Cardiovascular System:

Pulmonary circuit:
right ventricle → lungs → left atrium

Systemic circuit:
left ventricle → body → right atrium

Arteries = away from heart
Veins = toward heart
Capillaries = exchange vessels in between
Heart

-left of midline, between 2nd rib and 5th intercostal space, posterior to sternum, in pericardial cavity in mediastinum
-heart is fist sized, < 1 lb, beats 100,000 times/day moving 8000 L blood/day
-surrounded by pericardium (serous and fibrous layers)
-serous membranes (visceral & parietal) secrete pericardial fluid, reduce friction

Pericarditis = inflammation of pericardium, usually due to infection, causes friction
Cardiac tamponade = buildup of fluid in pericardial space, restricts heart movement
Four Chambers:

2 Atria:
-superior, thin walls, smooth posterior walls internally,
pectinate muscles (ridges) anteriorly

-each has expandable flap called an auricle lateral and superior
-left and right separated by interatrial septum
2 Ventricles:
- inferior, thick walls, lined with trabeculae carneae (muscular ridges)
- left and right separated by interventricular septum
Left ventricle 3X thicker, 5X more friction while pumping, same volume as right
Left is round, right is crescent

External divisions:
Coronary sulcus marks division between atria and ventricles

Anterior interventricular sulcus and posterior interventricular sulcus mark division between ventricles
Heart Wall Layers

1. Epicardium (thin) - visceral pericardium: serous membrane with loose CT attached to myocardium
2. Myocardium (thick) - cardiac muscle tissue with CT, vessels and nerves
3. Endocardium (thin) - simple squamous epithelium lining with basal lamina; continuous with endothelium of blood vessels
Cardiac Muscle Tissue
-muscle cells = cardiocytes
-uses actin and myosin sliding filaments to contract
-rich in mitochondria, resists fatigue but dependent on aerobic respiration
-cells connected by intercalated discs = desmosomes + gap junctions

-contraction is all or none
-longer contractile phase than skeletal muscle
-fibrous skeleton of the heart (tough CT) acts as the tendon
Heart Valves (one way, prevent backflow)

Atrioventricular valves - between atria and ventricles
(flaps = cusps)

Tricuspid valve:
right side, 3 cusps

Bicuspid/Mitral valve:
left side, 2 cusps

cusps attached to chordae tendineae from papillary muscles on ventricle wall

-contraction of papillary muscles prevent cusps opening backward during ventricle contraction

-cusps hang loose when ventricles not contracting, allow ventricles to fill with blood
Semilunar valves - between ventricles and arteries
-3 cusps
-no chordae tendineae or muscles
-forced open by blood from ventricular contraction
-snap closed to prevent backflow

Valvular heart disease - valve function deteriorates to extent that heart cannot maintain adequate circulation
   e.g. Rheumatic fever - childhood reaction to streptococcal infection, chronic carditis, VHD in adult

Heart murmur - leaky valve (audible to Dr.)

Mitral valve prolapse - murmur of left AV valve, cusps don’t close properly, blood regurgitates back into left atrium
Congestive heart failure - decreased pumping efficiency (diseased valves, damaged muscle), blood backs up, fluid leaks from vessels and collects in lungs and tissues

Blood Flow Through The Heart
(on handout)
Fetal Heart (adapted to bypass lungs)

- Foramen ovale in right atrium,
  ~25% of blood bypasses directly the left atrium,
closes at birth leaving scar called fossa ovalis.

- Ductus arteriosus connects pulmonary trunk to aorta,
  ~90% of blood bypasses lungs,
closes at birth leaving the ligamentum arteriosum

Failure of either to close = poor oxygenation of blood, cyanosis, “blue baby syndrome”
Coronary circulation

- heart: <1% body mass, requires 5% of blood
- too thick for diffusion
- coronary arteries originate at base of ascending aorta, branch to capillary beds for diffusion
- blood returns via cardiac veins that empty into right atrium
Coronary artery disease - partial or complete block of coronary circulation, results in coronary ischemia
Can lead to myocardial infarction (heart attack): heart tissue denied oxygen dies.
Common symptom of CAD: angina pectoralis pain in the chest, especially during activity, as a result of the ischemia
Coronary bypass surgery - use healthy veins (from legs) to create anastomoses around blockages
Most people have 4 major coronary arteries: “quadruple bypass”
Heart Beat

-1% myocardial cells autorhythmic: depolarize without neural or endocrine stimulation
-depolarization transmitted to other myocardial cells through cardiac conduction system:
  Sinoatrial (SA) node - right atrium wall near superior vena cava
  Atrioventricular (AV) node - inferior portion of interatrial septum above tricuspid valve

Conducting cells: AV bundle, Bundle branches, and Purkinje fibers: connect nodes and myocardium, run down interventricular septum and around apex
-cells of nodes cannot maintain resting membrane potential, drift to depolarization:
  SA node 80-100 action potentials/min ("natural pacemaker")
  AV node 40-60 action potentials/min
-Resting rate (sinus rhythm) ~75bpm set by SA node + parasympathetic stimulation
Heartbeat Chain of Events: Electrical Conduction and Contraction (on handout)

Normal average heart rate = ~ 70-80bpm
max = ~230bpm, but inefficient above 180
Bradycardia = heart rate slower than normal <60
Tachycardia = heart rate faster than normal >100
(Fun fact?: Lifespan theory 800 million beats)
Electrocardiogram (EKG) (Kardio- German) - recording of electrical events of heart (on handout)

P-wave = depolarization wave from SA node through atria ~80ms

QRS complex = atrial repolarization and ventricle depolarization ~80ms

T-wave = ventricle repolarization ~160ms
EKG used to diagnose heart problems:
- P-R longer than 200ms = damage to AV node or conducting cells
  Total heart block = damaged AV node, no impulses transmitted through, atria and ventricles beat independently (atria fast, ventricles slow)
- Large QRS = enlarged heart
- Q-T longer than 380ms = coronary ischemia or myocardial damage
- Cardiac arrhythmias = abnormal patterns of cardiac activity
- Fibrillation = rapid, irregular, out of phase contractions due to activity in areas other than SA node: defibrillation to stop all activity so SA node can resume control
Normal Sinus Rhythm

SA Node Not Functional, AV Node Only (Note No P Waves, Rate: 40-60 bpm)

Heart Block: some P waves not conducted through AV node

Ventricular fibrillation
Cardiac Cycle
-alternating contraction and relaxation

Systole = contraction, high pressure, blood gets pushed to next chamber

Diastole = relaxation, low pressure, chamber fills with blood
(on handout)

↑ heart rate = ↓ cycle time, ↓ diastole time = ↓ time to fill
Atrial contraction only adds ~30% more to ventricles (can live with bad atria)
Heart Sounds
“lubb” - S1: AV valves close at start of ventricular systole
“dupp” - S2: semilunar valves close at start of ventricular diastole

Cardiac Output
CO = amount of blood pumped by each ventricle in one minute, depends on heart rate and stroke volume:  CO = HR X SV
SV = amount of blood pumped by ventricle
Usually SV constant, change HR to increase CO as needed.

HR affected by:
1. autonomic nervous input
   sympathetic = ↑ HR
   parasympathetic = ↓ HR
2. hormones
3. venous return
   more blood return = ↑ HR (stretch receptors activate sympathetic)
4. other factors (ions, drugs)
@ 160-180 bpm CO at max:

↑ HR = ↓ time to fill ventricles,
if not full = ↓ SV and ↓ CO

Conditioning can ↑ SV and ↓ HR
Fit athletes can ↑ max CO by 700%, & ↓ resting HR by 50% with same CO due to ↑ SV
Heart Rate Effectors:
1. Autonomic Innervation
-SA node, AV node and atrial myocardium
   innervated by both sympathetic (NE) and
   parasympathetic (Ach) nerve fibers
equally.
-Sympathetic dominates in ventricles.
-Cardiac centers in medulla oblongata monitor
   BP and gasses to adjust HR:
   A. Cardioacceleratory center -sympathetic
   B. Cardiocinhibitory center -parasympathetic
-Parasympathetic tone reduce rate of SA node:
   72-80bpm female
   64-72bpm male
   40bpm athletes
2. Hormones
Epinephrine, Norepinephrine, Thyroxine --all
   increase HR by acting at SA node
-Beta blockers- drugs to treat hypertension,
   block B-receptors for E / NE thus
   preventing sympathetic stimulation
3. Other Heart Rate Effectors:
- Caffeine: rapid depolarization of SA node, increases HR
- Nicotine: stimulates sympathetic neurons, increases HR
- Hyperkalemia: high K\(^+\), inhibits repolarization, beats weak, heart can stop
- Hypokalemia: low K\(^+\), hyperpolarization, cells less responsive, decrease HR
- Hypercalcemia: high Ca\(^+\), muscle cells excitable, increase HR, can cause prolonged contraction, heart seizes
- Hypocalcemia: low Ca\(^+\), contractions weak, heart can stop
- Temperature: affects metabolic rate of cardiocytes
  - high temp = \(\uparrow\)HR
  - low temp = \(\downarrow\)HR