# ❤️ Cardiovascular Physiology

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## 🫀 Cardiac Output Basics

### 📌 Key Formula

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

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### 💥 Stroke Volume (SV)

### 📌 Determinants (HIGH-YIELD)

SV depends on:

- Contractility
- Preload

- Afterload

💡 Memory Trick: "SV CAP"

➡️ Contractility, Afterload, Preload

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🔄 Effects on Stroke Volume

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

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⚙️ Stroke Work (SW)

- Work done by ventricle to eject blood

📌 Relationship:

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

💡 Clinical:

- Heart failure → ↓ SV → ↓ stroke work

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💪 Contractility

📌 Definition

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

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🔼 Factors Increasing Contractility

Mechanism Flowchart:

$\beta 1$ receptor stimulation (catecholamines) → ↑ cAMP → Activation of protein kinase A (PKA) → Phosphorylation of phospholamban → ↑ $Ca^{2+}$ ATPase activity → ↑ $Ca^{2+}$ storage in SR → Phosphorylation of $Ca^{2+}$ channels

- ↑ $Ca^{2+}$ influx
- ↑ $Ca^{2+}$-induced $Ca^{2+}$ release

↑ Intracellular $Ca^{2+}$ → ↑ Contractility 💪

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## 💊 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

- $\beta$1-blockers
- Systolic heart failure
- Acidosis
- Hypoxia / hypercapnia
- Non-dihydropyridine $Ca^{2+}$ channel blockers (e.g., verapamil)

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## 🫀 Preload

### 📌 Definition

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

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### 🔄 Determinants

- Venous tone

- Blood volume

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## 💊 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)

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### 🔄 Mechanism (Laplace Relationship)

Increased pressure → ↑ wall tension → ↑ afterload

### 💊 Clinical Effects

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

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### 🧠 Adaptive Response

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

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## ❤️ Cardiac Oxygen Demand

### 📌 Increased by:

- ↑ Contractility

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

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## 🔄 Mechanism Flowchart

↑ Pressure or ↑ ventricular size → ↑ wall tension → ↑ myocardial $O_2$ demand 🫀

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## 📐 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:

- $\sigma$ = wall stress
- h = wall thickness

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## 🧠 Key Insights

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

💡 Explains why LV hypertrophy is compensatory

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## 🩸 Coronary Oxygen Extraction

- Coronary sinus contains most deoxygenated blood in the body 😮

  💡 Because myocardium extracts maximum $O_2$ at baseline

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## 💡 Final 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 = foundational concept for cardiology questions

-> The End <-