UNIT 3: THE HEART (OVERVIEW)

FROM GUYTON AND HALL TEXTBOOK

- The heart is composed of three major types of cardiac muscles
    1. Atrial muscle
    2. Ventricular muscle
    3. Specialized excitatory and conductive muscle fibers

- The muscle fiber layers of left ventricle allows the heart to contract in twisting motion during systole.
  The subepicardial (outer) layer spirals in leftward direction.
  The subendocardial (inner) layer spirals in rightward direction.
  This causes clockwise rotation of apex and counter clockwise rotation of base of left ventricle.

- Intercalated discs are cell membranes that separate individual cardiac muscle cells from one another

- At each intercalated disc, the cell membranes fuse with one another to form permeable communicating junctions, called gap junctions, that allow rapid diffusion of ions. And hence action potentials travel easily from one cardiac muscle cell to the next, past the intercalated discs. Thus cardiac muscle is a syncytium of many heart muscle cells in which cardiac cells are so interconnected that when one cell becomes excited, the action potential rapidly spreads to all of them.

- Heart is composed of two syncytia
    1. Atrial syncytium – constituting the walls of the two atria
    2. Ventricular syncytium – constituting the walls of the two ventricles

- Two types of channels in cardiac muscles
    1. Voltage activated fast sodium channels
    2. Slow calcium channels

  These slow calcium channels account for prolonged action potential and the plateau in cardiac muscle

- Immediately after the onset of action potential, the permeability of the cardiac muscle membrane for potassium ions decreases about fivefold, thus greatly decreasing the efflux of

positively charged potassium ions during action potential plateau and thereby preventing early return of action potential voltage to its resting level.

- PHASES OF CARDIAC MUSCLE ACTION POTENTIAL

  PHASE 0 (Depolarization)
  - Fast sodium channels open
  - Membrane potential reaches about +20mV before sodium channels close

  PHASE 1 (Initial Repolarization)

  - Fast sodium channels close
  - Potassium ions efflux through open potassium channels

  PHASE 2 (Plateau)

  - Calcium channels open
  - Fast potassium channels close

  PHASE 3 (Rapid Repolarization)

  - Calcium channels close
  - Slow potassium channels open

  PHASE 4 (Resting Membrane Potential)

  - -80 to -90mV

- Velocity of conduction
  In atrial and ventricular muscle fibers – about 0.3 to 0.5 m/s
  In purkinje fibers – as high as 4 m/s

- The refractory period of heart is the interval of time during which a normal cardiac impulse cannot re-excite an already excited area of cardiac muscle
  Refractory period of ventricle – 0.23 to 0.3 sec
  Refractory period of atrial muscle – 0.15 sec

- The term excitation-contraction coupling refers to the mechanism whereby the action potential causes the myofibrils of muscle to contract
  - Action potential enters the interior of cell through T tubules and cause the release of calcium ions into the sarcoplasm from sarcoplasmic reticulum
  - Calcium ions also diffuse into sarcoplasm from the T tubules at the time of action potential, which opens voltage dependent calcium channels in the membrane of T tubule.

- Cacium entering the cells then activates calcium release channels, also called ryanodine receptor channels, in sarcoplasmic reticulum membrane, triggering the release of calcium into sarcoplasm.
- Calcium ions in the sarcoplasm then interact with troponin to initiate cross-bridge formation and contraction.
- Inside the T tubules of cardiac muscles, a large quantity of mucopolysaccharides are present which are electronegatively charged and bind an abundant store of calcium ions, keeping them available for diffusion to the interior of the cardiac muscle fiber when a T tubule action potential appears.

- Duration of contraction
  0.2s in atrial muscle
  0.3s in ventricular muscle

- The cardiac events that occur from the beginning of one heartbeat to the beginning of the next are called cardiac cycle.

- Total duration of cardiac cycle - 0.833 sec/beat

- ECG
  P wave - spread of Depolarization through the atria
  QRS wave - appear 0.16 second aftet onset of P wave as a result of electrical depolarization of ventricles
  T wave - Repolarization of ventricles

- 80% of blood flows directly through atria into ventricles, even before the atria contract

Thus, atrial contraction usually causes an additional 20% filling of the ventricles.

- Pressure changes in atria
  a wave:
  - Caused by atrial contraction
  - Right atrial pressure increases 4-6mmHg
  - Left atrial pressure increases 7-8mmHg

  c wave:
  - Occurs when ventricles begin to contract
  - Caused
    1. Slight backflow of blood into atria at the onset of ventricular contraction
    2. By bulging of AV valves backward towards the atria due to increasing pressure in ventricles

  v wave:
  - Occurs towards end of ventricular contraction
  - Results from slow flow of blood into atria from veins while AV valves are closed during ventricular contraction

- Resting membrane potential of
  Sinus node = -55 to -60 mV
  Ventricular muscle fibers = -85 to -90 mV

- Membrane ion channels of cardiac muscle
    1. Calcium channels (particularly L type or slow calcium channels)
    2. Fast sodium channels
    3. Potassium channels

- Time of transmission of impulses
    - In intermodal pathway = 0.03 sec
    - In AV node = 0.09 sec
    - In AV bundle = 0.04 sec
    - In Purkinje fibers = 0.03 sec
    - In ventricular mass = 0.03 sec

- Action Potential Transmission Rate
    - In atrial muscle = 0.03 m/s
    - In several small bands of atrial muscles (such as Bachman's bundle or interatrial band) = 1 m/s
    - Purkinje fibers = 1.5 – 4 m/s
    - In ventricular muscle mass = 0.3 – 0.5 m/s

- Rhythmical stimulation of sinus node = 70 – 80 times per minute
  Rhythmical stimulation of AV node when not stimulated from outside source = 40 – 60 times per minute
  Rhythmical stimulation of purkinje fibers when not stimulated from outside source = 15 – 40 times per minute
  As Rhythmical stimulation of SA node is faster than that of any other part of heart so SA node controls the heart rhythm.

- P wave and QRS wave – Depolarization wave
  T wave – Repolarization wave
  P wave – contraction of atria
  QRS complex – contraction of ventricles
  T wave – Repolarization of ventricles

- The atria repolarize about 0.15 to 0.20 sec after termination of P wave

- Ventricles repolarize 0.2 sec after beginning of QRS complex but in many other fibers it takes 0.35 sec.

- When ECG recorded from electrodes on two arms or one arm and one leg, the normal voltages of
    - QRS complex = 1.0 – 1.5 mV (from top of R wave to bottom of S wave)
    - P wave = 0.1 – 0.3 mV
    - T wave = 0.2 – 0.3 mV

- 10 small divisions upward or downward represent 1mV

- Times for
    - PQ Interval or PR Interval= 0.16 sec

- QT interval = 0.35 sec

- PQ or PR Interval – from beginning of P wave to the beginning of QRS complex
  QT Interval – from beginning of Q wave to end of T wave

- Heart rate can be determined from R-R Interval of ECG
  Normal R-R Interval in adult = 0.83 sec
  Heart rate is reciprocal of R-R Interval
  So Heart Rate = $\frac{1\ beat}{0.83\ sec} = \frac{60\ beats}{0.83\ min}$ = 72 beats/min

- Lead I
  - Negative terminal on right arm
  - Positive terminal on left arm

  Lead II
  - Negative terminal on right arm
  - Positive terminal on left leg

  Lead III
  - Negative terminal on left arm
  - Positive terminal on left leg

- Einthoven's Law
  Lead I potential + Lead III potential = Lead II potential

- aV_R Lead – positive terminal on right arm
  aV_L lead – positive terminal on left arm
  aV_F Lead – positive terminal on left leg

- Diagnosis of cardiac arrhythmia depend mainly on the time relationship of different waves of the cardiac cycle

- Diagnosis of damage in ventricular or atrial muscle or in purkinje conducting system depends greatly on the leads because abnormalities of cardiac muscle contraction or cardiac muscle conduction change the patterns of ECG markedly in some leads yet may not affect other leads

- QRS Complex in leads
  - Leads I, II, III – positive deflection
  - Leads v_1, v_2 – negative deflection
  - Leads v_4, v_5, v_6 – positive deflection
  - Lead aV_R – negative deflection
  - Leads aV_F, aV_L – positive deflection

- A vector is an arrow that points in the direction of the electrical potential generated by the current flow, with the arrowhead in the positive direction. Also, by convention, the length of the arrow is drawn proportional to the voltage of the potential.

- Axis of leads
  Lead I → 0°

Lead II → +60°
Lead III → +120°
Lead aV_R → +210°
Lead aV_F → +90°
Lead aV_L → -30°

- One of the most common causes of decreased voltage of QRS complex is a series of old myocardial infarctions with resultant diminished muscle mass.

- One of the most important causes of decreased voltage in ECG leads is excessive fluid in the pericardium (pericardial effusion)

- Pulmonary emphysema can also decrease the voltage of QRS complex

- Prolonged QRS complex may be caused by ventricular hypertrophy
  Normal QRS complex = 0.06 – 0.08 sec
  QRS complex in ventricular hypertrophy = 0.09 – 0.12 sec

- Purkinje system block
  QRS complex in purkinje system block = 0.14 sec or larger

- Abnormalities that can cause a current of injury
  1. Mechanical trauma
  2. Infectious processes
  3. Ischemia of local areas of heart muscle caused by local coronary occlusions

- J Point is the zero reference level in ECG for current of injury and is the exact point at which wave of depolarization just completes its passage through the heart, which occurs at the end of QRS complex

- The injury potential in each lead is the difference between the voltage of the ECG immediately before the onset of the P wave and the zero voltage level determined from the J point

- One of the most important diagnostic features of the ECGs recorded after acute coronary thrombosis is the current of injury

- The positive end of the injury potential vector points towards the normal cardiac muscle, and the negative end points towards the injured portion of the heart that is emitting the current of injury

- T wave is normally positive in all bipolar limb leads

- Tachycardia – greater than 100 beats/min

- Some causes of tachycardia
  1. Increased body temperature
  2. Dehydration
  3. Blood loss anemia

4. Stimulation of heart by sympathetic nerves
5. Toxic conditions of heart

- Bradycardia – fewer than 60 beats/min

- Normal PR Interval = 0.16 sec
  When PR Interval increases to more than 0.2 sec, the patient is said to have first-degree incomplete heart block

- One means for determining the severity of heart diseases, such as acute rheumatic heart disease, is to measure the PR Interval.

- Incomplete atrioventricular block
  1. First Degree Block
     - Prolonged PR Interval i.e. greater than 0.2 sec
  2. Second Degree Block
     a) Mobitz Type I (Wenchback Periodicity)
        - PR Interval ranges from 0.25 – 0.45 sec
        - Dropped beats of ventricles observed
        - Almost always caused by abnormality of AV node
     b) Mobitz Type II
        - Usually there are a fixed number of non-conducted P waves for every QRS complex
        - Generally caused by an abnormality of the Bundle of His-Purkinje system
        - May require implantation of pacemaker to prevent progression to complete heart block or cardiac arrest

  3. Third Degree Block (Complete AV Block)
     - Ventricles establish their own signal, usually originating from AV node or AV bundle

- Possible causes of ectopic foci are as follows
  1. Local areas of ischemia
  2. Small calcified plaques at different points in the heart, which press against the adjacent cardiac muscle so that some of the fibers are irritated
  3. Toxic irritation of AV node, purkinje system or myocardium caused by infection, drugs, nicotine or caffeine

- Lack of blood flow to the brain for more than 5 to 8 minutes usually causes permanent mental impairment or even destruction of brain tissue

- Patients with atrial fibrillation are often placed on blood thinner medications (anti coagulants) to reduce the risk of embolism