Cardiovascular Physiology

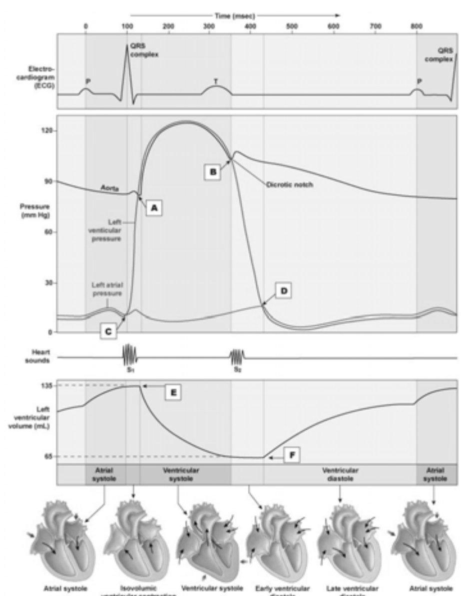
Part 2

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 Integrated Review



Heart Animation

Lecture Outline

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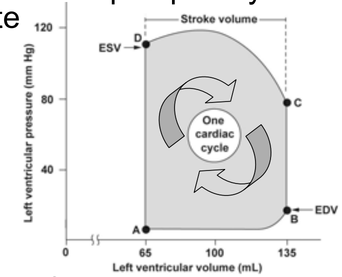
Cardiovascular Physiology

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 bmp
  - CO = SV x HR
    70ml/b 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?

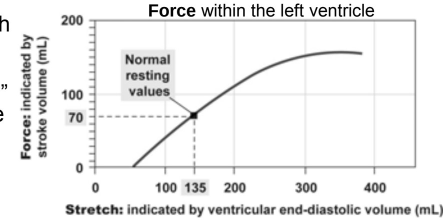


Cardiovascular Physiology

Cardiac Output

- Influencing stroke volume
  - Pre Load
    - The amount of stretch within the contractile myocardial fibers
    - Represents the "load" placed on the muscle fibers before they contract
    - They respond according to length-tension patterns observed in muscle tissue by Frank, then by Starling
      - Became known as the Frank-Starling Law of the Heart
      - "The heart will pump all the blood that is returned to it"

Relationship between **Stretch** and **Force** within the left ventricle



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

Cardiovascular Physiology

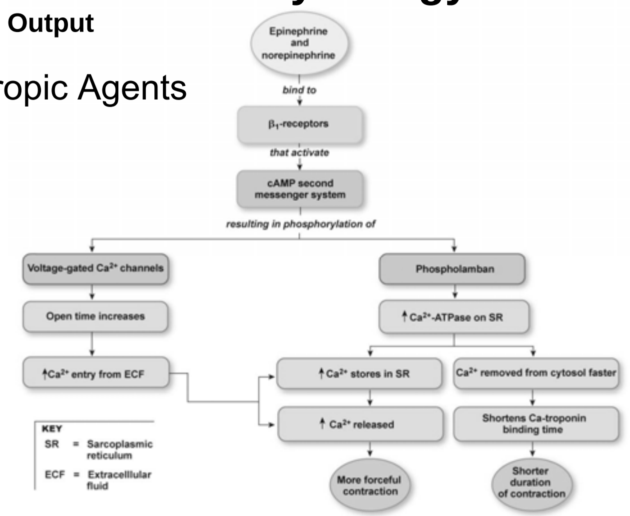
Cardiac Output

- Influencing stroke volume
  - 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 Ca^{2+} which creates a stronger graded contraction.

Cardiovascular Physiology
Cardiac Output

- Inotropic Agents



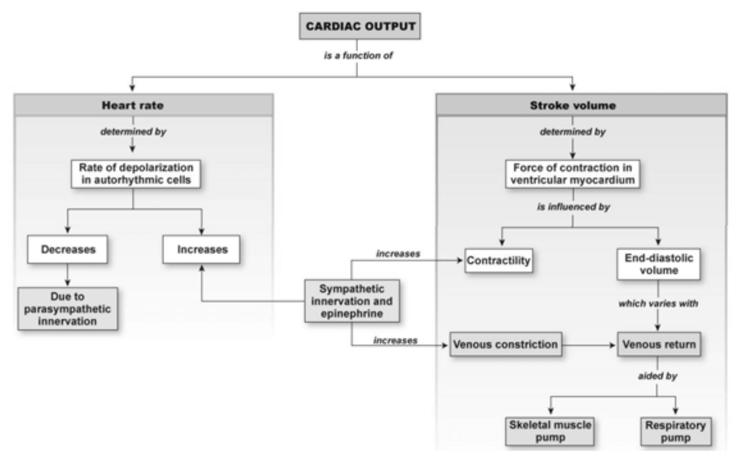
Cardiovascular Physiology
Cardiac Output

- 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)

Cardiovascular Physiology
Cardiac Output

- Influencing Heart Rate
  - Rate is set by pacemaker cells rate of depolarization
    - Chronotropic effects may be excitatory
      - Sympathetic activity
    - Or inhibitory
      - Parasympathetic activity

Cardiovascular Physiology
Cardiac Output Overview of Influences



Lecture Outline

- Review Integrated Cardiac Page
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- **Blood Flow & Blood Pressure Controls**
- Medullary Center for Cardiovascular Control & the Baroreceptor Reflex

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

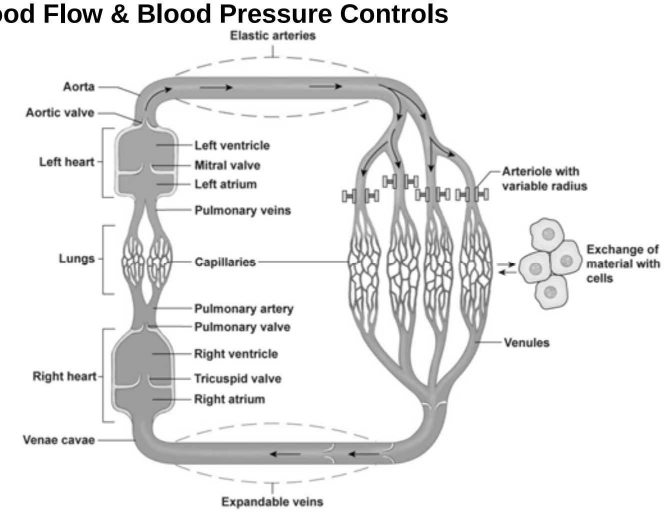
Cardiac Physiology

Blood Flow & Blood Pressure Controls

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

Cardiac Physiology

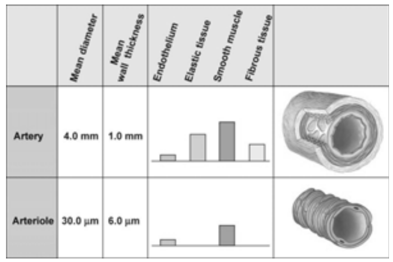
Blood Flow & Blood Pressure Controls



Cardiac Physiology

Blood Flow & Blood Pressure Controls

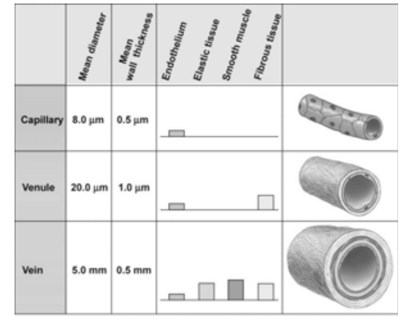
- Blood Vessel Structure enables specific functions
  - Aorta
    - absorb pulse pressure (systolic pressure – diastolic pressure) and release energy creating diastolic pulse
  - Large arteries
    - conduct and distribute blood to regional areas
  - Arterioles
    - Regulate flow to tissues and regulate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



Cardiac Physiology

Blood Flow & Blood Pressure Controls

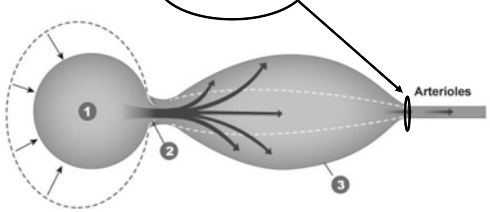
- Capillaries
  - Allow for exchange
- Venules
  - Collect and direct blood to the veins
- Veins
  - Return blood to heart and act as a blood reservoir



Cardiac Physiology

Blood Flow & Blood Pressure Controls

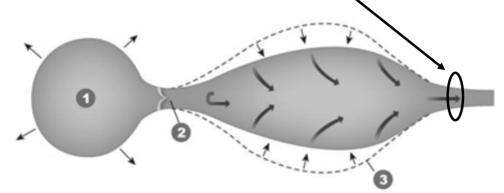
- Blood Vessels & Blood Pressure
  - Systolic Pressure
    - The pressure that is created when the ventricles contract
    - Usually around 120 mm Hg



Cardiac Physiology

Blood Flow & Blood Pressure Controls

- Blood Vessels & Blood Pressure
  - 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)

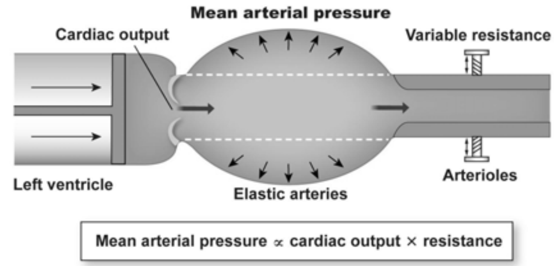
      MAP = diastolic Pressure + 1/3 Pulse Pressure

      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
  - 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

- 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(η) (thickness) of the blood and inversely proportional (to the 4th power) of the vessel radius, so….

$$R \propto L\,\eta/r^4$$

However as the L and η should remain relatively constant, we can determine that peripheral resistance is mainly a factor of the vessel diameter

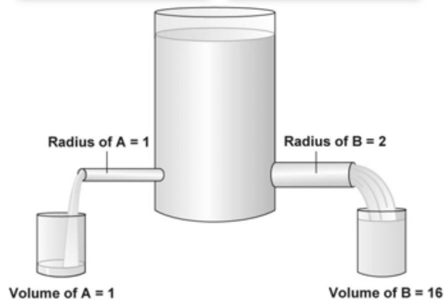
$$R \propto 1/r^4$$

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?

Resistance $\propto \frac{1}{radius^4}$	
Tube A	Tube B
$R \propto \frac{1}{1^4}$	$R \propto \frac{1}{2^4}$
$R \propto 1$	$R \propto \frac{1}{16}$

Flow $\propto \frac{1}{resistance}$	
Tube A	Tube B
Flow $\propto \frac{1}{1}$	Flow $\propto \frac{1}{\frac{1}{16}}$
Flow $\propto 1$	Flow $\propto 16$



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

vasoconstrictors:

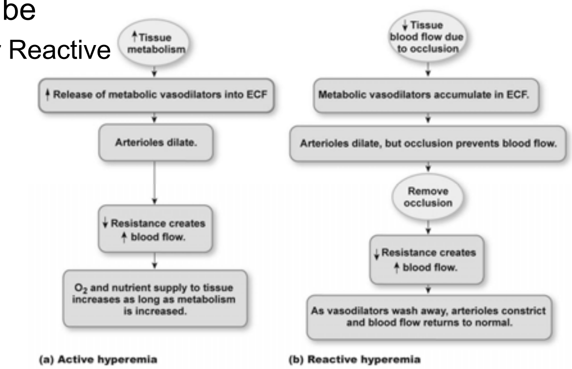
- Serotonin
  - » Secreted by activated platelets
- Endothelin
  - » secreted by vascular endothelium

vasodilators:

- 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
- $\downarrow O_2$, $\uparrow CO_2$, $\uparrow K^+$, $\uparrow H^+$, $\uparrow$temp

Cardiac Physiology
Blood Flow & Blood Pressure Controls

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

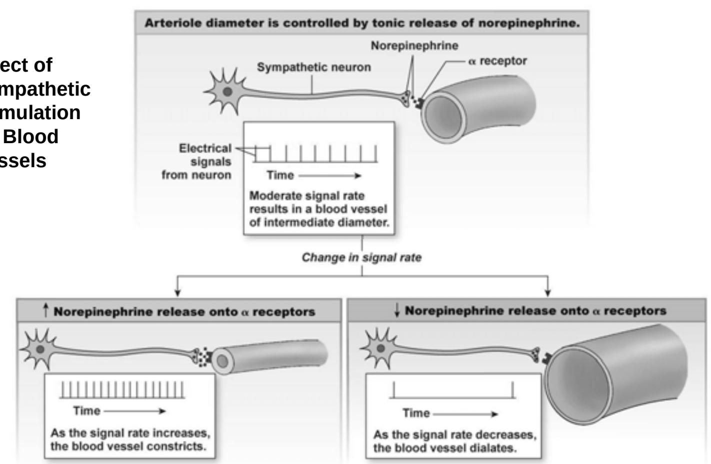


Cardiac Physiology
Blood Flow & Blood Pressure Controls

- The controls of vessel diameter are both local (intrinsic) and systemic (extrinsic)
  - Systemic controlling mechanisms for vasoconstriction include
    - NE – sympathetic postganglionic neurons
    - Serotonin – neurons
    - Vasopressin (ADH) – posterior pituitary
    - Angiotensin II – part of renin-antiogensin pathway
  - Systemic controls for vasodilation include
    - Beta-2 epinephrine – from adrenal medulla
    - ACH – parasympathetic postganglionic neurons
    - ANP (atrial natriuretic peptide) – from atrial myocaridum and brain
    - VIPs (vasoactive intestinal peptides) – from neurons

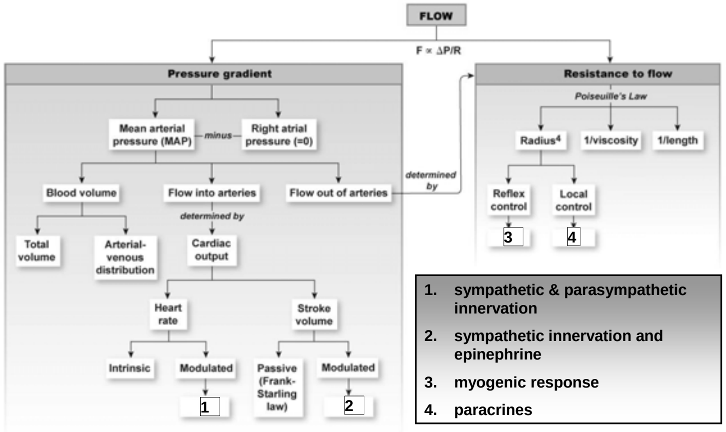
Cardiac Physiology
Blood Flow & Blood Pressure Controls

Effect of Sympathetic Stimulation on Blood Vessels



Cardiovascular Physiology

Review of Factors Influencing Blood Flow



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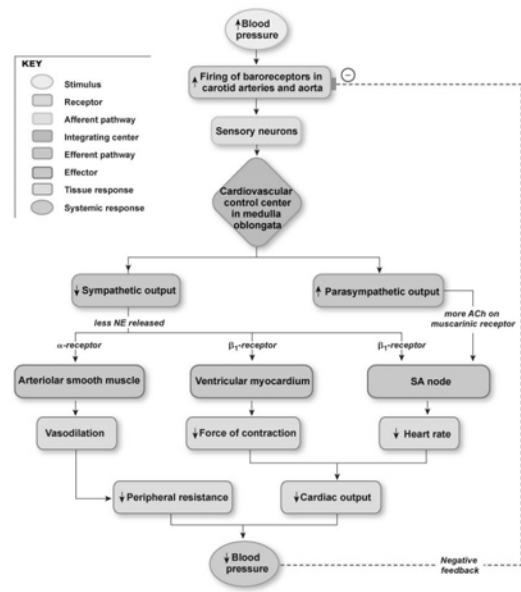
Cardiac Physiology

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**

The Baroreceptor Reflex Pathways



Next Time

- Capillary Exchange
- Blood