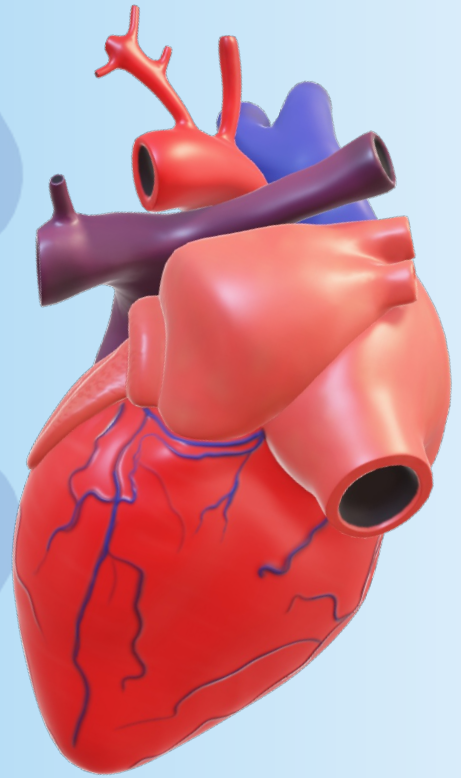
Coronary Circulation

By Niki Brzezinski



Arteries

- Anatomy
- variations
- Myocardial supply

Veins

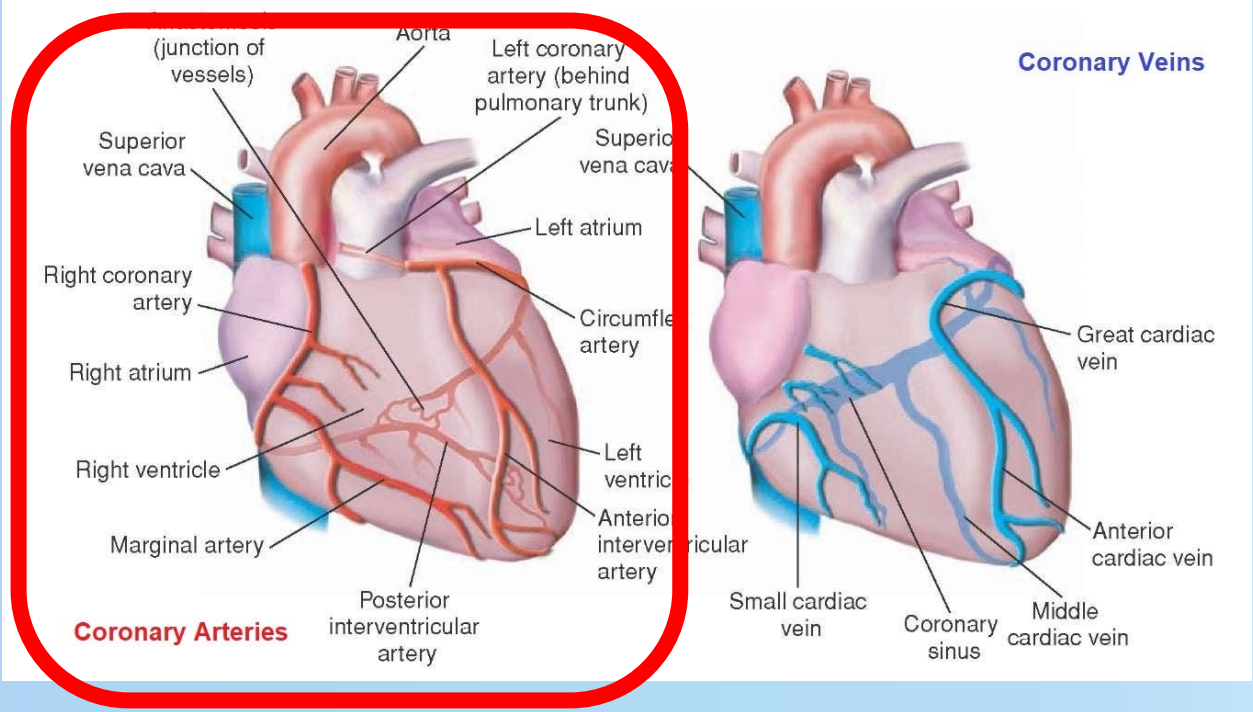
- Anatomy

Blood Flow Regulation

- Basics
- Regulation
- Myogenic
- Metabolic

Pathology

Arteries


(junction of vessels)
Aorta
Left coronary artery (behind pulmonary trunk)
Superior vena cava
Left atrium
Right coronary artery
Right atrium
Right ventricle
Marginal artery
Posterior interventricular artery
Coronary Arteries
Great cardiac vein
Anterior cardiac vein
Small cardiac vein
Coronary sinus
Middle cardiac vein
Coronary Veins

ARTERIES

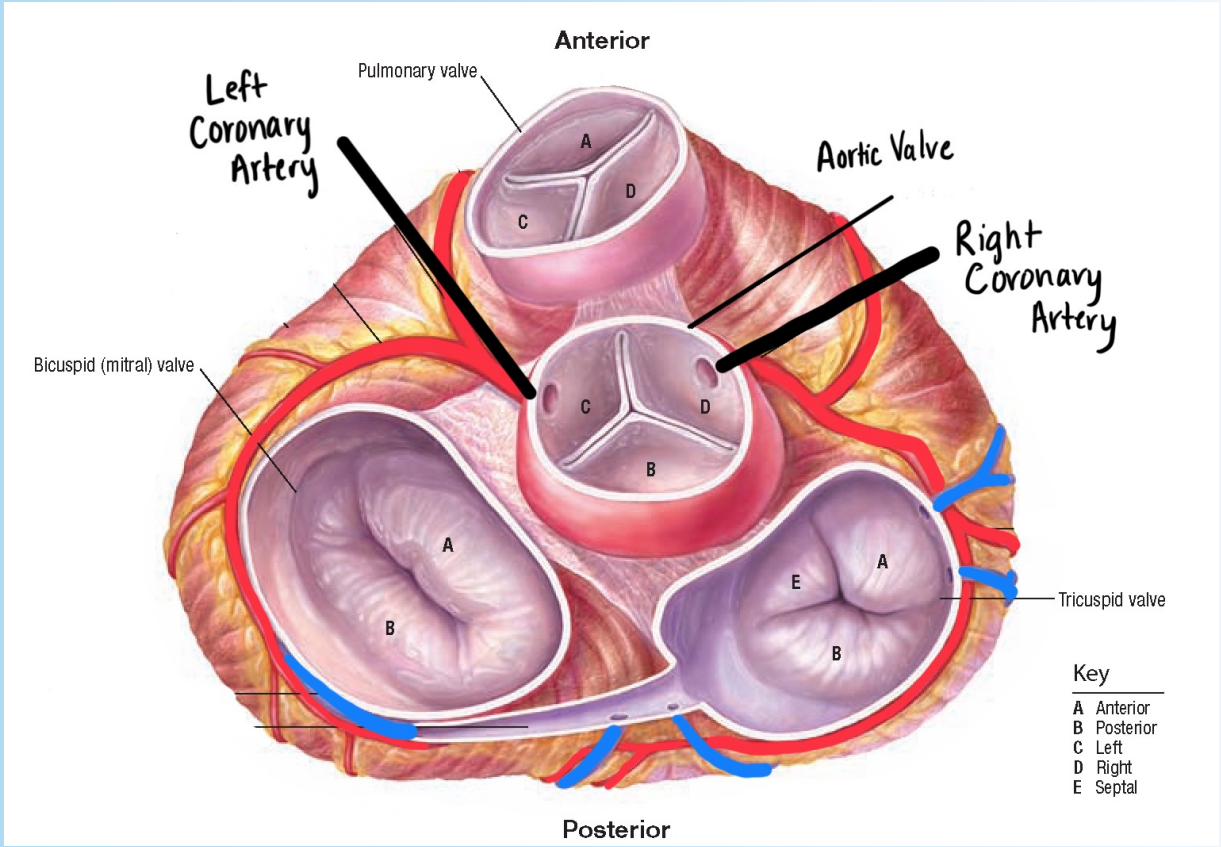
Begin at Aortic Root

Above semilunar valves

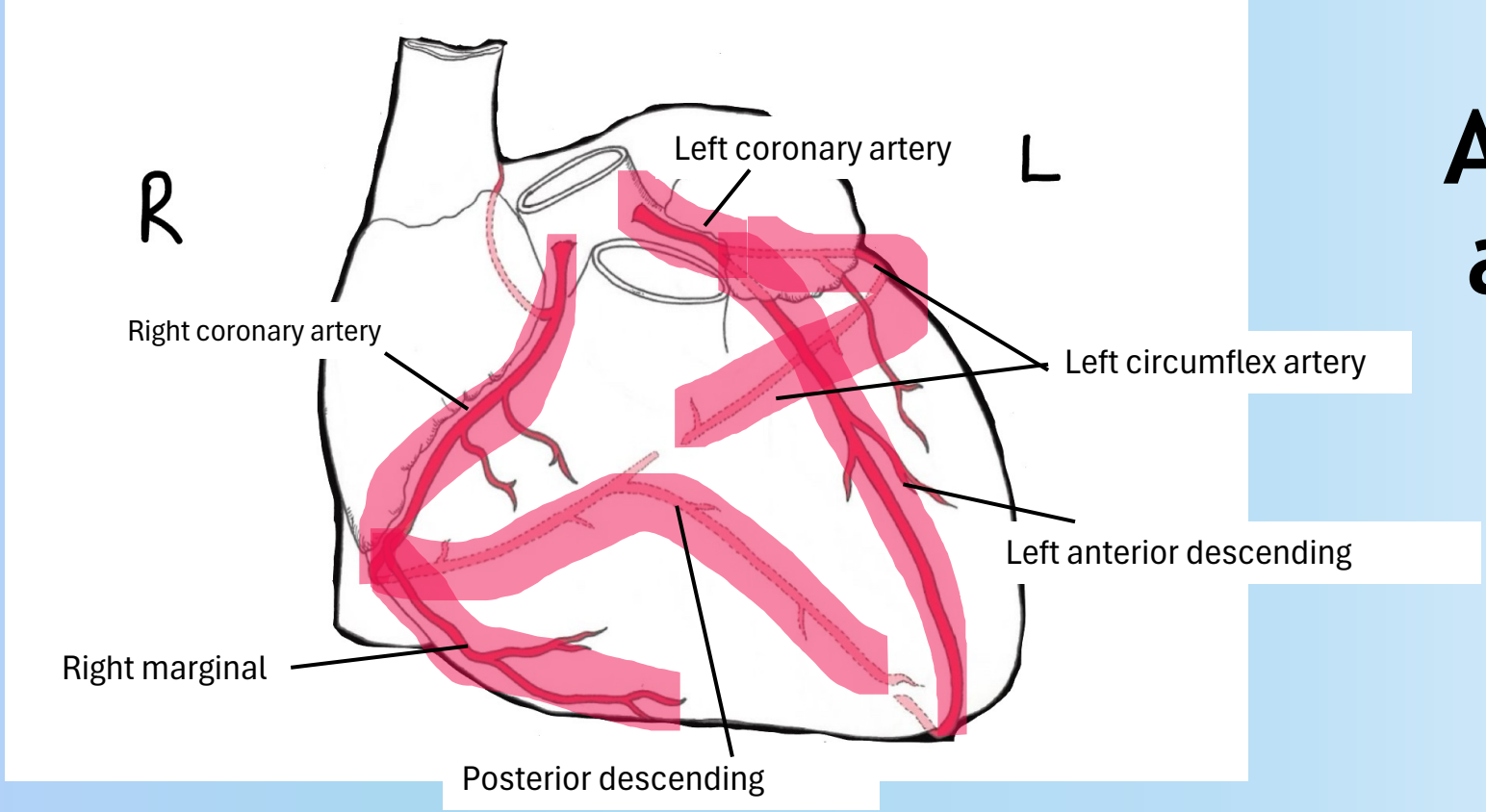
Left cusp = L coronary artery

Right cusp = R coronary artery

Provides 5% of cardiac output to myocardium. (O_2 delivery)



Anatomy arteries



Right Coronary artery (RCA)

- Right Marginal artery
- Posterior descending artery (PDA)
  Posterior intraventricular artery (PIA)

Left Coronary artery (LCA)

- Left Anterior Descending Artery (LAD)
  Anterior intraventricular artery (AIA)
- Left circumflex artery

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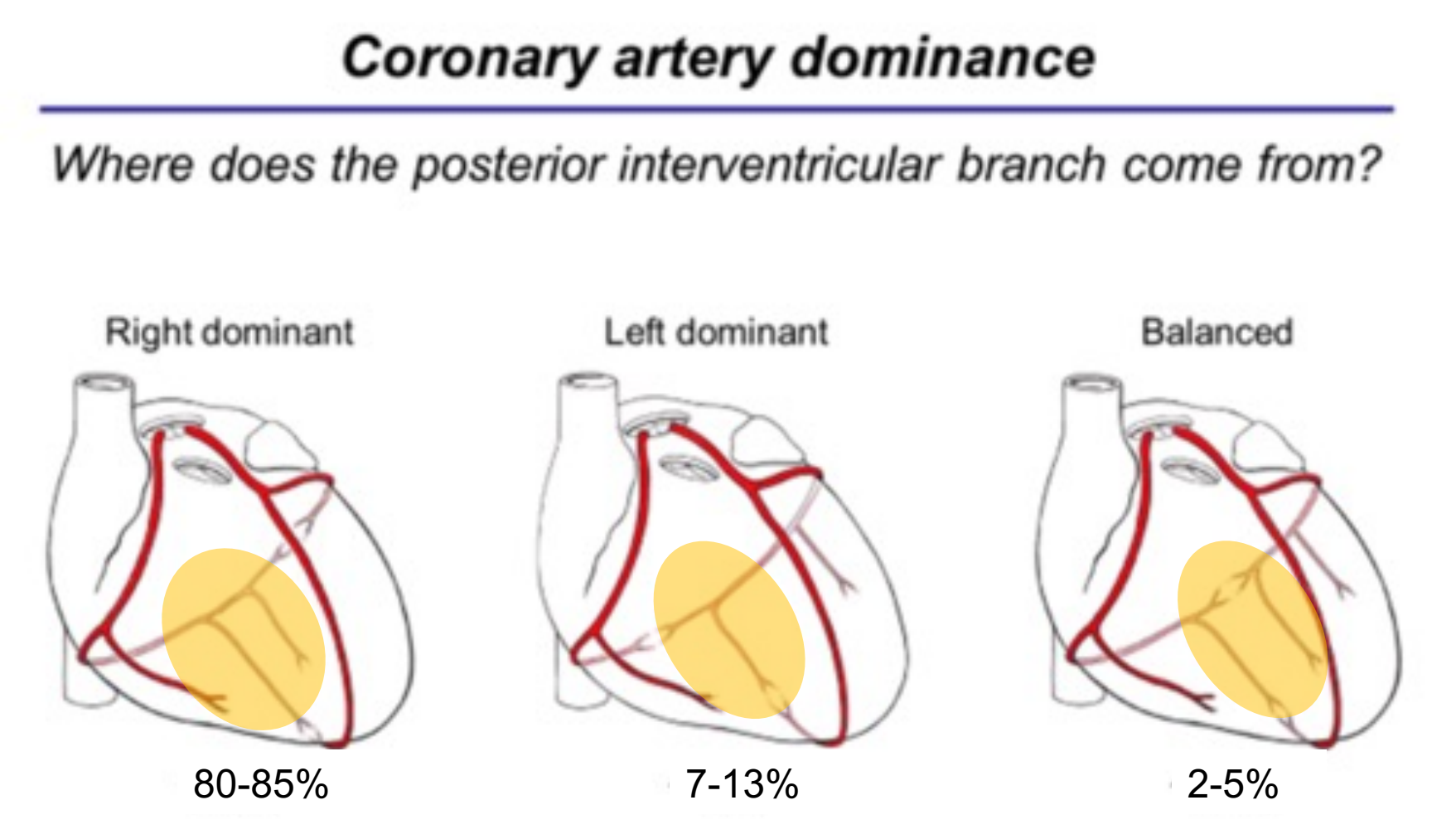
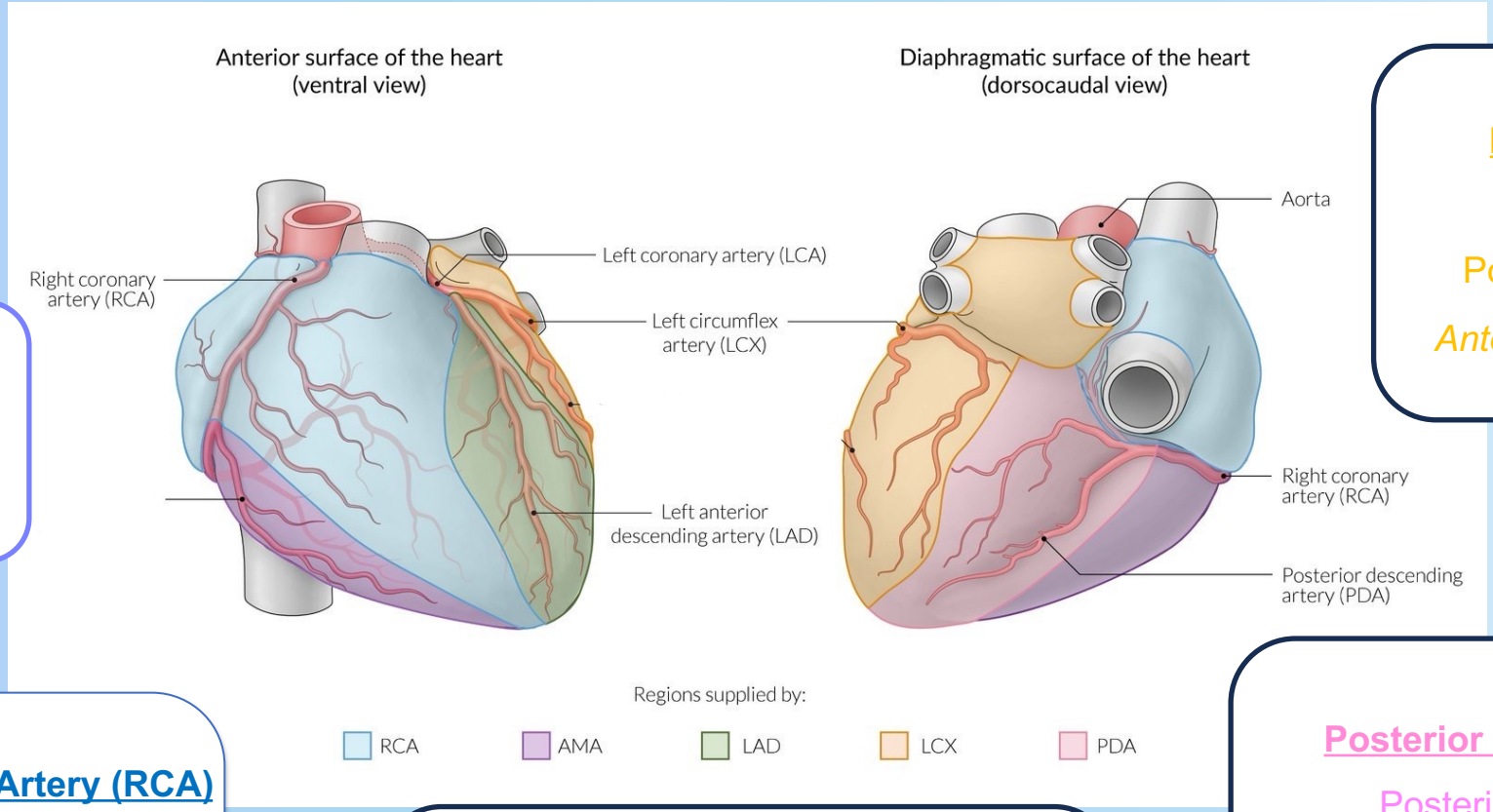
Posterior Descending artery variations



Myocardial Supply



Right Marginal Artery

Lateral Right ventricle

Left Circumflex Artery

Left atrium

Posterolateral left Ventricle

*Anterolateral papillary muscle**

Right Coronary Artery (RCA)

Right atrium

Anterior right ventricle

SA node (SA nodal artery)

AV node (AV nodal artery)

Left Anterior Descending Artery (LAD)

Anterior left ventricle

Apex

Anterior 2/3 of interventricular septum

*Anterolateral papillary muscle**

Posterior descending artery

Posterior right ventricle

Posterior 1/3 interventricular septum

Posteromedial papillary muscle

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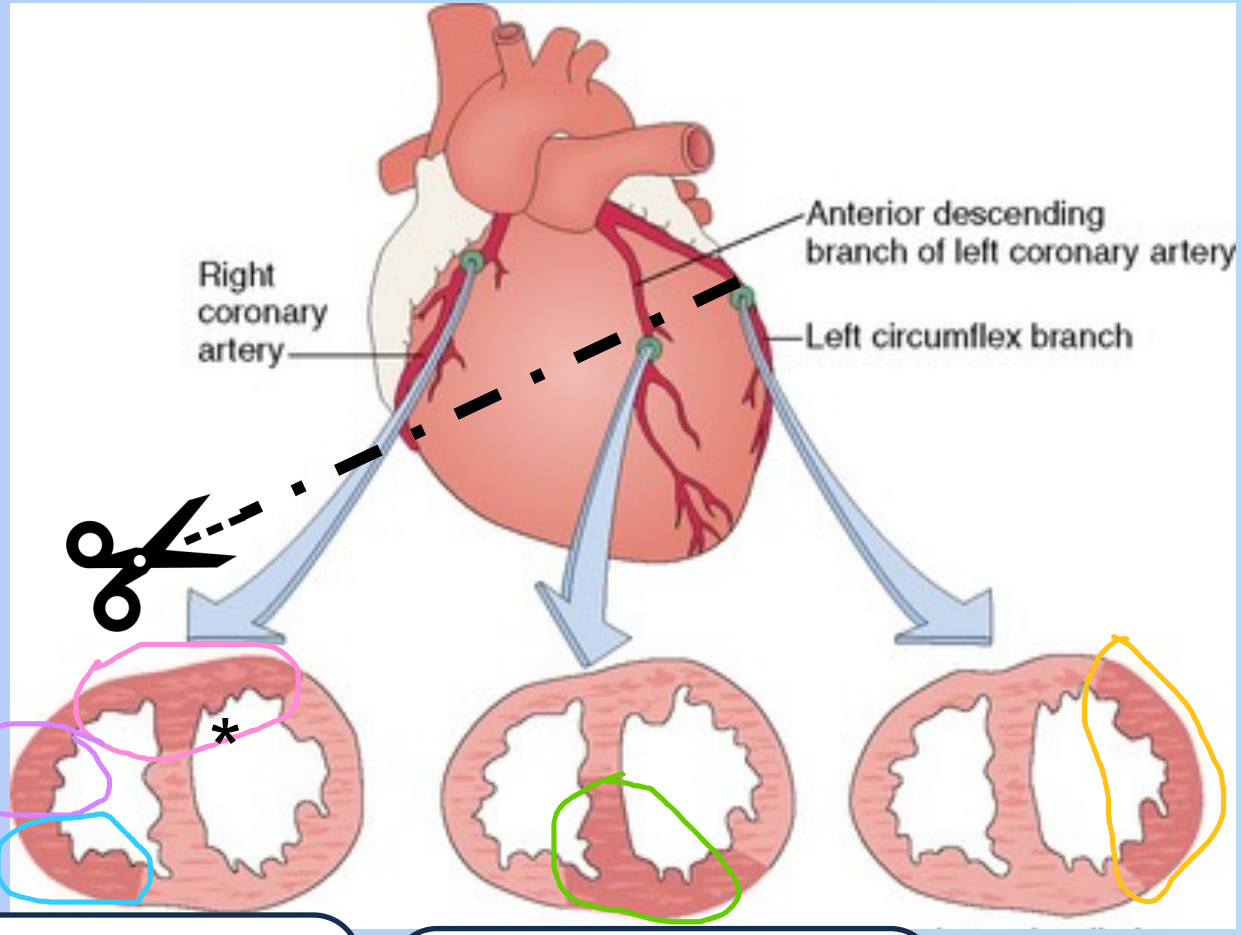
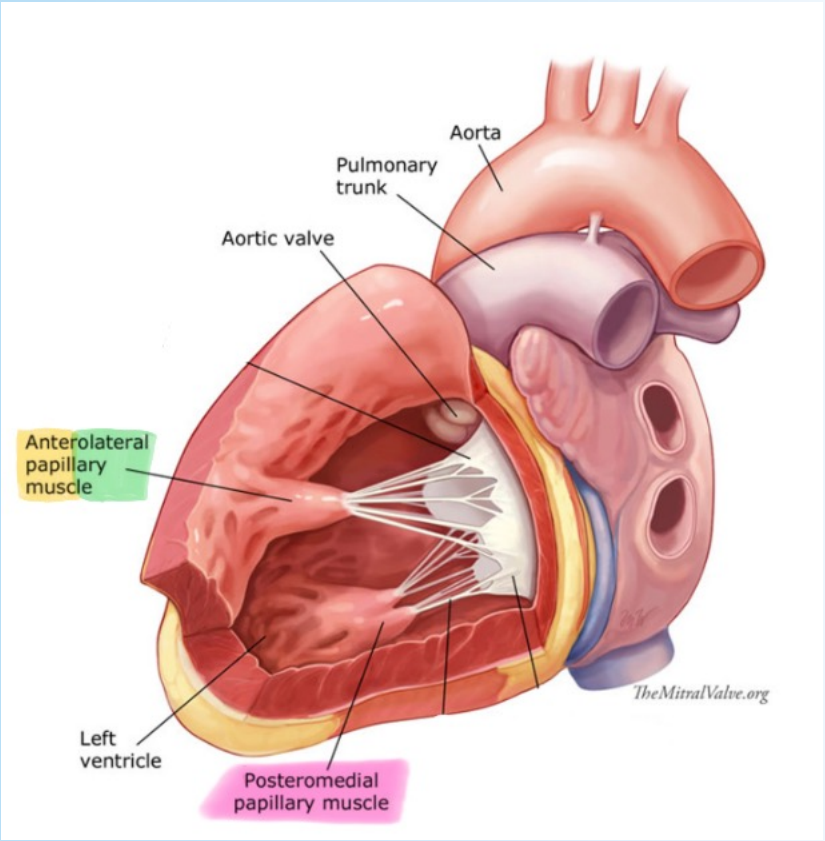
Myocardial Supply cross sections





Posterior Descending artery

Posterior 1/3 interventricular septum

Left Anterior Descending Artery (LAD)

Anterior 2/3 of interventricular septum

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Arteries

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Veins

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Blood Flow Regulation

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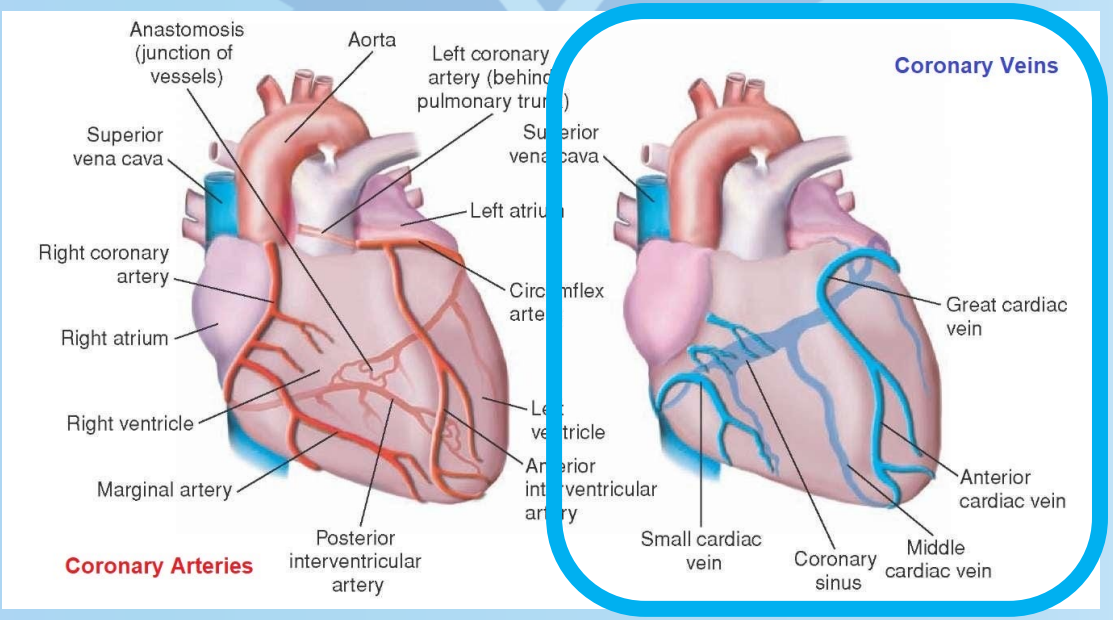
Pathology

Anatomy: veins

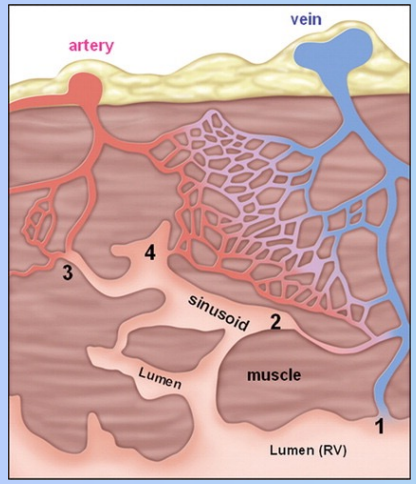


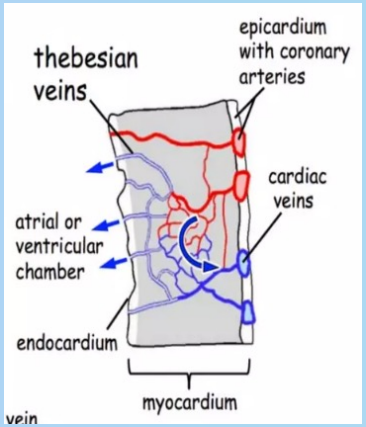
Veins

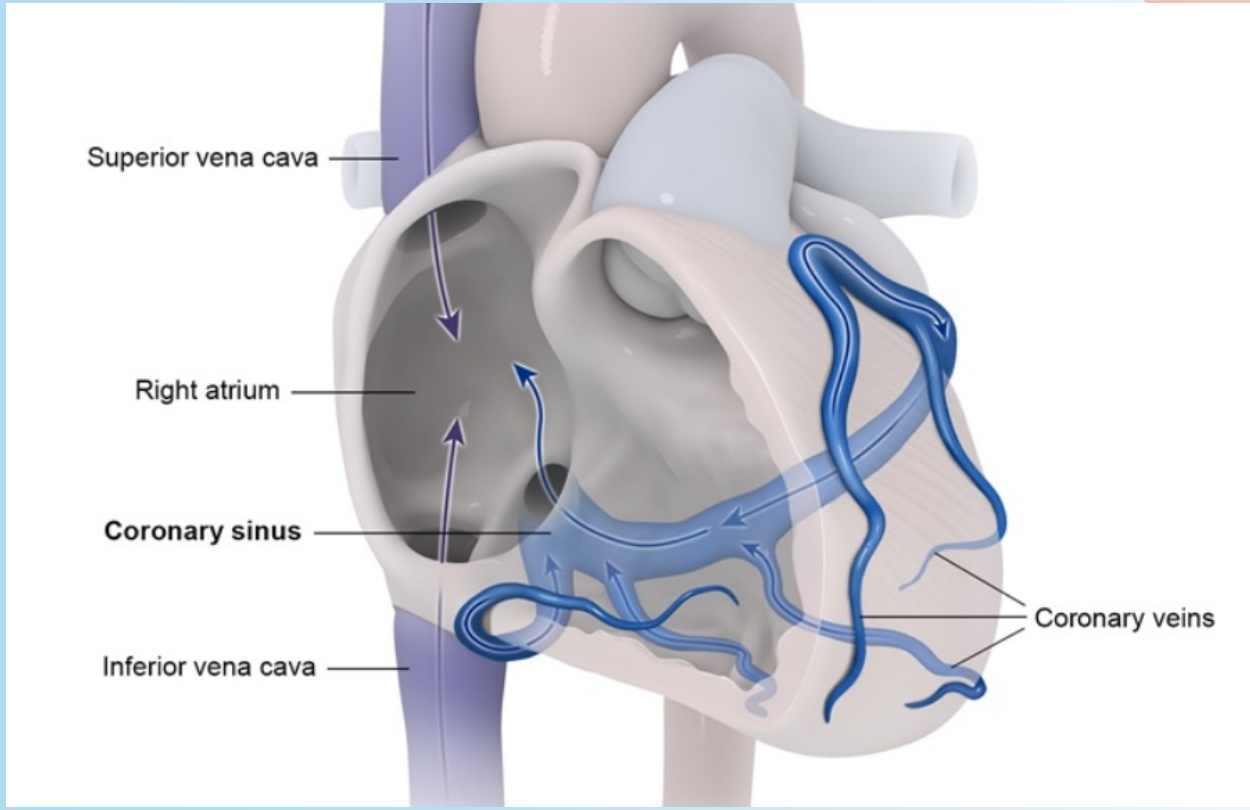
Drain into Right Atrium

The venous drainage occurs 3 ways

1. Coronary sinus → R. atrium(75% of draining)
2. Anterior cardiac veins → R. atrium
3. Venae cordis minimae/ Thebesian veins → numerous tiny veins that drain directly into heart







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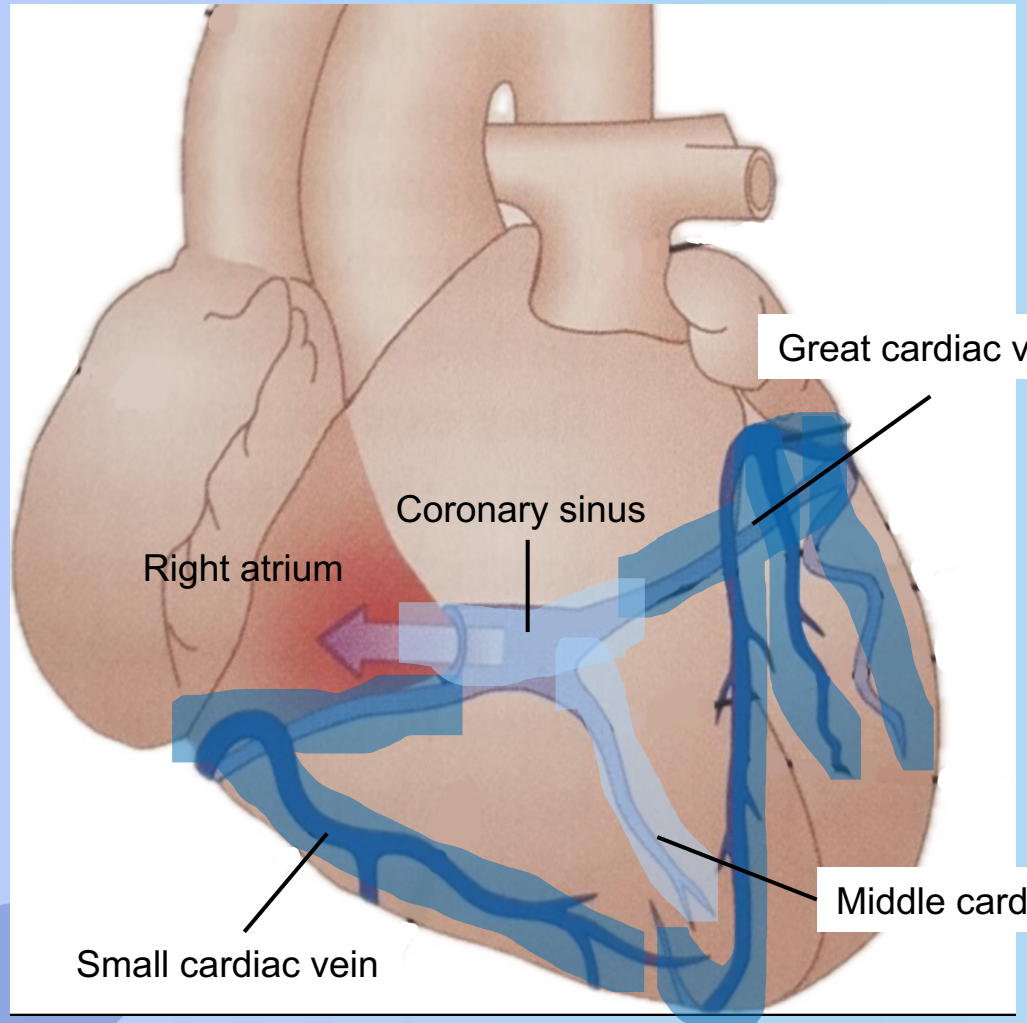
Veins



Great cardiac vein
- Anterior intraventricular
- Left marginal

Small cardiac vein

Middle cardiac vein

Coronary Sinus

Right Atrium

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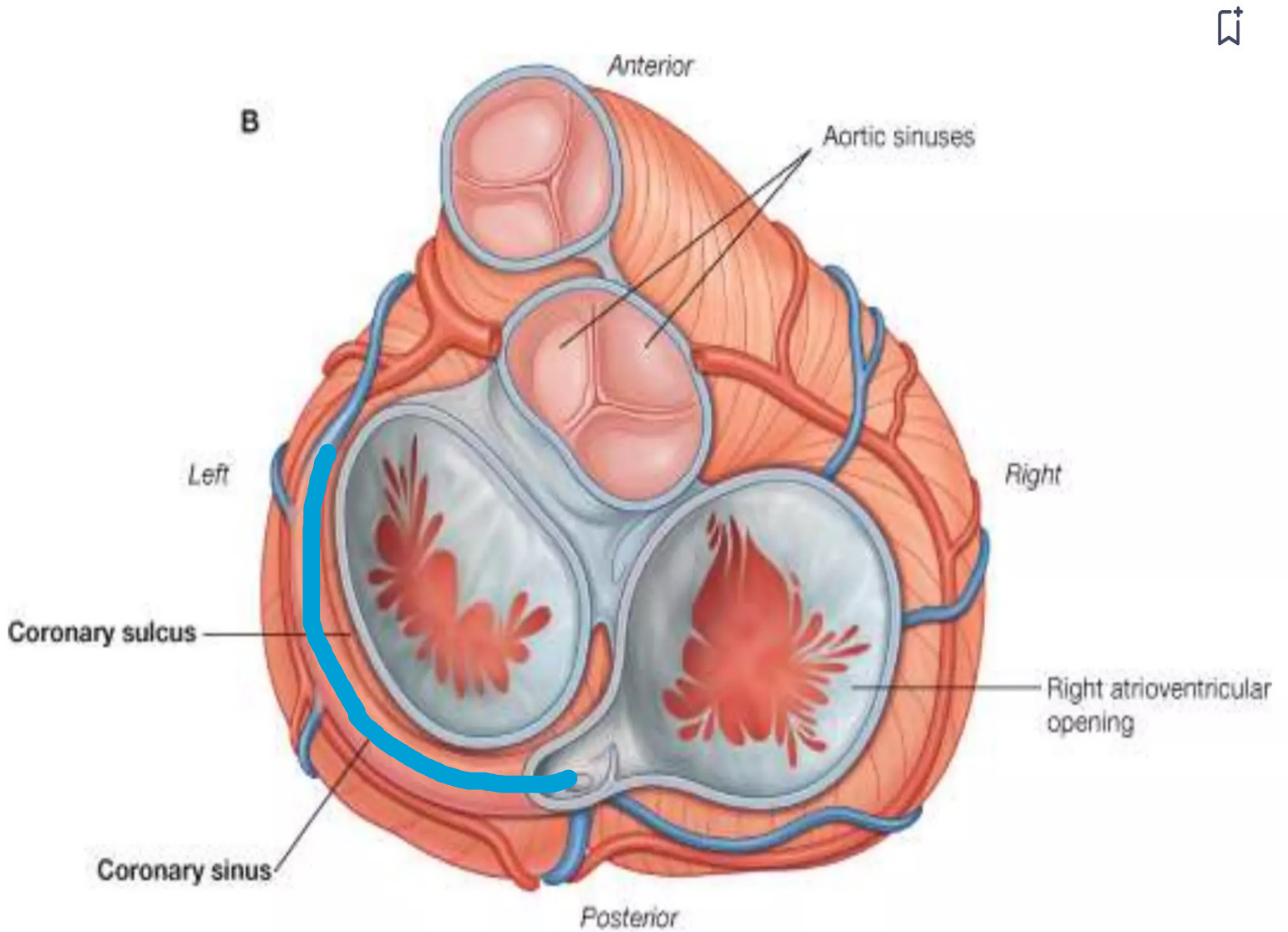
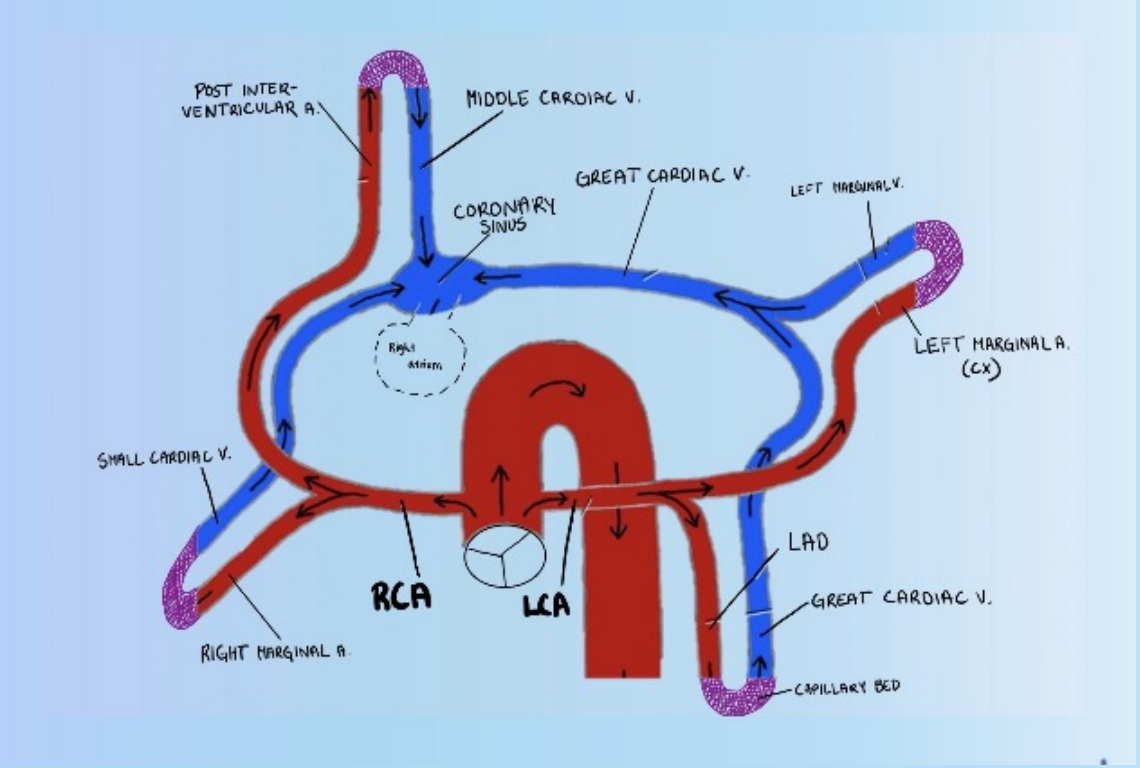
Coronary sinus

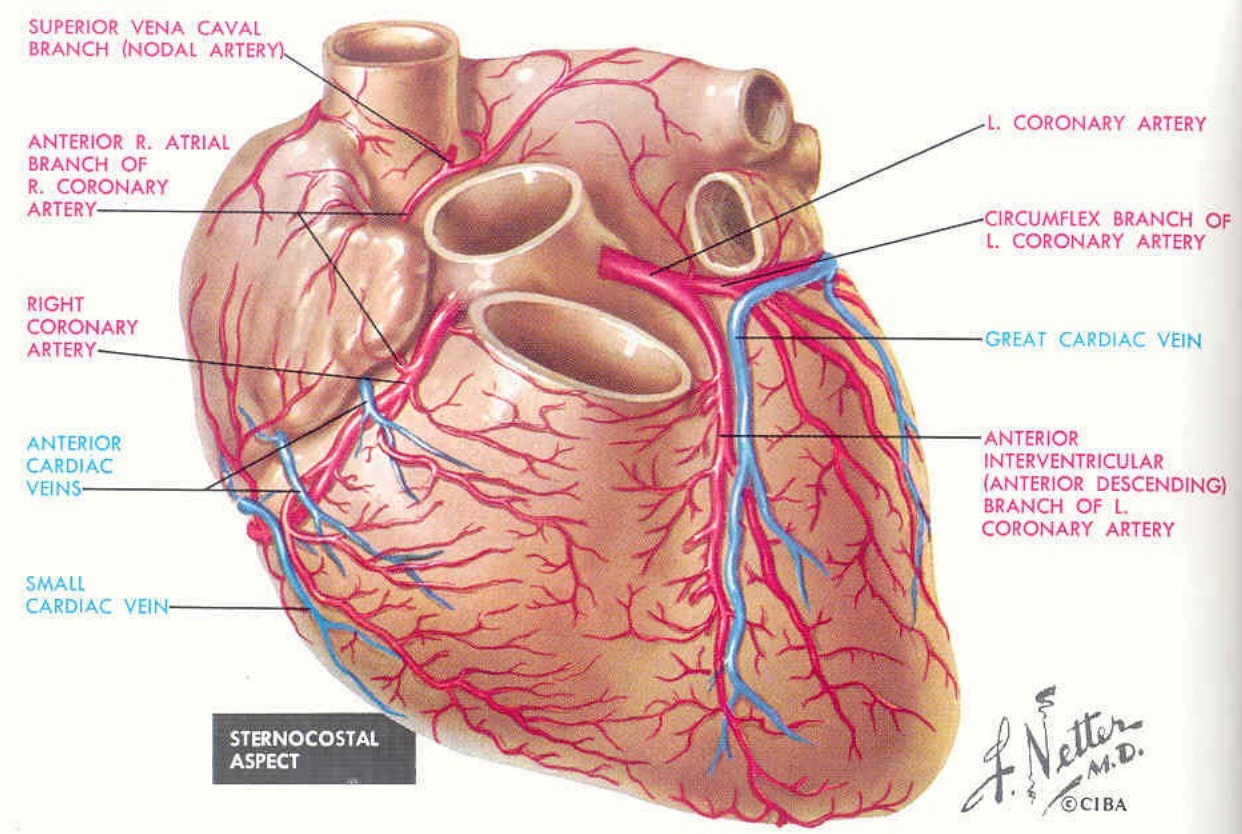
Thebesian valve

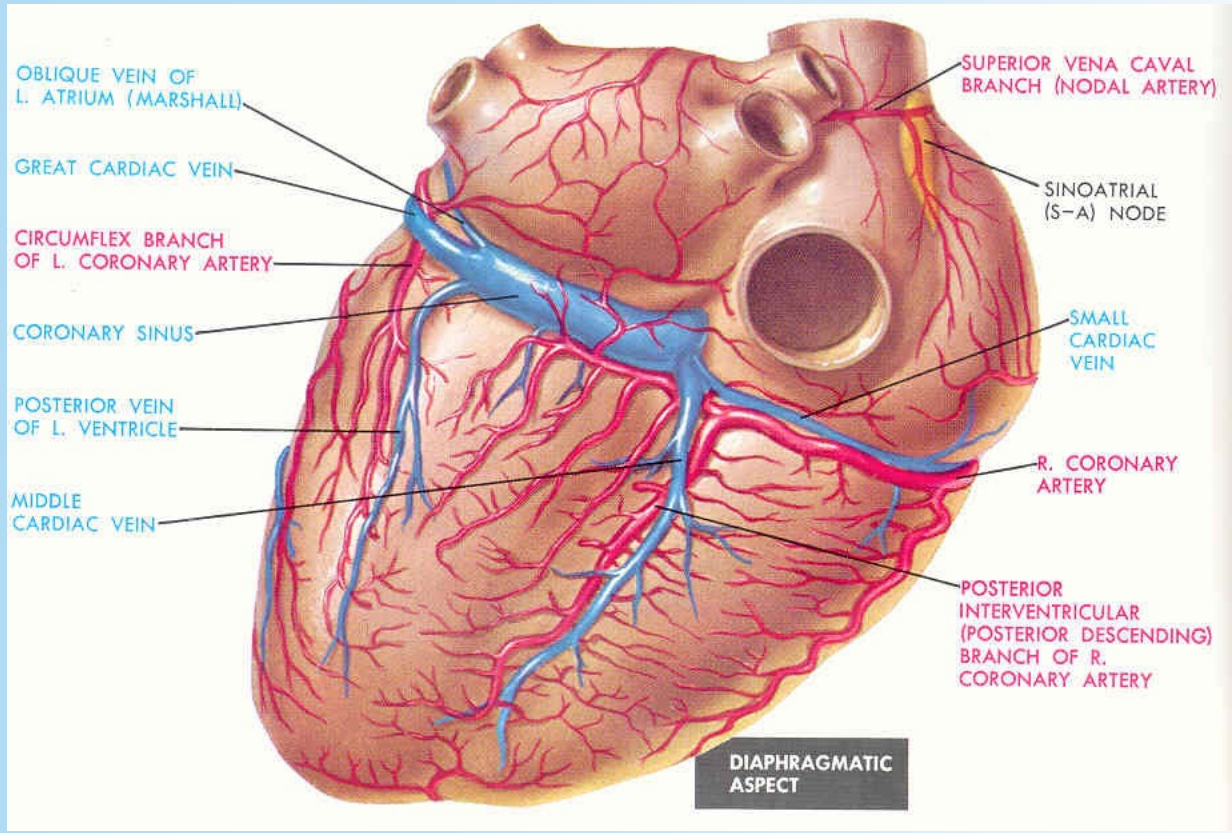


Arteries: O_2 in		Veins: CO_2 out
Right marginal artery	→	Small cardiac vein
Posterior interventricular artery/ Posterior ascending artery	→	Middle cardiac vein
Left Anterior Descending artery (LAD)/ Anterior interventricular artery	→	Great cardiac vein



Arteries and Veins





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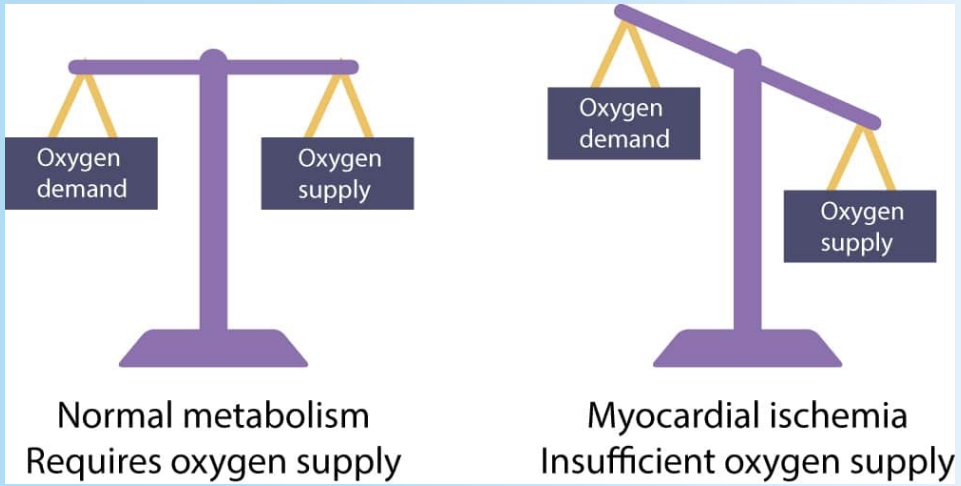
Blood flow & Regulation

Regulation

Myocytes are constantly contracting, which is why it needs oxygen to meet metabolic demands (i.e. ATP).

Therefore, coronary blood flow is tightly regulated by oxygen demand



Blood supply

Myocardial muscle has high oxygen extraction

- Cardiac muscle 60-80%
- Skeletal muscle 20-30%

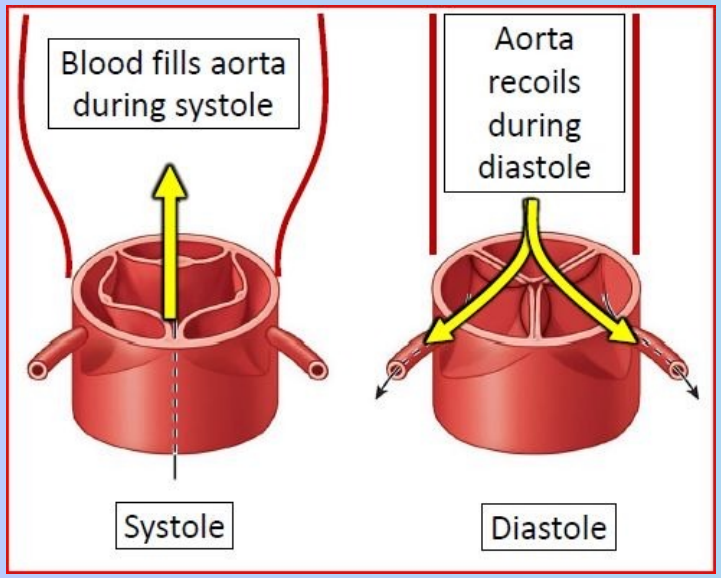
Resting coronary blood flow is ~250mL/min
During exercise can increase up to 2L/min

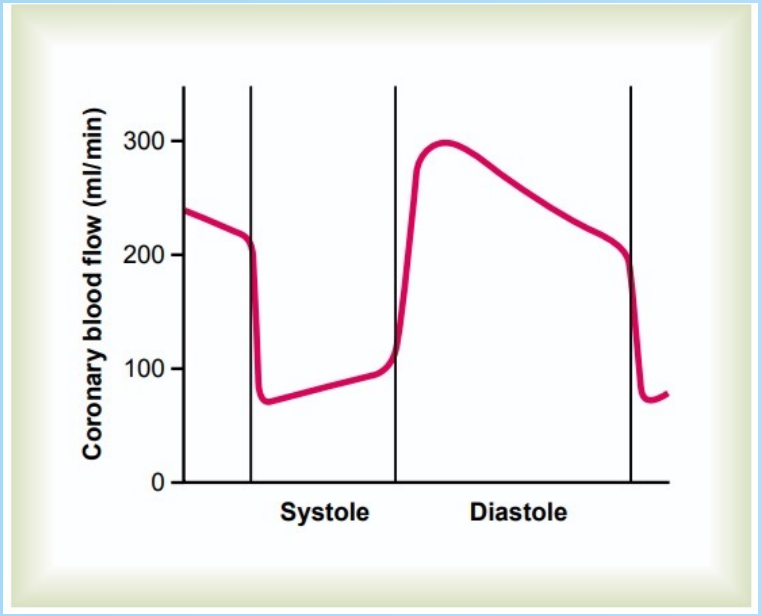
Coronary blood flow phases

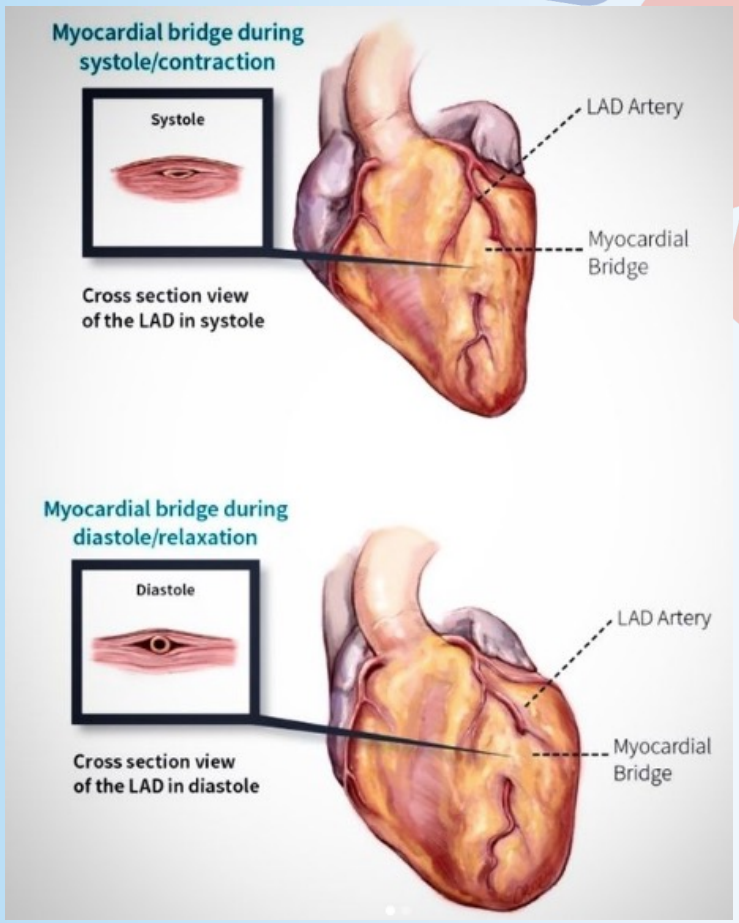
Coronary arteries fill during diastole

Coronary blood flow is cyclical (following systole/diastole)

- Systole is squeezing the heart muscle and will CONSTRICT vessels
  - *If artery is occluded = ischemia*
- Diastole is relaxing, vessels will return to normal







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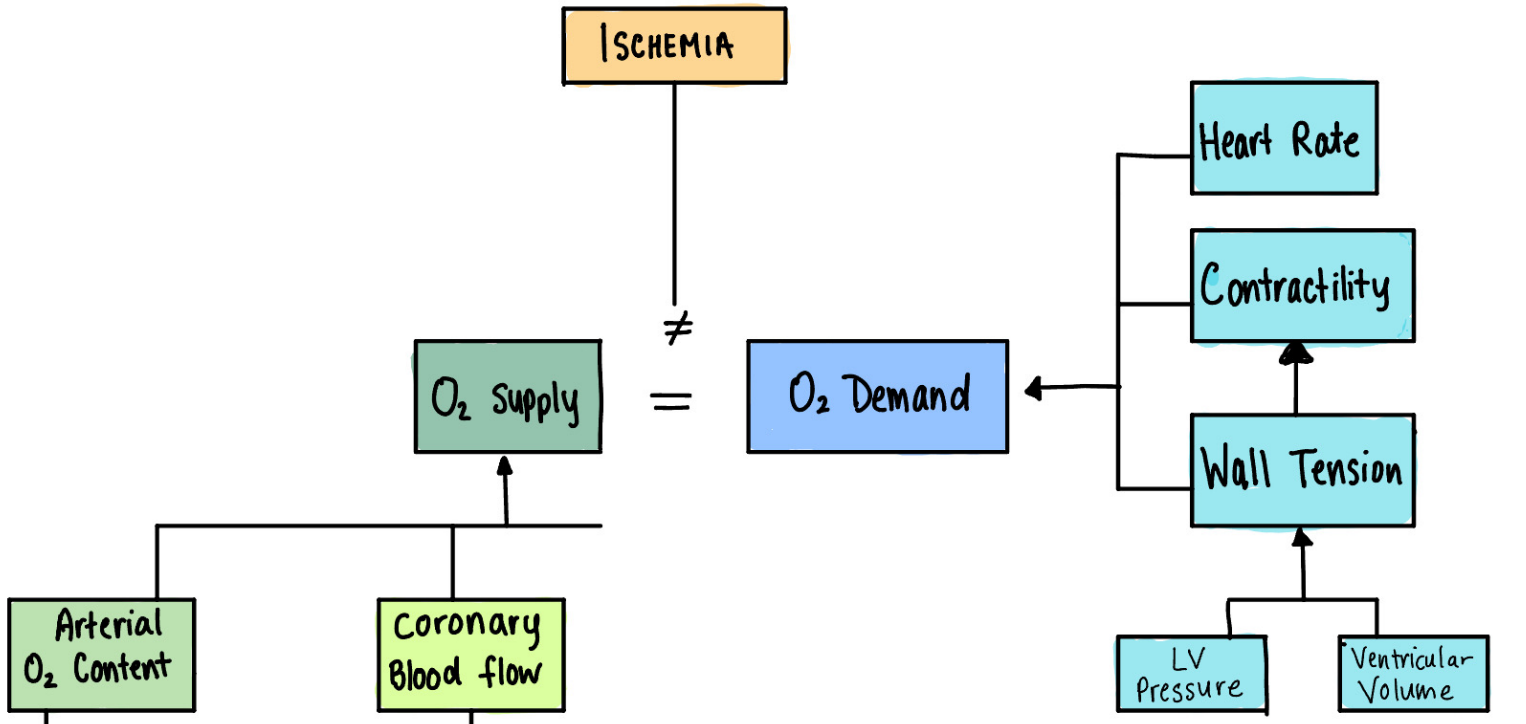


1. Diastolic Perfusion time

2. Coronary Perfusion Pressure

A_o P-LVED Gradient

- L. Ventricular end-diastolic pressure
- Aortic diastolic pressure

3. Coronary vascular Resistance

- Local Autoregulation
  - Myogenic
  - Metabolic
- Mechanical (compression)
- Neural – sympathetic system (ACh, NE on α and β)
- Endothelial (Endothelin-1, Angiotensin II, Thromboxane, Prostaglandin)

Local Autoregulation

Autoregulation is the ability to keep <u>constant</u> blood flow

1. Myogenic

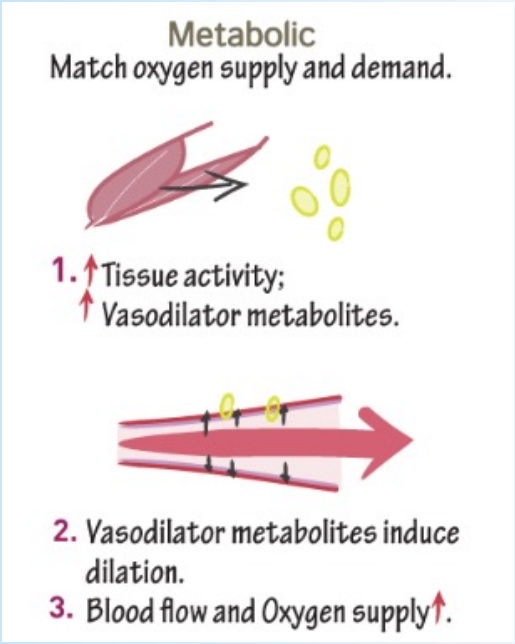
Sometimes called myogenic hypothesis

Intrinsic property of vascular smooth muscle (VSMC)

Pressure-dependent contractions

2. Local Metabolic → hyperemia

- Nitric Oxide (NO)
- O_2
- CO_2
- Adenosine



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1. Myogenic regulation

Autoregulation goal = Keep Flow Constant

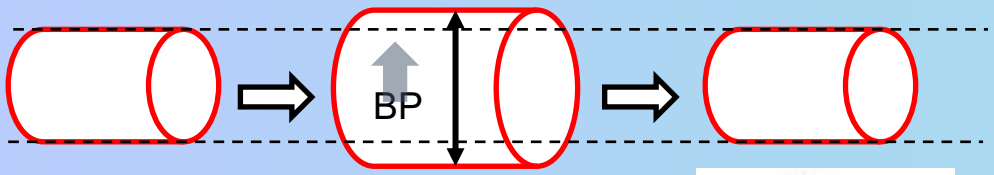
$$\textbf{Flow} = \frac{\Delta P}{R} = \Delta P \times \frac{\pi r^4}{8\eta L}$$

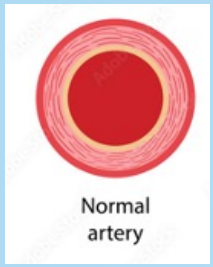
Increase in blood pressure

↑ Δ P = stretch of vessel

↓ decrease radius = vasoconstriction

↑ ↓ = net neutral effect



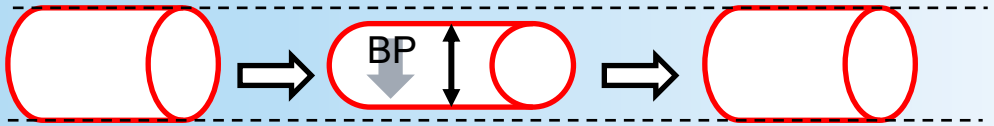


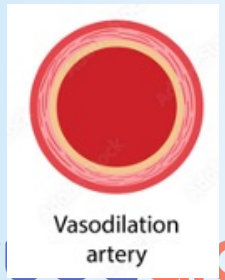
Decrease in blood pressure

↓ Δ P

↑r - increase radius = vasodilation

↓ ↑ = net neutral effect





2. Local metabolites regulation

Hypoxia and metabolic need for oxygen biggest driving force.

Low oxygen ($\downarrow O_2$) from

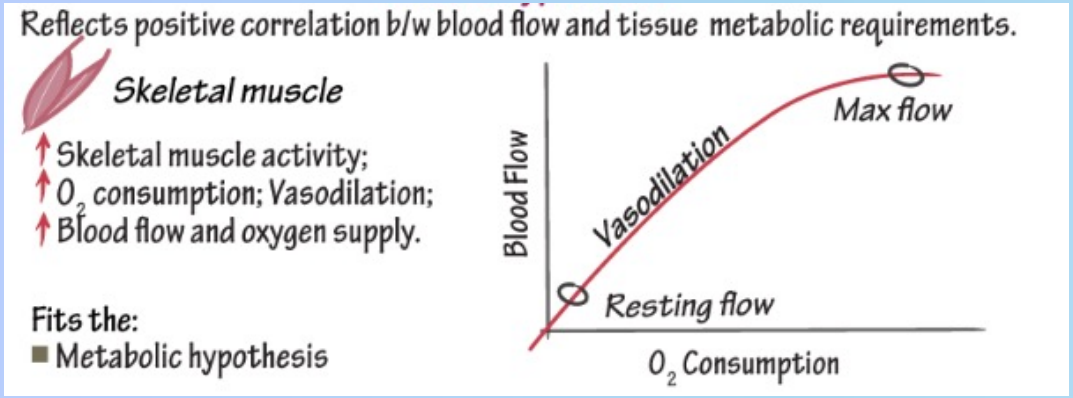
- increased metabolic need (i.e. exercise), used up available oxygen
- Obstruction, oxygen debt created

→ build up of vasodilating metabolites (NO, adenosine, $\uparrow CO_2$)

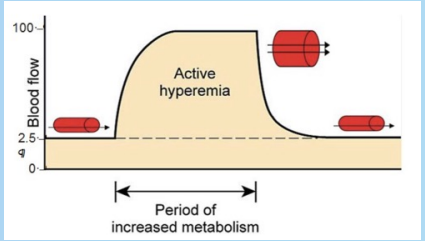
Increased blood flow washes away vasodilator metabolites and blood flow returns to baseline

Hyperemia = high blood flow

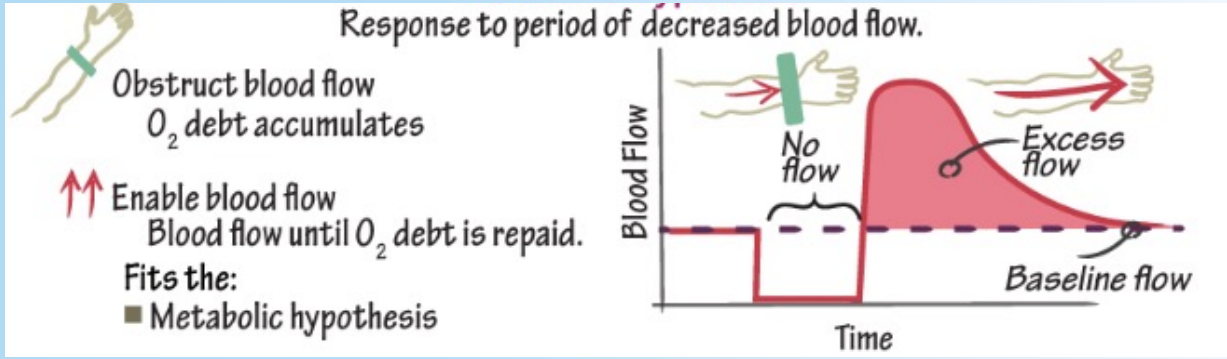
Active Hyperemia



- High blood flow to meet muscle's metabolic need for oxygen
- Proportionate!



Reactive hyperemia



- Something limits blood flow, period of low blood flow
- Remove restriction → High blood flow

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Pathology

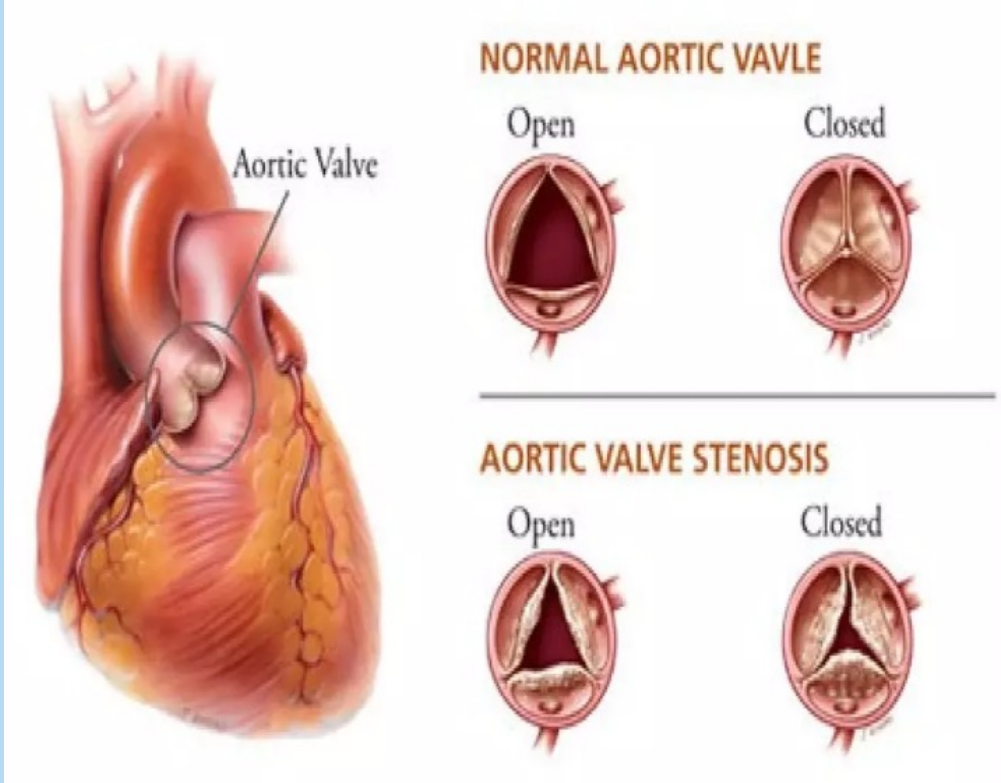
Decrease of blood flow = decrease oxygen

Remember: coronary arteries fill during diastole

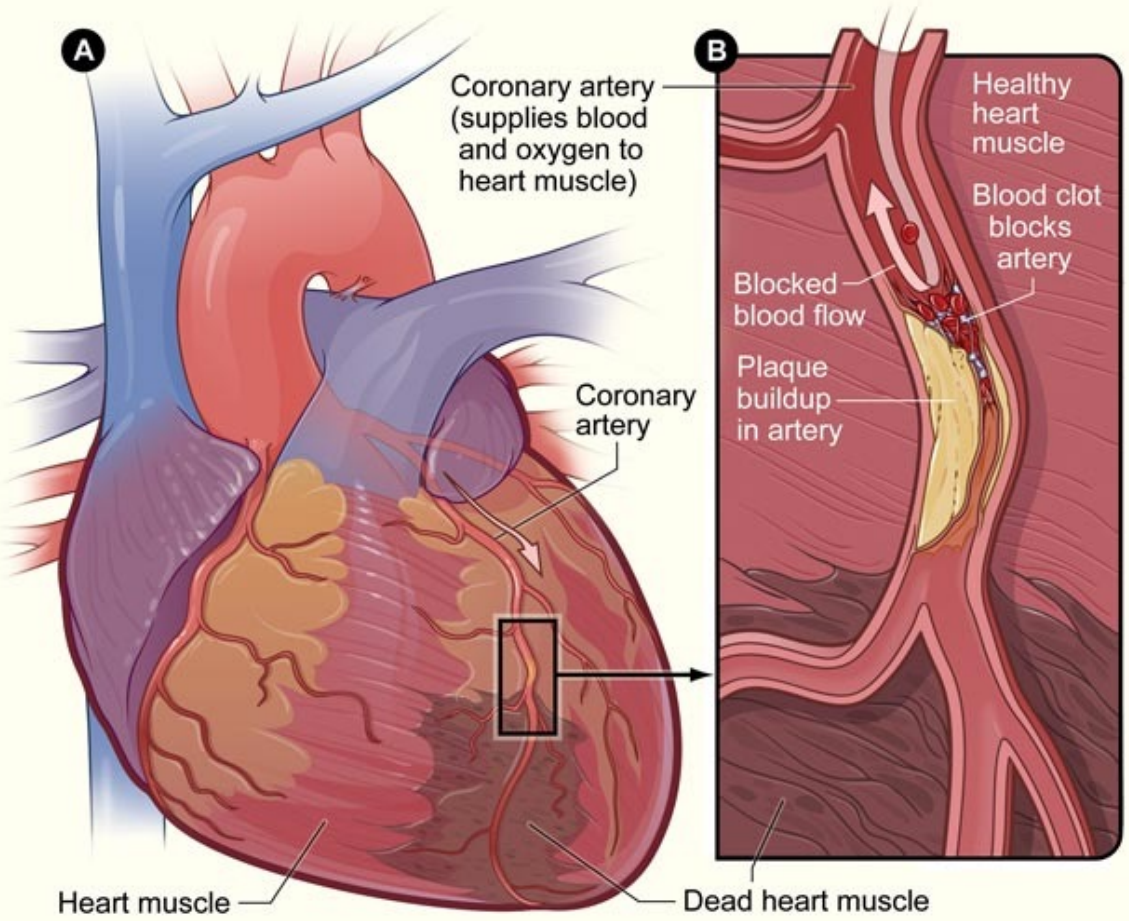
Aortic stenosis can limit coronary artery flow

Tachycardia and arrythmia's shorten coronary artery filling time since the heart muscle cannot fully relax

Clinical application: Important to remember in patients with coronary artery disease (CAD) who already have reduced coronary artery supply



MI: myocardial infarction



Narrowing of blood vessel

→ occlusion

→ O_2 deficit (hypoxemia)

→ Ischemia of heart muscle

= myocardial infarction

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