The Heart

Circulation
- Heart is the pump that circulates blood
- Systemic circulation
  - Left atrium, left ventricle, aorta, arteries, arterioles, capillaries, venules, veins, vena cava
- Pulmonary circulation
  - Right atrium, right ventricle, pulmonary trunk, pulmonary arteries, arterioles, capillaries, venules, pulmonary veins
- Oxygenated vs. deoxygenated blood

Location
- Within the mediastinum
- 2/3 of mass left of midline
- Enclosed in pericardium
  - Three layers - fibrous pericardium, and two layers of serous pericardium (parietal & visceral or epicardium)
  - Pericardial cavity between two serous layers
  - Pericardial fluid reduces friction
  - Pericarditis and cardiac tamponade
- Suspended by superior vessels and diaphragm

Heart Structure
- Three layers
  - Epicardium - visceral layer of pericardium
  - Myocardium - muscle layer, two networks separated by connective tissue
  - Endocardium - endothelial and connective tissue layer contiguous over chamber interiors, valve & endothelia of vessels
- Chambers - R/L atria, auricles, pectinate muscles (anterior), fossa ovalis, R/L ventricles, septum, trabeculae carneae

Heart Valves
- A-V valves
  - Tricuspid & bicuspid
  - Chordae tendinae, papillary muscles
- Semilunar valves - three cusps
- All are set in layer of dense connective tissue - supports, prevents over-stretching and insulates A & V
- Valve damage
  - Rheumatic fever (from group A, β-hemolytic Streptococcus pyogenes)
  - Phen Fen

Blood Supply to Heart
• Blood supply to myocardium
• Many anastomoses
• Most heart problems associated with coronary circulation (blood clots, atherosclerotic plaques, vascular spasms)
  – Angina pectoris - ischemia caused hypoxia associated with exertion
  – Myocardial infarction - interrupted blood supply due to thrombus or embolus
  • Use of thrombolytic agents, angioplasty, or bypass surgery

Conduction of Excitability
• Autorhythmicity controlled by autonomic NS and hormones
• Components include: SA node, AV node, bundle of His, R/L bundle branches, Purkinje fibers
• SA node rhythm at 90-100 APs/min
  – Resting HR reduced to 75 beats/min by parasympathetic control (ACh)
• Timing - 50 msec SA→AV, 100 msec delay at AV node (small diameter cells), 50 msec to all of ventricles = 200 msec total
• Ectopic focus - due to caffeine, nicotine, electrolytes, hypoxia, drugs

Inherent AP Rates
• SA node 90-100 APs/min
• AV node 40-50 APs/min
• AV conduction fibers 20-40 APs/min
• Artificial pacemakers
  – Activity sensitive pacemakers

Physiology of Cardiac Muscle
• AP from pacemaker, through conduction fibers, to cells connected by intercalated discs
• Depolarization initiates contraction
  – Voltage-gated fast Na⁺ channels
  – Voltage-gated slow Ca²⁺ channels
    • Certain drugs alter calcium flow e.g. epinephrine ↑
  – Voltage-gated K⁺ channels
  – Repolarization
• Sliding filament action similar to skeletal muscle
• Long refractory period

Electrocardiogram (ECG)
• Measures electrical current generated by heart
• Magnitude & direction of waveform depends on mass of myocardium contracting and direction of impulse relative to the leads
• Direction, magnitude and timing provide diagnostic information about:
  – Conduction pathway abnormalities, heart enlargement, regional damage
• Bigger P wave - enlarged atrium due to mitral stenosis
• Larger Q wave - myocardial infarction
• Larger R wave - enlarged ventricles
• Flattened T wave - insufficient oxygen (coronary artery disease)
• Enlarged T wave - increased K⁺ conc in blood
• P-Q interval longer - scarring reduced conduction

Cardiac Cycle
• See fig. 20.13
• Systole vs. diastole
• Pressure changes (mm Hg)
  – Vena cava 8-10
  – Pulmonary veins 5-20
  – Pulmonary artery 15-20
  – Aorta 80-120
• Other terms:
  – Ventricular filling, diastasis, isovolumetric contraction & relaxation, dicrotic wave
• Effect of changes in heart rate

Heart Sounds
• Lupp - closing of AV valves
• Duppp - closing of semilunar valves
• Heart murmur
  – Mitral stenosis, insufficiency or valve prolapse (genetic)
  – Aortic stenosis or insufficiency

Cardiac Output
• Amount of blood leaving L ventricle per minute
• CO=stroke volume x beats per min
  – 5250 ml/min = 70 ml/beat x 75 beats/min)
• Cardiac reserve=max CO/ CO at rest
  – 4-5 for most, 7-8 for conditioned athletes

Stroke Volume
• SV=EDV - ESV
  – 70ml = 130ml - 60ml
• Adjustments in SV
  – Preload (increased EDV) - stretching of muscle increases systolic force (Frank-Starling Law)
    • Related to duration of diastole and venous BP
    • Exercise decreases duration but increases BP
    • At above about 160 beats/min, (short diastole) preload & EDV decline
  • Contractility - controlled by release of Ca²⁺
    – Positive inotropic agents
• Stimulation by sympathetic ANS
  • Hormone (epinephrine based)
  • Increased extracellular Ca\(^{2+}\)
  • Drugs like Digitalis
    – Negative inotropic agents
      • Inhibition of sympathetic ANS
      • anoxia
      • acidosis
      • Some anesthetics
      • High extracellular K\(^+\)

• Afterload - arterial pressure ventricles must exceed to eject blood
  – Increase afterload, decrease SV (due to high BP or blockage)

• Decline in SV leads to congestive heart failure (CHF)
  – Caused by excessive stretching of ventricular muscle
  – If left side can’t keep up, pulmonary edema
  – If right side can’t, peripheral edema

**Regulation of Heart Rate**

• Important short-term control over CO and BP

• ANS
  – Cardiovascular center in medulla receives input from:
    • Higher brain centers (cerebrum, limbic) - anticipation
    • Proprioceptors in muscles
    • Chemoreceptors that detect \(O_2, CO_2\) and H\(^+\) in blood
    • Baroreceptors that measure pressure in aorta and carotid arteries
  – ANS has two divisions
    • Sympathetic via cardiac accelerator nerves to increase HR and force of contraction (increased Ca\(^{2+}\) flow into cells)
      – Max CO between 160-200 beats/min
    • Parasympathetic via vagus nerves decreases HR (via ACh)
      – Min HR is 20-30 beats/min

• Chemical control
  – Hypoxia and high H\(^+\) decrease HR
  – Hormones - epinephrine (adrenal glands) & thyroid hormones increase HR
  – Ions
    • Excess K\(^+\) and Na\(^+\) decreases HR and contractility
    • Moderate increase of Ca\(^{2+}\) increases HR & contractility

• Other factors
  – Age
  – Gender
  – Physical fitness
  – Body temperature

**Heart Disease Risk Factors**

• High blood cholesterol level
• High BP
• Smoking
• Obesity
• Lack of regular exercise
• Alcoholism
• Other, less controllable factors
  – Diabetes, gender, genetic predisposition

Plasma Lipids
• Moved to Chapter 25
• High blood cholesterol promotes growth of fatty plaques (atherosclerosis)
• Cholesterol important for cell membranes, steroid synthesis, and bile production
• Lipids transported via proteins
  – LDL, HDL, VLDL
  – LDL has highest % of cholesterol - transfer cholesterol to cells
  – HDL has lowest % of cholesterol - transfer cholesterol from cells to liver
  – VLDL transfer triglycerides from liver to fat storage
  • Upon release of triglyceride, becomes LDL
• Sources include diet and liver production
• Acceptable levels
  – TC less than 200 mg/dl
  – LDL less than 130 mg/dl (TC-HDL)
  – HDL greater than 40 mg/dl
  – Risk ratio (TC/HDL) less than 4

Effects of Exercise
• Aerobic exercise 20 mins 3-5 times/wk
  – Increased CO and hemoglobin
  – Cardiac hypertrophy, higher SV & lower HR
  – Increased HDL, lower triglycerides
  – Decreased blood pressure
  – Increased thrombolytic activity
  – Others - improved lung function, weight control, reduced incidence of diabetes, lower anxiety and depression

Heart Disorders
• On your own