Section 3a Cardiac A & P

Significance of Monitoring

- Must have adequate perfusion to meet metabolic demands of the body
- Circulation does not guarantee perfusion

Cardiovascular Monitoring

- Non-invasive indicators of peripheral perfusion
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Cardiovascular Monitoring

- Invasive indicators of peripheral perfusion
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Goal of Cardiovascular Monitoring

Optimize cardiac output and arterial blood pressure to maintain adequate tissue perfusion

Cardiovascular A & P - Circuit

- Elastic, fluid-filled
- Heart - pump
- Arteries - distribute & regulate amount of oxygenated blood to organs & tissues
- Capillaries - exchange gases, nutrients, metabolites
- Veins - return system to heart & lungs; reservoir for blood volume
Cardiovascular A & P - Heart

Factors responsible for the forward movement of blood =

2 circulatory systems

Cardiovascular A & P - Blood Flow

Pulmonary Circulation

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Systemic Circulation

Cardiovascular A & P - Cardiac Cycle

- Atria - entrance chambers for ventricles; also contract to augment ventricular filling
- Ventricles - supply the power that creates pressure gradients

Cardiovascular A & P - Cardiac Cycle

- Diastole -
- Systole -
Cardiac Output

- Amount of blood ejected by ventricles in 1 minute
- Normal:
- Varies with age, sex, size, tissue $O_2$ demand, blood viscosity

Cardiac Output

\[ \dot{Q}_T = \text{HR} \times \text{SV} \]

- Normal HR:
- Normal stroke volume:

Cardiac Index

- To compare people of different age, size, etc.

\[ CI = \frac{\dot{Q}_T}{\text{BSA}} \]

- Normal CI:
Factors Affecting Cardiac Output

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Heart Rate

- Bradycardia
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- Tachycardia
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Stroke Volume

- Determined by
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Section 3a Cardiac A&P
Preload

- Filling volume of ventricle which stretches the relaxed ventricular wall before contraction (end-diastole)
- Demonstrated by

Starling’s Law states -

- The greater the ventricular end-diastolic filling volume (i.e. the greater the stretch on the myocardial muscle fibers, the greater the force of the contraction and stroke volume)
Preload - Ventricular Function Curves

- Disease can alter the shape of the normal function curves:

  - Decreased Compliance
  - Normal Compliance

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Preload

- Influenced by 3 factors:
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Preload - Blood Volume

- Blood volume
Preload - Distribution of Blood Volume

- **Body position**
  - Blood to dependant areas

- **Intrathoracic pressure**
  - If ↑
    - Pneumothorax, Valsalva, coughing spells, CMV

- **Venous tone**
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Preload - Atrial “Kick”

- **Lost if**
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Afterload

- **Refers to the resistance or impedance to right or left ventricular ejection of blood**
- **Imposed by resistance to blood flow through pulmonary &/or systemic circulation (i.e. ventricles have to work harder to move blood)**
Afterload (Left Ventricle)

- ↑ SVR → ↑ afterload
- ↑ myocardial work
- □ SV (if heart diseased) → ↑ myocardial O2 requirement

\[ SVR = \frac{\text{mean art BP} - \text{RVEDP}}{\text{QT}} \]

Normal = ≤ 20 mmHg/L/min

Dynes•sec/cm\(^5\) = mmHg/L/min X 80 (900-1600)

Causes of Increased SVR

- Vasoconstriction

- □

- □

- □

- □

- □
Causes of Increased SVR

- Congested venous system
- Increased blood viscosity
- Hypervolemia
- Aortic stenosis

Treatment for Increased SVR

- Vasodilators
- Surgery

Afterload (Right Ventricle)

\[
PVR = \frac{\text{mean PAP} - \text{PCWP}}{\text{Qt}}
\]

Normal = ≤ 2.5 mmHg/L/min
Dynes•sec/cm<sup>5</sup> = mmHg/L/min X 80 (≤ 160)
Causes of Increased PVR

- Pulmonary vasoconstriction
- Histamine release

Causes of Increased PVR

- Loss, dysfunction or destruction of pulmonary vasculature
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Causes of Increased PVR

- Increased pulmonary blood flow
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Section 3a Cardiac A&P

Causes of Increased PVR

- Increased pulmonary venous pressure
- Increased blood viscosity

Contractility

- Contractility - strength and speed of the ventricular contraction

Contractility

- Normal
- Moderate dysfunction
- Severe dysfunction

OUTFLOW RESISTANCE

STROKE VOLUME
Causes of Decreased Contractility

- Loss of contracting muscle mass
- Loss of muscle size

Causes of Decreased Contractility

- Depressants
  - Procaine, Lidocaine, quinidine, Pronestyl (procainamide)
  - Barbiturates
  - Beta-blockers (Inderol)
  - Acidosis, hypoxia, hypercarbia

Treatment for Decreased Contractility

- Positive inotropic agents:
  - digitalis, (Digoxin, Lanoxin)
  - epinephrine
  - Dopamine
  - Dobutamine
  - Isuprel
  - caffeine (Cafergot)
  - calcium