

Cardiac Physiology – Part 2
Contraction & Electrical Conductivity
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Differences between skeletal muscle and cardiac muscle contraction

- Means of Contraction
 - Skeletal muscle fibers are stimulated by contact with a motor neuron
 - Cardiac cells are stimulated by nerves and are self-excitability. Automaticity
- Organ vs. Motor Unit Contraction
 - Skeletal muscle has all the cells of a specific motor unit are stimulated and contract at the same time. Impulses do not spread from cell to cell. Skeletal muscle can perform recruitment.
 - The heart follows the all-or-nothing principle. It either contracts as a unit or it does not contract at all.
- Length of Resting (Refractory) Period
 - In skeletal muscle, the resting period is only 1-2 ms and the contractions are 15-20 times longer.
 - In cardiac muscle, the resting period is about the same as the contraction period. The long resting period prevents tetanic contractions, which would stop the beating heart.

Starling's Law of the Heart

- The Frank-Starling Law states that the stroke volume of the left ventricle will increase as the left ventricular volume increases due to the myocyte stretch causing a more forceful systolic contraction.
- The Frank-Starling law states that the more the ventricular muscle cells are stretched, the more forcefully they contract.
- In a healthy individual, an overloading of blood in the ventricle triggers an increase in muscle contraction, to raise the cardiac output.

Electrical Activity of the Heart - Excitation of the Heart

- Pacemaker cells
 - The cells with the fastest intrinsic rates of activity = automaticity
 - The heart is myogenic, which means that it can generate its own impulse and does not need nerves to work.
 - Note: skeletal muscle is neurogenic because it needs nerves to work.

Myogenic Nerve Tissue

- Includes cardiac and visceral muscle
- Exhibits intrinsic activity = automaticity
- Spontaneous increase of sodium >>>
Spontaneous depolarization (called pacemaker potential) >>>
Action potential >>> CONTRACTION !!!
- Not all heart cells generate an action potential at the same speed. Some generate AP (action potentials) at a very fast speed (pacemaker cells) and some generate an AP (action potential) at a much slower speed.

Myogenic Nerve Tissue - continued

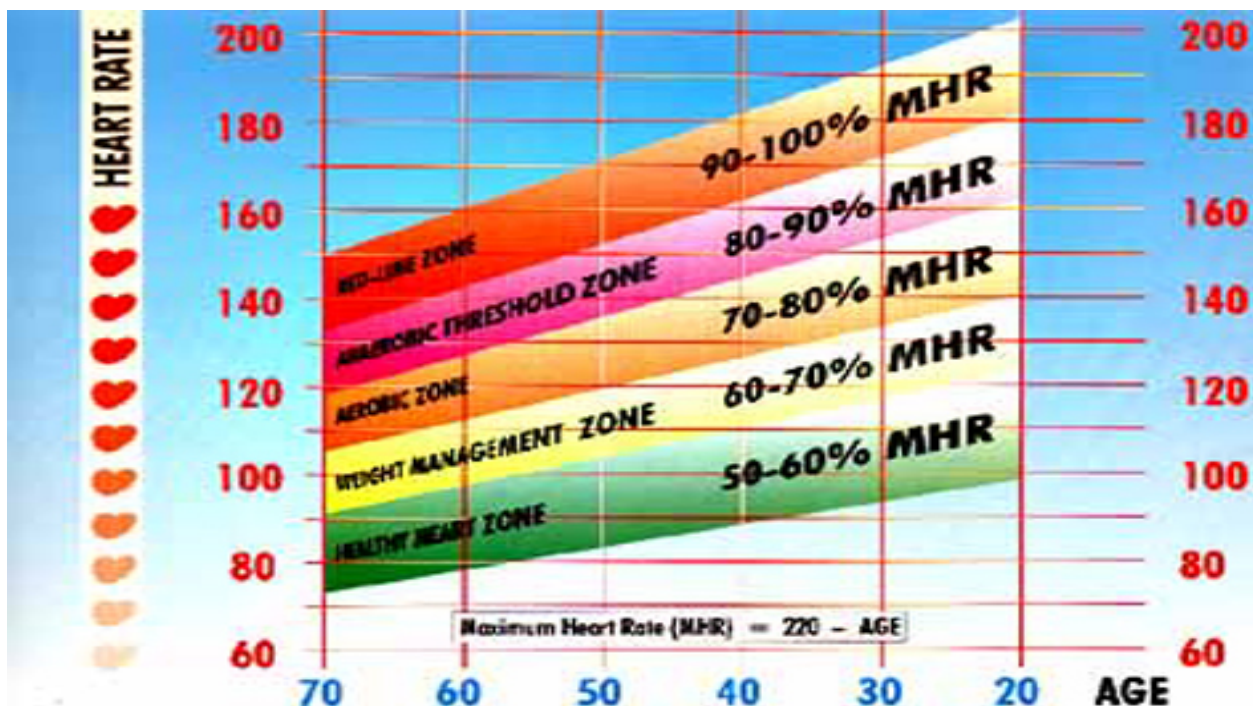
- Myogenic muscle tissue includes visceral and cardiac muscle.
 - Exhibits intrinsic activity = automaticity = myogenic

Electrical Activity of the Heart - Excitation of the Heart

- Cardiac muscle cells are electrically joined together by gap junctions.
 - This enables electricity to travel faster.
- AP (action potentials) generated by the pacemaker cells normally spread throughout the heart before the other cells have a chance to generate an action potential.
- The heart functions as a single unit.
- Spontaneous increase of the permeability of sodium >>>>>>
spontaneous depolarization (called pacemaker potential) >>>>>>
Action Potential >>>>>> contraction

Electrical Refractory (resting) Period of the Heart

- Minimal recovery period following an action potential
 - In the non-pacemaker cells is about .3 sec = 300 / millisecond
 - This is due to the sustained action potential (period of reversed polarity) in most cardiac cells.
- The heart can only “twitch”. It cannot exhibit a sustained (tetanic) contraction.
 - By the time the heart can generate a second action potential, it has almost fully relaxed.
- Theoretical Maximum Heart Rate = 200 BPM



Electrical Conduction System of the Heart

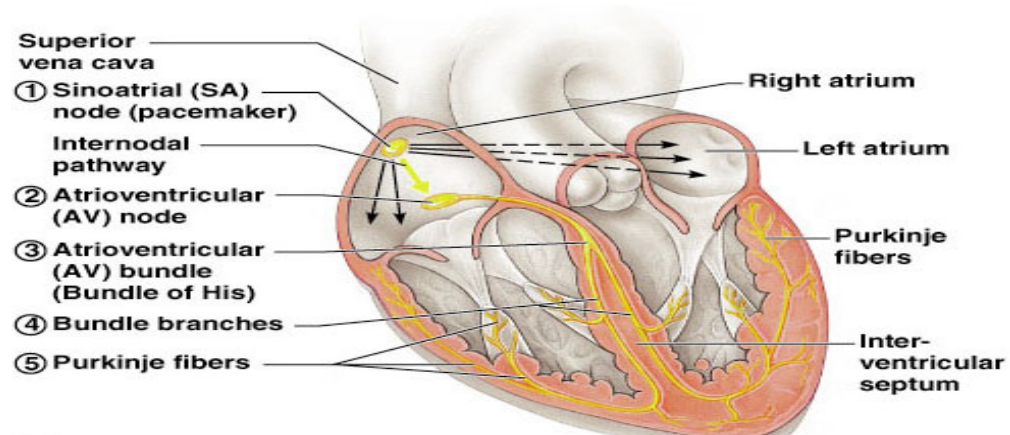
- Consists of specialized myocardial tissue that acts to generate and conduct action potentials rapidly through the heart.
- Sino-Atrial (SA) Node
 - Located in the wall of the right superior atrium, near the entrance of the SVC.
 - Functions as the normal heart pacemaker.
 - Innervated by autonomic motor neurons that influence the rate at which action potentials spontaneously generate.
- Atrio-Ventricular (AV) Node
 - Located in the lower part of the interatrial septum of the right atrium.
 - Functions as the only link between the atria and the ventricles.
 - The AV node is like an electrical “bottle-neck”.
 - AP (action potentials) are transmitted through the AV node at a slow velocity.
 - Myocardial ischemia in the AV node results in AV Nodal Block.
 - Also innervated by autonomic motor neurons that influences the velocity of the action potential through the AV node.
 - If the SA node stops functioning, the AV node becomes the pacemaker of the heart.
- The skeletal muscle has a sarcoplasmic reticulum and lateral sacs (called terminal cisterns) and these sacs store Calcium for use latter.
- Heart muscle does not store Calcium and that is why heart muscle cells need Calcium to flow in to cause a contraction
- Calcium Channel Blockers are medications given to control angina. They slow the heart rate down by decreasing the influx of Calcium into the cells.
 - Also called calcium antagonists, relax and widen blood vessels by affecting the muscle cells in the arterial walls.

Common Atrio-Ventricular (AV) Bundle (of His)

- Extends down the interventricular septum branching into the right and left bundle branches.
- Functions to conduct AP (action potentials) rapidly from the AV node down to the right and left ventricles.
- Myocardial ischemia in a bundle branch results in “Bundle Branch Block”.

Purkinje Fibers

- Branches off the right and left bundle branches
- Functions to conduct the AP (action potentials) through the ventricular myocardium.



Neural Innervation of the Heart

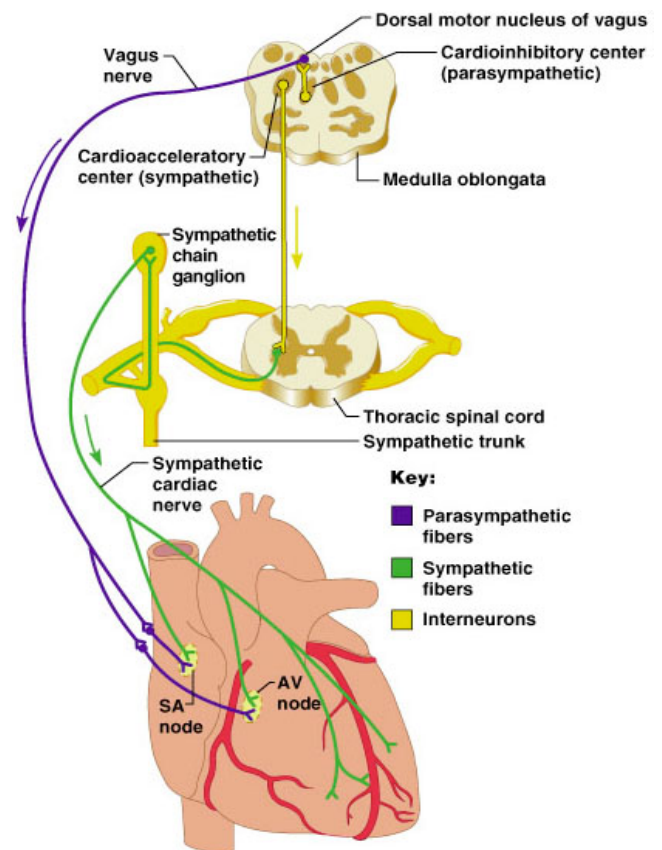
- Autonomic motor neurons
 - Innervates the SA node and AV node
 - Parasympathetic innervation
 - Via the Vagus nerve (X)
 - Causes bradycardia (< 60 BPM) during normal relaxed states
 - Also slows transmission of Aps through the AV node.
 - Mechanism of action:
 - ACh >>>>> Increases permeability of potassium >>>>> potassium rushes OUT of the cell >>>>> hyperpolarization >>>>> slows the rate of intrinsic activity

Extrinsic Innervation of the Heart

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

Sympathetic innervation

- Stimulates 3 changes during states of stress
 - Tachycardia (> 100 BPM)
 - Increased velocity of AP transmission through the AV node >>>>>
 - Increased myocardial contractility = increased force of contraction
 - Mechanism of action:
 - Catecholamines >>>>> increased permeability of sodium >>>>> sodium rushes INTO cells >>>>> depolarization >>>>> increased rate of activity



The Electrocardiogram (EKG)

- The 1924 Nobel Prize in Physiology and Medicine was awarded to William Einthoven (Holland) for first using a galvanometer to record the change in electrical activity of the heart.

- Clinical uses of EKG

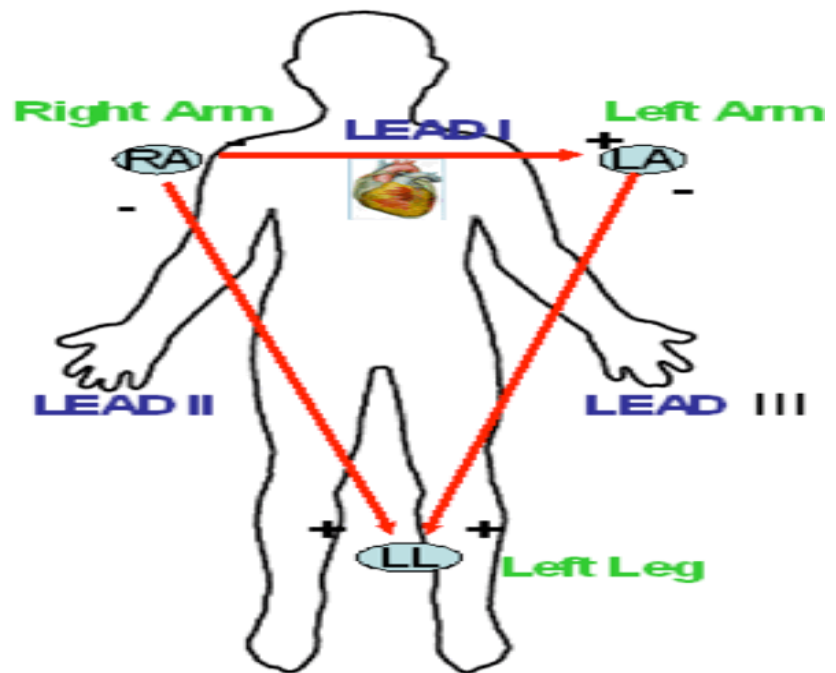
- Used to diagnosis MI and CAD
- Used to identify cardiac arrhythmias
- Used to identify cardiomegaly
- Used to identify electrolyte abnormalities (especially calcium & potassium)

- Surface electrodes are used to record the direction of electrical current (AP) through the heart.

- Bipolar Limb Leads

- Records the differences in voltages between two limbs
- Einthoven's Triangle
 - Lead 1 – measures electrical activity from right arm to left arm
 - Lead 2 – measures electrical activity from right arm to left leg. THE MOST IMPORTANT LEAD. This gives the information from the electrical flow from the SA node to the AV node.

- Lead 3 – measures the electrical activity between the left arm and the left leg



The Electrocardiogram (EKG)

- Unipolar (Augmented)

- 9 more leads that are used
- Records the difference in voltage between one limb (unipolar) and a ground electrode.
- Unipolar leads show larger (augmented) deflections than bipolar leads.
- Examples:
 - AVR – electrodes on right arm
 - AVL – electrode on left arm
 - AVF – electrode on left leg

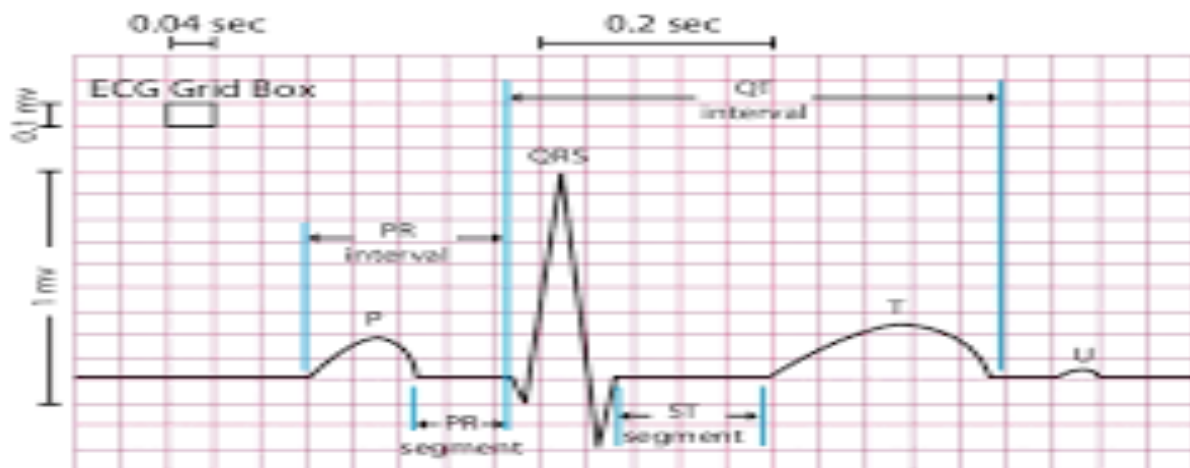
- Unipolar Chest (Precordial) Leads

- V1 – V6
- Records the difference in voltage between one state on the chest and a ground electrode.

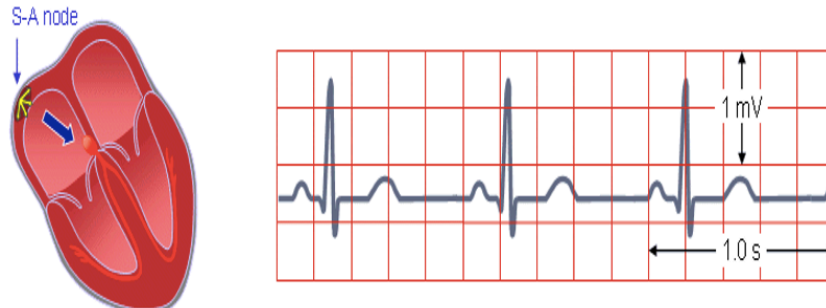
EKG Waves Related to Heart Anatomy

- **NSR = Normal Sinus Rhythm**
 - Every P, Q, R, S, T Cycle = 1 Heartbeat
- **P - Wave**
 - Corresponds to electrical depolarization
 - Enlarged P – wave indicates an enlarged atria
 - Example – mitral stenosis
- **P – R Interval**
 - Period of time between the start of the P – wave to the start of the Q – wave.
 - This is measuring the travel time from the SA Node to the AV node.
 - Cardiologist will examine this to see if it is too short or too long.
 - P – R Intervals longer than .2 sec indicated some degree of AV Nodal Block.
 - Will need a pacemaker
- **QRS Complex**
 - This is showing the depolarization of the ventricle muscle
 - This corresponds to ventricular muscle depolarization
 - It is examined to see how long or short it is
 - Enlarged R – wave means enlargement of the ventricles
- **ST Segment**
 - This is the refractory or resting time (300 msec)
- **T – Wave**
 - Corresponds to ventricular repolarization of the heart muscle cells
 - If it doesn't look normal, it is interpreted as the ventricles not normally repolarizing
 - A flat T – wave indicates ventricular myocardial ischemia
 - An inverted T – wave indicates Myocardial Infarction
 - A peaked T – wave indicates hyperkalemia (increased potassium)

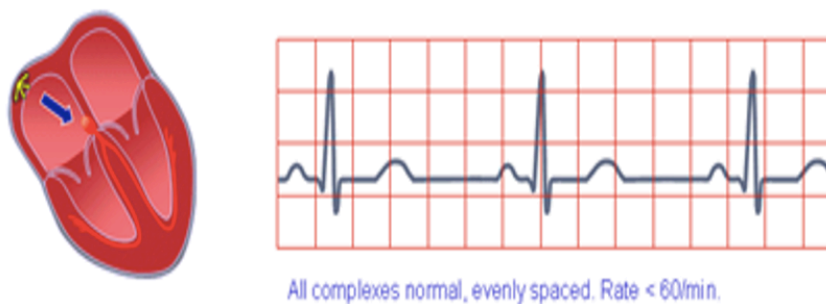
EKG INTERVALS



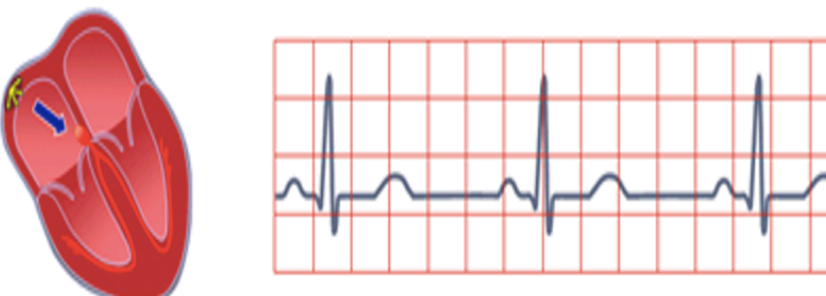
Normal Sinus Rhythm – Rate 60 to 100 bpm



Tachycardia – Rate > 100 bpm



Bradycardia - < 60 bpm



Cardiac Arrhythmias

- Premature atrial contractions (PAC)
 - Seen in normal people with too much caffeine, anxiety, alcohol, electrolytes, vomiting or diarrhea
- Premature ventricular contractions (PVC)
 - Often seen in middle age and of no concern
 - Can also occur with MI, CHF, hypoxia
- Paroxysmal atrial tachycardia (PAT)
 - Usually in women 20-25
 - May be congenital and start in first year of life
 - Called Wolf Parkinson White Syndrome which requires a catheter radio-ablation

Cardiac Arrhythmias - continued

- Atrial fibrillation and flutter (AF)
 - Not life-threatening, yet it is a common cause of hospitalization
 - Causes the heart to be sporadically with no rhythmic pattern
 - May be caused by COPD, alcohol, cardiac surgery, hyperthyroidism, or idiopathic
 - The inefficiency of the atrial contraction leads to a potential buildup of clots in the wall of the atria
 - Treated with electrical cardioversion with anticoagulation meds
 - Some are resistant to cardioversion which puts them at risk for strokes, ventricular fibrillation and sudden death
- Ventricular tachycardia (VT)
 - A normal response to exercise, stress reactions and sexual activity causing the heart to elevate up to 200/minute
 - In patient's with structural heart disease, VT can occur without provocation
 - S & S – pounding heart and lightheadedness
 - Treated with electrical cardioversion and beta blockers

Bradycardia

- Pulse less than 60/min or 46/min for athletes
- Extrinsic causes by drugs, hypothyroidism, CNS disorders
- Intrinsic causes by SA or AV node dysfunction
- Treat the cause, such as a pacemaker

Heart block

- Often caused by ischemia due to CAD or may be idiopathic
- Three degrees possible
 - 1st degree – impulses reach the ventricles and slow in the AV node – no treatment needed
 - 2nd degree – impulse slows so that not all beats get through the ventricle, causing bradycardia – may or may not need a pacemaker
 - 3rd degree (complete) – all impulses from the atria to the ventricles are blocked at the AV node – all need external pacemaker
- Sick sinus syndrome - a wide variety of alternate bradycardia and tachycardia

Other Cardiac Tests

- Standard chest x-ray
 - Normal views are PA and left lateral
 - Portable chest films used in ER and with very ill patients
- Doppler echocardiography
 - Evaluates chamber size, wall thickness, valve structure and motion, shunts, pressures and hemodynamics
- Holter monitor
 - 24-hour continuous heart tracing

Other Cardiac Tests - continued

- Treadmill ECG stress testing
 - Contraindications – unstable angina, uncontrolled CHF, myocarditis, severe valve disease, rapid arrhythmias
 - Healthiest response is to exercise for 9 minutes at 85% of age-predicted maximum heart rate
- Blood tests
 - Fasting lipid profile and glucose studies
 - Homocysteine – for atherosclerotic plaques
 - Fasting serum glucose – for diabetes control
 - Cardiac enzymes –diagnosing and MI
 - Serum electrolytes and thyroid profile
 - Hemoglobin - anemia
 - C-reactive protein – generalized body inflammation
- Cardiac catheterization
 - The “gold standard” of cardiac diagnosis

Dr. M's favorite line with Deb – “Honey, being around you causes me to have ventricular tachycardia.”

