Cardiovascular Physiology

Part 2
Cardiac Output & Control Systems

Lecture Outline

• Review Integrated Cardiac Page
• Cardiac Output & Controls
• Blood Flow & Blood Pressure Controls
• Medullary Center for Cardiovascular Control & the Baroreceptor Reflex
Cardiovascular Physiology
Cardiac Output

- Cardiac Output (CO) is the volume pumped by the left ventricle each minute
  - influenced by
    - Stroke Volume (SV)
      - EDV – ESV = SV
      - 135ml – 65ml = 70ml
    - Heart Rate (HR) bpm
      - 80 bpm
  - CO = SV x HR
    - 70ml/bpm x 72bpm = 5040 ml/min
      - 5.04L/min
- How is this controlled to account for changing conditions? (exercise, disease, stress…)
  - What influences SV?
  - What influences HR?

Cardiovascular Physiology
Cardiac Output

- Influencing stroke volume
  - Pre Load
    - operates under Frank-Starling Law of the Heart
    - What then influences the stretch applied to cardiac muscle tissue prior to contraction?
      - Venous return, driven by
        - Skeletal muscle pump
        - Respiratory pump
        - Atrial Suction
  - Contractility
    - Stronger contraction = larger stroke volume
    - Due to inotropic agents
      - Epinephrine, Norepinephrine, Digitalis* are (+) inotropic agents
      - ACh is a (-) inotropic agent
      - How do they work?

*digitalis – a cardiac glycoside (drug) that lowers Na⁺/K⁺ ATPase activity and therefore the NCX transporter activity, resulting in elevated ICF Ca²⁺ which creates a stronger graded contraction.
Cardiovascular Physiology

Cardiac Output

- **Inotropic Agents**

  - Influencing stroke volume
    - **Afterload**
      - This is the amount of pressure that is sitting on the semilunar valves that must be overcome before ventricular ejection can occur.
      - The more pressure that must be built up during isovolumetric ventricular contraction reduces the time that ejection can occur:
        - Reduces the ejection fraction (SV/EDV)
          - Normal 70ml/135ml = 52%
          - Elevated aortic pressure causes the reduction from normal
          - 60ml/135ml = 44%
        - **indirect relationship**
          - Higher aortic pressure = lower stroke volume
        - **Causes?**
          - Elevated blood pressure
          - Loss of compliance in aorta (loss of elasticity)
  
- **Influencing Heart Rate**
  - Rate is set by pacemaker cells rate of depolarization
    - **Chronotropic effects may be excitatory**
      - Sympathetic activity
    - **Or inhibitory**
      - Parasympathetic activity
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Cardiac Physiology

**Blood Flow & Blood Pressure Controls**

- CO tells us how much blood is ejected per minute and is influence by both intrinsic & extrinsic factors
- Extrinsic factors (besides ANS) include
  - blood vessels & blood pressure
  - blood volume & viscosity
  - capillary exchange & the lymphatic return
  - cardiovascular disease

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Cardiac Physiology

Blood Flow & Blood Pressure Controls

- Blood Vessels Function to
  - Provide route (arteries – away, veins – visit)
  - Allow for exchange (capillaries)
  - Control & regulate blood pressure

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Cardiac Physiology

Blood Flow & Blood Pressure Controls

[Diagram of blood circulation and cardiovascular system]
Cardiac Physiology
Blood Flow & Blood Pressure Controls

- Blood Vessel Structure enables specific functions
  - Aorta
    - absorb pulse pressure (systolic pressure – diastolic pressure) and release energy creating diastolic pulse
  - Large arteries
    - conduct and distribute blood to regional areas
  - Arterioles
    - Regulate flow to tissues and regulate MAP (mean arterial pressure)

### Blood Vessels & Blood Pressure

- **Systolic Pressure**
  - The pressure that is created when the ventricles contract
  - Usually around 120 mm Hg

- **Diastolic Pressure**
  - The pressure that is created by the recoil of the aorta AND the closure of the aortic semilunar valve
  - Usually around 80 mm Hg
Cardiac Physiology
Blood Flow & Blood Pressure Controls

• Blood Vessels & Blood Pressure
  – Pulse Pressure
    • The difference between the systolic and diastolic pressures
      – Usually 40 mm Hg (120 mm Hg – 80 mm Hg)
    • Only applies to arteries
  – Why do we care about systolic, diastolic and pulse pressures?
    • We can determine the average pressure within the arterial system = Mean Arterial Pressure (MAP)
      MAP = diastolic Pressure + 1/3 Pulse Pressure
      MAP = 80 mm Hg + 1/3( 120 mm Hg – 80 mm Hg)
      MAP = 93 mm Hg
    • Then we can determine general health of the cardiovascular system

Cardiac Physiology
Blood Flow & Blood Pressure Controls

• MAP is proportionate to the cardiac output and the amount of peripheral resistance
  – The opposition to blood flow in the arterioles
    • Resistance is directly proportional to the length (L) of the vessel, and the viscosity($\eta$) (thickness) of the blood and inversely proportional (to the 4th power) of the vessel radius, so….
    \[ R \propto L \frac{\eta}{r^4} \]
    However as the L and $\eta$ should remain relatively constant, we can determine that peripheral resistance is mainly a factor of the vessel diameter
    \[ R \propto \frac{1}{r^4} \]

Cardiac Physiology
Blood Flow & Blood Pressure Controls

• MAP is proportionate to the cardiac output and the amount of peripheral resistance
  – If CO increases but resistance to the outflow does not change
    • Then more blood is flowing into the system than out and arterial pressure must go up to allow inflows to equal outflows

Cardiac Physiology
Blood Flow & Blood Pressure Controls

• So… if resistance is affected by the radius, and flow is inversely proportionate to the resistance
  – What effect will vasoconstriction / vasodilation have on blood pressure and blood flow? And what controls it?
  – What will obesity do to blood pressure and blood flow & why?
Cardiac Physiology
Blood Flow & Blood Pressure Controls

• The controls of vessel diameter are both local and systemic
  – Enables tissues to control their own blood flow
  – Local controlling mechanisms include
    • Myogenic response by smooth muscle of arterioles
      – Increased stretch due to increasing blood pressure causes vessel constriction due to mechanically gated Ca\(^{2+}\) channel activation
    • Paracrines – local substances which alter smooth muscle activity
      – Serotonin
        » Secreted by activated platelets
      – Endothelin
        » Secreted by vascular endothelium
      – NO secreted by vascular endothelium
      – Bradykinin – from various sources
      – Histamine – from mast cells in connective tissues
      – Adenosine secreted by cells in low O\(_2\) (hypoxic) conditions
      – ↓O\(_2\), ↑CO\(_2\), ↑K\(^+\), ↑H\(^+\), ↑temp
  – Systemic controlling mechanisms for vasoconstriction include
    • NE – sympathetic postganglionic neurons
    • Serotonin – neurons
    • Vasopressin (ADH) – posterior pituitary
    • Angiotensin II – part of renin-angiotensin pathway
  – Systemic controls for vasodilation include
    • Beta-2 epinephrine – from adrenal medulla
    • ACH – parasympathetic postganglionic neurons
    • ANP (atrial natriuretic peptide) – from atrial myocardium and brain
    • VIPs (vasoactive intestinal peptides) – from neurons

Cardiac Physiology
Blood Flow & Blood Pressure Controls

• Hyperemia is locally mediated increases in blood flow, may be
  – Active or Reactive

Cardiac Physiology
Blood Flow & Blood Pressure Controls

Effect of Sympathetic Stimulation on Blood Vessels
Cardiovascular Physiology
Review of Factors Influencing Blood Flow

1. sympathetic & parasympathetic innervation
2. sympathetic innervation and epinephrine
3. myogenic response
4. paracrine

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Cardiac Physiology
Neural Regulation of Blood Pressure
- CNS contains the Medullary Cardiovascular Control Center
  - Receives inputs from carotid and aortic baroreceptors
  - Creates outflow to sympathetic and parasympathetic pathways
    - Sympathetic to SA & AV nodes and myocardium as well as to arterioles and veins
    - Parasympathetic to the SA Node
  - Baroreceptors initiate the baroreceptor reflex
Next Time

• Capillary Exchange
• Blood