy	Upward shift of cardiac curve ⬆️
↓ Inotropy (HF, β-blockade)	Downward shift ⬇️

🩸 Changes in Venous Return

🔄 Flowchart

↑ Blood volume / venous tone → ↑ RAP → ↑ preload → ↑ SV → ↑ CO

📊 Examples

Causes of increase in VR	Causes of decrease in VR
Fluid infusion 💧	Hemorrhage 🩸

Sympathetic tone	Spinal anesthesia
AV shunt	-

🧱 Changes in Total Peripheral Resistance (TPR)

🔄 Effects

↑ TPR (vasoconstriction) → ↓ CO → RAP effect variable

or

↓ TPR (exercise, AV shunt) → ↑ CO

📊 Examples

Change	Causes

↑ TPR	Vasopressors
↓ TPR	Exercise, AV shunt

🔄 Integrated Physiologic Responses

1. 🏃 Exercise

Exercise → ↑ inotropy → ↓ TPR → ↑ CO massively 🚀

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2. ❤️ Heart Failure Compensation

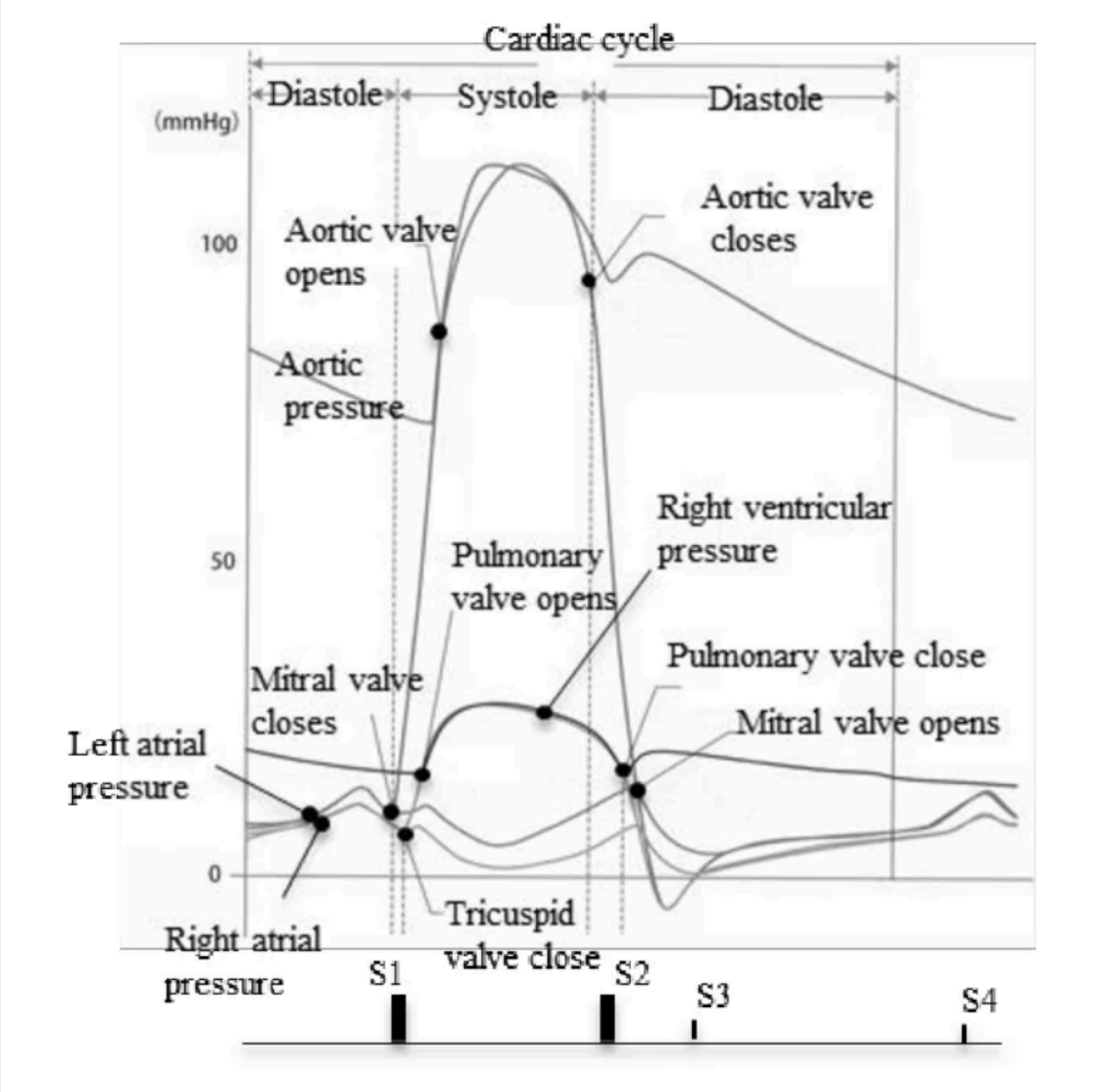
↓ Contractility → ↓ CO → Body compensates:

- Fluid retention
- ↑ preload
- Partial restoration of CO

💡 Takeaways

- Flow = velocity × area
- Resistance $\propto 1/r^4$
- Arterioles = TPR control; veins = blood reservoir
- Capillaries = slowest flow (exchange)
- Viscosity depends on hematocrit
- CO = VR at equilibrium point
- Inotropy, volume, TPR shift curves predictably

❤️ Cardiac Cycle, Heart Sounds & JVP



Source: researchgate.net

🔄 Cardiac Cycle (Left Ventricle Phases)

📌 Sequence Overview

Mitral valve closes (S1) → Isovolumetric contraction → Aortic valve opens → Systolic ejection → Aortic valve closes (S2) → Isovolumetric relaxation → Mitral valve opens → Rapid filling → Reduced filling (diastasis) → Repeat 🔁

🧠 Phase-by-Phase Breakdown

1️⃣ Isovolumetric Contraction

- Between mitral valve closure → aortic valve opening
- Volume constant, pressure rises sharply

💡 Highest O_2 consumption phase 🚨

2️⃣ Systolic Ejection

- Between aortic valve opening → closing

- Blood ejected from LV → aorta

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3 Isovolumetric Relaxation

- Between aortic valve closure → mitral valve opening
- Volume constant, pressure falls

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4 Rapid Filling

- Just after mitral valve opens
- Passive filling of LV

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5 Reduced Filling (Diastasis)

- Slow filling phase before atrial contraction

🔊 Heart Sounds

❤️ S1 — "Lub"

- Closure of mitral + tricuspid valves
- Loudest at apex (mitral area)

❤️ S2 — "Dub"

- Closure of aortic + pulmonary valves
- Loudest at left upper sternal border

⚠️ S3 — Early Diastole

📌 Timing

- During rapid filling phase

🔄 Mechanism

Rapid ventricular filling → Blood hits dilated ventricle → Turbulence → S3 sound

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🧠 Associations

- Volume overload states:
  - Mitral regurgitation
  - Aortic regurgitation
  - Heart failure
  - Thyrotoxicosis

💡 Can be normal in:

- Children
- Young adults
- Athletes
- Pregnancy

⚠️ S4 — Late Diastole

📌 Timing

- During atrial contraction ("atrial kick")

🔄 Mechanism

Atrial contraction → Blood forced into stiff LV →

Turbulence → S4 sound

🧠 Associations

- Decreased compliance:
  - LV hypertrophy
  - Chronic hypertension

💡 Always pathologic if palpable

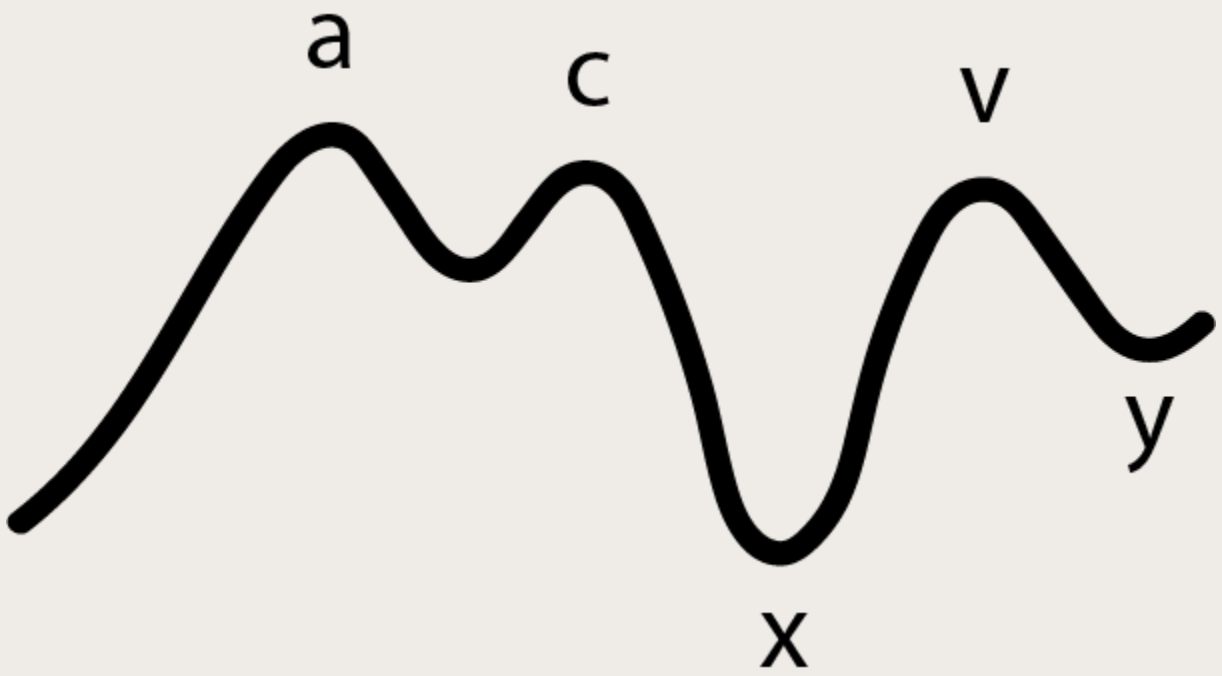
🆚 S3 vs S4 (Exam Favorite!)

Feature	S3	S4
Timing	Early diastole	Late diastole
Cause	Volume overload	Stiff ventricle
Normal?	Sometimes	Usually abnormal
Mnemonic	"Ken-tuc-ky" (S1-S2-S3)	"Ten-nes-see" (S4-S1-S2)

🩺 Jugular Venous Pulse (JVP)

Reflects right atrial pressure changes — frequently tested 📈

🔄 JVP Waveform



Source: clinicianrevision.com

a wave — Atrial contraction

- ↑ in AV dissociation → cannon a waves 💥
- Absent in atrial fibrillation

c wave — RV contraction

- Bulging of closed tricuspid valve into RA

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x descent — Atrial relaxation

Mechanism:

RA relaxation → ↓ RA pressure + downward pull of tricuspid valve during RV systole

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⚠️ Clinical

- ↓/absent in:
  - Tricuspid regurgitation
  - Right heart failure

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v wave — Venous filling of RA

- Occurs against closed tricuspid valve
- Reflects ↑ RA pressure

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y descent — RA emptying

Tricuspid valve opens → RA empties into RV → ↓ RA pressure

⚠️ Clinical Correlations

Finding	Condition
Prominent y descent	Constrictive pericarditis
Absent y descent	Cardiac tamponade 🚨

🧠 Integrated Clinical Insights

1. 🫀 Cardiac Tamponade

- ↓ y descent
- Equalization of pressures
- Impaired filling

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2. 🧱 Constrictive Pericarditis

- Prominent y descent
- Rapid early filling, then abrupt stop

💡 Final High-Yield Takeaways

- S1 = AV valve closure; S2 = semilunar closure

- S3 = volume overload (can be normal)
- S4 = stiff ventricle (pathologic)
- Isovolumetric contraction = highest O2 demand
- JVP reflects RA pressure dynamics

⚠️ Abnormal JVP Patterns

🩺 Jugular Venous Pulse (JVP) Patterns

📌 Normal JVP Components

a wave → atrial contraction

c wave → RV contraction (tricuspid bulging)

x descent → atrial relaxation

v wave → venous filling of RA

y descent → RA emptying into RV

1️⃣ Atrial Fibrillation

❌ Absent a waves

Why?

No organized atrial contraction → No "a wave"

2️⃣ Tricuspid Regurgitation

❌ Absent x descent

RV contracts → Blood leaks back into RA → RA pressure does NOT fall normally → Loss of x descent

💡 Often also see prominent v waves

3️⃣ Constrictive Pericarditis

⬆️ Prominent y descent

Tricuspid valve opens → Rapid early filling → Abrupt stop (rigid pericardium) → Sharp y descent

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4️⃣ Cardiac Tamponade 🚨

❌ Absent y descent

Fluid compresses heart → Impaired ventricular filling → RA cannot empty → No y descent

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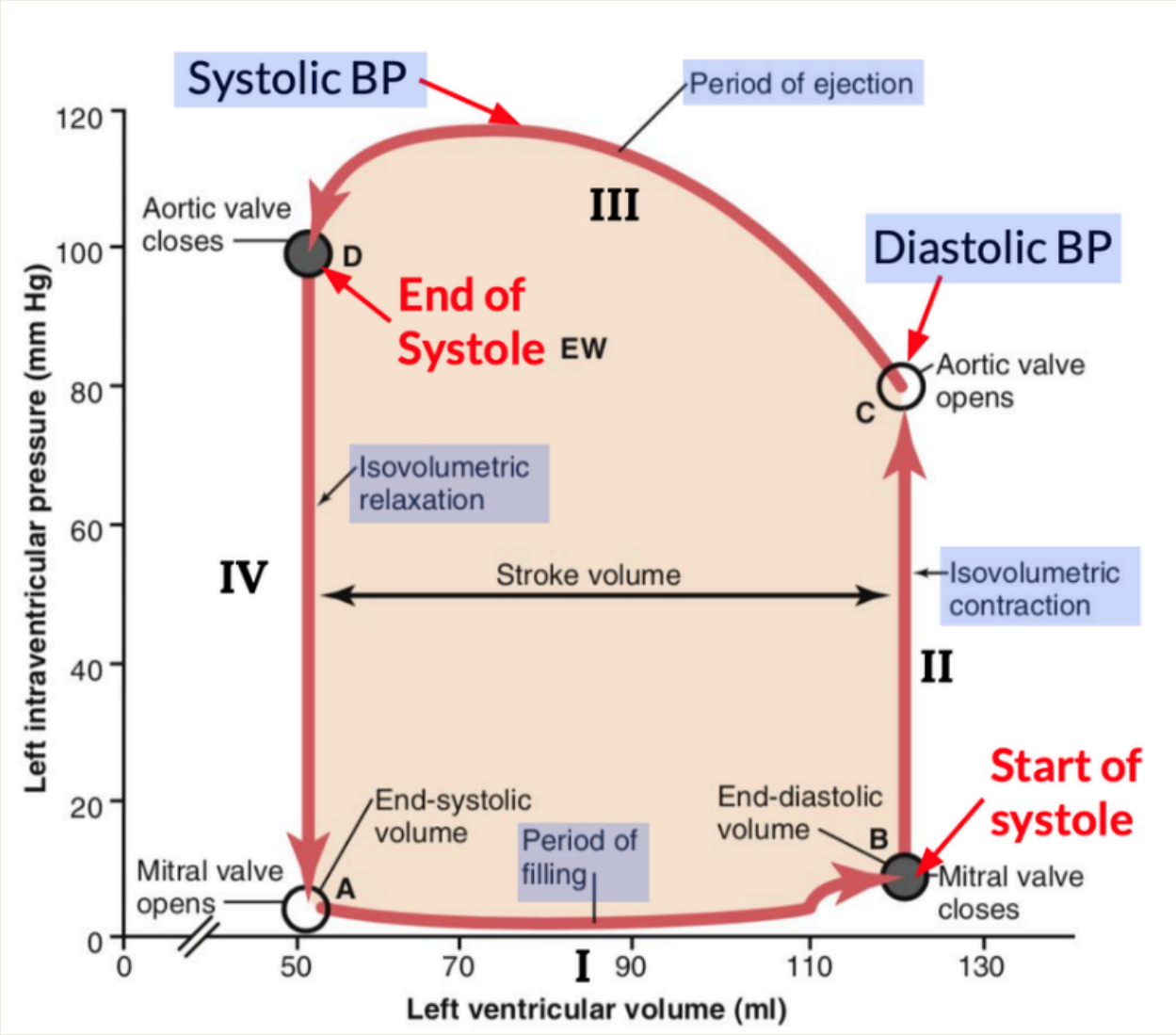
🧠 Quick Comparison Table

Condition	Key JVP Finding
Atrial fibrillation	Absent a wave

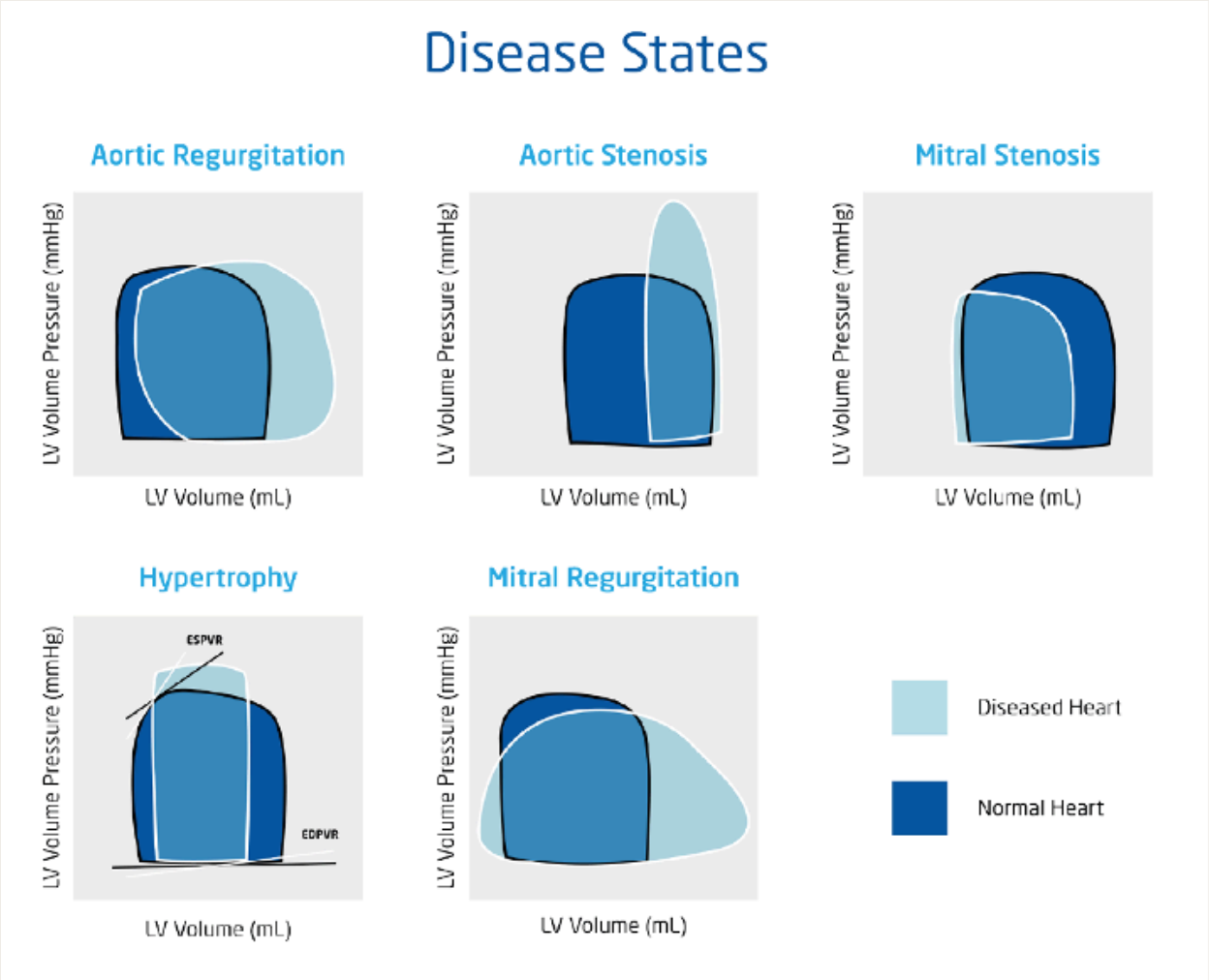
Tricuspid regurgitation	Absent x descent
Constrictive pericarditis	Prominent y descent
Cardiac tamponade	Absent y descent

📈 Pressure-Volume (PV) Loops & Valvular Disease

Normal:



Source: researchgate.net



Source: en.rattibha.com

🔴 Aortic Stenosis (AS)

🔄 Pathophysiology Flowchart

Narrow aortic valve → ↑ resistance to ejection → ↑ LV pressure needed

📊 PV Loop Changes

- ↑ LV pressure 🚨
- ↑ ESV (can't empty well)
- Normal EDV (early)
- ↓ SV

🧠 Adaptation

↑ Afterload → LV hypertrophy → ↓ compliance → ↑ end-diastolic pressure (EDP)

🔵 Aortic Regurgitation (AR)

🔄 Pathophysiology Flowchart

Aortic valve incompetence → Blood leaks back into LV during diastole → Volume overload

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📊 PV Loop Changes

- ↑ EDV (volume overload)
- ↑ SV (forward + backward flow)
- ❌ No true isovolumetric phases
- Wide pulse pressure 💥
- Loss of dicrotic notch

🟡 Mitral Stenosis (MS)

🔄 Pathophysiology Flowchart

Narrow mitral valve → Impaired LV filling → ↑ LA pressure

📊 PV Loop Changes

- ↑ LA pressure
- ↓ EDV (less filling)
- ↓ ESV
- ↓ SV

🟢 Mitral Regurgitation (MR)

🔄 Pathophysiology Flowchart

Mitral valve incompetence → Blood flows back into LA during systole → Volume overload of LA + LV

📊 PV Loop Changes

- ↑ EDV (more filling)

- ↓ ESV (less resistance)
- ↑ total SV (forward + backward)
- ❌ No true isovolumetric phases

🧠 Classic Finding

- Tall V waves in JVP 🚨

🔥 High-Yield Comparison Table

Disease	EDV	ESV	SV	Special Feature
Aortic stenosis	Normal	↑	↓	↑ LV pressure
Aortic regurgitation	↑	—	↑	Wide PP, no iso phases
Mitral stenosis	↓	↓	↓	↑ LA pressure

Mitral regurgitation	↑	↓	↑	Tall V wave

🧠 Exam Tips

- No isovolumetric phase → think regurgitation 🚨
- Pressure overload → stenosis → ↑ ESV
- Volume overload → regurgitation → ↑ EDV
- Wide pulse pressure → aortic regurgitation

💡 Final High-Yield Takeaways

- JVP patterns = diagnostic clues
- Tamponade vs constriction → y descent difference
- PV loops test:
  - Pressure vs volume changes
  - Presence/absence of isovolumetric phases

- Regurgitation = volume overload
- Stenosis = pressure overload

❤️ Splitting of S2 (A2 & P2)

📣 What is S2?

- S2 = closure of semilunar valves:
  - A2 → Aortic valve closure
  - P2 → Pulmonic valve closure

💡 Normally: A2 occurs before P2

🌬️ Physiologic Splitting (NORMAL)

🔄 Mechanism (Flowchart)

Inspiration → ↓ Intrathoracic pressure → ↑ Venous return to right heart → ↑ RV filling → ↑ RV stroke volume → ↑ RV ejection time → Delayed P2

🧠 Additional Factor

- ↓ Pulmonary impedance during inspiration → further delays P2

📌 Result

- Split S2 during inspiration
- Single S2 during expiration

🔥 Wide Splitting

📌 Definition

- Exaggeration of normal physiologic splitting

🔄 Mechanism

Delayed RV emptying → Delayed P2 (more than normal)

⚠️ Causes

- Pulmonic stenosis
- Right bundle branch block (RBBB)

🧠 Key Feature

- Split present in both inspiration & expiration
- Wider during inspiration

🟡 Fixed Splitting 🚨

📌 Classic Cause

- Atrial Septal Defect (ASD)

🔄 Mechanism (Flowchart)

Left → right shunt → ↑ RA volume → ↑ RV volume →

↑ flow through pulmonic valve → Delayed P2 (constant)

📌 Result

- Split S2 does NOT change with respiration ❗

💡 This is a buzzword finding for ASD

🔴 Paradoxical (Reversed) Splitting 🚨

📌 Definition

- P2 occurs before A2 (reverse of normal)

🔄 Mechanism

Delayed LV emptying → Delayed A2

⚠️ Causes

- Aortic stenosis
- Left bundle branch block (LBBB)

🔄 Unique Pattern (Flowchart)

Expiration → Split heard (A2 delayed)

Inspiration → P2 delayed → Moves closer to A2 → Split disappears ("paradoxical")

🧠 Quick Comparison Table

Type	Cause	Key Feature
Physiologic	Normal	Split on inspiration only
Wide	Delayed RV emptying (RBBB, PS)	Always split, wider on inspiration
Fixed	ASD	No change with respiration 🚨
Paradoxical	Delayed LV emptying (AS, LBBB)	Split on expiration only

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🎯 Exam Tips

- ASD → fixed splitting (automatic answer)
- RBBB → wide splitting
- Aortic stenosis → paradoxical splitting
- If split disappears on inspiration → paradoxical

💡 Takeaways

- Inspiration delays P2 → physiologic split
- Right-sided problems → wide splitting
- ASD → fixed splitting (no respiratory variation)
- Left-sided delay → paradoxical splitting

❤️ Auscultation of the Heart

📍 Where to Listen (APT M Rule)

💡 Mnemonic: "APT M" (from right → left → apex)

Area	Location	Valves Best Heard
Aortic (A)	Right 2nd intercostal space	Aortic valve
Pulmonic (P)	Left 2nd intercostal space	Pulmonic valve
Tricuspid (T)	Left lower sternal border	Tricuspid valve
Mitral (M)	Apex (5th ICS, midclavicular line)	Mitral valve ❤️

🔊 Murmurs by Location

🟠 Aortic Area

Systolic murmurs:

- Aortic stenosis 🚨
- Flow murmur (physiologic)
- Aortic valve sclerosis

🔵 Pulmonic Area

Systolic murmurs:

- Pulmonic stenosis
- Atrial septal defect (flow murmur)
- Physiologic flow murmurs

🟢 Tricuspid Area

Holosystolic murmurs:

- Tricuspid regurgitation
- Ventricular septal defect (VSD)

Diastolic murmurs:

- Tricuspid stenosis

🔴 Mitral Area (Apex)

Systolic murmurs:

- Mitral regurgitation
- Mitral valve prolapse (MVP)

Diastolic murmurs:

- Mitral stenosis

🟣 Left Sternal Border (LSB)

Systolic:

- Hypertrophic cardiomyopathy (HCM) 🚨

Diastolic:

- Aortic regurgitation
- Pulmonic regurgitation

🧠 High-Yield Pattern Recognition

Murmur Type	Common Cause
Holosystolic	MR, TR, VSD
Crescendo-decrescendo systolic	Aortic stenosis
Diastolic rumble	Mitral stenosis
Blowing diastolic	Aortic regurgitation

🔄 Maneuvers & Murmur Changes 🚨

🔻 ↓ Preload Maneuvers

Standing / Valsalva (strain phase)

Effect: ↓ venous return → ↓ LV volume

🔼 Murmurs that INCREASE:

- Hypertrophic cardiomyopathy (HCM)
- Mitral valve prolapse (MVP)
  - Earlier click ⏱️

🔽 Murmurs that DECREASE:

- Most others (AS, MR, AR, etc.)

🔺 ↑ Preload Maneuvers

Passive Leg Raise → ↑ venous return → ↑ LV volume

🔼 Murmurs that INCREASE:

- Most murmurs (AS, MR, AR, VSD)

🔽 Murmurs that DECREASE:

- HCM
- MVP (later click)

🟫 Squatting → ↑ preload + ↑ afterload

🔼 Murmurs that INCREASE:

- Most murmurs

🔽 Murmurs that DECREASE:

- HCM
- MVP

❓ Why HCM/MVP are Special

Feature	Most Murmurs	HCM / MVP
Effect of ↓ preload	↓ velocity/gradient → softer	LV size ↓ → obstruction/prolapse ↑ → louder
Mechanism	Less flow → less turbulence	Geometry-dependent, dynamic obstruction

✊ Handgrip → ↑ ↑ afterload

🔼 Murmurs that INCREASE:

- Mitral regurgitation
- Aortic regurgitation
- VSD

💡 Why? → ↑ resistance → ↑ backward flow

🔽 Murmurs that DECREASE:

- Aortic stenosis
- HCM

🌬️ Inspiration

↑ venous return to right heart + ↓ to left heart

🔼 Murmurs that INCREASE:

- Right-sided murmurs (TR, PS)

🔽 Murmurs that DECREASE:

- Left-sided murmurs

🧠 Concept

Handgrip

↑ Afterload → Harder for LV to eject forward → More blood regurgitates backward → ↑ MR / AR intensity 🔊

🎯 Exam Strategy

- Handgrip → think regurgitation louder
- Inspiration → right-sided murmurs louder
- Valsalva → HCM louder (classic question)

💡 Takeaways

- APT M = auscultation map
- Location + timing = diagnosis clue
- Maneuvers change preload/afterload → change murmur intensity
- HCM & MVP = exceptions (increase with ↓ preload)

- Right-sided murmurs ↑ with inspiration

🎧 Heart Murmurs

🔊 SYSTOLIC MURMURS

🔴 Aortic Stenosis (AS)

📌 Auscultation

- Crescendo-decrescendo ejection murmur
- Loudest at aortic area (base)
- Radiates to carotids 🚨

🧠 Key Findings

- Soft/absent S2
- ± Ejection click
- Pulsus parvus et tardus (weak, delayed pulse)

🔄 Pathophysiology

Narrow aortic valve → LV must generate high pressure

→ LV pressure > aortic pressure during systole

⚠️ Causes

- Elderly → calcific degeneration
- Young → bicuspid aortic valve

🚨 Classic Triad

- Syncope
- Angina
- Dyspnea on exertion

  👉 "SAD"

🔵 Mitral Regurgitation (MR) / Tricuspid Regurgitation (TR)

📌 Auscultation

- Holosystolic, blowing murmur

Valve	Best Heard	Radiation
MR	Apex	Axilla
TR	Tricuspid area	—

🔄 Mechanism

Valve incompetence → Backflow during systole →

Continuous murmur throughout systole (holosystolic)

⚠️ Causes

MR:

- Ischemic heart disease (post-MI)
- Mitral valve prolapse
- LV dilation
- Rheumatic fever

TR:

- RV dilation

Both:

- Infective endocarditis

🟡 Mitral Valve Prolapse (MVP)

📌 Auscultation

- Mid-systolic click (MC)
- Followed by late systolic murmur
- Best heard at apex

🔄 Mechanism

Mitral valve prolapses into LA → Sudden tension on chordae → Click sound → Regurgitation → murmur

---

🧠 Associations

- Usually benign
- Can lead to infective endocarditis

💡 Causes

- Myxomatous degeneration
- Connective tissue disorders
- Rheumatic disease

🟢 Ventricular Septal Defect (VSD)

📌 Auscultation

- Holosystolic, harsh murmur
- Loudest at tricuspid area

🧠 Key Insight

- Small VSD → louder murmur 🔊
- Large VSD → softer murmur

💡 Why? → Large defect = less turbulence

💡 Mnemonic: "Sometimes, *I watch MMA on TV*"

- S = Systolic
- MVP
- MR
- AS
- TR
- VSD

🔊 DIASTOLIC MURMURS

🔴 Aortic Regurgitation (AR)

📌 Auscultation

- Early diastolic, decrescendo blowing murmur
- Heard at:
    - Base (aortic root dilation)
    - Left sternal border

🔄 Mechanism

Aortic valve incompetence → Blood leaks back into LV during diastole

---

⚠️ Causes (BEAR)

- Bicuspid valve
- Endocarditis
- Aortic root dilation
- Rheumatic fever

🧠 Classic Signs

- Wide pulse pressure 💥
- Bounding pulses ("water hammer")
- Quincke pulse (nail bed pulsation)
- Head bobbing (severe cases)

⚠️ Complication → Left heart failure

🟣 Mitral Stenosis (MS)

📌 Auscultation

- Opening snap (OS) after S2
- Followed by rumbling mid-late diastolic murmur

🧠 Key Insight

- Shorter S2–OS interval = more severe MS 🚨

🔄 Mechanism

Narrow mitral valve → ↑ LA pressure → ↓ LV filling

⚠️ Associations

- Rheumatic fever (classic cause)

🧠 Complications

- LA enlargement
- Atrial fibrillation
- Pulmonary congestion

- Hemoptysis
- Ortner syndrome (hoarseness)
- Right heart failure

🔁 CONTINUOUS MURMUR

⚙️ Patent Ductus Arteriosus (PDA)

📌 Auscultation

- Continuous "machine-like" murmur 🤖
- Best heard at left infraclavicular area
- Loudest at S2

🔄 Mechanism

Persistent connection → Aorta → Pulmonary artery → Continuous flow

---

⚠️ Causes

- Prematurity
- Congenital rubella

💡 Memory Trick

👉 "You need a patent for that machine"

🧠 Summary Table

Murmur	Timing	Key Feature

AS	Systolic	Crescendo-decrescendo systolic murmur with pulsus parvus et tardus, i.e. weak, delayed carotid upstroke, carotid radiation
MR	Systolic	Holosystolic murmur that radiates to the axilla
MVP	Systolic	Mid-systolic click, often followed by a late systolic murmur
VSD	Systolic	Harsh holosystolic murmur, loudest at the left lower sternal border
AR	Diastolic	High-pitched, decrescendo diastolic murmur, best heard at the left sternal border with the patient leaning forward, wide PP
MS	Diastolic	Low-pitched, mid- diastolic rumble following an Opening Snap (OS)
PDA	Continuous	Machine-like
TR	Systolic	Holosystolic murmur that increases with inspiration, Carvallo's sign

🎯 Takeaways

- Systolic = AS, MR, MVP, VSD,TR
- Diastolic = AR, MS (always pathologic!) 🚨
- Click = MVP; Snap = MS
- Wide pulse pressure = AR
- Machine murmur = PDA

❤️ Myocardial Action Potential

⚡ Ventricular (Non-Pacemaker) Action Potential

🔄 Phases Overview (0 → 4)

Phase 4 (resting) → Phase 0 (rapid depolarization) → Phase 1 (initial repolarization) → Phase 2 (plateau) → Phase 3 (repolarization) → Back to Phase 4 🔁

🧠 Phase-by-Phase Breakdown

0️⃣ Phase 0 — Rapid Depolarization

- Opening of fast voltage-gated Na^+ channels → Massive Na^+ influx → Rapid upstroke ⚡

⚡ Phase 1 — Initial Repolarization

- Na^+ channels inactivate
- K^+ channels begin to open
- Brief drop in membrane potential

⚡ Phase 2 — Plateau ("Platwo") 🚨

- Ca^{2+} influx (L-type channels)
- Balanced by K^+ efflux

🔄 Mechanism

Ca^{2+} enters cell → Triggers Ca^{2+} release from SR → Excitation-contraction coupling 💪

⚡ Phase 3 — Rapid Repolarization

- Ca^{2+} channels close
- K^+ channels fully open
- Massive K^+ efflux
- Return to negative potential

⚡ Phase 4 — Resting Potential

- High K^+ permeability
- Stable resting membrane (~ -85 mV)

⛔ Effective Refractory Period (ERP)

"The ERP is the time during which a cardiac cell cannot generate another action potential, no matter how strong the stimulus is"

- Occurs during phases 0 → early 3
- Prevents tetany

⚡ Why does it happen?

- Due to inactivation of fast Na^+ channels
- During plateau (Ca^{2+} influx), membrane stays depolarized → Na^+ channels remain inactive

- Cell cannot re-excite

🚫 Key function: Prevents tetany

- In skeletal muscle → repeated stimulation = tetany
- In cardiac muscle → ERP is long, so:
  - No summation
  - No sustained contraction

👉 Heart must relax before next beat

💡 Why important?

- Allows proper filling & coordinated contraction

🧠 Key Differences vs Skeletal Muscle

Feature	Cardiac Muscle	Skeletal Muscle

Plateau phase	Present 🚨	Absent
Ca^{2+} source	ECF + SR	SR only
Tetany	Impossible	Possible
Cell connection	Gap junctions	Independent

⚡ Pacemaker Action Potential (SA & AV Nodes)

📌 Key Differences

- No phases 1 & 2 ❌
- No fast Na^{+} channels ❌
- Automatic depolarization ✔️

🔄 Pacemaker Phases

⚡ Phase 4 — Spontaneous Depolarization 🚨

- Due to If ("funny current")

🔄 Mechanism

If channels open → Na^+ slowly enters → Gradual depolarization → Reaches threshold

---

🧠 Clinical Importance

- Slope of phase 4 = heart rate ❤️

🧪 Modulation

Factor	Effect
Sympathetic (catecholamines)	↑ slope → ↑ HR
Parasympathetic (ACh, adenosine)	↓ slope → ↓ HR

⚡ Phase 0 — Depolarization

- Opening of Ca^{2+} channels (NOT Na^{+}!) → Slow upstroke

💡 Explains:

- Slow conduction in AV node
- Allows ventricular filling time

⚡ Phase 3 — Repolarization

- Ca^{2+} channels close

- K^+ efflux increases
- Repolarization

🧠 Ventricular vs Pacemaker AP

Feature	Ventricular	Pacemaker
Phase 0 ion	Na^+	Ca^{2+}
Plateau	Present	Absent
Automaticity	No	Yes
Phase 4	Flat	Sloped 🚨

🔄 Integrated Concept

Sympathetic Stimulation

↑ β1 activity → ↑ If channel opening → ↑ slope of phase 4 → ↑ HR

Parasympathetic Stimulation

ACh release → ↓ If current → ↓ slope → ↓ HR

💡 Takeaways

- Phase 0 (ventricle) = Na^+; Phase 0 (node) = Ca^{2+} 🚨
- Phase 2 plateau = Ca^{2+} influx → contraction
- ERP prevents tetany in cardiac muscle
- Pacemaker cells = automatic due to If current
- Slope of phase 4 determines heart rate
- Sympathetic ↑ HR; parasympathetic ↓ HR

❤️ Electrocardiogram (ECG)

⚡ Cardiac Conduction Pathway

🔄 Sequence

SA node → Atria → AV node → Bundle of His → Right & Left bundle branches → Purkinje fibers → Ventricles ❤️

🧠 Key Anatomy

🔹 SA Node (Pacemaker)

- Located near SVC opening (crista terminalis)
- Fastest intrinsic rate → dominant pacemaker
- Blood supply: RCA

🔹 AV Node

- Located in interatrial septum near coronary sinus
- Blood supply: usually RCA

⏱️ Critical Function

AV node delay (~100 ms) → Allows ventricular filling before contraction

---

⏱️ Pacemaker Rates (Hierarchy)

Structure	Rate
SA node	60–100 bpm ❤️

AV node	40–60 bpm
His-Purkinje	20–40 bpm

💡 Fastest wins → SA node normally controls rhythm

⚡ Conduction Velocity

📌 Order (FAST → SLOW)

His-Purkinje > Atria > Ventricles > AV node

💡 Mnemonic: "He Parks At Ventura Avenue" 🚗

📈 ECG Wave Components

🔹 P Wave

- Atrial depolarization

🔹 PR Interval

- From start of P → start of QRS

📌 Normal:

- 120–200 ms

🧠 Represents:

Atrial depolarization

- AV node delay

🔹 QRS Complex

- Ventricular depolarization

📌 Normal:

- < 100 ms

💡 Wide QRS → think bundle branch block

🔹 QT Interval

- Entire ventricular activity:
  - Depolarization
  - Contraction
  - Repolarization

🔹 T Wave

- Ventricular repolarization

⚠️ Clinical Clue

- T-wave inversion → ischemia or recent MI 🚨

🔹 ST Segment

- Isoelectric (flat)
- Ventricles fully depolarized

⚠️ Clinical

- ST elevation → MI
- ST depression → ischemia

🔹 J Point

- Junction between:
    - End of QRS
    - Start of ST segment

🔹 U Wave

📌 Seen in:

- Hypokalemia
- Bradycardia

💡 Mnemonic: "hypo-K → U wave" ("*hypo-UK*")

🔄 Integrated ECG Flow

SA node fires → Atrial depolarization (P wave) → AV delay (PR interval) → Ventricular depolarization (QRS) →

Ventricular contraction → Ventricular repolarization (T wave)

🧠 Clinical Connections

⚠️ AV Node Blood Supply

- Usually from RCA → if RCA infarct → heart block

⚠️ Bundle Branch Blocks

- Cause wide QRS (>100 ms)

⚠️ Electrolyte Changes

Condition	ECG Change

Hypokalemia	U waves
Ischemia	T-wave inversion
MI	ST elevation

💡 Takeaways

- SA → AV → His → Purkinje → ventricles
- AV node delay = critical for filling
- PR = AV conduction time
- QRS = ventricular depolarization
- QT = total ventricular activity
- U wave = hypokalemia clue
- AV node supplied by RCA → infarct = conduction issues

❤️ ANP, BNP & Reflex Control Systems

💧 Atrial Natriuretic Peptide (ANP)

📌 Source

- Released from atrial myocytes

🔄 Trigger (Flowchart)

↑ Blood volume / ↑ atrial pressure → Atrial stretch → ANP release

⚙️ Mechanism

- Acts via cGMP

🎯 Effects

🫀 Cardiovascular

- Vasodilation ↓ BP

🧂 Renal

- ↓ Na^+ reabsorption (collecting tubule) → ↑ Natriuresis → ↑ Diuresis 💧

🧠 Renal Hemodynamics

Afferent arteriole dilation

- Efferent arteriole constriction → ↑ GFR → ↑ urine output

💡 Special Concept

Aldosterone Escape 🚨 → ANP overrides aldosterone → Prevents excessive Na^+ retention

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🧠 B-type Natriuretic Peptide (BNP)

📌 Source

- Released from ventricular myocytes

🔄 Trigger

↑ Ventricular wall tension → BNP release

🎯 Effects

- Same as ANP:
  - Vasodilation
  - ↑ Natriuresis
  - ↓ Blood volume

🧪 Clinical Importance 🚨

- BNP test → Heart failure diagnosis

💡 Key Point:

- High BNP → suggests HF
- Low BNP → rules OUT HF (high negative predictive value)

🧠 ANP vs BNP Table

Feature	ANP	BNP
Source	Atria	Ventricles
Trigger	Volume ↑	Pressure/tension ↑
Half-life	Short	Longer 🚨
Clinical use	Limited	HF diagnosis

🧠 Baroreceptors & Chemoreceptors

📍 Key Locations

Receptor	Location	Nerve
Carotid sinus	Carotid bifurcation	CN IX (Glossopharyngeal)

Aortic arch	Aortic arch	CN X (Vagus)

🧠 Central Integration

- Signals → Nucleus Tractus Solitarius (medulla)

⚡ Baroreceptor Reflex

🔻 Hypotension Response 🚨

🔄 Flowchart

↓ BP → ↓ arterial stretch → ↓ baroreceptor firing →
↑ sympathetic + ↓ parasympathetic →

- Vasoconstriction
- ↑ HR ❤️

- ↑ Contractility

Result: ↑ BP

🧠 Clinical Relevance

- Critical in hypovolemic shock

✋ Carotid Massage

🔄 Mechanism

↑ carotid sinus pressure → ↑ baroreceptor firing →
↑ vagal tone → ↓ AV node conduction → ↓ HR

⚠️ Clinical Use

- Can terminate supraventricular tachycardia (SVT)

⚠️ Risk

- Syncope (especially elderly/atherosclerosis)

🫁 Chemoreceptors

🔹 Peripheral Chemoreceptors

📍 Location

- Carotid bodies
- Aortic bodies

🔄 Stimuli

- ↑ PCO_2
- ↓ pH
- ↓ PO_2 (< 60 mmHg) 🚨

🔹 Central Chemoreceptors

📍 Location

- Medulla

🔄 Stimulus

↑ CO_2 → Diffuses into brain → ↓ pH (via H^+ generation) → Stimulates receptors

---

❗ Important Point

- DO NOT directly respond to O_2

⚠️ COPD Adaptation

Chronic ↑ CO_2 → Central receptors become less sensitive → Body relies on low O_2 (peripheral receptors)

🚨 Cushing Reflex

🔄 Flowchart

↑ Intracranial pressure (ICP) → ↓ cerebral perfusion → ↑ CO_2 + ↓ pH → Sympathetic activation → Hypertension

---

Then:

↑ BP → ↑ baroreceptor firing → Bradycardia

🧠 Triad

- Hypertension
- Bradycardia
- Respiratory depression

🎯 Final High-Yield Takeaways

- ANP/BNP → ↓ volume, ↓ BP, ↑ Na^+ excretion 💧
- BNP = best lab for ruling out HF 🚨
- Baroreceptors respond to stretch (BP)
- Chemoreceptors respond to CO_2 , pH, O_2 (<60)
- Carotid massage → ↓ HR via vagal stimulation

- Cushing reflex = hypertension + bradycardia + ↓ respiration

---

💧 Capillary Fluid Exchange — Starling Forces

🧪 Starling Forces

Fluid movement across capillary membranes is determined by the balance of hydrostatic and oncotic pressures:

Symbol	Name	Direction / Effect
Pc	Capillary hydrostatic pressure	Pushes fluid out of capillary (filtration)

Pi	Interstitial hydrostatic pressure	Pushes fluid into capillary (reabsorption)
π c	Plasma oncotic pressure	Pulls fluid into capillary (reabsorption)
π i	Interstitial fluid oncotic pressure	Pulls fluid out of capillary (filtration)

⚖️ Net Fluid Flow Equation

$Jv = Kf [(Pc - Pi) - \sigma (\pi c - \pi i)]$

Where:

- Jv = net fluid movement (ml/min)
- Kf = capillary permeability to fluid
- σ = reflection coefficient (0–1; 1 = impermeable to protein)

💡 Interpretation: Positive Jv → fluid leaves capillary; negative Jv → fluid enters capillary.

🫀 Capillary Dynamics

- Arteriole end: Pc dominates → net filtration
- Venous end: πc dominates → net reabsorption

Most of the 20 L/day filtered fluid is returned via lymphatics.

⚠️ Causes of Edema

Edema occurs when net fluid outflow > lymphatic return, commonly due to:

Mechanism	Example

↑ Capillary hydrostatic pressure (↑ Pc)	Heart failure, fluid overload
↑ Capillary permeability (↑ Kf)	Infections, burns, toxins
↑ Interstitial oncotic pressure (↑ π i)	Lymphatic blockage, inflammation
↓ Plasma oncotic pressure (↓ π c)	Nephrotic syndrome, liver failure, malnutrition

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Summary

- Filtration = Arterial capillary end → fluid leaves
- Reabsorption = Venous capillary end → fluid returns
- Net fluid flow depends on Starling forces
- Edema arises from imbalance in these forces

-> The End <-