Cardiac Anatomy and Physiology

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Disclosures

I do not have anything to disclose

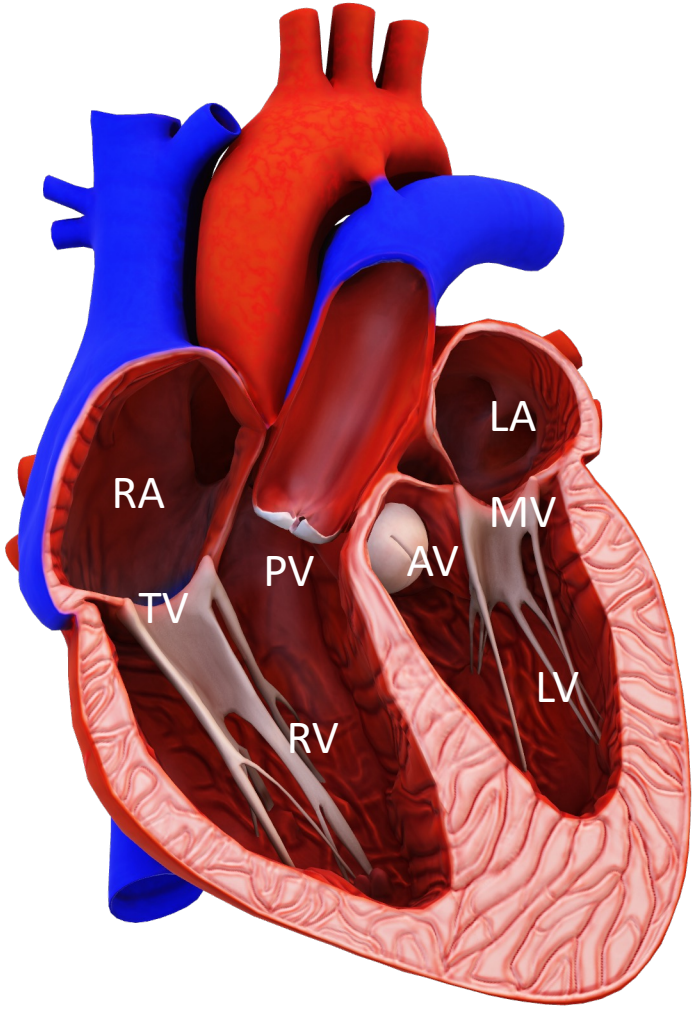
Outline and Learning Objectives

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

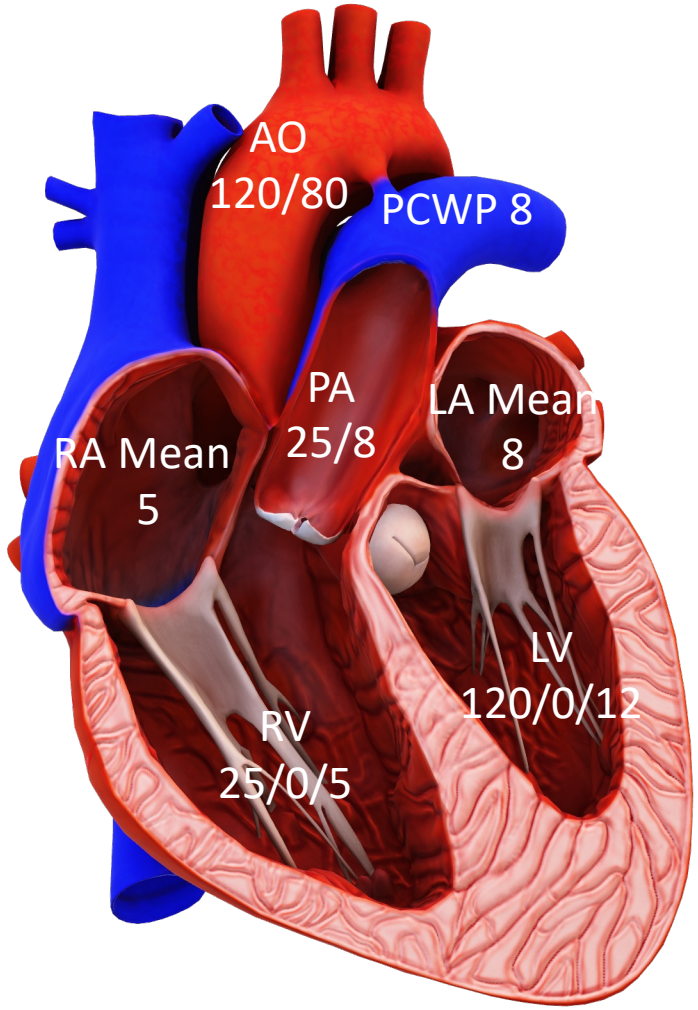
- Understand the basics of cardiac anatomy and coronary circulation
- Understand cardiac action potentials and the conduction pathway
- Understand the Frank-Starling Relationship and how contractility influences it
- Calculate cardiac output and understand the factors that influence it
- Describe the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

Cardiac Anatomy

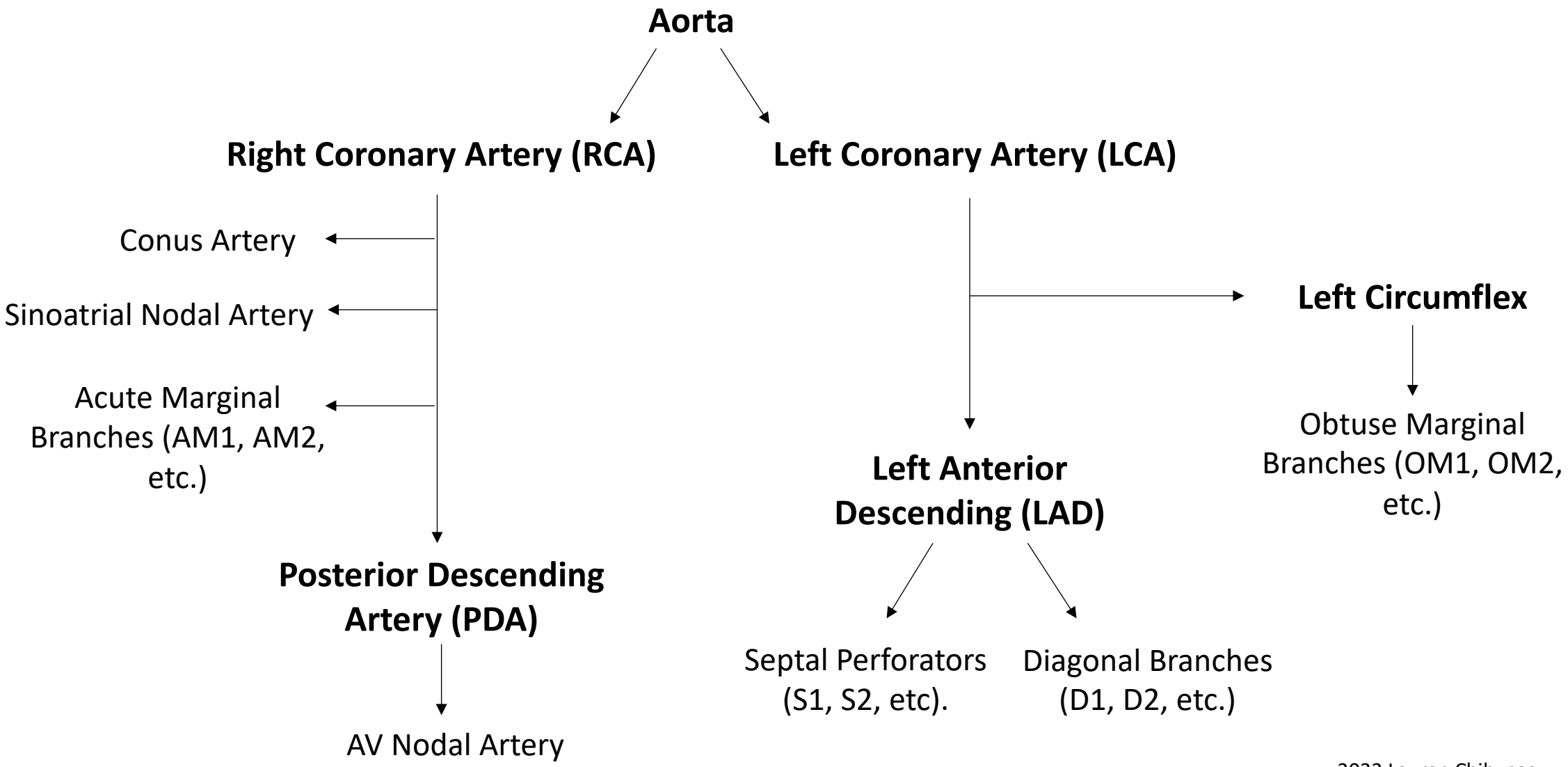
Cardiac Anatomy



Normal Cardiac Pressures



Coronary Circulation

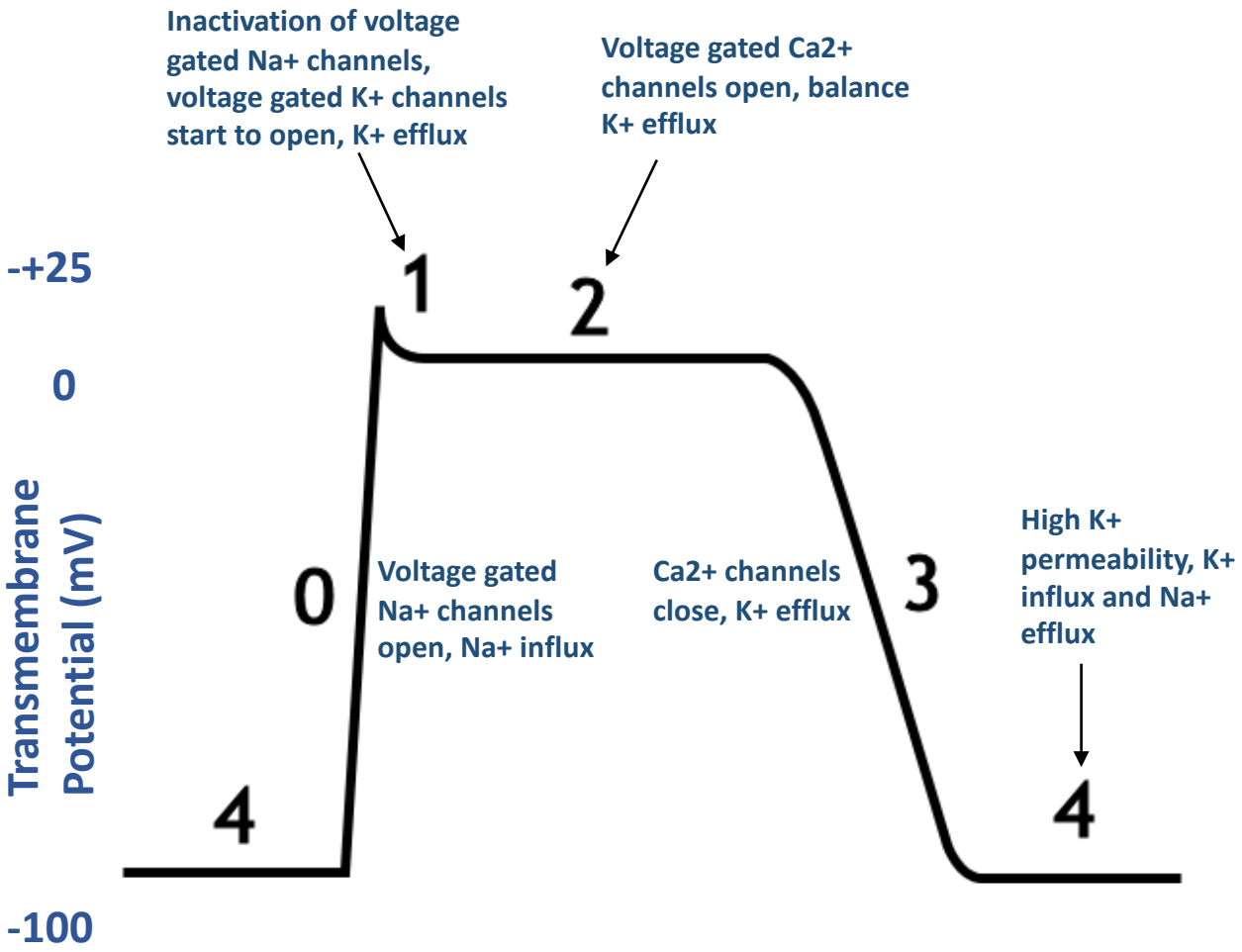


Coronary Dominance

- 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)

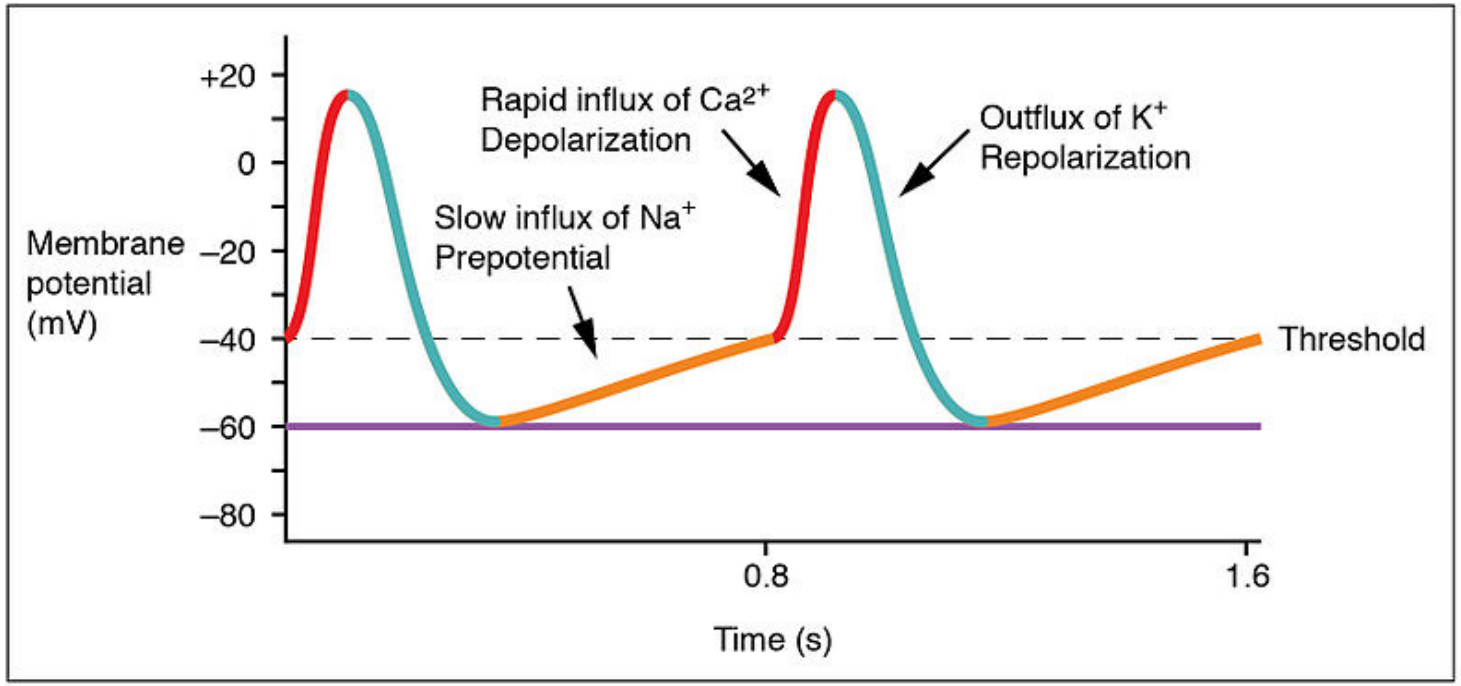
Action Potential and Conduction Pathway

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

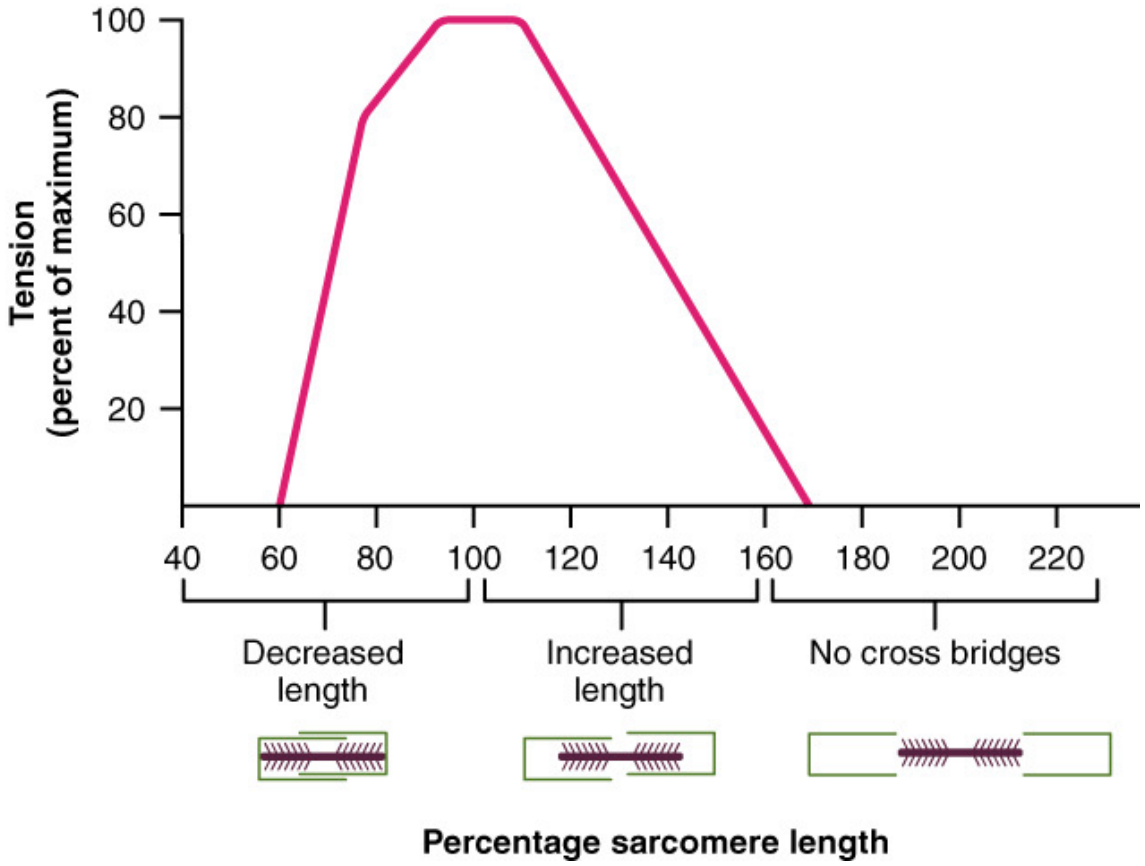
- Can think of preload and afterload as the wall stress present at the end of diastole and during LV ejection, respectively
- Can estimate wall stress with Laplace's Law:

$$\sigma = PxR/2h$$

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

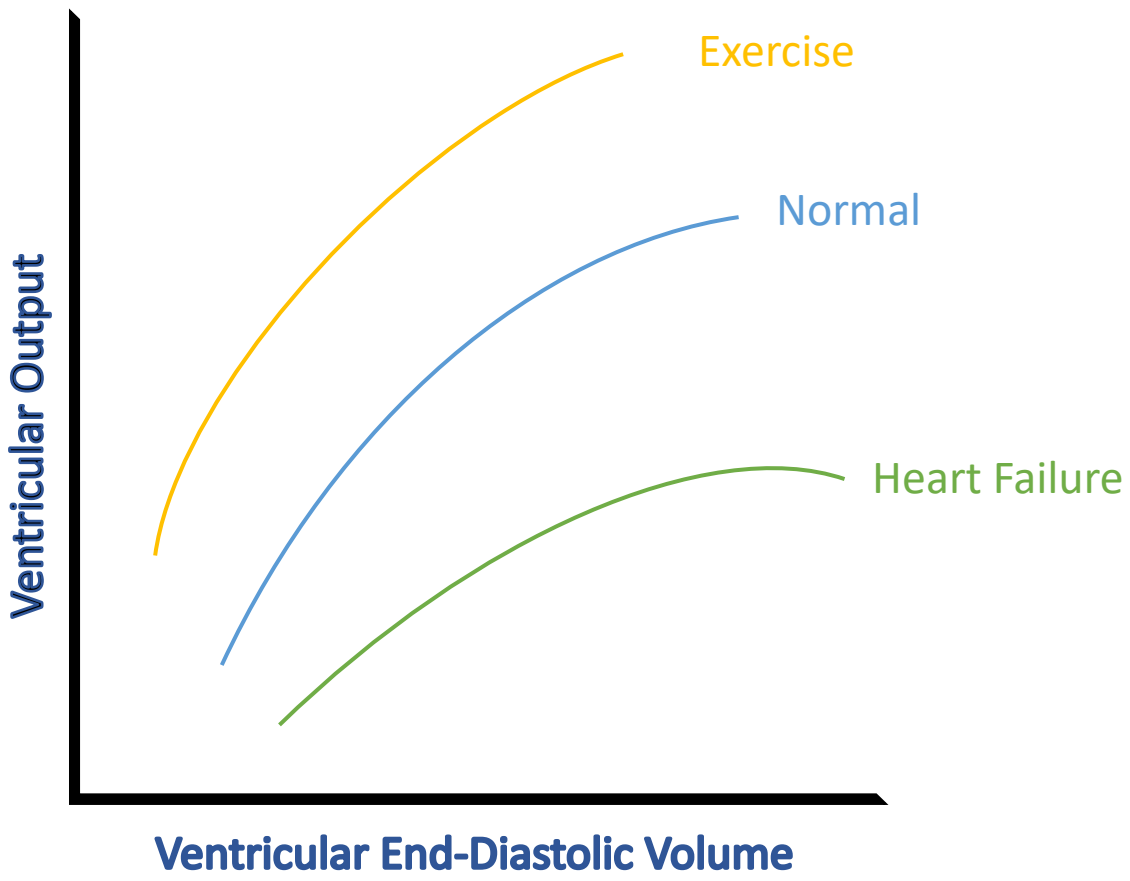
- Clinical example: aortic stenosis
  - Pressure increased secondary to AS, so in order to maintain similar wall stress LV thickness increases (hypertrophy)

Frank-Starling Relationship



- There is a linear 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, digoxin) 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 digoxin, 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)

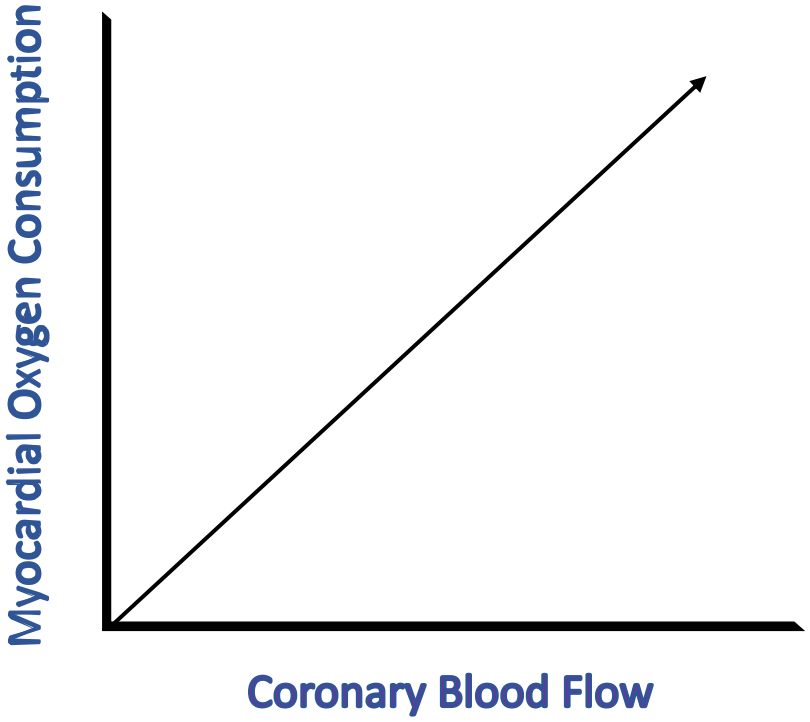
SV impacted by contractility, afterload, and preload

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

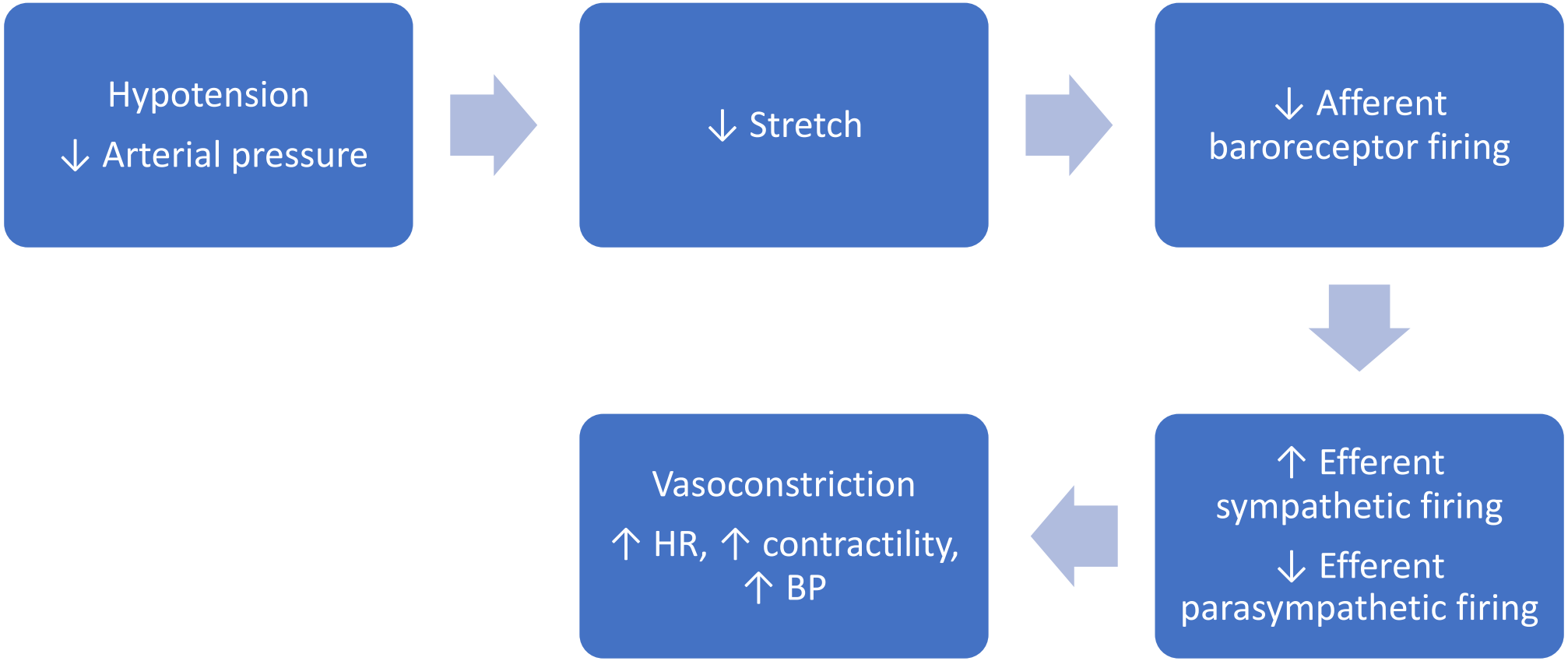
Myocardial Oxygen Utilization

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

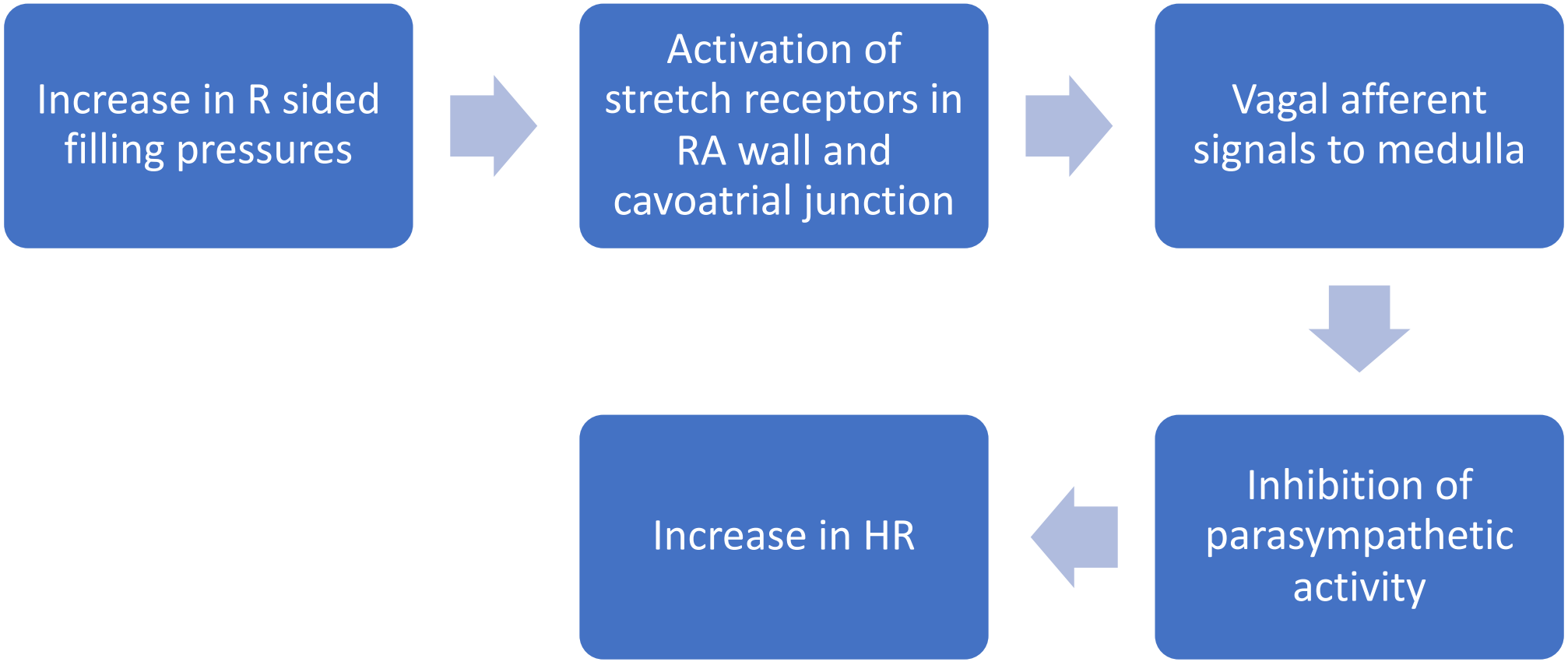
Baroreceptor Reflex (Carotid Sinus Reflex)



Chemoreceptor Reflex

- Both the carotid body and the aortic body have chemosensitive cells
- These cells are stimulated by PaO2 <50 and acidosis and send signals via the glossopharyngeal nerve to the medulla
- Medulla then stimulates the respiratory centers → increase respiratory drive (to increase PaO2 and resolve acidosis)
- Additionally, the parasympathetic nervous system is activated → reduced HR and contractility

Bainbridge Reflex



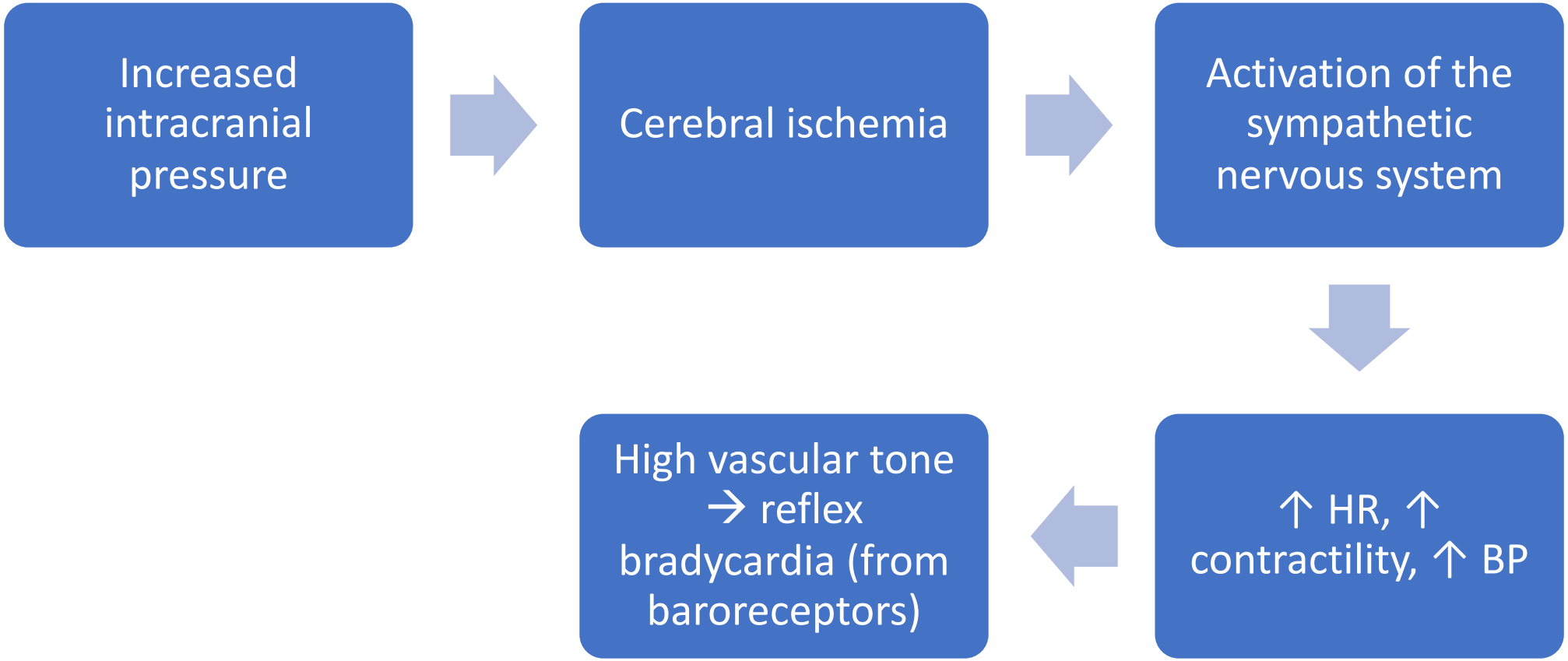
Bezold-Jarisch Reflex

- Chemoreceptors and mechanoreceptors in the 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



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

Questions?

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