Chapter 14 Outline

- Cardiac Output
- Blood Volumes
- Vascular Resistance to Blood Flow
- Blood Flow to the Heart and Skeletal Muscles
- Blood Flow to the Brain and Skin
- Blood Pressure
- Hypertension, Shock, and Congestive Heart Failure
Is volume of blood pumped/min by each ventricle

Stroke volume (SV) = blood pumped/beat by each ventricle

Heart rate (HR) = the number of beats/minute

Cardiac Output (CO)

\[ \text{CO} = \text{SV} \times \text{HR} \]

Regulation of Cardiac Rate

Without neuronal influences, SA node will drive heart at rate of its spontaneous activity

Normally Sympathetic and Parasympathetic activity influence HR (chronotropic effect)

Autonomic innervation of SA node is main controller of HR

Sympathetic and Parasympathetic nerve fibers modify rate of spontaneous depolarization

Norepinephrine and epinephrine stimulate opening of pacemaker HCN channels

- This depolarizes SA node faster, increasing HR

ACh promotes opening of K+ channels

- The resultant K+ outflow counters Na+ influx, slowing depolarization and decreasing HR
Regulation of Cardiac Rate

- Cardiac control center of medulla coordinates activity of autonomic innervation
- Sympathetic endings in atria and ventricles can stimulate increased strength of contraction

Table 14.1 Effects of Autonomic Nerve Activity on the Heart

<table>
<thead>
<tr>
<th>Region Affected</th>
<th>Sympathetic Nerve Effects</th>
<th>Parasympathetic Nerve Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>SA node</td>
<td>Increased rate of diastolic depolarization; increased cardiac rate</td>
<td>Decreased rate of diastolic depolarization; decreased cardiac rate</td>
</tr>
<tr>
<td>AV node</td>
<td>Increased conduction rate of contraction</td>
<td>Decreased conduction rate</td>
</tr>
<tr>
<td>Atrial muscle</td>
<td>Increased strength of contraction</td>
<td>No significant effect</td>
</tr>
<tr>
<td>Ventricular muscle</td>
<td>Increased strength of contraction</td>
<td>No significant effect</td>
</tr>
</tbody>
</table>
Stroke Volume

- Determined by 3 variables:
  - **End diastolic volume (EDV)** = volume of blood in ventricles at end of diastole
  - **Total peripheral resistance (TPR)** = resistance to blood flow in arteries
  - **Contractility** = strength of ventricular contraction

Regulation of Stroke Volume

- **EDV** is workload (preload) on heart prior to contraction
  - SV is directly proportional to preload and contractility
- Total peripheral resistance = **afterload** which impedes ejection from ventricle
- **Ejection fraction** is SV/EDV
  - Normally is 60% ; useful clinical diagnostic tool
- Strength of contraction varies directly with EDV

Frank-Starling Law of the Heart

- States that strength of ventricular contraction varies directly with EDV
  - Is an intrinsic property of myocardium
  - As EDV increases, myocardium is stretched more, causing greater contraction and SV
Frank-Starling Law of the Heart

- (a) is state of myocardial sarcomeres just before filling
- Actins overlap, actin-myosin interactions are reduced and contraction would be weak
- In (b, c and d) there is increasing interaction of actin and myosin allowing more force to be developed

Extrinsic Control of Contractility

- At any given EDV, contraction depends upon level of sympathoadrenal activity
- Norepi. and Epi. produce an increase in HR and contraction (positive inotropic effect)
- Due to increased Ca²⁺ in sarcomeres

\[ \text{Cardiac output} = \text{Cardiac rate} \times \text{Stroke volume} \]
Venous Return

- Is return of blood to heart via veins
- Controls EDV and thus SV and CO
- Dependent on:
  - Blood volume and venous pressure
  - Vasoconstriction caused by Symp NS
  - Skeletal muscle pumps
  - Pressure drop during inhalation

Venous Return

- Veins hold most of blood in body (~70%) and are thus called capacitance vessels
  - Have thin walls and stretch easily to accommodate more blood without increased pressure (higher compliance)
  - Have only 0-10 mm Hg pressure

Blood Volume
Blood Volume

- Constitutes small fraction of total body fluid
- 2/3 of body H2O is inside cells (intracellular compartment)
- 1/3 total body H2O is in extracellular compartment
  - 80% of this is interstitial fluid; 20% is blood plasma

Exchange of Fluid between Capillaries and Tissues

- Distribution of ECF between blood and interstitial compartments is in state of dynamic equilibrium
- Movement out of capillaries is driven by hydrostatic pressure exerted against capillary wall
  - Promotes formation of tissue fluid
  - Net filtration pressure = hydrostatic pressure in capillary (17-37 mm Hg) - hydrostatic pressure of ECF (1 mm Hg)

Exchange of Fluid between Capillaries and Tissues

- Movement also affected by colloid osmotic pressure
  - = osmotic pressure exerted by proteins in fluid
  - Difference between osmotic pressures in and outside of capillaries (oncotic pressure) affects fluid movement
    - Plasma osmotic pressure = 25 mm Hg; interstitial osmotic pressure = 0 mm Hg
Overall Fluid Movement

- Is determined by net filtration pressure and forces opposing it (Starling forces)
  
  \[(P_c + \pi_i) - (P_i + \pi_p)\]  
  
  [fluid out] – [fluid in]

- \(P_c\) = Hydrostatic pressure in capillary
- \(\pi_i\) = Colloid osmotic pressure of interstitial fluid
- \(P_i\) = Hydrostatic pressure in interstitial fluid
- \(\pi_p\) = Colloid osmotic pressure of blood plasma

Forces Acting across Capillary Walls
Edema

- Normally filtration, osmotic reuptake, and lymphatic drainage maintain proper ECF levels
- Edema is excessive accumulation of fluid resulting from:
  - High arterial blood pressure
  - Venous obstruction
  - Leakage of plasma proteins into interstitial fluid

Regulation of Blood Volume by Kidney

- Urine formation begins with filtration of plasma in glomerulus
- Filtrate passes through and is modified by nephron
- Volume of urine excreted can be varied by changes in reabsorption of filtrate
  - Adjusted according to needs of body by action of hormones

ADH (vasopressin)

- ADH released by Post Pit when osmoreceptors in hypothalamus detect high osmolality
  - From excess salt intake or dehydration
  - Causes thirst
  - Stimulates H₂O reabsorption from urine
- Homeostasis maintained by these countermeasures
**Aldosterone**

- Is steroid hormone secreted by adrenal cortex
- Helps maintain blood volume and pressure through reabsorption and retention of salt and water
- Release stimulated by:
  - salt deprivation
  - low blood volume
  - low blood pressure

**Renin-Angiotension-Aldosterone System**

- When there is a salt deficit, low blood volume, or pressure, angiotensin II is produced
- Angio II causes a number of effects all aimed at increasing blood pressure:
  - Vasoconstriction, aldosterone secretion, thirst
Atrial Natriuretic Peptide (ANP)

- Expanded blood volume is detected by stretch receptors in left atrium and causes release of ANP
- ANP inhibits aldosterone, promoting salt and water excretion to lower blood volume
- And promotes vasodilation

Atrial Natriuretic Peptide (ANP)

- ANP, together with decreased ADH, acts in a negative feedback system to lower blood volume and venous return

Blood Pressure
Blood Pressure (BP)

- Arterioles play role in blood distribution and control of BP
- Blood flow to capillaries and BP is controlled by diameter of arterioles
- Capillary BP is decreased because they are downstream of high resistance arterioles

Blood Pressure (BP)

- Capillary BP is also low because of large total cross-sectional area

Blood Pressure (BP)

- Is controlled mainly by HR, SV, and peripheral resistance
  - An increase in any of these can result in increased BP
  - Sympathoadrenal activity raises BP via arteriole vasoconstriction and by increased CO
  - Kidney plays role in BP by regulating blood volume and thus stroke volume
Baroreceptor Reflex

- Is activated by changes in BP
  - Which is detected by baroreceptors (stretch receptors) located in aortic arch and carotid sinuses
  - Increase in BP causes walls of these regions to stretch, increasing frequency of Act. Pot.
  - Baroreceptors send Act. Pot. to vasomotor and cardiac control centers in medulla
- Is most sensitive to decrease and sudden changes in BP
Atrial Stretch Receptors

- Are activated by increased venous return and act to reduce BP and in response:
  - Stimulate reflex tachycardia (slow HR)
  - Inhibit ADH release and promote secretion of ANP

Measurement of Blood Pressure

- Via auscultation (to examine by listening)
- No sound is heard during laminar flow (normal, quiet, smooth blood flow)
- Korotkoff sounds can be heard when sphygmomanometer cuff pressure is greater than diastolic but lower than systolic pressure
  - Cuff constricts artery creating turbulent flow and noise as blood passes constriction during systole and is blocked during diastole
  - 1st Korotkoff sound is heard at pressure that blood is 1st able to pass thru cuff; last occurs when one can no long hear systole because cuff pressure = diastolic pressure

Measurement of Blood Pressure continued

- Blood pressure cuff is inflated above systolic pressure, occluding artery
- As cuff pressure is lowered, blood flows only when systolic pressure is above cuff pressure, producing Korotkoff sounds
- Sounds are heard until cuff pressure equals diastolic pressure, causing sounds to disappear
The indirect, or auscultatory, method of blood pressure measurement:

1. **Pulse Pressure**
   - **Pulse pressure** = (systolic pressure) – (diastolic pressure)
   - **Mean arterial pressure (MAP)** represents average arterial pressure during cardiac cycle
     - Has to be approximated because period of diastole is longer than period of systole
     - \[ MAP = \text{diastolic pressure} + \frac{1}{3} \text{pulse pressure} \]