Cardiodynamics

Components

Regulation: Intrinsic & Extrinsic mechanisms and feedback
Definitions

- **Cardiac output** = \( CO \) (mL/min): volume of blood pumped by heart each minute
- **Stroke volume** = \( SV \) (mL/beat): volume of blood pumped by heart with each ventricular contraction
- **Heart rate** = \( HR \) (beats/min)
- **Starlings law**: the degree to which the ventricular walls are stretched by returning blood determines the stroke volume (more in = more out)
Physical basis of Starling’s Law

- Myocardium is interwoven with **elastic protein fibers**
  - This connective tissue, & pericardial connective tissue, restrict expansion and encourage recoil of the heart.
Effects on cardiac output

- Autonomic innervation
- Hormones
- End-diastolic volume
- End-systolic volume

Heart rate and stroke volume affect cardiac output.
Regulation of cardiodynamics

• **Intrinsic**: within the heart (SV)
  – *force of contraction* related to degree of stretch of myocardium
    • Lots of stretch = increased force production

• **Extrinsic**: outside the heart (NS: Autonomic or Hormonal)
  – **Heart rate** influenced by both *sympathetic* and *parasympathetic* (autonomic) nervous system
  – **Stroke volume** influenced by blood pressure
Autonomic Control: Reflexes

- Cardioregulatory center in MO
  - Acceleratory: **Sympathetic** branch
  - Inhibitory: **Parasympathetic** branch
  - Some input from hypothalamus
Cardiac Reflexes

- **Feedback mechanisms**
  - Cranial nerves IX (Glossopharyngeal) & X (Vagus) bring *afferent sensory input* (from chemo- and baroreceptors)
    - Where are these receptors located?
    - What are they measuring?
  - *Cardioregulatory center* integrates information & responds appropriately
    - Increasing or decreasing stimulation of nodal system
Extrinsic: baro & chemoreceptors influence cardioregulatory center

**Carotid:** $O_2$ & BP
**Aorta:** BP
Cardioregulatory center.

- **Parasympathetic fibers** slow HR
- **Sympathetic fibers** speed HR
- **Sympathetic fibers** induce adrenal gland to release NE & E, which speed HR
Mechanisms of autonomic control

- **Parasympathetic** neurons release ACh, which opens K⁺ channels on myocardial walls.
  - Slows rate of depolarization. *Why?*

- **Sympathetic** neurons release Norepinephrine (NE), which opens Na²⁺ - Ca²⁺ channels on myocardial walls.
  - Increases rate of depolarization. *Why?*
Baroreceptor reflex: BP

The cardioregulatory center increases parasympathetic stimulation of the heart and decreases sympathetic stimulation of the heart and adrenal medulla.

- Decreased heart rate and stroke volume result from the changed ANS stimulation of the heart.
- Decreased heart rate and stroke volume result from the decreased release of epinephrine and norepinephrine from the adrenal medulla.

A sudden increase in blood pressure is detected by the baroreceptors in the internal carotid arteries and aorta, which affect the baroreceptor reflex.

The blood pressure decreases because of the decreased cardiac output resulting from the decreased heart rate and stroke volume.

Follow the green arrows when blood pressure increases.

Blood pressure (normal range)

Blood pressure homeostasis is maintained.
Hormones

• Epinephrine, Norepinephrine, Thyroid hormone
  – All *increase HR* by stimulating cells of the SA node
  – Bind to and open Na\(^{2+}\) - Ca\(^{2+}\) channels.
Stroke Volume
Factors affecting Stroke Volume

- **EDV**: End Diastolic Volume
- **Preload** = the degree of stretch experienced by ventricles during diastole.
  - As stretch increases, myofilament overlap increases.
  - Preload is proportional to EDV.
  - At rest, preload is low. During exercise, EDV & preload increase.
- **ESV**: End Systolic Volume
- **Afterload** = amount of tension that ventricles must produce to open semilunar valves.
  - Afterload is inversely proportional to ESV.
Factors affecting Stroke Volume

• **Contractility:** Amount of forced produced, at a given preload.
  
  – Autonomic control
    
    • Sympathetic - NE, E; stimulate muscle cell metabolism; stimulate Ca^{2+} entry
    
    • Parasympathetic - ACh; hyperpolarizes myocardium & inhibits stimulation
Factors affecting End Diastolic Volume

Venous Return + Preload + EDV
Filling time + Preload
Factors affecting **End Systolic Volume**

- **Sympathetic stimulation**
- **Thyroid Hormone, NE, E, glucagon**
- **Vasoconstriction**
- **Vasodilation**

**Contractility**

**Afterload**

**Preload**

**ESV**
Summary

- Atrial reflex
- Autonomic innervation
- Hormones
- Venous return
- Filling time
- Autonomic innervation
- Hormones
- Preload
- Contractility
- End-diastolic volume
- End-systolic volume
- Vasodilation or vasoconstriction
- Afterload
- Stroke volume

(a) Factors affecting heart rate
(b) Factors affecting stroke volume