Heart Physiology
Electrical Events of the Heart
Intrinsic Conduction System

• It is a function of
  – gap junctions
  – non-contractile cardiac muscle cells
    (conduction system)

• Produces a coordinated contraction of heart
  for efficient blood movement.
Cardiac Muscle Fibers (cells)

- Striated and sliding filament mechanism of contraction
- Each fiber covered with endomysium
- Endomysium is highly vascular
- Endomysium is woven by collagen fibers into the fibrous skeleton of heart
Cardiac Muscle Fibers (cells)

- Intercalated discs provide site of desmosomes and gap junctions
- Heart is a functional syncytium
- Abundant mitochondria
- Highly branched
- System of Ca++ delivery is less extensive; no triads (only one T tube per sarcomere) and no terminal cisternae (lateral sacs)
Heartbeat – Cardiac Cycle

- Events involved in the sequential excitation and contraction of a single beat
- Involves two types of cells
  - Contractile cells
  - Conduction cells (pathway)
- Regulation of heartbeat is ANS
Action Potentials of Pacemaker Cells (Conduction Cells)

- Autorhythmic cells
  - resting potential is unstable. It continually depolarizes
  - unstable resting potential is called a pacemaker potential
  - interior of cell becomes more positive until cell reaches threshold and then complete depolarization is reached
Excitation Sequence

- Conduction sequence is
  - sinoatrial (SA) node
  - atrioventricular (AV) node
  - atrioventricular (AV) bundle (bundle of His)
  - right and left bundle branches
  - Purkinje fibers
Sinoatrial (SA) node

- Located just inferior to opening of superior vena cava
- Depolarizes to threshold at about 75 times/minute (without neural inhibition rate is about 100 times/minute)
- Fastest of all autorhythmic areas; thus, sets pace of heart
- Referred to as pacemaker of the heart and determines what is called “sinus rhythm”
Atrioventricular (AV) Node

• Located in inferior portion of interatrial septum
• Produces a slight delay (0.1 sec) which allows completion of atrial contraction; thus, filling of ventricles before their contraction.
Atrioventricular bundle (bundle of His)

- Only electrical connection between atria and ventricles; thus, serves as an atrial-ventricular pathway
Bundle branches (right and left)

- Branches from atrioventricular bundle which lead to corresponding sides of the heart
Purkinje Fibers

- Final conduction pathway into ventricular myocardium.
- Purkinje fibers and gap junctions ensure complete excitation of ventricular myocardial cells.
- Ventricular contraction is described as a “wringing” contraction which ejects some (not all) of ventricular blood.
Defects in Conduction

• Arrhythmias are irregular heart rhythms which may result in events such as uncoordinated atrial and ventricular contractions

• Fibrillation is rapid and irregular or out-of-phase contractions. Fibrillation of ventricles would produce little output.
Defects in Conduction

• Ectopic focus (or abnormal pacemaker)
  – might be due to defective SA node. If AV node assumed role rate would drop to about 40 - 60 beats/minute
  – may be due to hyperexcitable tissue (too much caffeine or nicotine. Result is premature contraction (PVCs)

• Heart block may result due to damage to AV node. Total heart block results when there is lack of conduction to ventricles Ventricular pacemaking is too slow to sustain life. Fixed rate pacemakers may be implanted
Extrinsic Innervation of Heart

• Intrinsic rate is modified by nervous system
  – parasympathetic slows (resting-digesting)
  – sympathetic increases rate and force of contraction (fight-or-flight)

• Stimuli for modification of heart rate
  – Cortical influences - hypothalamus
  – Hypothalamus (ANS)
  – Hormones (NE, E)
  – Baroreceptors (aortic arch, carotid)
  – Chemoreceptors (aortic arch, carotid)
Cardiac Centers

• Cardiac centers located in medulla oblongata
  – Cardioacceleratory center (sympathetic) exits spinal cord at T₁ - T₅, (synapses with postganglionic neurons and runs through cardiac plexus to heart. Targets SA, AV, and ventricular myocardium)
  – Cardiocinhibitory center (parasympathetic) innervates heart by branches of vagus nerve. Mostly targets SA and AV nodes
Heart’s Autonomic Innervation

Cardioacceleratory; sympathetic by way of sympathetic cardiac nerve (increases rate and force of contraction)

Cardioinhibitory; parasympathetic by way of vagus nerve (slows rate of contraction)

Medulla oblongata
Electrocardiography

- Electrocardiogram is a recording of the electrical activity of the heart
- Three distinctive waves (deflection waves) are the P, QRS complex, and T
P wave (SA node)

P wave begins with firing of SA node and starts the contraction (systole) of the atria.
P wave (atrial depolarization)

P wave continues with depolarization of both atria and results in their contraction (atrial systole) which moves blood into the ventricles.

Atrial pressure is greater than ventricular pressure so AV valves are open.
QRS Complex

QRS begins (the Q wave) with the depolarization of the AV node, AV bundle (bundle of His), bundle branches (right and left), and interventricular septum.
QRS Complex

QRS continues (the RS waves) with the depolarization of the ventricular myocardium. Contraction (systole) which “begins” at the apex of the ventricles pushes some blood upward and out into the pulmonary trunk and aorta.
QRS Complex (con’t)

When ventricular pressure is greater than atrial pressure the AV valves close. This produces first heart sound, lub. When ventricular pressure exceeds vascular pressure semilunar valves open.
T wave is produced by ventricular repolarization and represents the beginning of ventricular relaxation (diastole). Ventricular pressure drops below vascular pressure and the semilunar valves close. This produces the second heart sound.
Cardiac Cycle

- Systole refers to the physical contraction of the heart
- Diastole refers to the physical relaxation of the heart
- Cardiac cycle is one heart beat and represents
  - atrial systole and diastole
  - ventricular systole and diastole
Cardiac Cycle

- At the beginning of a cardiac cycle the heart
  - was in atrial and ventricular diastole (quiescent period of about 0.4 sec)
  - 70% of atrial filling (and ventricular filling) is passive and occurs during the interval from the previous T wave to the subsequent P wave
  - AV valves are open and semilunar valves are closed
Atrial Systole

- Atrial systole is contraction of the atria (0.1 sec)
  - provides final 30% push for ventricular filling
  - AV valves are open and semilunar valves are closed
Ventricular Systole

- Period of isovolumetric contraction
- AV valves close when ventricular pressure exceeds atrial pressure
  - first heart sound, lub, is produced
- Semilunar valves are still closed as ventricular pressure has not exceeded pulmonary trunk and aorta pressures
Ventricular Systole

- Ejection phase occurs when ventricular pressure exceeds pulmonary trunk and aorta pressures (aortic back pressure pressure would be about 80mm Hg. - this back pressure is called afterload)
- Pressure in aorta (systolic) pressure would reach about 120mm Hg.
- Ventricular systole = 0.3 sec
Ventricular Diastole

- Ventricular relaxation results in drop of pressure to “O” mm Hg.
- Semilunar valves closed when blood started to backflow toward ventricles – second heart sound, dup, produced
- Heart is now in quiescent period until next cycle (which lasts for about 0.4 s)
Heart Sounds

- Auscultation refers to “listening”
- Sounds associated with closure of valves
  - #1 aortic semilunar valve best heard at 2nd intercostal space at right sternal margin
  - #2 pulmonary semilunar valve best heard at 2nd intercostal space at left sternal margin
  - #3 tricuspid best heard at 5th intercostal space at right sternal margin
  - #4 mitral (bicuspid) best heard at 5th intercostal at left mid-clavicular line
Cardiac Output

- Cardiac output is output of each ventricle in 1 minute (both ventricles pump the same volume)
- Calculated by the product of heart rate (HR) and stroke volume (SV)
- \( CO = HR \times SV \)
  - \( CO = 75 \times 70 = 5250 \text{ ml/min} \) or 5.25 liters/minute
  - a person’s total blood volume passes through the heart in one minute (assuming the normal blood volume is 5 liters)
- Changed by adjusting rate and/or volume
Stroke Volume

- End-diastolic volume amount of blood in ventricle at the end of its diastole
- End-systolic volume amount of blood remaining in ventricle after systole
- Stroke volume is determined by difference between end-diastolic volume (EDV) and end-systolic volume (ESV)
  - \( SV = EDV \ (120\text{ml}) - ESV \ (50\text{ml}) \)
  - \( SV = 70 \text{ ml/beat} \)
Changes in Stroke Volume

• Three important factors which affect stroke volume are:

1. Preload; the amount the ventricles are stretched by the blood they contain

2. Contractility; the force produced by cardiac muscle fibers

3. Afterload; back pressure in the vessels which leave the heart (pulmonary trunk and aorta)
Preload; Frank-Starling Law

- The more cardiac muscle fibers are stretched, within limits, the greater the force of contraction.
  - Cardiac muscle fibers are shorter than their optimal length
  - Thus, stretching allows more interaction between the number of active cross-bridge interactions
Changes in Preload

• Most important factors influencing preload is amount of blood returning to heart – or venous return (increases EDV)

• Increases may be caused by
  – Exercise increases rate and promotes the action of the skeletal muscle pump. Slow heart rate allows more time for filling of ventricles

• Decreases may be cause by
  – Blood loss which reduces blood volume. High heart rate does not allow ventricles time to fill with blood

• Cardiac output by each ventricle is balanced. If right pump receives more blood, then left heart will accommodate for the change.
Contractility

- Independent of EDV and muscle stretch
- Due mostly to influx of extracellular calcium and the ER (SR)
- Sympathetic stimulation involves not only intrinsic conduction system but the myocardium, especially the ventricular myocardium.
  - Epinephrine and norepinephrine promote the up-take of calcium into cardiac cells (as do glucagon, thyroxine, and drugs such as digitalis). Calcium promotes cross-bridge binding by removal of regulator proteins
  - Negative agents include calcium channel blockers (verapamil), excessive potassium, and acidosis.
- Result is lower ESV; thus, greater SV
Afterload – “Back-Pressure”

• Afterload is the pressure the ventricles must overcome inorder to eject blood; thus, the pressure of the pulmonary trunk and the aorta.
• Increased afterload means that ESV is increased; thus, SV is reduced.
• Hypertension is a major contributor to increased afterload.
Heart Rate (HR)

- Heart rate is influenced mostly by hormones and CNS
- Sympathetic releases epinephrine and norepinephrine which causes threshold to be reached quicker; thus, increasing rate. (Also, increases contractility by increasing calcium ion entry.)
- Parasympathetic causes decrease in rate by opening potassium channels and causing hyperpolarization. It little effect on contractility.
- Heart exhibits vagal tone, which means that the most dominant control is parasympathetic. Loss of vagal stimulation will increase rate by about 25 beats/minute (SA node intrinsic rate of 100 beats/minute.)
Heart Rate (HR)- Cardiovascular Center

• Cardiovascular center is located in the medulla oblongata. Through the action of the sympathetic and parasympathetic autonomic systems, it adjusts blood pressure by controlling cardiac output and blood vessel diameter.

• Major sensory monitors include
  – Baroreceptors (pressure receptors) which respond to changes in arterial blood pressure and stretch
  – Chemoreceptors which respond to changes in carbon dioxide, oxygen, and pH
  – Higher brain centers such as the hypothalamus
Chemical Regulation of HR

- Epinephrine from adrenal medulla increases heart rate and force of contraction.
- Hypocalcemia (reduced calcium) reduces heart activity
- Hypercalcemia (increased calcium) couples excitation-contraction and leads to prolonged action potential and increase heart irritability (spastic contractions)
- Hyperkalemia (excessive potassium) lowers resting membrane potential and may lead to heart block and arrest
- Hypokalemia (decreased potassium) increases resting membrane potential and decreased beat and causes abnormal rhythms
Heart Rates

- Bradycardia – heart beats less than 60 beats/minute
- Tachycardia – heart beats more than 100 beats/minute
Congestive Heart Failure (CHF)

- Heart is not able to maintain adequate blood flow to tissues of body.
  - Coronary atherosclerosis; clogging of coronary arteries. Heart becomes hypoxic
  - Hypertension; 90 mm Hg. or more as afterload. Heart undergoes hypertrophy and cardiac muscle becomes weakened.
  - Myocardial infarcts; normal muscle is replace by non-functional tissue
Pulmonary Congestion

• Left heart failure results in pulmonary congestion
  – Right side pumps normally
  – Left side has failed
  – Result is increased blood pressure in vessels of lungs. Edema results and person can suffocate.
Peripheral Congestion

• Results when right side of heart fails
• Blood pools, edema occurs; especially in extremities.
• Cardiac flow to pulmonary circuit is decreased as well as output to tissues (from left side of heart)