Chapter 18
The Heart

Heart Overview
- Heart anatomy
- Cardiac muscle cells
- Heart chambers, valves and vessels
- Conducting system
- EKG
- Cardiac cycle
- Contractile and pacemaker cells
- Cardiodynamics
- Cardiac disorders

Organization of the Cardiovascular System

Heart
- Pump of cardiovascular system
- Approximate size of clenched fist
- Made of cardiac muscle
- Beats ~100,000 times/day
- Pumps 8,000L of blood/day (how much do you have?)
  - Top (base): ½ inch to left of midline
  - Bottom (apex): 3 inches to the left of midline
  - Rotated slightly so that right side faces anteriorly

Two Circuits
- **Pulmonary circuit**: Carries blood to and from gas exchange surfaces of lungs
  - Right heart
- **Systemic circuit**: Carries blood to and from the body
  - Left heart

Blood alternates between pulmonary circuit and systemic circuit (has to go through both, then starts over again)
Pericardium

- Pericardium – a double-walled sac around the heart composed of:
  - A superficial fibrous pericardium
  - A deep two-layer serous pericardium
    - Parietal pericardium (outer) lines the internal surface of the fibrous pericardium
    - Visceral pericardium (inner) or epicardium lines the surface of the heart
  - They are separated by the fluid-filled pericardial cavity filled with pericardial fluid
- Protects and anchors the heart
- Prevents overfilling of the heart with blood
- Allows for the heart to work in a relatively friction-free environment

Pericardium

- Pericarditis: Complication of viral infections that causes infection of the pericardium
- Risk of cardiac tamponade

The Heart Wall

- Epicardium: outer layer
- Myocardium: middle layer
- Endocardium: inner layer

Heart Wall

- Epicardium
  - Visceral pericardium, covers the heart
- Myocardium = muscular wall of the heart
  - Concentric layers of cardiac muscle tissue
  - Atrial myocardium wraps around great vessels
  - 2 divisions of ventricular myocardium
    - Superficial ventricular muscles: surround ventricles
    - Deep ventricular muscles: spiral around and between ventricles
- Fibrous skeleton of the heart – crisscrossing, interlacing layer of connective tissue
- Endocardium – endothelial layer (tissue type?)

Cardiac Muscle Cells

- Small with one, central nucleus
- Branching interconnections between cells
- Intercalated discs:
  - Interconnect cardiac muscle cells
  - Secured by desmosomes to convey force of contraction
  - Linked by gap junctions to propagate action potentials

Characteristics of Cardiac Muscle Cells
Cardiac Cells vs. Skeletal Fibers

<table>
<thead>
<tr>
<th>Feature</th>
<th>Cardiac Cells</th>
<th>Skeletal Fibers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shape</td>
<td>Multinucleate</td>
<td>Mononucleate</td>
</tr>
<tr>
<td>Myofilaments</td>
<td>Actin, myosin</td>
<td>Actin, myosin</td>
</tr>
<tr>
<td>Cytoskeleton</td>
<td>Myofilaments</td>
<td>Myofilaments</td>
</tr>
<tr>
<td>Type of Movement</td>
<td>Contraction</td>
<td>Contraction</td>
</tr>
<tr>
<td>Number of Mitochondria</td>
<td>Many</td>
<td>Few</td>
</tr>
<tr>
<td>Number of Adipocytes</td>
<td>Few</td>
<td>Many</td>
</tr>
<tr>
<td>Ability to Reproduce</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Ability to Proliferate</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>

General Anatomy of the Heart
- Great veins and arteries at the **base**
- Pointed tip is **apex**
- Surrounded by **pericardial sac**

Specific Heart Anatomy

**Anterior View**
- Right atrium: collects blood from systemic circuit
- Right ventricle: pumps blood to pulmonary circuit
- Left atrium: collects blood from pulmonary circuit
- Left ventricle: pumps blood to systemic circuit

**Posterior View**

Blood Vessels
- **Arteries**: carry blood away from heart
- **Veins**: carry blood to heart
- **Capillaries**: networks between arteries and veins. Also called exchange vessels because only in capillaries exchange materials (dissolved gases, nutrients, wastes) between blood and tissues

Heart Anatomy Overview
- **4 chambers** (RA, RV, LA, LV)
- **4 valves**
  - 2 at exit from ventricles (from atriya)
  - 2 at exit from ventricles (to great vessels)
  - None at entry to atria
- **4 major vessels at the base**
  - Superior vena cava (entry)
  - Inferior vena cava (entry)
  - Aorta (exit)
  - Pulmonary trunk (exit)
- 4 Pulmonary veins (entry) = not major vessels
Movie
• Heart Anatomy

Heart dividing lines

Sulci
– Grooves in the heart that divide it
– contain blood vessels of cardiac muscle

• Coronary sulcus
– divides atria and ventricles
– Closer to base (top) than apex

• Anterior and posterior interventricular sulci
– separate left and right ventricles

Chambers of the heart

• Right Atrium
  – receives deoxygenated blood through vena cavae

• Left Atrium
  – receives oxygenated blood through pulmonary veins

• Right Ventricle
  – pumps blood to lungs through the pulmonary arteries

• Left Ventricle
  – pumps blood into the systemic circuit through the aorta

Atria

• Small, thin-walled
• Expandable outer auricles = flaps on anterior surface
• Fill with blood passively
• Separated by interatrial septum
• Connected to ventricles via atrioventricular valves
• Internally covered with pectinate (comb) muscles, ridges on anterior atrial wall and inner surfaces of right auricle

Ventricles

• Right ventricle wall is thinner; LV develops 4-6 times more pressure than left ventricle
• Right ventricle is pouch-shaped, left ventricle is round
• Similar internally, but right ventricle has moderator band
• How do volumes compare?

Trabeculae Carneae

• Muscular ridges on internal surface of ventricles
• Includes moderator band (in RV):
  – ridge contains part of conducting system
  – coordinates contractions of cardiac muscle cells
Atroventricular (AV) Valves

- Right AV valve (tricuspid): between RA and RV
- Left AV valve (bicuspid or mitral): between LA and LV
- Have 3 or 2 fibrous flaps, respectively
- Permit blood flow in 1 direction: atria to ventricles
- Free edges of flaps attach via chordae tendineae to papillary muscles of ventricle
- Blood pressure closes valve cusps during ventricular contraction
  - muscles tense chordae tendineae, preventing valves from swinging into atria (opening backward)

Semilunar Valves

- Pulmonary valve
  - between RV and pulmonary trunk
- Aortic valve
  - between LV and aorta
- Prevent backflow from great vessels (pulmonary trunk and aorta) into ventricles
- Have no muscular support
- Both have 3 crescent-shaped cusps, support like a tripod
Valvular Heart Disease (VHD)
- Genetic, or complication of carditis (inflammation of heart muscle)
- Rheumatic fever is a common cause
- Decreases valve function to point where adequate circulation is no longer possible

Great Vessels - Veins
- Vena Cavae deliver systemic circulation to right atrium (oxy or deoxy?)
  - Superior vena cava: receives blood from head, neck, upper limbs, and chest
  - Inferior vena cava: receives blood from trunk, and viscera, lower limbs
- Right and left pulmonary veins return blood from lungs (oxy or deoxy?)

Great Vessels - Arteries
- Aorta receives blood from LV (through which valve?) (oxy or deoxy?)
  - Ascending aorta curve to form aortic arch sends off three branches and turns down to become descending aorta
- Pulmonary trunk splits into left and right pulmonary arteries to send blood from (chamber) through (valve) to lungs. (oxy or deoxy?)

Movie
- Heart valves

Regurgitation
- Failure of valves
- Causes backflow of blood into atria
- Can cause heart murmur
**Pulmonary Circuit**
- To RA from superior and inferior vena cavae
- RA through open tricuspid valve to RV
- Conus arteriosus (superior right ventricle) through pulmonary valve to pulmonary trunk
- Pulmonary trunk divides into left and right pulmonary arteries, to right and left lungs, respectively

**Systemic Circuit**
- Blood returns from lungs to left atrium through 2 left and 2 right pulmonary veins
- Left atrium to left ventricle through mitral valve
- Left ventricle through aortic SL valve into ascending aorta
- Ascending aorta turns (aortic arch) and becomes descending aorta

**Complete Pathway of Blood Through the Heart and Lungs**
- Right atrium → tricuspid valve → right ventricle → pulmonary semilunar valve → pulmonary arteries → lungs → pulmonary veins → left atrium → bicuspid valve → left ventricle → aortic semilunar valve → aorta → systemic circulation → vena cavae → repeat

**Foramen Ovale**
- Before birth, is an opening through interatrial septum
- Connects the 2 atria
- Seals off at birth, forming fossa ovalis

- Why connect the two atria?
- **PFO** – can cause problems in adulthood but usually only with strenuous exercise or high altitude

**Aortic Sinuses**
- Dilations at the base of ascending aorta
- Prevent aortic semilunar valve cusps from sticking to aorta when open
- Origin points of right and left coronary arteries

**Internal Heart Dividing Lines**
- **Septa**
  - **Interatrial septum:**
    – separates atria
  - **Interventricular septum:**
    – separates ventricles
Coronary Circulation

- Coronary arteries
  - Left and right
  - Originate at aortic sinuses
  - Elastic rebound forces blood through coronary arteries only between contractions
- Cardiac veins
  - Return blood to coronary sinus, opens into right atrium

Coronary Arteries

- Right Coronary Artery. Supplies blood to:
  - Right atrium, portions of both ventricles, cells of sinoatrial (SA) and atrioventricular (AV) nodes
  - Branches include:
    - Marginal arteries (surface of right ventricle)
    - Posterior interventricular artery
- Left Coronary Artery. Supplies blood to:
  - Left ventricle, left atrium, interventricular septum
  - Main branches:
    - Circumflex artery
    - Anterior interventricular artery

Arterial Anastomoses

- Interconnect anterior and posterior interventricular arteries
- Stabilize blood supply to cardiac muscle by providing collateral circulation
- E.g. RCA meets with the circumflex artery (which is a branch of the LCA)

2 Types of Cardiac Muscle Cells

- Contractile cells:
  - Account for 99% of heart tissue
  - Activated by change in the membrane potential (just like skeletal muscle cells)
  - Produce contractions, generate force
- Conducting system:
  - Initiate and distribute electrical activity
  - Consists of nodes and internodal pathways
  - Controls and coordinates heartbeat

Action Potentials in Skeletal and Cardiac Muscle
Cardiac activity – contractile cells

- Resting Potentials
  - Ventricular cells: -90 mV. Threshold = -75 mV
  - Atrial cells: -80 mV
- Signal to depolarize comes from:
  - Conducting system
  - Adjacent myocytes (via gap junctions)
- Threshold is usually reached in the latter manner in a portion of membrane near intercalated discs
- Then...

Cardiac Action Potential

1. Rapid depolarization:
   - voltage-regulated sodium channels (fast channels) open
2. Plateau:
   - At ~+30mV sodium channels close and inactivate, but no net loss of positive ions occurs because...
   - voltage-regulated calcium channels (slow channels) open and calcium ion entry roughly balances Na⁺ ion loss
   - Holds membrane at 0 mV plateau for ~175msec
3. Repolarization:
   - slow calcium channels close
   - slow potassium channels open
   - rapid repolarization restores resting potential

The Refractory Periods

- Absolute refractory period:
  - long
  - cardiac muscle cells cannot respond
- Relative refractory period:
  - short
  - response depends on degree of stimulus
- Length of cardiac action potential in ventricular cell is 250–300 msecs
  - 30 times longer than skeletal muscle fiber
  - long refractory period prevents summation and tetany

Calcium and Contraction

- Contraction of a cardiac muscle cell is produced by an increase in calcium ion concentration around myofibrils
  - 20% of calcium ions required for a contraction enter cell membrane through slow channels during plateau phase
  - This extracellular Ca²⁺ triggers release of calcium ion reserves from sarcoplasmic reticulum (80%)
- This is why heart is so sensitive to blood calcium

Pacemaker potentials

- At special site in the heart, cells have unstable resting potential
- These pacemaker cells depolarize spontaneously to initiate heartbeat = automaticity
- The SA and AV nodes have the greatest concentrations of autorhythmic cells
- Spontaneous electrical activity caused by special channels:
  - Na⁺ channels slowly open, allowing Na⁺ in
  - K⁺ channels close
  - Ca²⁺ channels open to start, sustain AP
- No fast Na⁺ channels at all!
The Cardiac Cycle

- Period from the start of one heartbeat to the start of the next
- Includes:
  - 370msec for heart contraction
  - A ~400msec delay
- Begins with action potential at SA node
- Transmitted through conducting system
- Produces action potentials in cardiac muscle cells (contractile cells) → force

The Heartbeat

- A single contraction of the heart
- Lasts about 370msec (cf. neurons?)
- The entire heart contracts in series:
  - first the atria
  - then the ventricles

The Conducting System

- A system of specialized cardiac muscle cells that initiates and distributes electrical impulses that stimulate contraction
- Cells display automaticity: contract automatically (without need for any external stimulation from nerves or other muscles)
- SA and AV nodes are the pacesetters

Sinoatrial (SA) Node

- In posterior wall of right atrium
- Contains pacemaker cells
- Connected to AV node by internodal pathways
- Begins atrial activation (Step 1)
Atrioventricular (AV) Node
- In floor of right atrium
- Receives impulse from SA node @ 50msec (Step 2)
- Takes ~100msec for impulse to travel through the AV node, delays impulse (Step 3)
- Atrial contraction begins (@150 msec)
- Delay limits maximum HR to ~230bpm

Pacemaker potential
- An unstable resting potential of conducting cells in SA and AV node that gradually depolarizes toward threshold
- SA node depolarizes faster (80-100 APs/min) than AV node (40-60 per minute) and so SA fires first, establishing heart rate (hence we call these the pacemaker cells)
- Why is your resting heart rate not 80 - 100bpm?

Conducting Cells
- Interconnect SA and AV nodes
- Distribute stimulus through myocardium
- In the atrium, called internodal pathways
- In the ventricles: AV bundle and bundle branches

The AV Bundle (bundle of His)
- Only electrical connection between A and V
- Travels in the septum
- Carries impulse to left and right bundle branches, which conduct to Purkinje fibers at 175 msec (Step 4), and to the moderator band, which conducts to papillary muscles

The Purkinje Fibers
- Distribute impulse through ventricles (Step 5) to contractile cells
- Trigger ventricular contraction to begin (@ 225msec) after atrial contraction is completed

Impulse Conduction through the Heart
Abnormal Pacemaker Function

- **Bradycardia:**
  - abnormally slow heart rate
- **Tachycardia:**
  - abnormally fast heart rate
- **Ectopic Pacemaker**
  - Abnormal cells generate high rate of action potentials
  - Bypass conducting system
  - Disrupt ventricular contractions

Heart Excitation Related to ECG

Electrocardiogram (ECG)

- Electrical events in the cardiac cycle can be recorded at the surface of the body using an electrocardiogram (ECG)
- Abnormal patterns diagnose cardiac arrhythmias, (abnormal patterns of cardiac electrical activity) due to damage or disease

Features of an ECG

- **P wave:**
  - SA node and atria depolarize (begin contraction ~25msec after P wave starts)
- **QRS complex:**
  - ventricles depolarize (begin contracting just after R peak)
- **T wave:**
  - ventricles repolarize

Time Intervals

- **P–R interval:**
  - from start of atrial depolarization to start of QRS complex
- **Q–T interval:**
  - from ventricular depolarization to ventricular repolarization
**EKG problems**

- Large QRS – caused by hypertrophy
- Small QRS – reduced heart muscle mass
- Small T – low energy reserves, ischemia
- Long P-R interval – damage to conducting pathways
- Long Q-T interval – conduction problems, myocardial damage, ischemia, congenital defect

**The Cardiac Cycle**

- The period between the start of one heartbeat and the beginning of the next
- Includes both contraction and relaxation
- Each chamber undergoes:
  - systole (contraction) → pressure rises
  - diastole (relaxation) → pressure falls
- **Blood flows from high to low pressure,** controlled by **timing** of contractions and directed by one-way valves

**Phases of the Cardiac Cycle**

1. Atrial systole
2. Atrial diastole
3. Ventricular systole
4. Ventricular diastole

**Cardiac Cycle and Heart Rate**

- At 75 beats per minute, each cardiac cycle lasts about 800 msecs
- When heart rate increases **all phases** of cardiac cycle shorten, but particularly ventricular diastole (less time spent resting)
8 Steps in the Cardiac Cycle

Begin: all relaxed, ventricles 70% filled

1. Atrial systole begins
   - atrial contraction begins
   - rising pressure forces open right and left AV valves
   - no venous flow into atria but little backflow either

2. Atria eject blood into ventricles:
   - filling ventricles (topping them off)

3. Atrial systole ends (100msec):
   - ventricles contain maximum volume = end-diastolic volume (EDV) which is normally about 130ml

4. Ventricular systole begins:
   - AV valves close as $P_{vent}$ quickly exceeds $P_{atria}$
   - pressure in ventricles continues to rise
   - All valves closed: isovolumetric ventricular contraction

5. Ventricular ejection:
   - When pressure in ventricles exceeds arterial pressure in great vessels, semilunar valves forced open
   - blood flows into pulmonary and aortic trunks (isotonic contraction)
   - Stroke volume (SV) = 80ml. Percent of end-diastolic volume that is ejected = ejection fraction around 80%

6. Ventricular pressure falls near end of systole:
   - backflow from vessel trunks closes semilunar valves
   - ventricles contain end-systolic volume (ESV), about 40% of end-diastolic volume or 50ml
   - Aortic elastic recoil causes dicrotic notch (double beat). ESV = 50ml or 40%

7. Ventricular diastole (starts at 370msec):
   - ventricular pressure is still higher than atrial pressure
   - all heart valves are closed
   - ventricles relax (isovolumetric relaxation)
   - Lasts for remaining 430msec of cycle plus 100msec of the next cycle

8. Falling ventricular pressure drops below atrial pressure:
   - forces AV valves open
   - passive atrial filling (continuous during atrial diastole)
   - passive ventricular filling (to 70% full)
   - cardiac cycle ends
   - Repeat

Movie

- Cardiac cycle
- Note that:
  - AV valves close early (step 3), open late (step 8)
  - SL valves open and close in between (step 5 open, step 6 closed)
Heart Failure

- Lack of adequate blood flow to peripheral tissues and organs due to inadequate cardiovascular output (usually due to ventricular damage)
- Atrial damage can be inconsequential, or problematic depending on what is damaged.

Auscultation - Heart Sounds

- “lub-dup”
- S₁: loud sounds produced by AV valves closing, signifies beginning of systole
- S₂: loud sounds produced by semilunar valves closing at the beginning of ventricular diastole
- Sounds are actually produced by blood changing flow patterns

Aerobic Energy of Heart

- From mitochondrial breakdown of fatty acids and glucose (lots of mitochondria in cardiac myocytes)
- Oxygen from circulating hemoglobin
- Cardiac muscles store oxygen in myoglobin

Important Cardiodynamics Terms

- **End-diastolic volume (EDV)**
  - Max filling after atrial systole (usually about 130ml)
- **End-systolic volume (ESV)**
  - Residual volume after ventricular systole (usually about 50ml)
- **Stroke volume (SV)**: \( SV = EDV - ESV \)
  - Volume (ml) of blood ejected per beat

Stroke Volume
Important Cardiodynamics Terms

- **Ejection fraction:**
  - the percentage of EDV represented by SV
- **Cardiac output (CO):**
  - the volume pumped by each ventricle in 1 minute (equals how much blood gets to the tissues every minute)
  - \( CO = HR \text{ (in bpm)} \times SV \)
- What is CO for a HR of 80 bpm and a SV of 80 ml/beat?

Adjusting CO to Conditions

- **Cardiac output:**
  - Can be adjusted by changes in heart rate or stroke volume
- Stroke volume can be increased 2x:
  - adjusted by changing EDV or ESV
- Heart rate can be increased 2.5:
  - adjusted by autonomic nervous system or hormones (How?)

Factors Affecting Stroke Volume

- Changes in **EDV or ESV**
  - Slow heartbeat and exercise increase venous return to the heart, increasing SV
  - Blood loss and extremely rapid heartbeat decrease SV
- EDV affected by:
  - filling time (duration of ventricular diastole)
  - rate of venous return (rate of blood flow during ventricular diastole)
- ESV affected by:
  - Preload: degree of ventricular stretching during diastole due to amount of blood (increased by greater venous return)
  - Contractility: force produced during contraction, at a given preload
  - Afterload: tension the ventricle must produce to open the semilunar valve and eject blood to the great vessels

Preload and Afterload

- At rest:
  - EDV is low
  - myocardium stretches little (low preload)
  - stroke volume is low, ESV is high (lots left)
- With exercise:
  - EDV increases (increased venous return)
  - myocardium stretches more
  - stroke volume increases, ESV decreases

EDV and Stroke Volume
The Frank–Starling Principle

- Preload, or degree of stretch, of cardiac muscle cells before they contract is the critical factor controlling stroke volume
- As EDV increases, stroke volume increases (and vice versa)
- Basically, if there is a lot of blood in the ventricles (increased venous return), stretching causes them to pump more blood out. More in → more out
- Keeps the two sides of the heart in balance

ESV and Stroke Volume

- Contractility is affected by:
  - Sympathetic activation
    - NE released by postganglionic fibers of cardiac nerves
    - E and NE released by adrenal medullae
    - Cause ventricles to contract with more force
    - Increases ejection fraction and decreases ESV
  - Parasympathetic activity
    - Acetylcholine released by vagus nerves reduces force of cardiac contractions
  - Hormones
  - Drugs mimic hormone actions:
    - Stimulate or block beta 1 receptors (beta-blockers)
    - Affect calcium ions e.g., calcium channel blockers (negative inotropic effects) decrease contractility

ESV and Stroke Volume

- Afterload
  - As afterload increases, takes longer before SL valves open and thus less blood will be ejected: ESV increases and stroke volume decreases
  - Increased by any factor that restricts arterial blood flow (like atherosclerosis)
  - Extremely high afterload can cause heart failure

Heart Rate Control Factors

1. Autonomic nervous system: sympathetic and parasympathetic
   - Note: we just saw how the autonomic NS can affect contractility, which affects stroke volume
2. Circulating hormones (thyroxine, E and NE in blood all increase HR)
3. Venous return and stretch receptors

Autonomic NS Innervation

- Heart is stimulated by the sympathetic cardioacceleratory center
- Heart is inhibited by the parasympathetic cardioinhibitory center

Autonomic Innervation

- Vagus nerves (X) carry parasympathetic preganglionic fibers to small ganglia in cardiac plexus → SA and AV nodes
- Sympathetic postganglionic fibers → cardiac plexus → SA and AV nodes
- Autonomic tone:
  - Dual innervation maintains resting tone by releasing ACh and NE (which dominates?)
  - Fine adjustments meet needs of other systems
Cardiac reflexes

- Cardiac centers in medulla monitor:
  - baroreceptors (blood pressure)
  - chemoreceptors (arterial oxygen and carbon dioxide levels)
- Cardiac centers adjust cardiac activity via sympathetic (NE increases HR) and parasympathetic (ACh decreases HR) activity

Atrial (Bainbridge) Reflex

- Sympathetic reflex initiated in response to increased venous return (remember that increased venous return also caused increased stroke volume, called?)
- Stretch receptors in right atrium trigger increase in heart rate through increased sympathetic activity to "pump out the excess" blood and decrease venous pressure

Autonomic NS Regulation of Pacemaker Cells in SA, AV Nodes

- Membrane potential of pacemaker cells is less negative than other cardiac cells (-60mv)
- Sympathetic and parasympathetic stimulation are both greatest at SA node (heart rate)
- Rate of spontaneous depolarization depends on:
  - resting membrane potential (where you start)
  - ACh (para NS) opens K+ channels. Result?
  - NE binds to Beta adrenergic receptors, opens sodium-calcium ion channels. Result?

KEY CONCEPT

- Cardiac output:
  - the amount of blood pumped by the left ventricle each minute
  - adjusted by the ANS in response to:
    - circulating hormones
    - changes in blood volume
    - alterations in venous return
- Most healthy people can increase cardiac output by 300–500% = cardiac reserve

Summary

- Heart anatomy
- Cardiac muscle cells
- Heart chambers, valves and vessels
- Conducting system
- EKG
- Cardiac cycle
- Contractile and pacemaker cells
- Cardiodynamics
### Heart Failure
- Inadequate cardiac output to meet demand
- Due to left ventricular insufficiency
- If LV not pumping enough, blood backs up in the pulmonary circulation. Elevated pressures cause increased loss of plasma to interstitial fluid of lungs = CHF (congestive heart failure). If severe, can cause pulmonary edema (fluid fills air spaces)
- Treatment? Digoxin – positive inotrope to increase contraction force. Blood pressure reducers: diuretics, vasodilators, spironolactone (ald antagonist)

### Cardiomyopathy
- Cardiac muscle degeneration and fibrosis
- Can lead to heart failure
- Hypertrophic cardiomyopathy; thickened left ventricular walls

### Heart Block
- Damage to conduction pathways
- Often show abnormal ECG
- Escape
  - when conducting pathways damaged, ventricles can “escape” control of SA or AV node usually by autorhthymicity of Purkinje fibers. Ventricles continue to beat but only at 40-50bpm (which the intrinsic rate of the Purkinje fibers)

### Tachycardias
- Atrial fibrillation:
  - atrial depolarization occurs so fast that the atrium appears to quiver. Ventricles cannot follow this rate
- Not usually life threatening because of escape
- Ventricular arrhythmias
  - Ventricular tachycardia (V-tach)
  - Ventricular fibrillation (V-fib) = cardiac arrest
    - No rhythm. Defibrillator can restore by unifying cell activity

### Cardiac technologies
- Pacemakers: treat chronic bradycardia due to conduction deficits, etc.
- External defibrillators (clear!): can rescue from cardiac arrest
- Implantable defibrillators: for people with chronic heart conditions (often congenital)
- LVAD: Left ventricular assist device
  - Implantable pump in parallel with the left ventricle for those with LV insufficiency
  - Boosts cardiac output by allowing some blood to bypass the left ventricle and enter the aorta at sufficient pressure

### Examining the heart
- Coronary angiogram: catheter is threaded up through femoral artery into aortic sinus. Dye released that can be seen on a series of high speed X-rays
- Echocardiogram: heart ultrasound
Ionic imbalances

• Potassium
  – Hyperkalemia – decreases K+ gradient, inhibits repolarization
  – Hypokalemia – increases K+ gradient, causes hyperpolarization of resting membranes

• Calcium
  – Hypercalcemia: cardiac muscle cells become very excitable
  – Hypocalcemia

Coronary artery disease

• Can cause coronary ischemia (reduced blood flow) to heart
• Atherosclerosis: plaques caused by fatty deposits in vessels cause narrowing of artery
• Angina pectoris: pain associated with heart ischemia
  • Treatments:
    – Vasodilators (nitroglycerin)
    – Beta blockers (propanolol)
    – Calcium channel blockers
    – Balloon angioplasty with stents—recently shown to be no better at preventing death from a second heart attack than drugs alone
    – Coronary artery bypass – leg vein grafts

MIs

• Myocardial infarction – blockage in coronary circulation causes cells to die
• Most due to CAD, usually thromboses
• Blood tests for markers of anaerobic metabolism, like lactate dehydrogenase, can indicate a heart attack
• About 25% die before receiving any medical attention
• About 50% die within the first year
• Women have fewer MIs but their mortality rate is higher. Recent evidence indicates that men and women have different kinds of CAD