Cardiovascular System Physiology of Circulation

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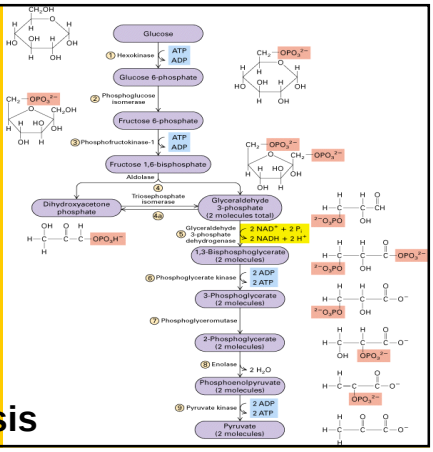
Metabolism

- Nurses think at organism level, doctors think at organ system level, shock is a cellular phenomena
- Metabolism is series of chemical reactions that maintain life
- Primary reaction glucose converted into adenosine triphosphate (ATP)
  - Glycolysis
  - Oxidative phosphorylation



Glycolysis

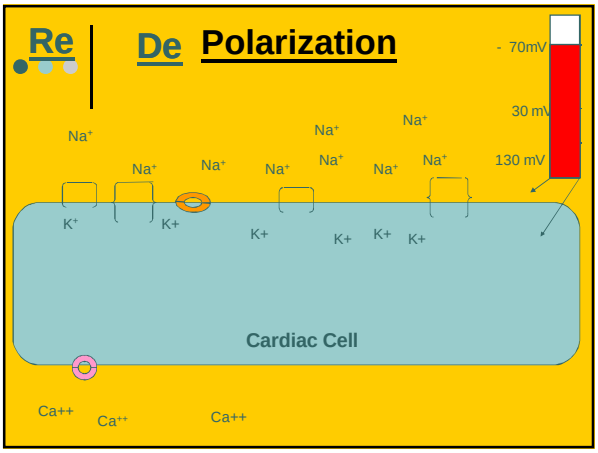
Glycoloysis

- First step of glucose metabolism
- Occurs in cytosol of cell
- Doesn't require oxygen
- Yields 2 molecules of ATP
- Waste products are pyruvic acid which changes into lactic acid

Oxidative Phosphorylation

- Occurs in mitochondria of cells
- Requires oxygen
- Total of 32 ATP molecules produced per glucose molecule
- Waste products are water and carbon dioxide

Re De Polarization



Cardiac Cycle

- Involves all events associated with one heart beat
  - Atrial systole/ ventricular diastole
  - Ventricular systole/ atrial diastole
- Contraction initiated by electrical stimulus

Diastole

- **Events:** Ventricular relaxation/ atrial contraction
- **Pressure:** Atrial greater than ventricular
- Valve: Semi-lunar closed; AV open
- **Blood volume:**
  - 70% of ventricular filling occurs before atria contracts
  - Atrial contraction accounts for remaining volume

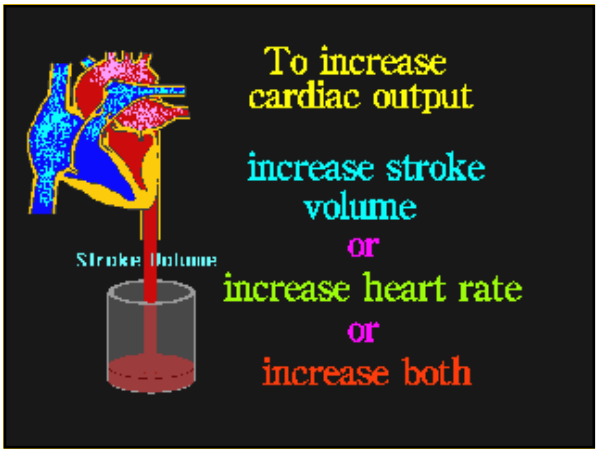
Systole

- Events: Atrial relaxation/ ventricular contraction
- **Pressure:** Ventricular pressure greater than atrial and arterial pressures
- **Valves:** AV valves snap closed/ semilunar valves open
- **Blood Volume:**
  - Approx. 70 -80 ml of blood pumped out (SV)
  - Small amount remains to prime the pump (ESV)

Hemodynamics of Blood Flow

- **Cardiac Output = Stroke Volume x Heart Rate**
  - Stroke volume: Amount of blood ejected from ventricles with each contraction
  - Heart rate: Number of contractions

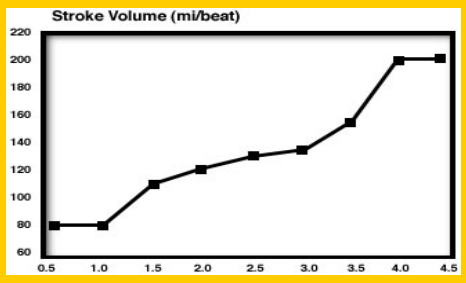
Ex: 70 ml/beat x 75 beats/ min = 5250 ml/min



Hemodynamics

- Total blood volume passes through heart every minute
- CO varies depending on need for oxygen by cells
- Cardiac reserve: Ratio of rest CO and max. CO
- Ejection Fraction: Percentage of EDV ejected with each contraction

Cardiac Reserve



Stroke Volume

- Definition: End diastolic volume - end systolic volume
  - Volume at start – volume at end = SV
- Changes in either EDV or ESV alters SV and CO

Ex: ESV = 120 ml
EDV = 50 ml

What is the stroke volume?

What is happening?

- Predict the effect each change will have on cardiac output.
  - **Increased EDV**
  - **Increased ESV**
  - **Decreased EDV**
  - **Decreased ESV**
- What factors affect EDV and ESV?

Three Factors

- Preload
  - Stretch in heart wall prior to contraction
- Afterload
  - Pressure against semilunar valves
- Inotropy (Contractility)
  - Strength of contraction

Preload

- The greater the preload of muscle fibers, the greater the strength of contraction
- Frank- Starling Law- Within physiologic limits, the greater the preload, the stronger the contraction will be
- Think myosin and actin overlap

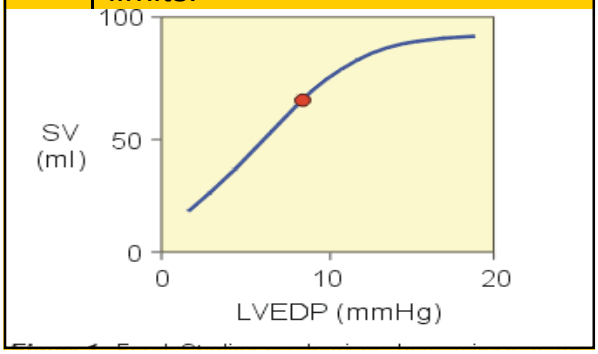
How do you stretch cardiac muscle?

- Venous Return

Increased venous return
- •Slower heart rate
- •Exercise
- •Fluid bolus
- •Positioning

Decreased return
- •Blood loss
- •Tachycardia

Remember physiologic limits!



Remember

This is normal physiology, ventricles in failure do not respond like a healthy ventricle!!

Afterload

- "Load" heart must pump against
- Increased afterload = increased end systolic volume = decreased stroke volume
- Normal hearts- afterload remains relatively constant
- Failing hearts- very sensitive to changes in afterload

How Do You Change Afterload?

Increased Afterload

- Hypertension
- Aortic Stenosis
- Valvular Defects
- Fluid Retention

Decreased Afterload

- Left ventricular hypertrophy
- Valvular Defects
- Vasodialators

Inotropy

- Strength of contraction of myocytes
- Affected by influx of calcium ions into cytoplasm of cell

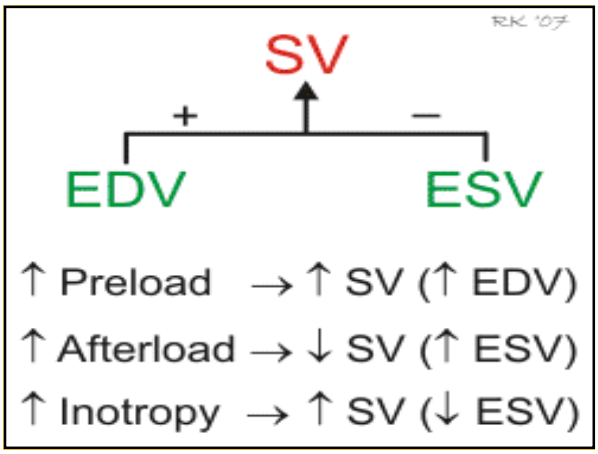
Inotropy

Increased

- Sympathetic agonism
- Parasympathetic antagonism
- Hormones (Thyroxine, epinephrine)
- Medications (Digitalis)
- Slightly increased heart rate

Decreased

- Parasympathetic agonism
- Sympathetic antagonism
- Hyperkalemia
- Acidosis
- Medications (Calcium channel blockers)



Considerations

- Changes in EDV, ESV, and SV are synergistic
- You cannot change one without affecting the others
- Must be carefully considered when treating patients with failing hearts

Remember

This is normal physiology, ventricles in failure do not respond like a healthy ventricle!!

Heart Rate

- Normal = 60 – 100 bpm
- Bradycardia = < 60 bpm
- Tachycardia = > 100 bpm

- Remember:

  CO = SV x HR

Regulation of Heart Rate

- Under Autonomic Nervous System Control
  - Can be sped up
  - Can be slowed down
  - Depends upon physiologic need for oxygen at the cellular level

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Sympathetic Control

- Increases Heart Rate by release of norepinephrine
- Norepinephrine binds to beta-1 adrenergic receptors of autorhythmic cells and opens Na^{+} and Ca^{++} channels
- Causes cells to reach threshold more quickly
- Decreases time at AV junction

Parasympathetic Control

- Decreases Heart Rate
- Vagus nerve releases AcH
- AcH interacts with muscarinic cholinergic receptors in myocyte membranes
- Opens potassium channels in membranes and results in hyperpolarization of cells
- Decreases SA node conduction and AV excitability

Hormonal Control

- Epinephrine- Released by adrennal medulla increases rate and contractility
- Thyroxine- increases rate

Ionic Effects

- Calcium
  - High = Increased rate
  - Low = Decreased rate
- Potassium
  - High = Decreased rate
- Sodium
  - High = Decreased rate

Circulation

- Under normal conditions, blood flow = cardiac output
- Blood flow= Volume of blood that flows through a vessel (ml/min)

Terms to Understand

- **Velocity-** Speed of fluid (mm/min)
  - Affected by size of vessels; inversely proportional to cross sectional area of vessels
  - Greatest in large vessels
  - Lowest in capillaries
- **WHY????**

Cross Sectional Area and Velocity

- Small changes in vessel radius creates large changes in flow
  - 3 mm vessel has 16 x more flow than 2 mm
- Vessels that are connected in parallel have less resistance than vessels in series

Terms to Understand

- **Hydrostatic Pressure-** Pressure exerted by a fluid against the walls of its container
- **Blood Pressure-** Hydrostatic pressure of blood in arterial system
- **Venous Pressure-** HP of blood in venous system

Terms to Understand

- **Vascular Resistance-** Resistance from friction of blood against endothelium
- **Peripheral Vascular Resistance-** Arterial resistance
  - Affected by vascular resistance and blood viscosity
- **Total Peripheral Vascular Resistance-** Resistance of total cardiovascular system

Normal Pressures Within System

- **Arterial Hydrostatic Pressure-** Varies from 100-35
- **Capillary Hydrostatic Pressure-**
  - Arterial side = 35
  - Venous side = 18
- **Venous Hydrostatic Pressure-** 18-0
- **Blood Flow Depends on Pressure Gradient**

Factors Affecting Resistance

- Major factor affecting resistance is arteriole diameter
- Vasoconstriction- Increases resistance and decreases blood flow
  - Causes
    - Sympathetic stimulation
    - Hormones (Epinephrine, ADH, angiotensin)

Factors Affecting Resistance

- Vasodialation- Decreased resistance and increased flow
  - Causes
    - Blocked sympathetic stimulation (no direct parasympathetic innervation of arteries)
    - Medications (Nitroglycerin, Lasix)

Factors Affecting Resistance

- Blood viscosity- Thickness of blood
  - Increased viscosity caused by polycythemia, or dehydration
  - Decreased viscosity caused by fluid overload

Factors Affecting Resistance

- Length of blood vessels
  - One extra pound of fat adds 200 miles of blood vessels

Management of Flow and Pressure

- Factors that control pressure and flow
  - Cardiac output
    - SV
    - Heart rate
  - Blood volume
  - Blood viscosity
  - Peripheral resistance
    - Constriction
    - Dialation

Management of Flow and Pressure

- Control of flow and pressure can be regulated centrally or locally
- Some mechanisms are fast acting, others are long term and are slow to respond

Intrinsic Control

- Autoregulation
  - Managed by stretch receptors in arterioles
- Metabolic
  - If demand for oxygen is greater than supply carbon dioxide is produced
  - Carbon dioxide caused arteriole vasodialation

Central Control

- Cardiovascular centers in medulla oblongata measure flow and pressure
  - Cardiac center
    - Acceleratory center
    - Inhibitory center
  - Vasomotor center

Central Control

- Cardiovascular centers interpret messages from receptors in carotid arteries and aorta
- Two types of receptors
  - Baroreceptors
    - Detect stretch
  - Chemoreceptors
    - Detect pH and oxygen levels

Pathways that control blood pressure and flow!