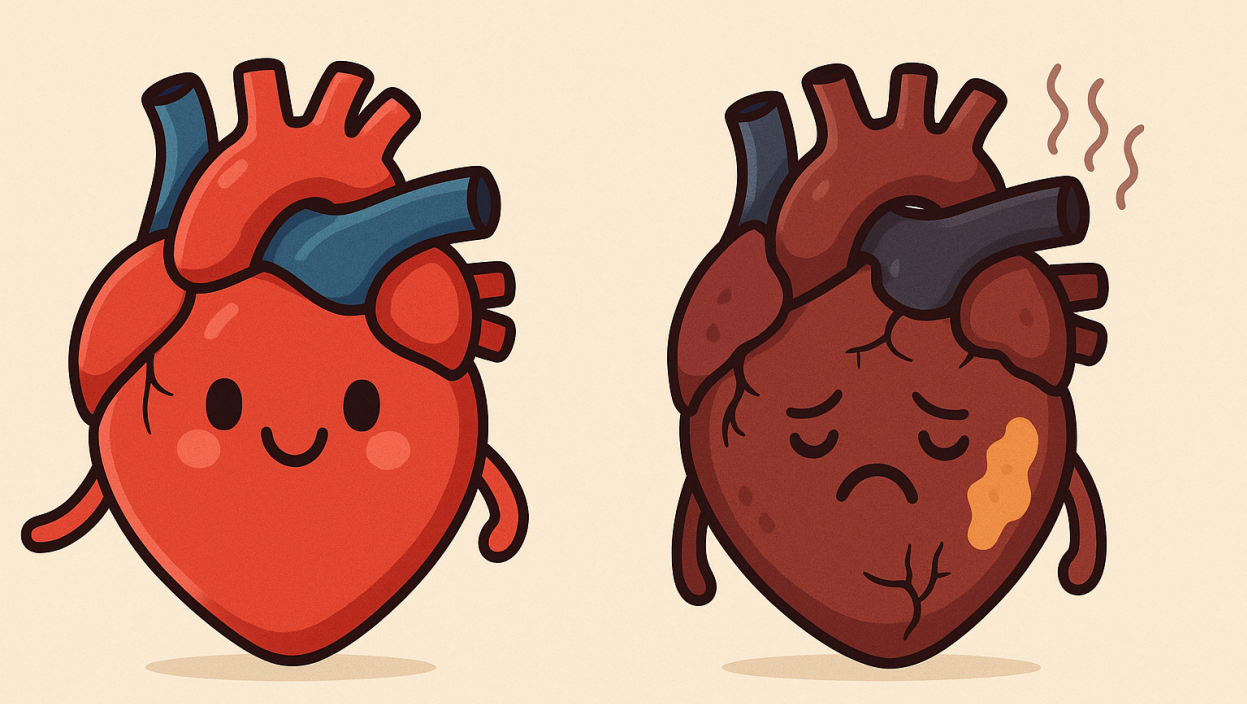
Cardiac Review & Ischemic Heart Disease



This guide includes an overview of basic cardiac anatomy, perfusion, and ischemic heart disease.

1. Basic Anatomy of the Heart

The human heart is a muscular organ located in the mediastinum, made up of four chambers: two atria and two ventricles. The heart pumps blood through the pulmonary and systemic circulations.

Atria

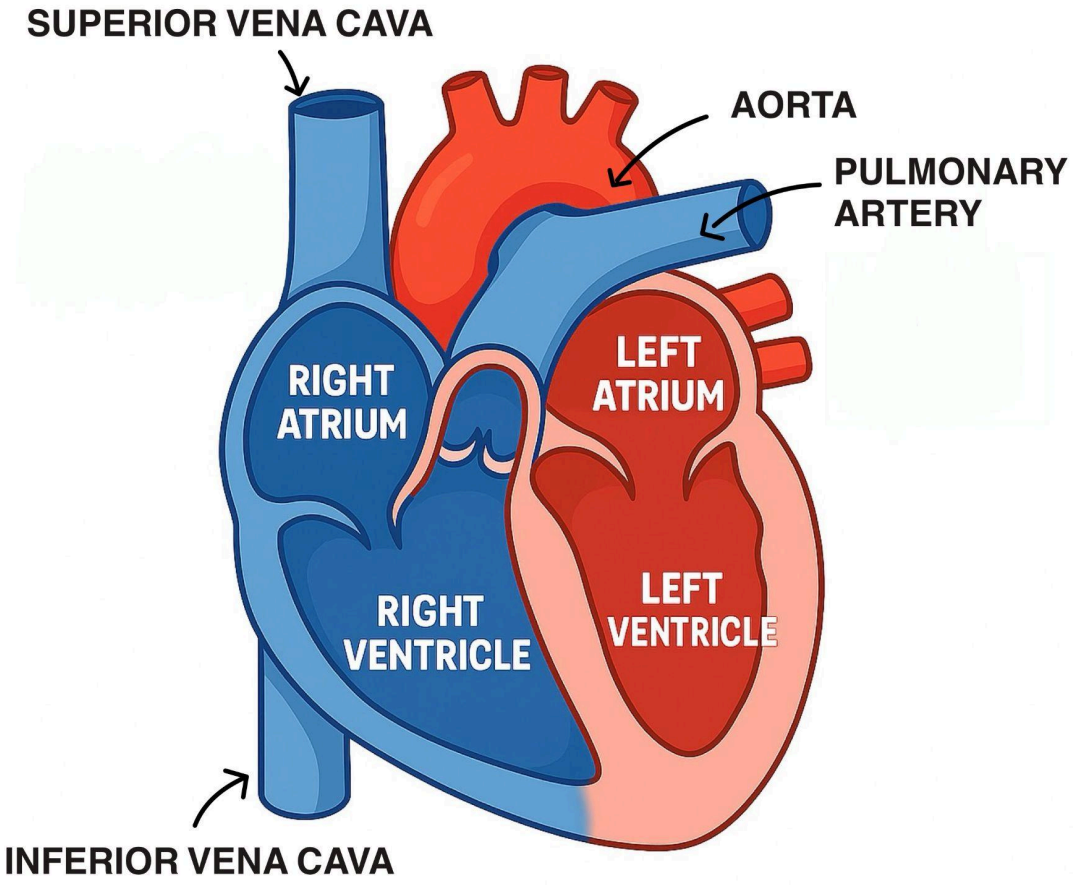
- **Right Atrium (RA)**
  - Receives **deoxygenated** blood from the superior and inferior vena cava and coronary sinus

- **Left Atrium (LA)**
  - Forms the base of the heart
  - Receives **oxygenated** blood from the pulmonary veins

Ventricles

- **Right Ventricle (RV)**
  - Pumps **deoxygenated** blood to the lungs through the pulmonary arteries

- **Left Ventricle (LV)**
  - Pumps **oxygenated** blood through the aorta and into systemic circulation



2. Cardiac Valves

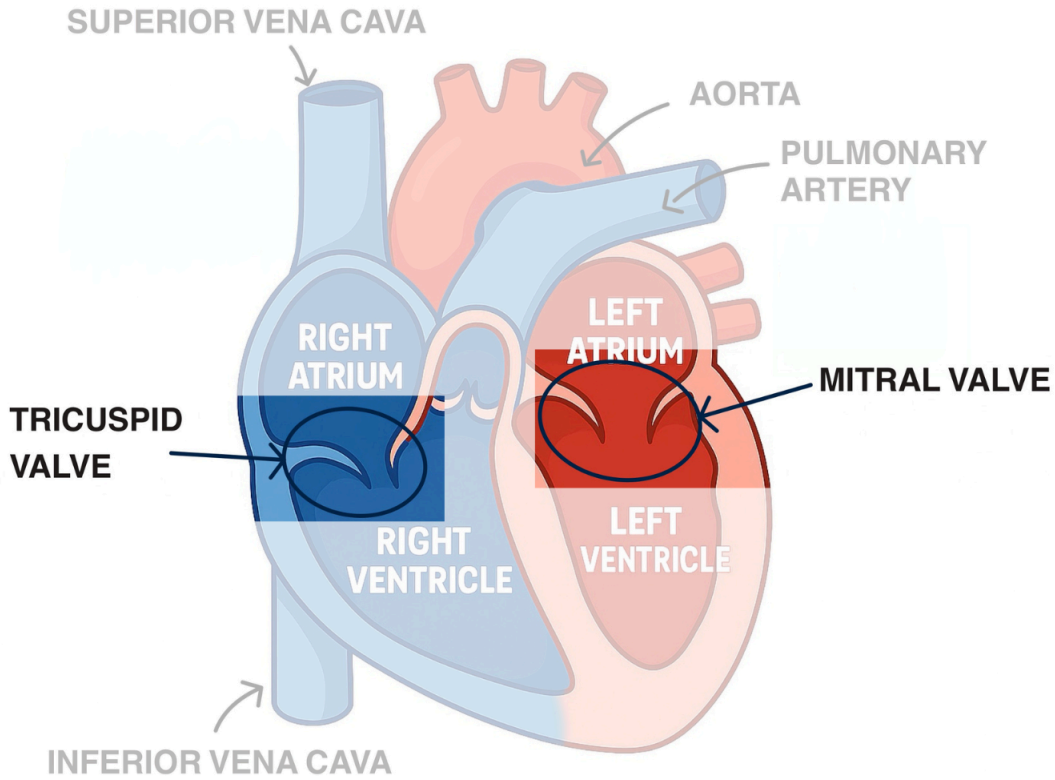
The cardiac valves prevent backflow of blood through the heart. When the ventricles contract, blood should flow into the pulmonary artery or the aorta, and not 'backwards' into the right and left atria.

Both the pulmonary artery and aorta have **semi-lunar valves**. The valves between the atria and ventricles are the **tricuspid** and **bicuspid (mitral)** valves for the right and left side of the heart, respectively.

Valve	Location	Function	Components
Tricuspid	Between RA and RV	Prevents backflow into RA	3 leaflets
Pulmonary	RV to pulmonary trunk	Prevents backflow into RV	3 semilunar cusps
Bicuspid (Mitral)	Between LA to LV	Prevents backflow into LA	2 leaflets
Aortic	LV to aorta	Prevents backflow into LV	3 semilunar cusps

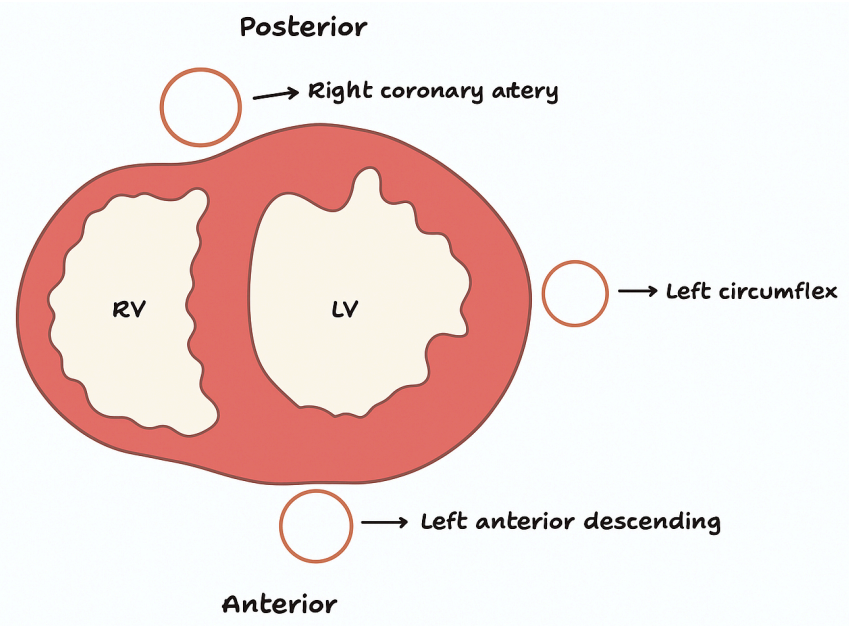
→ **Memory Hack**: "Try (Tri) before you Buy (Bi)"

- **Tricuspid** valve comes **first** in blood flow (on the **right** side of the heart, between the RA and RV)
- **Bicuspid (mitral)** valve comes **after** it (on the **left** side of the heart, between the LA and LV)



3. Coronary Arteries

The coronary arteries supply blood to the heart.



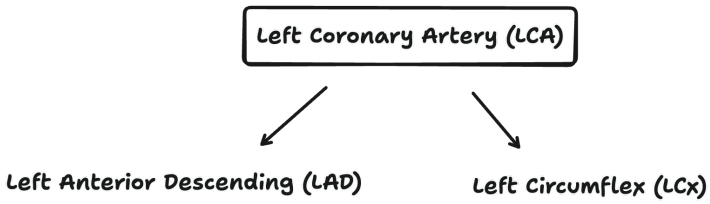
*LCA not depicted

Right Coronary Artery (RCA)

- **Supplies**
  - Entire free wall of the RV
  - Right dominant hearts only
    - Posterobasal wall of the LV and the posterior 1/3 of the ventricular septum

Left Coronary Artery (LCA)

- Divides into the left anterior descending artery and the left circumflex artery



Left Anterior Descending (LAD)

- **Supplies:**
  - Anterior LV wall
  - Apex of the heart
  - Anterior 2/3 of the ventricular septum

Left Circumflex (LCx)

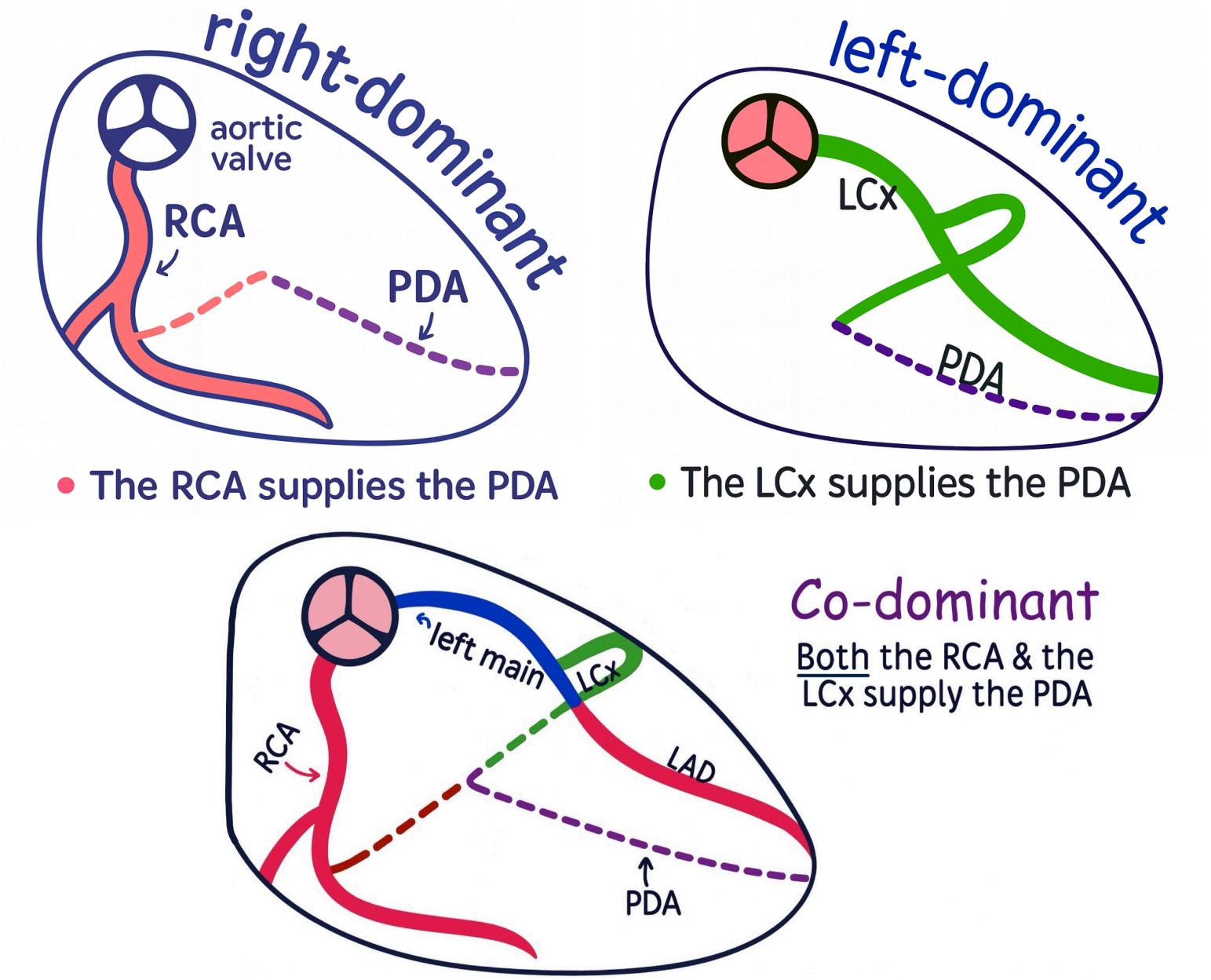
- **Supplies:**
  - Lateral left ventricular wall
  - Left dominant hearts only
    - Posterobasal wall of the LV and the posterior 1/3 of the ventricular septum

4. Coronary Dominance

Dominance is determined by which artery gives rise to the **Posterior Descending Artery (PDA)**. The PDA supplies the posterior basal wall of the LV as well as the posterior 1/3 of the ventricular septum. In the majority of individuals, the PDA branches off the RCA.

Because of this dominance pattern, RCA occlusions can lead to LV damage.

Type	PDA Origin	Prevalence	Areas Supplied
Right Dominant	RCA	~80%	Posterior septum, inferior LV by RCA
Left Dominant	LCx	10%	Posterior septum, inferior LV by LCx
Co-Dominant	RCA & LCx	10–20%	Shared supply to posterior LV and septum



5. Ischemic Heart Disease

Definition and Overview

Ischemic heart disease (IHD) encompasses a group of related syndromes caused by myocardial ischemia, an imbalance between cardiac blood supply (perfusion) and myocardial oxygen demand. Because the cardiac myocardium is constantly working, its demand for oxygen, nutrients, and waste removal is high, making it very susceptible to ischemia.

The most common underlying cause of IHD is **coronary artery disease (CAD).** CAD is characterized by **atherosclerosis of the epicardial coronary arteries**, leading to progressive luminal narrowing and, in some cases, acute plaque rupture with thrombosis.

When an obstruction in the coronary artery blocks 70% of the lumen (point of "critical stenosis"), it typically represents the onset of symptomatic ischemia related to increased physical demand. Coronary artery obstruction of 90% of the lumen can lead to symptomatic ischemia even while at rest.

Pathogenesis

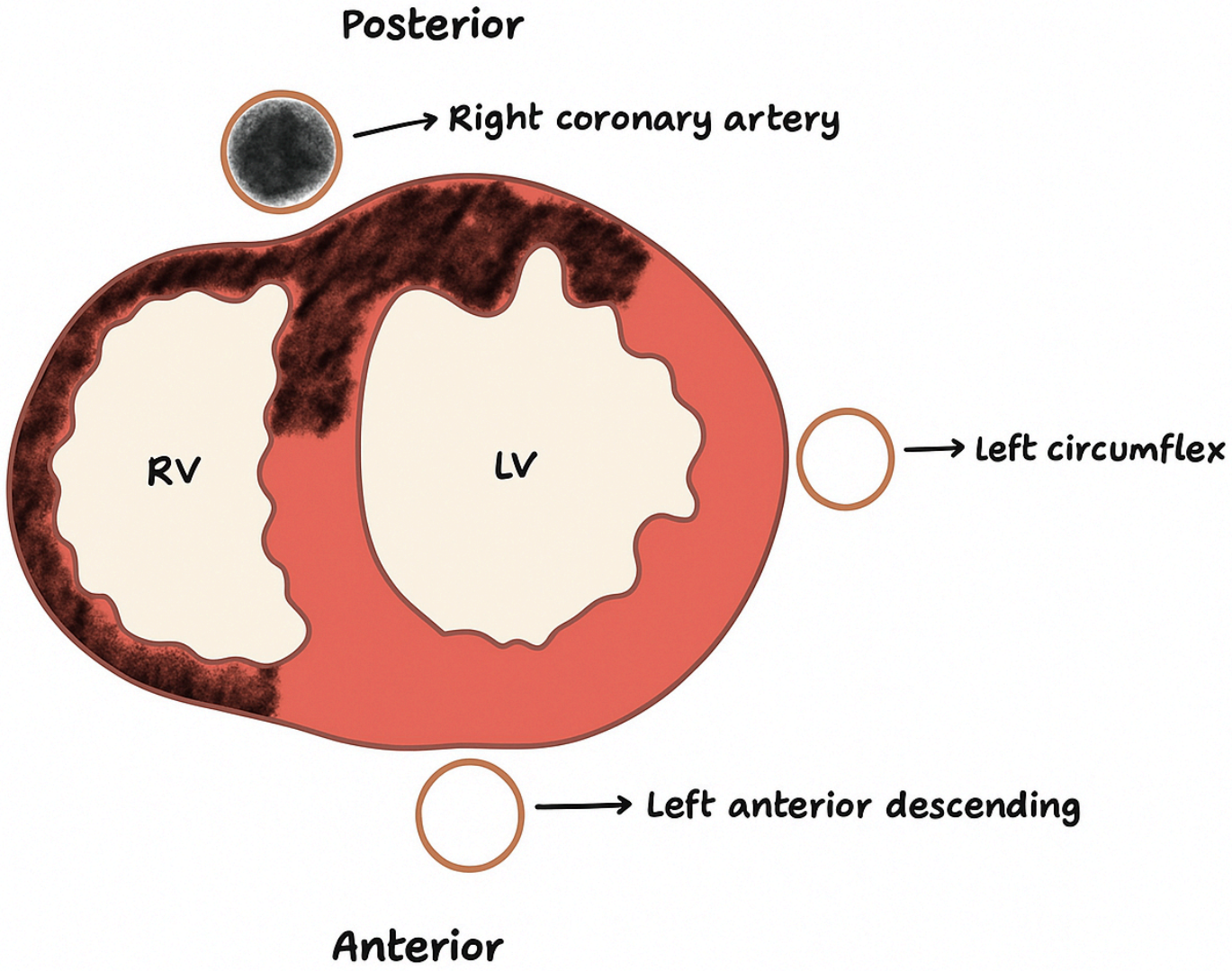
- **Primary Mechanism:** Reduced coronary blood flow from atherosclerotic obstruction
- **Critical Stenosis:** Fixed narrowing of ≥ 70% of the coronary lumen produces ischemia during exertion; > 90% obstruction can cause ischemia at rest
- **Acute Plaque Change:** Rupture, erosion, or hemorrhage into atherosclerotic plaques can trigger thrombosis and vasospasm, leading to acute coronary syndromes
- **Other Causes:** Coronary emboli, vasculitis, vasospasm (e.g. cocaine use), global hypotension (shock), hematologic abnormalities (e.g. sickle cell disease), dissection
    - Anything that causes a significant reduction/cessation in blood flow through a coronary artery causing the myocardium it supplies to infarct

Common Arterial Involvement

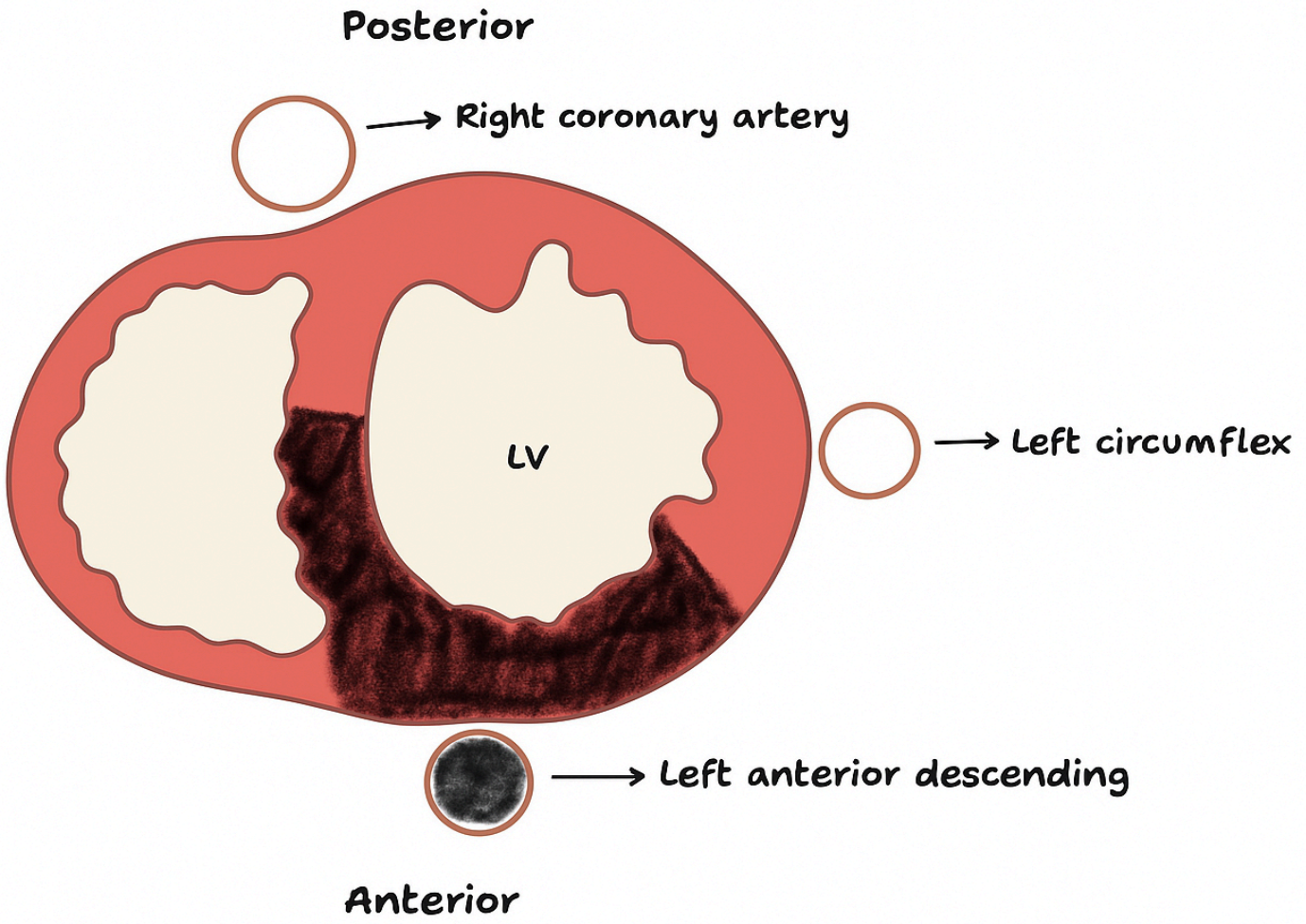
Knowing which parts of the heart muscle get blood from each coronary artery helps us understand which area could be damaged if one of those arteries gets blocked.

Affected Coronary Artery	Typical Infarct Location
RCA	Posterior LV, posterior ⅓ of septum, RV wall
LAD	Anterior LV wall, anterior ⅔ of septum, apex
LCx	Lateral LV wall

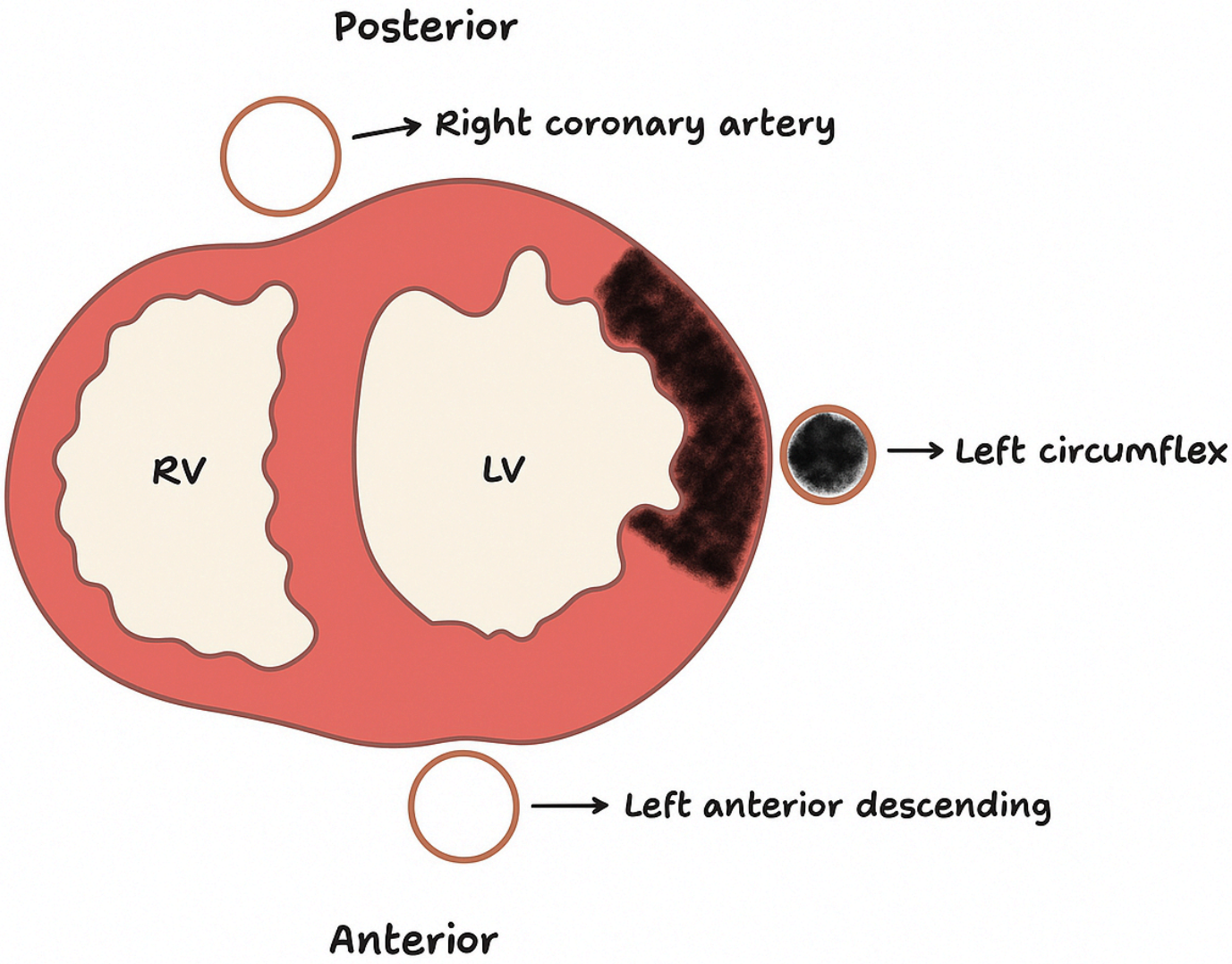
RCA Obstruction (R dominant shown)



LAD Obstruction



LCx Obstruction (R dominant shown)



Major Clinical Syndromes

1. **Angina Pectoris** – Episodes of chest pain caused by temporary myocardial ischemia without infarction
    - **Subtypes:**
        - **Stable Angina:** Predictable onset of chest discomfort with exertion or stress; worsens in colder weather or after heavy meals → lasts ≤ 15 minutes
            - ↳ Due to fixed (>70%) atherosclerotic narrowing
            - ↳ Treated with rest, nitroglycerin, oxygen
        - **Unstable Angina:** Unpredictable onset of chest discomfort; episodes may become increasingly frequent or cause severe pain; can occur at rest
            - ↳ Usually caused by plaque rupture and thrombosis → considered a pre-infarction state
            - ↳ Treated with rest, nitroglycerin, cardiac medications, oxygen
        - **Variant/Prinzmetal Angina:** Unpredictable onset, usually occurring at night; can occur at rest
            - ↳ Caused by coronary vasospasm, though atherosclerosis is usually present as well
            - ↳ Treated with calcium channel blockers

2. **Myocardial Infarction (MI)** – Prolonged ischemia leading to irreversible myocyte necrosis (infarction)
    - Most commonly due to acute thrombosis superimposed on a disrupted atherosclerotic plaque

3. **Chronic Ischemic Heart Disease (IHD with Heart Failure)** – Progressive heart failure secondary to accumulated ischemic myocardial damage and compensatory hypertrophy of non-infarcted myocardium

4. **Sudden Cardiac Death (SCD)** – Unexpected cardiac death due to fatal arrhythmia
    - Often due to acute ischemia or pre-existing scar tissue

Consequences of Myocardial Ischemia

Syndrome	Pathophysiology	Typical Cause
Stable Angina	Fixed atherosclerotic stenosis limiting blood flow	Chronic plaque with >70% narrowing
Unstable Angina	Acute plaque change and partial thrombosis	Plaque rupture with superimposed thrombus
Myocardial Infarction	Prolonged ischemia → necrosis	Complete arterial occlusion due to thrombus
Sudden Cardiac Death	Fatal arrhythmia from ischemia	Acute plaque change or scar-related conduction block
Chronic IHD	Progressive pump failure from ischemic injury	Prior infarcts and ongoing hypoperfusion

Patterns of Infarction

- **Transmural Infarction:** Full-thickness necrosis of ventricular wall
    - Usually from acute occlusion of an epicardial coronary artery

- **Subendocardial Infarction:** Limited to inner one-third to half of wall
    - Due to partial occlusion or global hypoperfusion

- **Multifocal Microinfarction:** Small, scattered necroses
    - Due to vasculitis, microemboli, or catecholamine-induced spasm

Complications of Myocardial Infarction

Complication	Mechanism	Timing / Features
Arrhythmias	Conduction disturbances, ischemic irritability	First 24 hrs; most common cause of early death
Contractile Dysfunction	LV failure, shock	Proportional to infarct size
Papillary Muscle Rupture	Ischemic necrosis → mitral regurgitation	3–7 days post-MI
Ventricular Free Wall Rupture	Weakening of necrotic myocardium	3–7 days; leads to tamponade
Ventricular Septal Rupture	Septal necrosis	3–7 days; causes L→R shunt
Mural Thrombus	Endocardial injury and stasis	Days to weeks; risk of embolism
Ventricular Aneurysm	Large transmural scar bulging	Late; risk of HF, thrombus, arrhythmia
Pericarditis	Inflammation overlying infarct	2–3 days (acute) or weeks later (Dressler)
Chronic Heart Failure	Progressive remodeling	Late stage of IHD

Morphologic Evolution of MI

Early gross morphologic changes of myocardial infarction are difficult or impossible to detect, and the same is true for histologic changes in the first several hours.

If a patient dies from the infarction, gross and histologic changes will be entirely absent from the myocardium. As time progresses, these changes become more evident. This is due to the progression of coagulative necrosis.

Time Post-MI	Gross Findings	Microscopic Features
0–4 hrs	None	Usually none → however, wavy fibers *may* be detected at the borders
4–12 hrs	Occasional dark mottling	Early coagulative necrosis; edema; hemorrhage
12–24 hrs	Dark mottling	Coagulative necrosis; hypereosinophilia; neutrophilic infiltrate; pyknosis of nuclei; contraction band necrosis
1–3 days	Mottled yellow-tan center	Peak neutrophilic infiltration; coagulative necrosis with loss of nuclei and striations
3–7 days	Hyperemic border; central yellow-tan softening	Macrophage phagocytosis of dead cells → patients are most susceptible to myocardial rupture during this phase as macrophages remove necrotic tissue and fibrosis/scar formation has not yet occurred
7–10 days	Maximally yellow-tan and soft, red-gray borders (healing is just starting)	Well developed phagocytosis of dead tissue; granulation tissue formation at the margins
10–14 days	Red-grey depressed infarct borders	Well established granulation tissue with new blood vessels and collagen deposition
2–8 weeks	Gray-white scar formation, progressing towards core of infarct	Increased collagen deposition, reduced cellularity
>2 months	Firm white scar	Dense fibrosis