Chapter 8 Physics of the Cardiovascular System

- Cardiovascular system (CVS): blood, vessels, heart
  - Supply energy (fuel from food)
  - Supply O₂ from the air we breathe
  - Dispose by-products of combustions (CO₂, H₂O, heat, etc)
- Blood: 7% of body mass: 4.5 kg or 4.4 liters in a 64 kg person
- Fetal heart
  - Start blood circulation at the eighth week after conception
  - Obtain oxygenated blood from mother via umbilical cord
  - An opening between RA and LA
    - 90% of blood flows from RA to LA through the opening
    - 10% circulates through fetal lungs
    - Within minutes after birth, the opening is closed in effect
    - Complete closure takes several months
    - Blue baby: inadequate closure of the opening, requires a surgery
- Specialists
  - Hematologist
  - Cardiologist
  - Heart surgeon

1. Heart

- Heart: Fig. 8.1
  - Double pump
  - Provides the force needed to circulate the blood
  - Two major circulatory system (Fig. 8.2)
    - Pulmonary circulation (right side pump): RV (25 mmHg) ⇒ pulmonary artery ⇒ pulmonary capillary ⇒ pulmonary vein ⇒ LA (7 ~ 8 mmHg) ⇒ LV
    - Systemic circulation (left side pump): LV (125 mmHg) ⇒ arteries ⇒ arterioles ⇒ capillary bed (for a few seconds) ⇒ venules ⇒ veins ⇒ superior vena cava and inferior vena cava ⇒ RA (5 ~ 6 mmHg) ⇒ RV
• Four chambers
  □ Atrium collects returning blood
  □ Ventricle generates high pressure

○ Cardiac muscle
    • Shares characteristics of skeletal and smooth muscle
      □ Strong tension, all-or-none response (like skeletal muscle)
      □ Not under direct conscious control, influenced by autonomic nervous system (like smooth muscle)
    • Cardiac muscle cells exhibit rhythmic self-excitation
    • Long refractory period (~ 250 ms): prevents tetanus (sustained contraction) and limits heart rate

○ Cardiac output
    • Cardiac output = heart rate × stroke volume
    • Cardiac output is determined by heart rate, contractility (contraction strength), and peripheral flow resistance
    • Regulation of heart rate and contractility is very complex and involves CNS and hormonal influences, intrinsic cardiac mechanisms
    • For typical adult, about 5 l/min
    • Stroke volume ~ 80 ml
    • About 1 min for the average RBC to make one complete cycle of the body
    • Maximum cardiac output
      □ 25 l/min for young men
      □ 40 l/min for trained athletes

○ Heart valves and artificial heart valves (Fig. 8.18)
    • One-way flow
    • Valve closing and turbulent blood flow ⇒ heart sound

○ Blood volume: total volume of about 5 l
    • 80% (4 l) in systemic circulation and 20% (1 l) in pulmonary circulation
    • Systemic circulation: 15% in arteries, 10% in capillaries, 75% in veins
    • Pulmonary circulation: 7% in pulmonary capillaries, 46.5% in pulmonary arteries, 46.5% in pulmonary veins

2. Physics of Blood Flow

○ Conservation of flow rate
Flow rate = blood volume/time, l/min

Blood flow rate is continuous under three assumptions
- Blood is incompressible
- Vessels are rigid
- There are no sinks (leakage or hemorrhage) or sources (transfusion) of blood flow

Continuous flow \( \Rightarrow F_{\text{total}} = \text{cardiac output} = F_{\text{arteries}} = F_{\text{arterioles}} = F_{\text{caps}} = F_{\text{venules}} = F_{\text{veins}} \)

Flow velocity
- Flow rate = cross-sectional area \times \text{velocity} \((F = Av)\)
- For a group of \(n\) parallel blood vessels,
  - \(A_{\text{total}} = n A_{\text{average}}\)
  - \(F = A_{\text{total}} \times v_{\text{average}}\)
  - \(F = A_{\text{arteries}} \times v_{\text{arteries}} = A_{\text{arterioles}} \times v_{\text{arterioles}} = A_{\text{caps}} \times v_{\text{caps}} = A_{\text{venules}} \times v_{\text{venules}} = A_{\text{veins}} \times v_{\text{veins}} = \text{constant}\)
- Flow velocity is inversely related to total cross-sectional area (Fig. 8.11)
- \(v_{\text{arteries}} > v_{\text{arterioles}} > v_{\text{caps}} < v_{\text{venules}} < v_{\text{veins}}\)

Flow resistance (Fig. 8.15 and 8.16)
- Laminar flow \((v < \text{critical velocity})\)
  - Silent
  - Maximum velocity at the center
  - Zero velocity at the wall
  - Ponceuille's law: \(F = \Delta p \frac{\pi}{8} \frac{1}{\eta} \frac{r^4}{L}\)
  - \(r\) is dominant factor for controlling flow through vessel
  - Smooth muscle in vessel walls (except capillaries) controls \(r\)
- Turbulent flow \((v > \text{critical flow})\)
  - Audible \(\Rightarrow\) heart sound
  - Higher flow resistance than laminar flow
  - Most likely to occur at constrictions, further reducing flow

3. Blood Vessels and Flow Regulation

Different vessels have distinct anatomy and physiology. This is due in part to large changes in blood pressure throughout the circulation (Fig. 8.5).
- Combined length of all blood vessels > earth’s circumference (\(~ 25,000\) miles)
Arteries
- Thick, compliant walls
  - Must withstand high BP
  - Puncture can be fatal
  - High BP damages arterial walls, leading to vessel disease
- Large diameter
  - Low flow resistance
  - Transmits entire heart pressure to arterioles

Arterioles
- Large pressure drop ⇒ controls flow distribution
- Local control
  - Meet local metabolic rate
  - Increase flow to muscles during exertion
- Reflex (global) control
  - Regulate arterial BP
  - Maintain flow to vital organs (brain)
- Role of arterioles in hypertension
  - Autonomic nervous system innervates smooth muscle surrounding blood vessels.
  - Excessive constriction of arterioles is primary mechanism responsible to high BP.
  - Constriction of arterioles ⇒ high peripheral resistance ⇒ BP increases to maintain flow

Capillaries
- Maximize exchange
  - Huge surface area (~ football field)
  - Thin walls (no smooth muscle) to facilitate diffusion
  - Low blood velocity (~ 1 mm/s) gives more time for exchange
- Flow is regulated by precapillary sphincter (on-off)
- Only 1 of 30 capillaries is open in resting muscle

Veins and venules
- Veins: very large radius and thin wall
  - Largest cross-sectional area
  - Negligible flow resistance
  - Large volume: holds 75% of blood and acts as blood reservoir
- Pressure in veins governs ventricular filling (i.e. volume of blood filling heart),
an important determinant of cardiac output. If ventricular filling increases, the stretched heart responds with increased contractive force (Starling’s law), which increases stroke volume and cardiac output.

- Veins have one-way valve. Constriction of the veins drives blood toward heart, increasing cardiac output via Starling mechanism.

4. Arterial Blood Pressure

- BP during cardiac cycle: Fig. 8.17
  - Systole: ventricles contract, arteries expand, hold 2/3 of stroke volume
  - Diastole: ventricles relax, arteries recoil, squeezing blood to arterioles

- Effect of gravity on arterial blood pressure: Fig. 8.8

- Arterial BP affected by gravity \((P = \rho gh)\)
  - Resistive losses are small
  - No blood to head for upward acceleration of \(a > 3g\)
  - Adaptation to counteract gravity: one-way valve in veins, skeletal muscles squeeze veins, forcing blood back to heart

- Measurement of arterial BP: Fig. 8.6 and 8.7

- Sphygmomanometer: inflatable cuff and pressure gauge
  - Inflate cuff at heart level until \(P > P_{\text{systole}} \Rightarrow \text{no blood flow}\)
  - Slowly deflate. Listen for Korotkoff sounds (K sounds, due to turbulent blood flow)
    - First sound \(\Rightarrow P = P_{\text{systole}}\)
    - Last sound \(\Rightarrow P = P_{\text{diastole}}\)

- Work of heart
  - Work = area under \(P(V)\) curve
  - Heart works harder for higher BP
  - Power = \(\Delta P \frac{dV}{dt} = 100 \text{ (mmHg)} \times 80 \text{ (ml/s)} = 1.3 \times 10^4 \text{ (Pa)} \times 8 \times 10^5 \text{ (m3/s)} \sim 1.1 \text{ W (7 W peak)}\)
  - \(E_{\text{consumed}} = 10 \text{ W (assuming efficiency of 10\%)}\)

5. Cardiovascular Disease
Congestive heart failure (CHF)

- Normally heart increases contractility in two ways
  - By increasing nervous stimulation of the heart
  - By increasing ventricular filling (Starling mechanism)
- CHF is characterized by a heart that is weak and enlarged due to greatly increased ventricular filling. But for a very high ventricular filling, cardiac output falls.
- Backward failure of the heart: the heart cannot pump out all the blood returning from veins. Blood, therefore, backs up in the veins, which increases pressure in the veins and capillaries, causing fluid to accumulate in the tissues (edema)
  - Right ventricle failure: back-up in systemic circulation (swollen legs)
  - Left ventricle failure: back-up in pulmonary circulation (pulmonary congestion, very serious)

Treatment
- Rest
- Drugs to increase contractility, excretion
- Heart transplant

Risk factors, promoters of vessel disease

- Sex, age, weight, family history, general health, …
- Hypertension: 60 million in US, 50% are aware, silent killer
  - Systolic BP > 140 mmHg
  - Diastolic BP > 90 mmHg
  - Leading cause of stroke and heart disease
  - Arterioles constricted, regulation goes awry
  - 10% (50% in US) are sodium sensitive (salt elevates BP)
  - No obvious symptoms ⇒ silent killer
  - Damages arteries ⇒ hardening of arteries ⇒ narrowing (atherosclerosis)
- Cholesterol
  - Found only in animal tissue
  - Synthesized by liver from saturated fat
  - Important for metabolism, precursor to hormones
  - Two types: LDL (low density lipoprotein) and HDL
  - LDLs deposit on vessel walls ⇒ bad cholesterol
  - HDLs reduce the amount of LDLs ⇒ good cholesterol
- Smoking
  - Increases BP
Smoke contains CO ⇒ stresses heart
Increases platelet clumping ⇒ promotes atherosclerosis
Effects are reversible
- Other factors
  - Salt: not certain except effect on hypertension
  - Caffeine: no strong link to heart disease
  - Chronic stress: may increase risk in some individuals
  - Moderate alcohol: appears to be beneficial

Vessel disease
- Arteries surrounded by tough connective tissue. However, high blood pressure can damage the tissue, leading to vessel disease.
- Arteriosclerosis: hardening of arteries
- Atherosclerosis: plaque (fat, calcium) deposits on and within lining of damaged vessel
- Vessel wall damage ⇒ blood clots ⇒ clog arteries

Heart attack
- 0.5 million deaths annually in US
- Infarction: death of heart muscle due to blockage of coronary arteries
  - Heart uses 5/6 of blood oxygen
  - Often little or no collateral circulation
  - Coronary arteries are first to suffer complete blockage (SaO₂ ~ 0% in venous return from heart)
- Partial blockage
  - Angina pectoris: pain during exertion
  - Develop collateral circulation in a few days
- Sudden (acute) blockage
  - Blood clot forms rapidly on rough surface
  - Clot or deposit breaks off, blocks vessel downstream
- Symptoms of heart attack
  - The most common symptom (> 80%) is a heavy, persistent pressure in the center of the chest, lasting 10 min or more.
  - The pain often radiates upward to the neck, jaw, or left arm and shoulder. Weakness and profuse sweating are also common.
  - Sometimes the pain is a burning sensation, similar to heartburn.
  - Rarely associated symptoms: brief and sharp pain, pain on breathing
- Precautions for high-risk persons
- Sensible diet, life style
- Avoid overexertion, especially after eating

- Treatments
  - Angina: nitroglycerin (vasodilator) can relieve symptoms
  - Drugs ⇒ dissolve clot
  - Angioplasty ⇒ clean out vessel
  - By-pass surgery

- Stroke, cerebrovascular accident (200,000 deaths annually in US)
  - Blockage or rupture of brain blood vessel
  - Stroke symptoms (varied, often lateralized)
  - One-sided numbness or weakness
  - Blurred or decreased vision
  - Sudden severe headache
  - Problems speaking or understanding
  - Dizziness, loss of balance