Cardiac Anatomy and Physiology

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Disclosures

I do not have anything to disclose

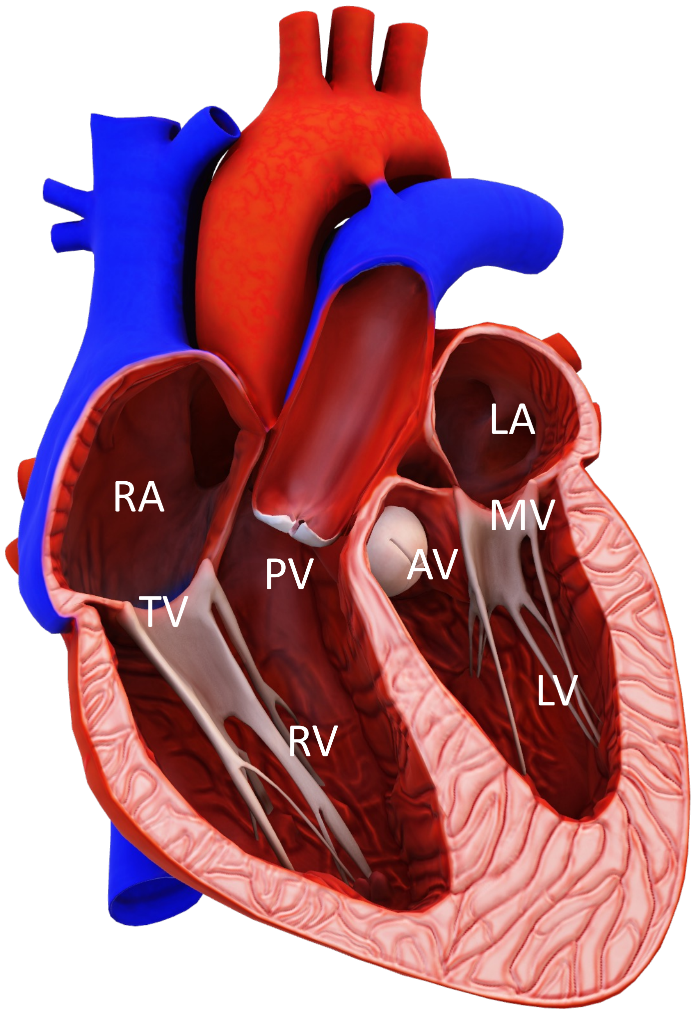
Outline and Learning Objectives

By the end of the lectures, learners should be able to:

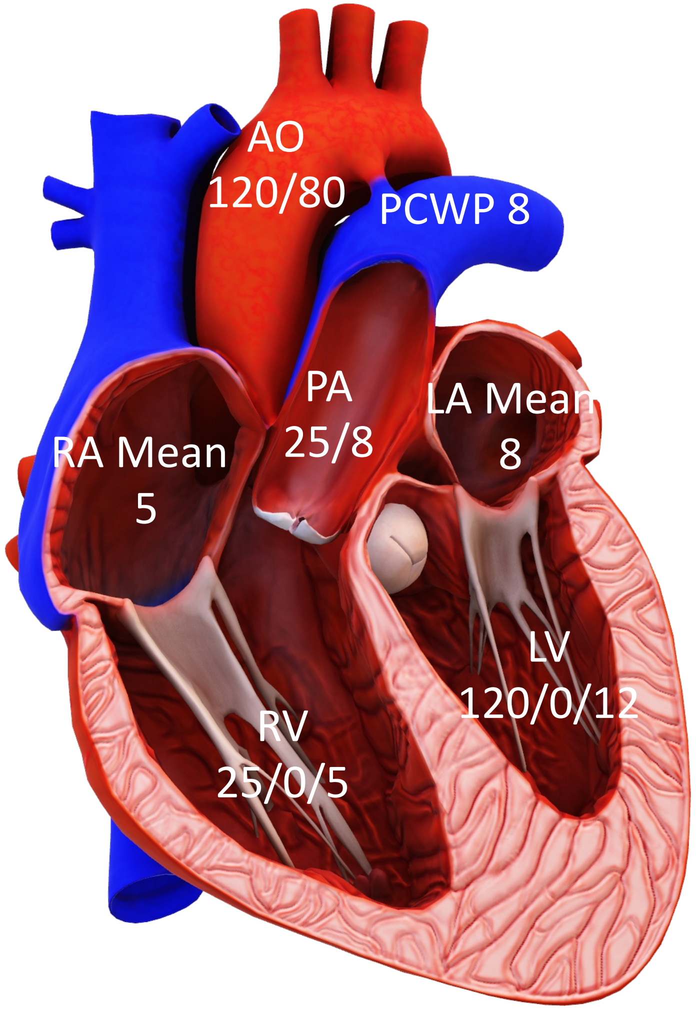
- Understand the basics of cardiac anatomy and coronary circulation
- Understand cardiac action potentials and the conduction pathway
- Understand the Frank-Starling Relationship and how contractility influences it
- Calculate cardiac output and understand the factors that influence it
- Describe the key cardiac reflexes

Cardiac Anatomy

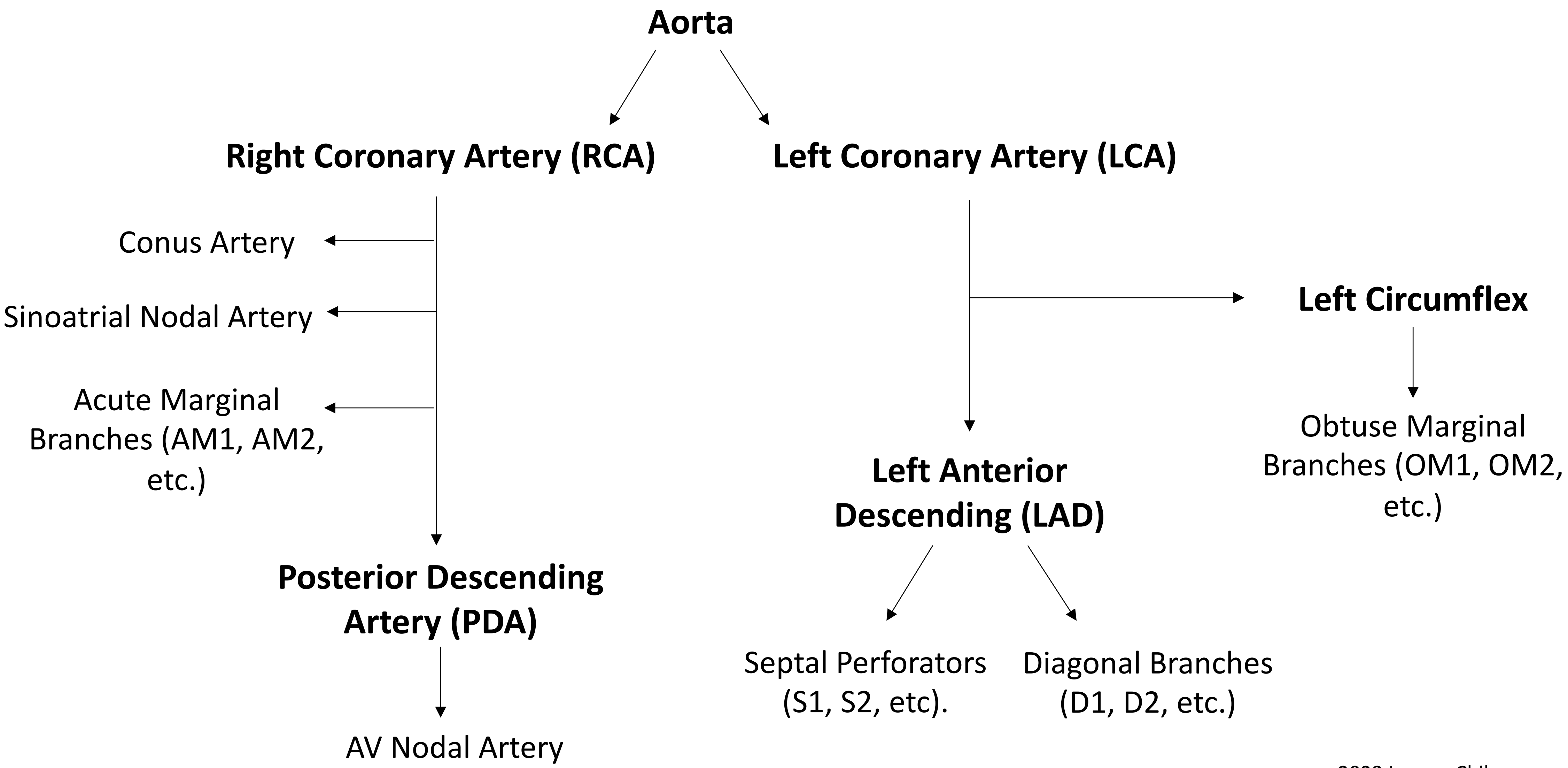
Cardiac Anatomy



Normal Cardiac Pressures



Coronary Circulation



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Coronary Dominance

- Right Dominance: PDA develops from RCA (70% of the population)
- Co-Dominance: PDA develops from both the RCA and left circumflex artery (20% of the population)
- Left Dominance: PDA develops from the left circumflex artery (10% of the population)

Practice Question

75-year-old M with known left dominant circulation presents with an MI and is found to have new complete heart block. Which artery is most likely occluded?

A. RCA
B. LAD
C. Left Circumflex
D. SA nodal artery

Practice Question

75-year-old M with known left dominant circulation presents with an MI and is found to have new complete heart block. Which artery is most likely occluded?

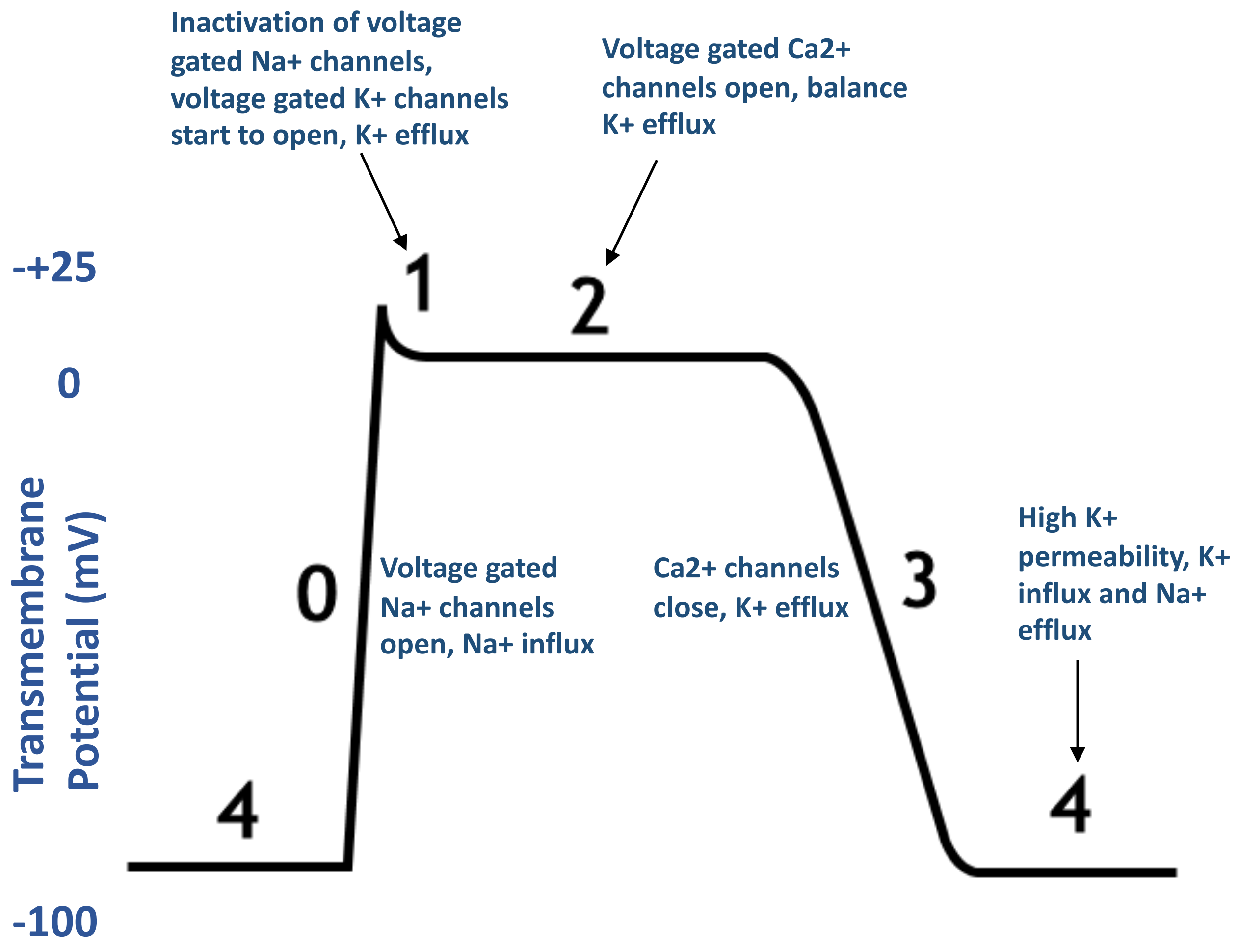
A. RCA
B. LAD
C. Left Circumflex
D. SA nodal artery

In a left dominant circulation, the PDA arises from the left circumflex. The PDA supplies the AV node and disruption of blood supply to the AV node can lead to complete heart block

Disruption of the SA nodal artery would lead to junctional escape rhythm vs sinus arrest

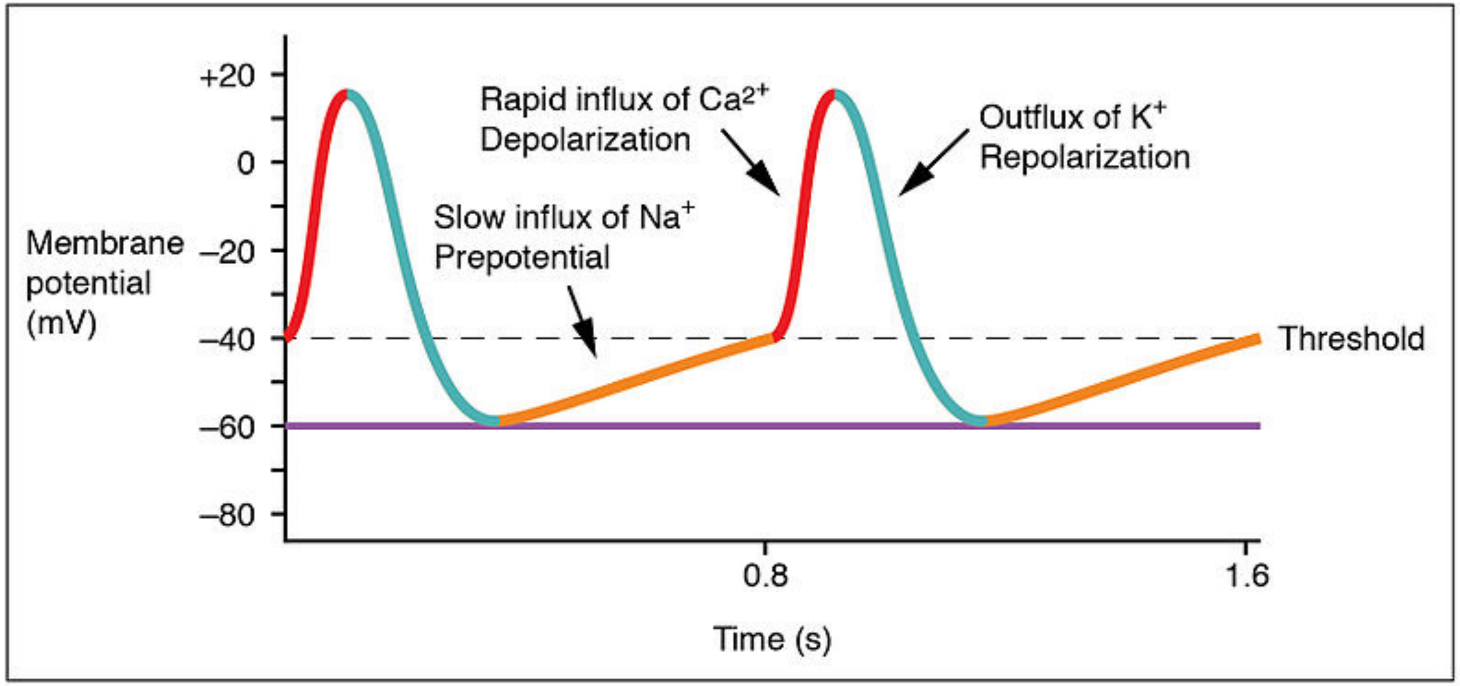
Action Potential and Conduction Pathway

Myocardial Action Potential



- Phase 0: Rapid upstroke and depolarization
- Phase 1: Initial repolarization
- Phase 2: Plateau
  - Ca^{2+} influx triggers Ca^{2+} release from SR and myocyte contraction
- Phase 3: Rapid repolarization
- Phase 4: Resting potential

Pacemaker Action Potential



- Phase 0: Upstroke (Ca^{2+} in)
- Phase 3: Repolarization (Ca^{2+} channels close, K^{+} out)
- Phase 4: slow spontaneous diastolic depolarization due to I_f (slow, mixed Na^{+}/K^{+} inward current)

Conduction Pathway and EKG

SA Node- ***no EKG deflection***

↓

Atria -*p wave*

↓

AV Node- ***no EKG deflection***

↓

Bundle of His- ***no EKG deflection***

↓

Right and Left Bundle Branches- ***LV free wall activation→ QRS***

↓

Purkinje Fibers- ***no EKG deflection***

↓

Ventricles- ***no EKG deflection***

↓

Ventricle repolarization- ***T wave***

Cardiac Cycle, Frank-Starling, and Cardiac Output

Ventricular Systole (2 Phases)

1. Isovolumic Contraction: Phase between start of ventricular systole and opening of the aortic/pulmonic valve

2. Ejection: Phase after aortic/pulmonic valve have opened

Ventricular Diastole (4 Phases)

1. Isovolumic Relaxation: Phase between closure of aortic/pulmonic valve and opening of MV/TV

2. Rapid Filling Phase: After opening of MV/TV

3. Slow Filling Phase (Diastasis)

4. Final Filling Phase during Atrial Systole

Preload and Afterload

- Preload: ventricular load at the end of diastole, before contraction has started
  - Clinically, we use pulmonary wedge pressure or CVP to estimate preload

- Afterload: systolic load on the LV after contraction has begun
  - Clinically, we use systolic blood pressure to approximate afterload

Laplace's Law

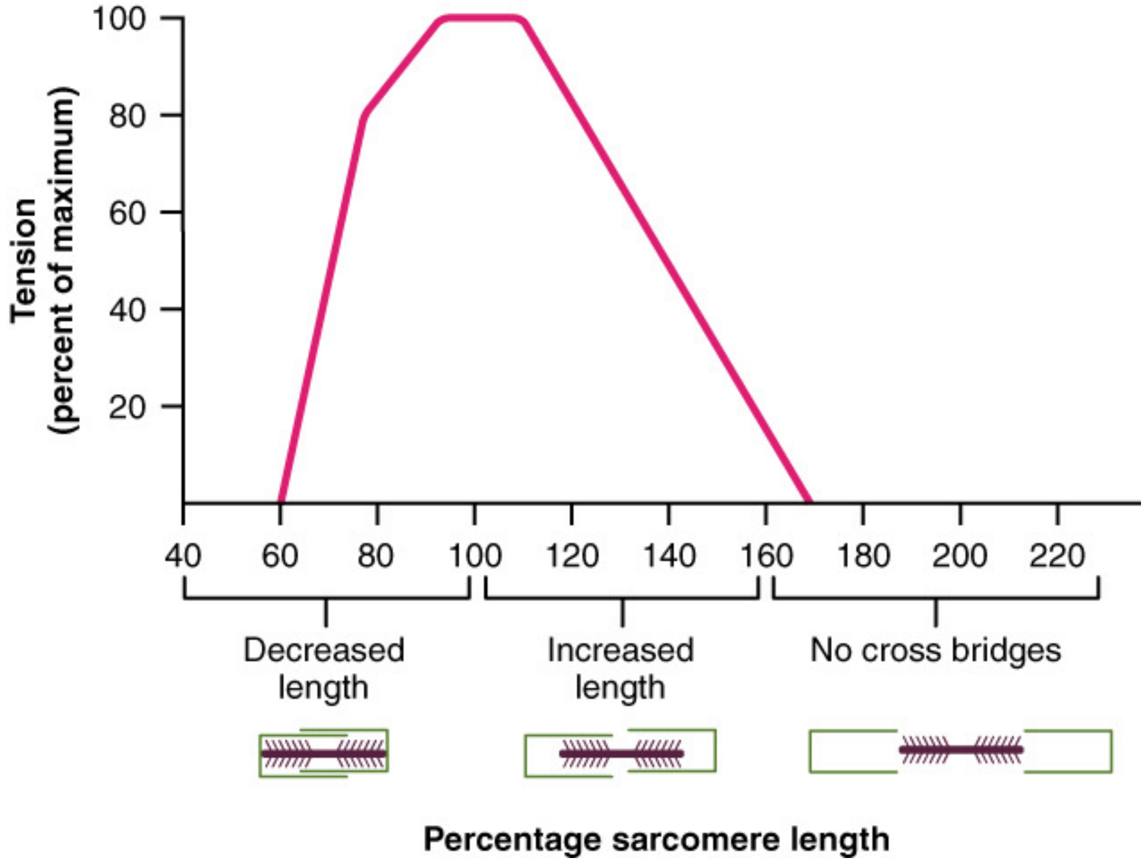
- Can think of preload and afterload as the wall stress present at the end of diastole and during LV ejection, respectively
- Can estimate wall stress with Laplace's Law:

$$\sigma = P x R / 2h$$

Where σ=wall stress, P= pressure, R= radius, h= wall thickness

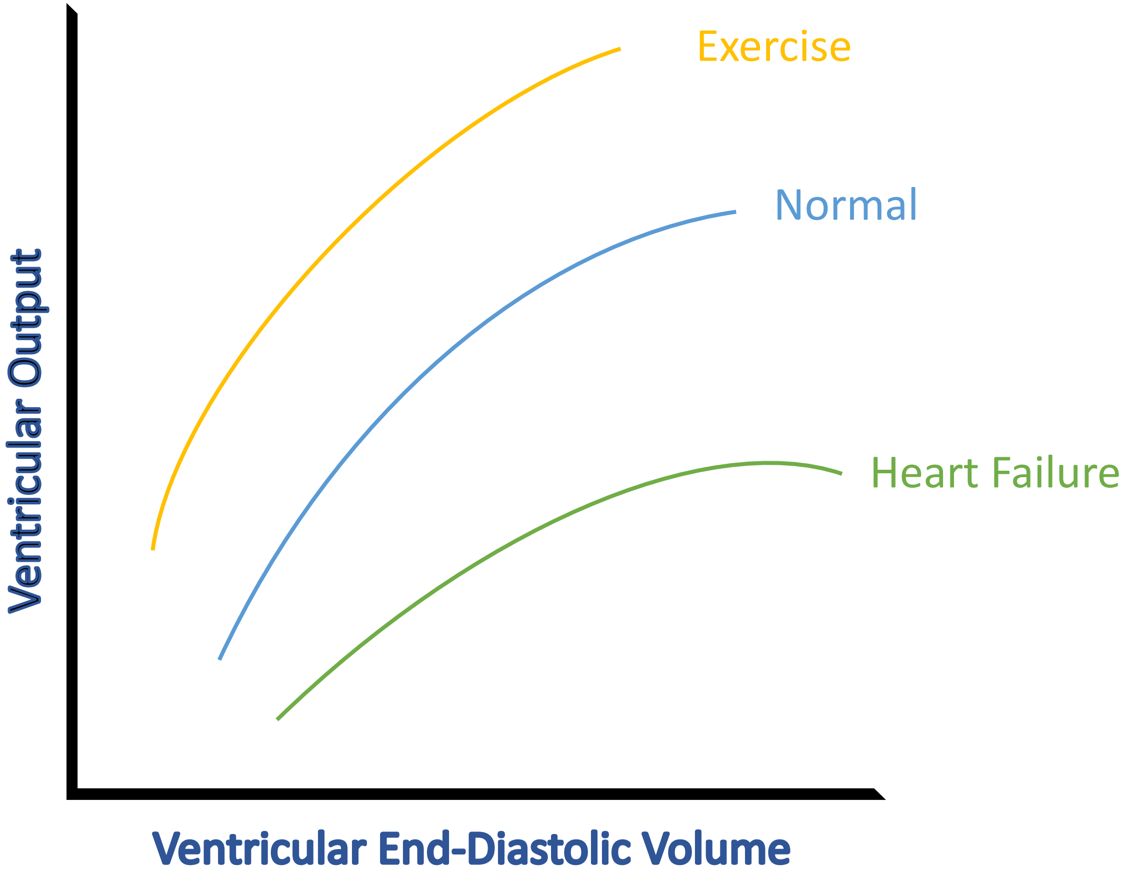
- Clinical example: aortic stenosis
  - Pressure increased secondary to AS, so in order to maintain similar wall stress LV thickness increases (hypertrophy)

Frank-Starling Relationship



- There is a relationship between end-diastolic sarcomere length (preload) and myocardial force of contraction
- Increased preload → optimal sarcomere length → improved contractions → increased SV

Frank-Starling Curves



- Frank Starling Curves are dependent on the level of contractility of the heart
- Factors that increase contractility (such as catecholamines) shift the curve to the left
- Factors that decrease contractility (such as HF, beta blockers) shift the curve to the right

Myocardial Contractility

- Contractility can be defined as the strength of contraction of myocardial fibers at a given preload and afterload

Factors that ↑ Contractility	Factors that ↓ Contractility
• SNS activation • Catecholamine stimulation • Inotropic agents (such as milrinone, calcium) • Increased intracellular calcium • Increased heart rate	• Beta Blockade • Acidosis • Hypoxia/Hypercapnia • Non-dihydropyridine Ca2+ channel blockers

Cardiac Output

- Cardiac output (CO) is the amount of blood pumped by the heart per unit time

CO= Stroke Volume (SV) x Heart Rate (HR)

- Stroke volume (SV) is the amount of blood pumped out of the LV during a systolic contraction

SV= end-diastolic volume (EDV)- end-systolic volume (ESV)

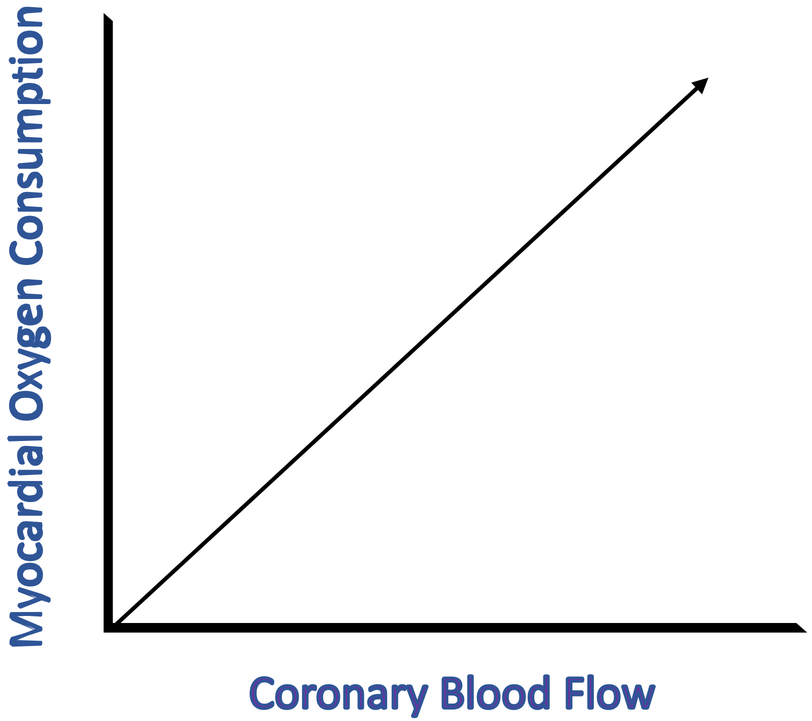
SV impacted by contractility, afterload, and preload

Cardiac Output: Fick Principle

- Based on the law of conservation of mass
  - O2 delivered to the pulmonary capillaries via the pulmonary artery (q1) and from the alveoli (q2) must equal the amount of O2 caried away by the pulmonary veins (q3)

- CO= Rate of O2 consumption (q2)/ (arterial O2 content- venous O2 content)

Myocardial Oxygen Utilization



- Myocardial Oxygen Demand is increased by:
  - ↑ Contractility
  - ↑ Afterload
  - ↑ Heart Rate
  - ↑Preload

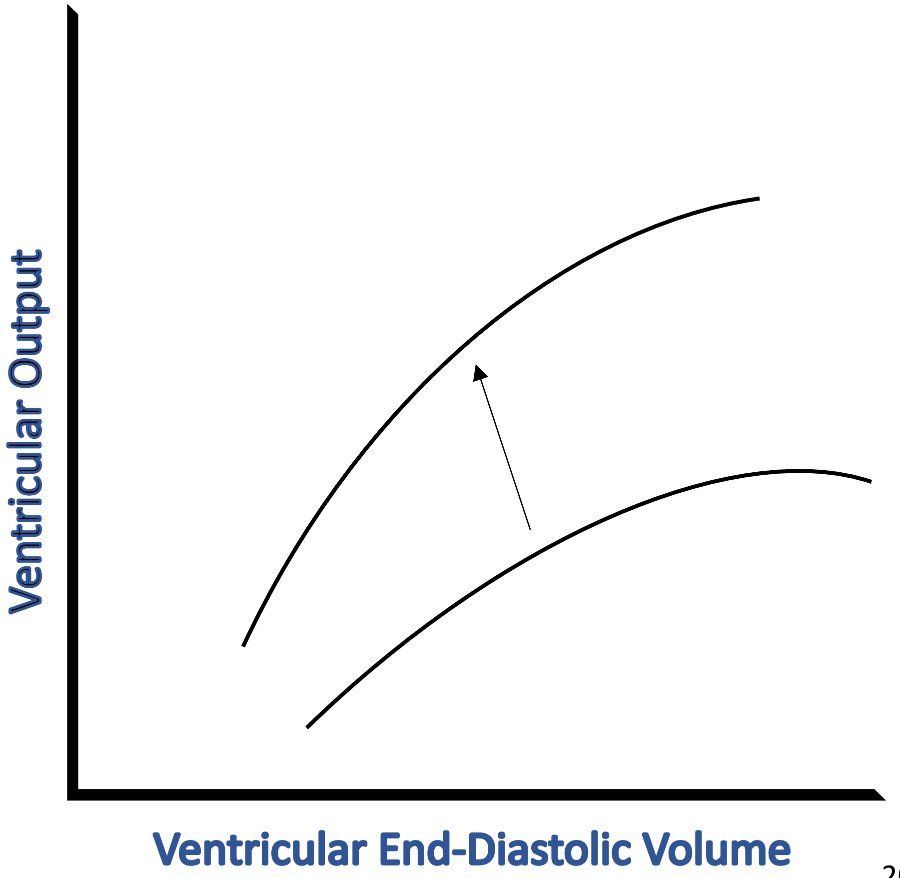
- Myocardial oxygen consumption and coronary blood flow have a positive relationship

Myocardial Oxygen Utilization

Practice Question

Administering which medication would cause the following change to the Frank Starling Curve for a patient with heart failure with reduced ejection fraction?

A. Furosemide
B. Labetalol
C. Digoxin
D. Phenylephrine



Practice Question

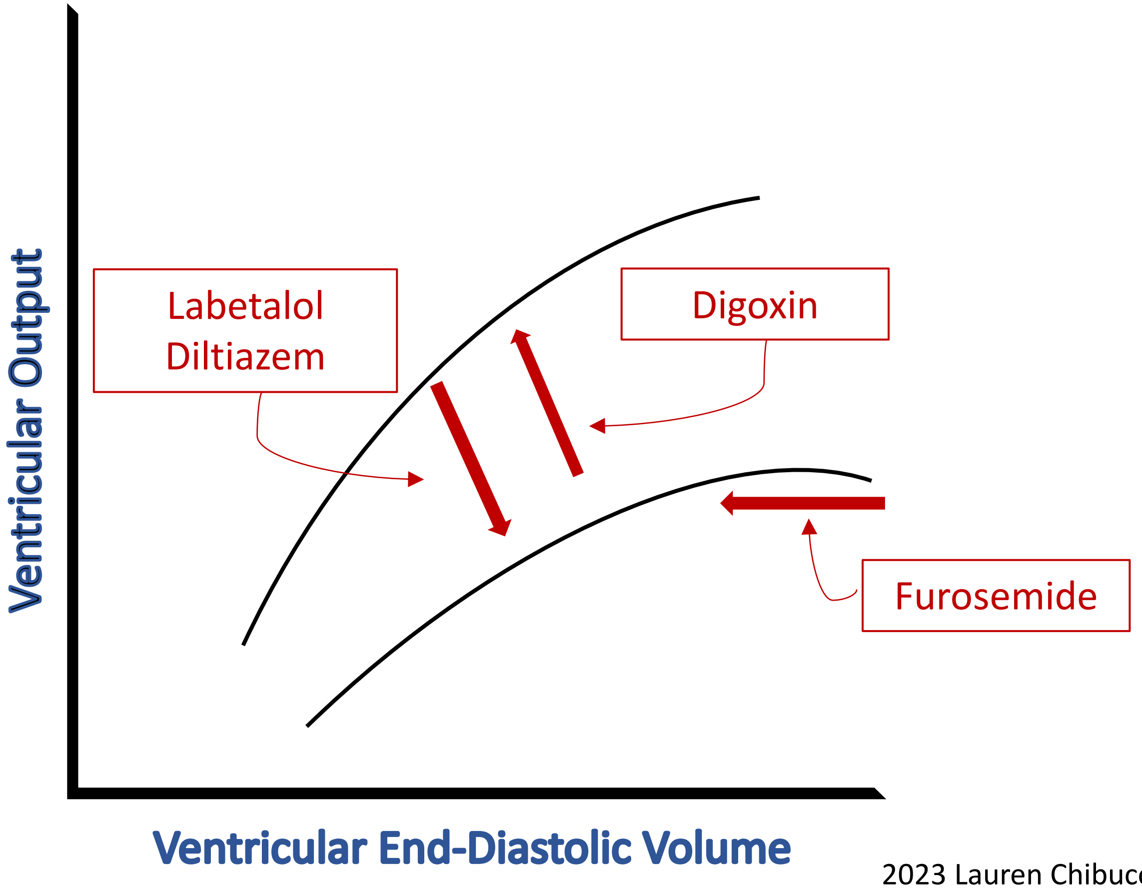
Administering which medication would cause the following change to the Frank Starling Curve for a patient with heart failure with reduced ejection fraction?

A. Furosemide

B. Labetalol

C. Digoxin

D. Diltiazem

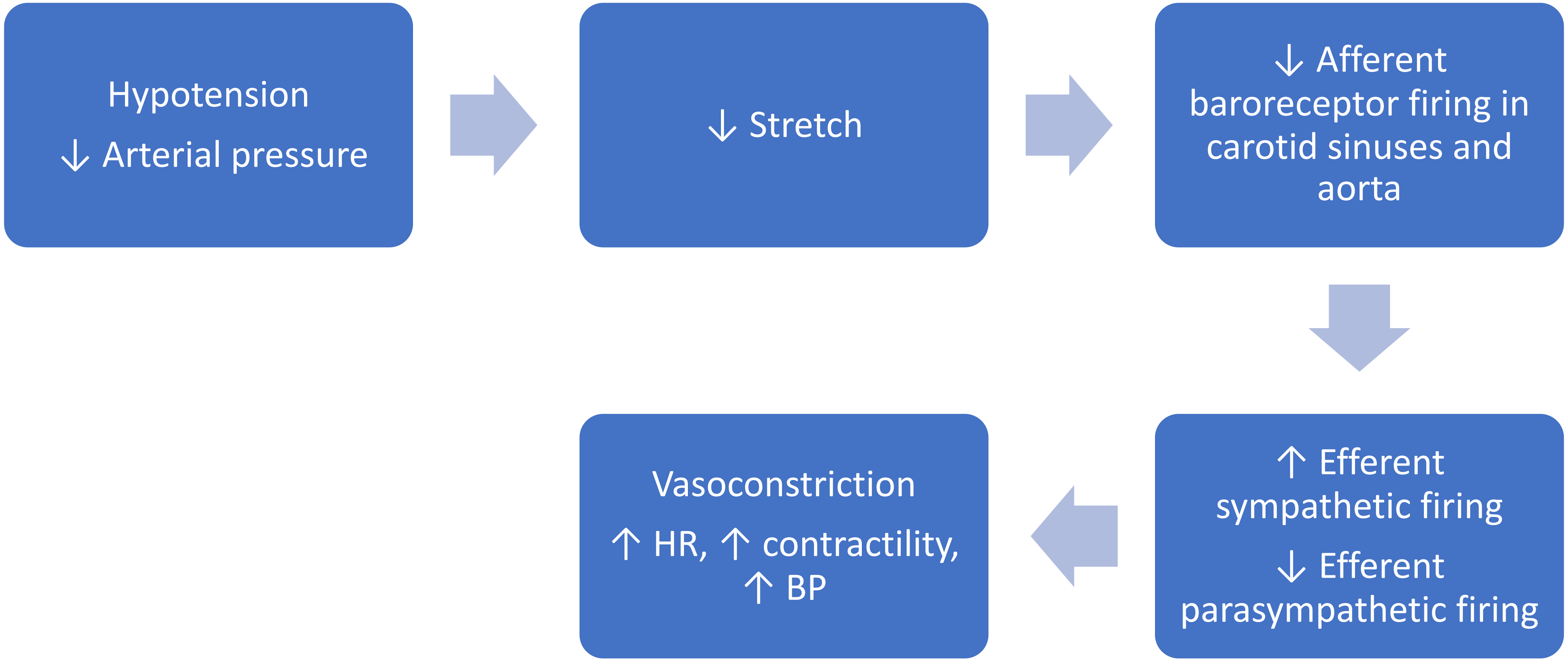


Cardiac Reflexes

Cardiac Reflexes

- Fast-acting reflex loops between the heart and CNS
- Cardiac Receptors:
  - Within atria, ventricles, pericardium, and coronary arteries
- Extracardiac Receptors:
  - Aortic arch and carotid sinus
- Sympathetic and parasympathetic nerve input is processed in the CNS and then efferent fibers to the heart (SA or AV nodes) or the systemic circulation provoke a reaction

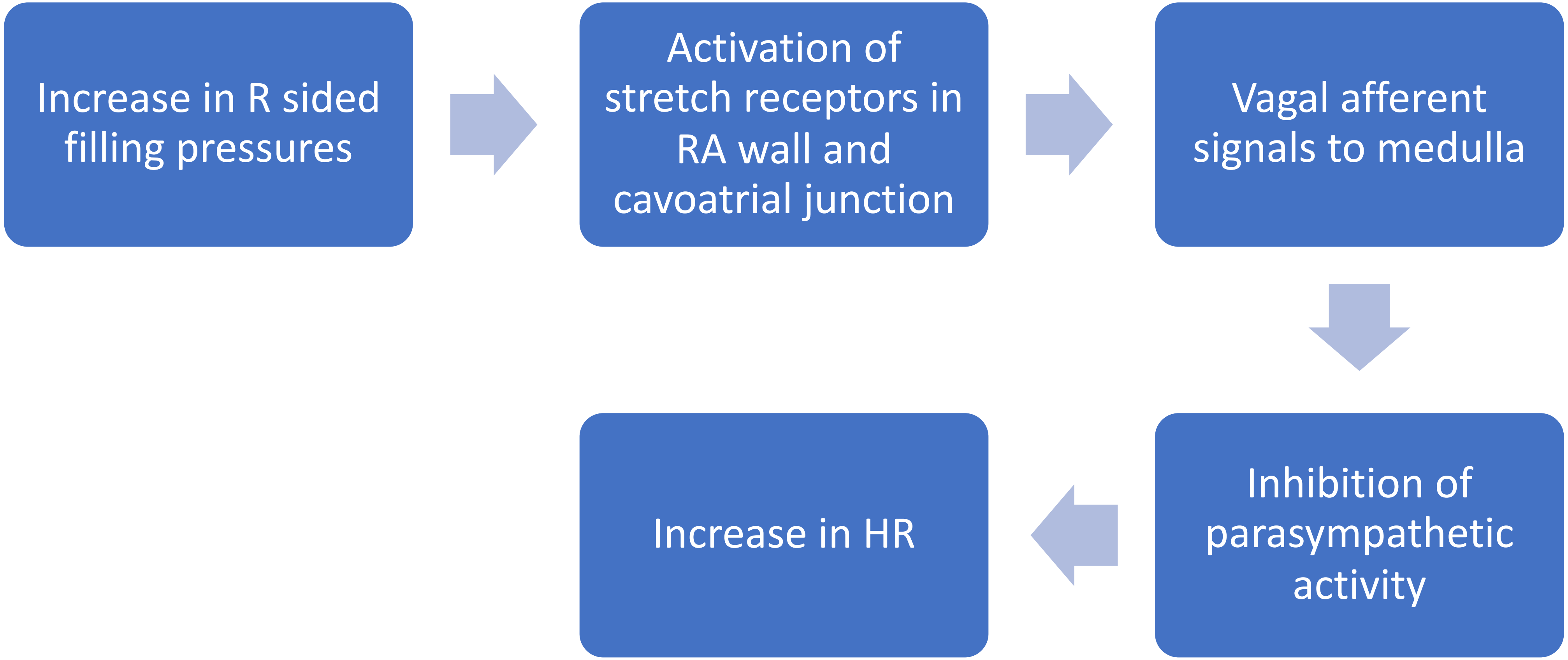
Baroreceptor Reflex (Carotid Sinus Reflex)



Chemoreceptor Reflex

- Both the carotid body and the aortic body have chemosensitive cells
- These cells are stimulated by $PaO_2 < 50$ and acidosis and send signals via the glossopharyngeal nerve to the medulla
- Medulla then stimulates the respiratory centers → increase respiratory drive (to increase PaO_2 and resolve acidosis)
- Additionally, the parasympathetic nervous system is activated → reduced HR and contractility

Bainbridge Reflex



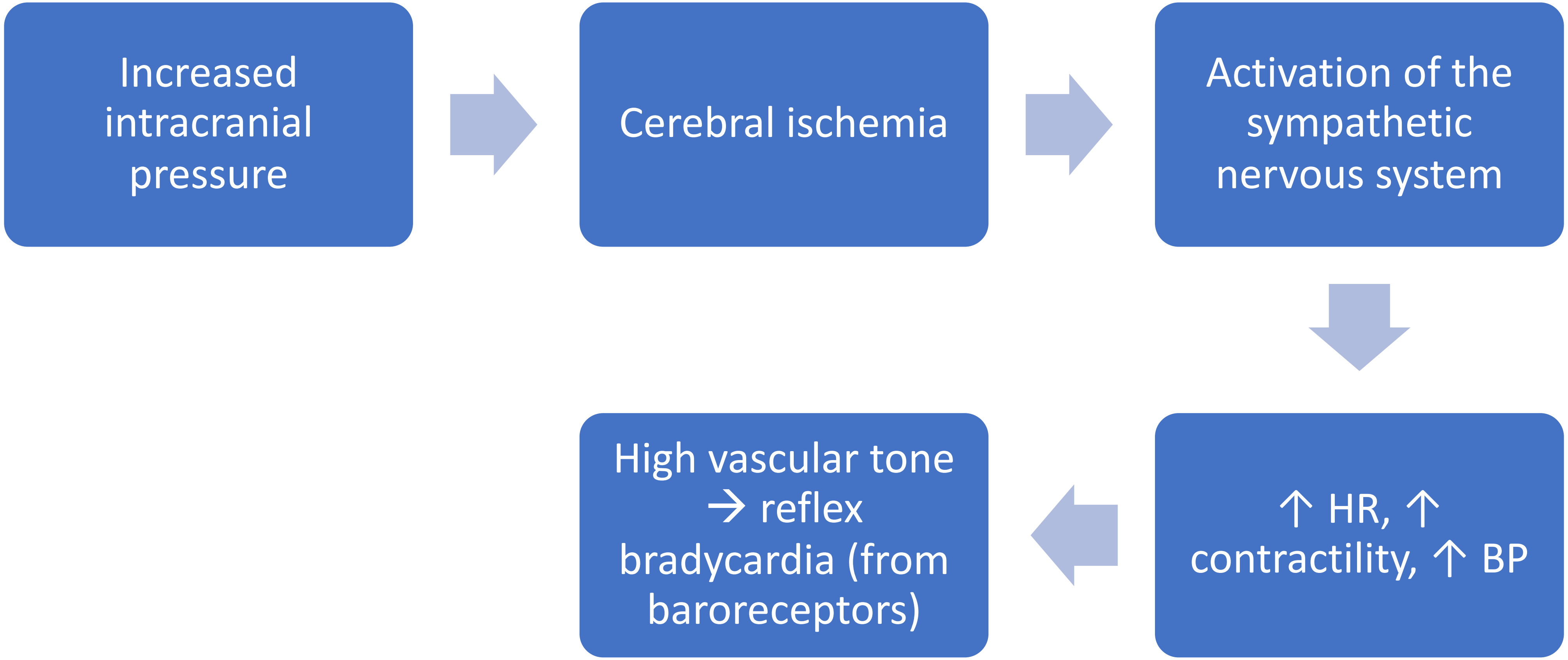
Bezold-Jarisch Reflex

- Chemoreceptors and mechanoreceptors within the LV wall respond to noxious ventricular stimuli
- Activated receptors communicate via vagal afferent type C fibers → increased parasympathetic tone
- Induces the triad of hypotension, bradycardia, and coronary artery dilation
- Thought to be related to the physiologic response to: MI, thrombolysis, revascularization, and syncope

Valsalva Maneuver

- Valsalva maneuver causes increased intrathoracic pressure → increased CVP and decreased venous return
- This leads to decreased CO and BP → increase in HR and contractility through sympathetic activation
- When the maneuver is stopped, venous return is increased and blood pressure is increased → baroreceptors sense this increase in blood pressure which stimulates parasympathetic activation and decrease in HR

Cushing Reflex



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Oculocardiac Reflex

- Provoked by pressure applied to the globe of the eye or traction to surrounding structures → activates stretch receptors → increased parasympathetic tone and subsequent bradycardia
- Incidence ranges from 30-90% of ophthalmic surgeries

Practice Question

A patient is in SVT with heart rates in the 170s. You perform a carotid sinus massage, which decreases his HR. What cardiac reflex is occurring?

A. Bainbridge Reflex
B. Baroreceptor Reflex
C. Bezold-Jarisch Reflex
D. Cushing Reflex
E. Chemoreceptor Reflex

Practice Question

A patient is in SVT with heart rates in the 170s. You perform a carotid sinus massage, which decreases his HR. What cardiac reflex is occurring?

A. Bainbridge Reflex
B. Baroreceptor Reflex ←
C. Bezold-Jarisch Reflex
D. Cushing Reflex
E. Chemoreceptor Reflex

Carotid sinus massage → ↑ stretch in carotid sinus pressure receptors → ↑ afferent baroreceptor firing → ↓ efferent sympathetic firing and ↑ efferent parasympathetic firing → ↓ HR

Questions?

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