Chapter 14 – Cardiovascular Physiology

A. Overview of cardiovascular system (Fig. 14-1)
   a. Heart is pump, vessels carry blood, and blood is the medium
   b. Circuits
      i. Pulmonary – takes blood between the lungs and the heart. Lungs oxygenate the blood.
      ii. Systemic – takes blood between tissues and the heart. Tissues take up oxygen from blood.
      iii. Arteries take blood away from heart, veins take blood to the heart.

B. Fluid Dynamics
   a. Pressure
      i. Pressure gradients – fluid flows from area of high pressure to low pressure (Fig. 14-2, 14-4). Pressure drops with distance (Fig. 14-3b)
      ii. Hydrostatic pressure – pressure from fluid on surroundings when not moving (Fig. 14-3a).
      iii. Flow – volume of fluid that passes a given point
   b. Resistance – forces that oppose flow
      i. Radius of tube – smaller creates more resistance (Fig. 14-5). Therefore, vasoconstriction reduces blood flow while vasodilation increases it.
      ii. Length of tube – greater creates more resistance
      iii. Viscosity of fluid – greater creates more resistance
   c. Velocity – speed (distance traveled) of fluid.
      i. Given a constant flow rate, velocity increases with smaller vessel (Fig. 14-6)
   d. Mean arterial pressure – cardiac output (flow rate of heart) X peripheral resistance (resistance at blood vessels).

C. Cardiac Muscle and the Heart
   a. Heart structure (Fig. 14-7)
      i. Pericardium surrounds heart and allows movement. Cardiac muscle is in heart wall.
      ii. Chambers
         1. Upper chambers are left and right atria
         2. Lower chambers are left and right ventricles
         3. Blood enters each atria and exit ventricle on same side
      iii. Valves (Fig. 14-9)
         1. Tricuspid and bicuspid valves (AV valves) separate the right and left atria and ventricles, respectively.
         2. Pulmonary and aortic (semilunar valves) valve separate the right and left ventricles and arteries.
      iv. Vessels
         1. Systemic – venae cavae bring blood from body to right atrium. Aorta takes blood from left ventricle to body.
         2. Pulmonary – pulmonary vein brings blood from lungs to left atrium. Pulmonary artery takes blood from right ventricle to lungs.
      v. Coronary arteries supply blood to heart and cardiac veins take away blood. Heart attacks occur when these are blocked
   b. Cardiac Muscle
      i. Contractile cells
1. Similar structure to skeletal muscle but not innervated and have intercalated disks for stronger connection and communication through gap junctions (Fig. 14-10)
2. EC coupling similar to skeletal except calcium moves from T-tubules through L-type calcium channels to activate RyR on SR (Fig. 14-11)
3. Action potential
   a. Individual contractile cells are spreading the potential, not nerves
   b. Influx of calcium lengthens the potential and changes the shape (Fig. 14-13)
   c. Prevention of tetanus
      i. Important to allow relaxation and filling of ventricles
      ii. Long refractory period prevents summation (Fig. 14-14)
ii. Autorhythmic cells
   1. These cells spontaneously produce action potentials at regular intervals.
   2. Do not contain a resting potential, but a pacemaker potential that steadily increases.
   3. Action potential (Fig. 14-15)
      a. $I_f$ channels open letting Na and K slowly come in. Some Ca channels open.
      b. Once threshold is hit, more Ca channels open.
      c. At peak of depolarization, Ca channels close and K channels open to let out K.
4. Speed is modulated by neurotransmitters (Fig. 14-16)
   a. Sympathetic neurotransmitters NE and epinephrine increase ion flow through $I_f$ channels. This quickens pace.
   b. Parasympathetic neurotransmitter ACh decreases ion flow through Ca channels. They also increase K flow so that cells begin hyperpolarized. Together, this causes lengthening pacemaker potential
D. Cardiac Function
   a. Conduction
      i. Pathways and pacemakers (Fig. 14-18)
         1. SA node $\rightarrow$ internodal pathways $\rightarrow$ AV node $\rightarrow$ AV bundle $\rightarrow$ AV branches $\rightarrow$ Purkinje fibers
         2. The sinoatrial (SA) node is the main pacemaker that sends signals to the AV node. SA node also triggers contractile cells of the atria. Fires at 70 bpm resting.
         3. The atrioventricular (AV) node sends signals down to Purkinje fibers. The AV node also helps slow the speed of signal so that atria contracts before ventricles. Is also a pacemaker at 50 bpm.
         4. Purkinje fibers activate ventricular contractile cells in an upward direction. Also a pacemaker at 35 bpm.
      ii. Electrocardiogram (Fig. 14-20, 14-21)
         1. 3 stages of a beat
            a. P wave – depolarization of atria
            b. QRS complex – ventricular depolarization
            c. T wave – ventricular repolarization
         2. ECG is the sum of electrical activity transmitted to surface of body. Action potential is a single cell’s activity (Fig. 14-22).
         3. Fibrillation may be repaired by an electric shock, shutting down the SA node. Other abnormalities (Fig. 14-23)
b. Cardiac Cycle
   i. Systole – contraction, diastole – relaxation.
   ii. When atria contract, ventricles relax and vice-versa.
   iii. Valves open and close to allow blood to move and to prevent backflow. “Lub-dup” sound is AV and semilunar valves closing, respectively.
   iv. 5 stages (Fig. 14-24)
      1. Late diastole – all chambers relaxed. Ventricles fill passively
      2. Atrial systole – atria contract and push blood into ventricles
      3. Isovolumic ventricular contraction – early phase of contraction. AV valves close.
      4. Ventricular ejection – late phase of contraction. Semilunar valves open and blood is pushed into arteries.
      5. Isovolumic ventricular relaxation – blood fills atria, semilunar valves close. When AV valves open, this is start of stage 1.
   v. Cardiac performance
      1. Stroke volume = volume pumped out during one contraction. This is the difference between volume at end of diastole and end of systole (Fig. 14-26)
      2. Cardiac output = stroke volume X heart rate (Fig. 14-31)

c. Regulation
   i. Autonomic control of heart rate. Sympathetic increases heart rate by increasing sodium and calcium influx. Parasympathetic decreases by increasing potassium efflux and decreasing calcium influx (Fig. 14-27)
   ii. Epinephrine and NE increase heart contraction force and lower duration.

Chapter 14 Problems: 2-8, 10, 12-16, 20, 22, 26-28

Chapter 15 – Blood Flow

A. Blood Vessels
   a. Types (Fig. 15-1)
      i. Arteries and arterioles
         1. Carry blood away from heart.
         2. They have thick layers of smooth muscle and lack valves.
         3. They experience high blood pressure.
      ii. Veins and venules
         1. Carry blood toward the heart.
         2. They have thin layers of smooth muscle and have valves to prevent backflow.
         3. They experience low blood pressure.
      iii. Capillaries
         1. Very permeable
         2. Consist of single layer of endothelial cells.
         3. Greatest volume and lowest velocity (Fig. 15-18)
   b. Angiogenesis – growth of new vessels
      i. Occurs during development and during wound repair.
      ii. Cytokines such as VEGF (vascular endothelial growth factor) cause angiogenesis. Tumors can release VEGF so vessels can help feed them.
      iii. Cytokines such as angiostatin can block angiogenesis. It could be used to treat some cancers.

B. Blood Pressure
a. Blood pressure highest in arteries right outside of the heart. It is lowest in veins near heart (Fig. 15-5).
b. Veins are aided by skeletal movement and one-way valves (Fig. 15-6).
c. Systolic – during ventricular contraction (120 mm Hg). Diastolic – during ventricular relaxation (80 mm Hg)
d. Velocity highest in largest vessels.
e. Sphygmomanometry – measuring blood pressure by a cuff (Fig. 15-7)
   i. Cuff is inflated above pressure needed to cut off artery. Cuff is deflated and as pressure drops, blood begins to flow. This is systolic pressure. Blood flow can be heard.
   ii. When pressure drops to where full flow returns, blood flow cannot be heard. This is diastolic pressure.
f. Pressures above 140/90 are considered hypertensive.
g. Factors that influence pressure: volume, cardiac output, resistance, distribution (Fig. 15-10)
h. Regulation
   i. Autoregulation – smooth muscle stretch receptors cause influx of calcium causing contraction.
   ii. Paracines – signaling molecules that increase or decrease blood flow (Tab. 15-2)
   iii. Sympathetic pathways – exert tonic control on arteriolar diameter
   iv. Baroreceptor reflex (Fig. 15-22)
      1. Stretch mechanoreceptors (baroreceptors) monitor pressure in carotid and aortic arteries.
      2. They send messages to medullary cardiovascular control center in brain.
      3. It sends signals through sympathetic and parasympathetic neurons to control SA node, ventricles, and vessels
C. Exchange at Capillaries
   a. Types of capillaries (Fig. 15-17)
      i. Continuous capillaries – have leaky junctions between cells. (Exception is brain with tight junctions to form blood-brain barrier.)
      ii. Fenestrated capillaries – have fenestrations (pores) that allow large volumes to pass.
   b. Mechanism of exchange
      i. Diffusion
      ii. Transcytosis – use of vesicles for transport
      iii. Bulk flow
         1. Movement of fluid. Absorption is net flow in and filtration is net flow out.
         2. Forces
            a. Hydrostatic pressure – blood pressure. Highest at arterial end, lowest at venous end.
            b. Osmotic pressure is constant
            c. Net flow (Fig. 15-19)
               i. Arterial side – hydrostatic pressure > osmotic pressure filtration
               ii. Venous side – osmotic pressure > hydrostatic pressure absorption
               iii. There is more net filtration than absorption. Lymphatic vessels absorb excess fluid (3L/day). Edema occurs if fluid is not moved.
D. Cardiovascular Disease
a. Atherosclerosis
   i. Mechanism (Fig. 15-25)
      1. Fatty streak - LDL gets under endothelial cells of arteries. Macrophages
         ingest LDL. Smooth muscle cells move into streak.
      2. Stable fibrous plaque – lipid core forms. Scar tissue forms around lipid
         core. Calcifications begin
      3. Vulnerable plaque – begins to break down. Inflammation and clotting
         begin. Enzymes destabilize clot.
   ii. Risk factors
      1. High LDL – from diet or hypercholesterolemia (uptake of LDL into cells
         is disrupted)
      2. Smoking
      3. Diabetes
      4. Lack of exercise
   iii. Complications
      1. Heart attack – a plaque breaks off and cuts off circulation in coronary
         arteries
      2. Stroke - a plaque breaks off and cuts off flow to brain
b. Hypertension
   i. Risk for cardiovascular disease doubles for each 20/10 mm Hg over 115/75 (Fig.
      15-26)
   ii. Can accelerate atherosclerosis.
   iii. Cause is usually hereditary
   iv. Treatment
      1. Decrease fluid retention by lowering salt intake or taking diuretics
      2. Vasodilation by taking vasoconstriction inhibitors (ACE and angiotensin
         receptor inhibitors)
      3. Lower cardiac output by “beta-blockers” that block beta receptors for
         cardiac stimulation
      4. Calcium channel blockers prevent calcium from stimulating cardiac and
         smooth muscle contraction.

Chapter 15 Problems: 2, 4, 7-9, 12, 16, 18, 19a-d, 20, 22-24, 26, 27, 29, 30, 32, 34

Chapter 17 – Breathing

A. Respiratory System
   a. Overall Functions (Fig. 17-1)
      i. Exchange of gases – oxygen in, carbon dioxide out
      ii. Regulation of pH – carbon dioxide lowers blood pH
      iii. Conditioning of air – filtration of pathogens and pollutants, warming, and
         moistening.
      iv. Vocalizations
   b. Components (Fig. 17-2)
      i. Nasal cavity ➔ pharynx ➔ larynx ➔ trachea ➔ bronchi ➔ bronchioles ➔
         alveoli.
      ii. Upper respiratory system is larynx and above. Lower is trachea and below.
      iii. Lungs consist of alveoli and bronchioles. Each is surrounded by pleural sac that
         seals lungs and helps in breathing and keeping lungs inflated. (Fig. 17-12)
      iv. Air is conditioned before entering lungs. Mucus along entire length plays a large
         role. Cilia on epithelium move mucus towards pharynx. (Fig. 17-5)
v. Alveoli make up the majority of surfaces for gas exchange.

B. Gas Laws
   a. Pressure
      i. Partial pressure – each gas contributes to pressure of a system. E.g. air = 760 mm Hg, but N₂=593, O₂=160, CO₂=0.25. (Tab. 17-2)
      ii. Gases move down their individual pressure gradients.
   b. Boyle’s Law
      i. Describes the relationship between pressure and volume (Fig. 17-6)
      ii. \( P_1V_1 = P_2V_2 \)

C. Ventilation
   a. Respiratory volume and capacity (Fig. 17-8)
      i. Tidal volume – amount of air entering (or exiting) during a single normal breath.
      ii. Residual volume – amount of air left in lung after full expiration.
      iii. Vital capacity – maximum amount of air that can be taken in (or blown out).
      iv. Total lung capacity – total amount of air that can fit in the lung (vital capacity plus residual volume).
   b. Mechanism
      i. Inspiration – inhalation.
         1. Diaphragm muscle contracts to pull on pleural membrane. This draws in air like an accordion (Fig. 17-9).
         2. Maximal inspiration requires pectoral and sternal muscles to contract.
         3. Inspiration causes negative alveolar pressure, allowing air to enter. Intrapleural pressure drops to a minimum at end of inspiration (Fig. 17-11)
      ii. Expiration – exhalation.
         1. Diaphragm muscle relaxes and elastic recoil of lung tissues pushes out air.
         2. Maximal expiration requires abdominal muscles to contract
         3. Expiration causes positive alveolar pressure, pushing air out. Intrapleural pressure rises to a maximum at end of expiration.
   c. Factors facilitating ventilation
      i. Compliance (stretchiness) and elasticity (shrinking back from stretching)
      ii. Surfactants – phospholipids and proteins that reduce surface tension in alveoli. This allows less force in breathing to stretch alveoli.
      iii. Airway resistance – normally, bronchii and trachea contribute to the most resistance. However, bronchioles are under autonomic control. Bronchoconstriction can occur under parasympathetic control. Bronchodilation occurs under sympathetic control or high CO₂.

D. Efficiency and local control
   a. Depth
      i. Total pulmonary ventilation = air taken in at each breath. 500ml.
      ii. Alveolar ventilation is only 350ml because of anatomic dead space (air that never makes it out of conducting airways) (Fig. 17-14)
   b. Rate – higher rate will increase volume/time
   c. Gas composition
      i. Under normal breathing, alveolar partial pressures of O₂ and CO₂ does not change much during inspiration and expiration
      ii. However hyperventilation or hypoventilation does change them (Fig. 17-15)
   d. Blood flow matches ventilation
      i. Blood flow and alveolar ventilation are matched
If blood is not properly oxygenated, vessels constrict. This helps divert blood to other vessels for proper oxygenation (Fig. 17-16)

E. Pulmonary Disorders
   a. Diagnostic tools
      i. Spirometer – measures ventilation performance (Fig. 17-7)
      ii. Auscultation – listen to breathing sounds
   b. Restrictive lung disorders
      i. Compliance is reduced
      ii. E.g. asbestosis. Asbestos fibers lodge in alveoli an prevents expansion
   c. Obstructive lung disorders
      i. Airway is restricted. Compliance if fine
      ii. E.g. asthma. Inflammation leading to bronchoconstriction. Usually associated with allergies.

Chapter 17 Problems: 1, 4-6, 8, 10, 11, 14, 15a-d, 18, 21, 23-25, 27-29, 31

Chapter 18 – Gas Exchange

A. Exchange at Lungs and Tissues
   a. Diffusion increases with surface area, concentration gradient, and thinness of membrane
   b. Gases diffuse down their partial pressure gradients (Fig. 18-3)
      i. \( \text{O}_2 \) goes from alveoli to blood to tissues
      ii. \( \text{CO}_2 \) goes from tissues to blood to alveoli
   c. Several conditions can lead to low blood oxygen (hypoxia) (Fig. 18-4)

B. Transport in Blood (Fig. 18-6)
   a. Oxygen
      i. Bound to hemoglobin
         1. 98% of oxygen carried this way
         2. Hemoglobin contains 4 subunits, each with a heme group (Fig. 18-8)
         3. Dissociation curves express amount of hemoglobin-oxygen binding (Fig. 18-9)
         4. High pH, low temp, and low \( \text{CO}_2 \) increase \( \text{O}_2 \) binding (Fig. 18-10)
         5. Fetal hemoglobin has higher affinity than maternal hemoglobin (Fig. 18-12)
         6. Carbon monoxide (CO) kills because it binds hemoglobin preventing oxygen binding.
      ii. Dissolved in plasma (2%)
   b. Carbon Dioxide (Fig. 18-14)
      i. Bound to hemoglobin (23%)
      ii. Dissolved in plasma (7%)
      iii. Dissolved as bicarbonate in plasma (70%)
         1. \( \text{CO}_2 \) enters red blood cell and is converted to carbonic acid (\( \text{H}_2\text{CO}_3 \)).
            This will form bicarbonate (\( \text{HCO}_3^- \)) and \( \text{H}^+ \)
         2. Bicarbonate is excreted into plasma and \( \text{H}^+ \) is bound to hemoglobin

C. Regulation of Ventilation
   a. Control centers
      i. Medulla oblongata (Fig. 18-17)
         1. Quiet breathing is controlled by the dorsal respiratory group (DRG).
         2. Some voluntary and forceful breathing is controlled by the ventral respiratory group (VRG). Also acts a breathing pacemaker.
1. Pontine respiratory group (PRG) coordinates rhythm.

b. Sensing
   i. Chemoreceptors
      1. Peripheral – glomus cells in arteries activate medullary centers by low oxygen, pH, or high carbon dioxide. (Fig. 18-19)
      2. Central – high CO\textsubscript{2} and acid levels are detected in cerebrospinal fluid at medulla. (Fig. 18-20)
   ii. Mechanoreceptors
      1. Irritants can trigger protective reflexes including coughing and sneezing.

c. Higher brain centers
   i. Voluntary breathing – controlled by cerebellum
   ii. Limbic system – emotional and autonomic activities affecting breathing rate.

Chapter 18 Problems: 2-8, 11-13, 16-18, 20, 23-26, 30