Electrocardiogram

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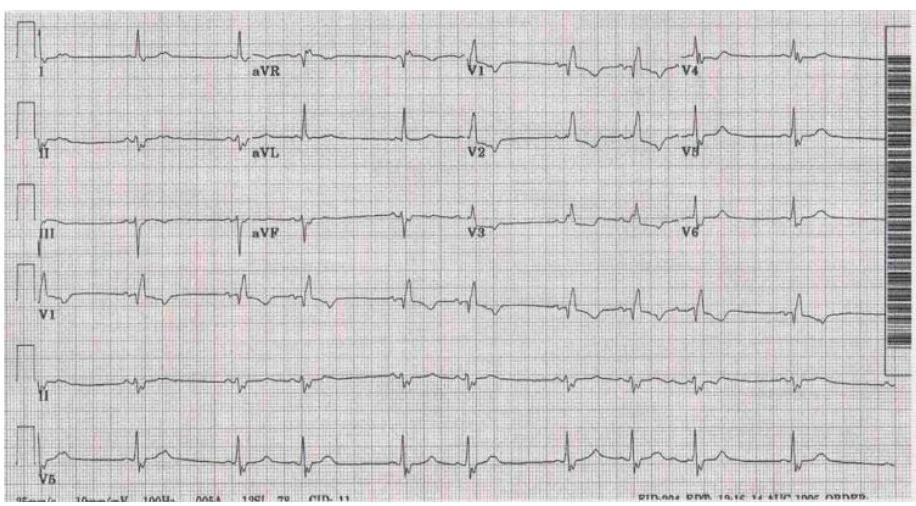
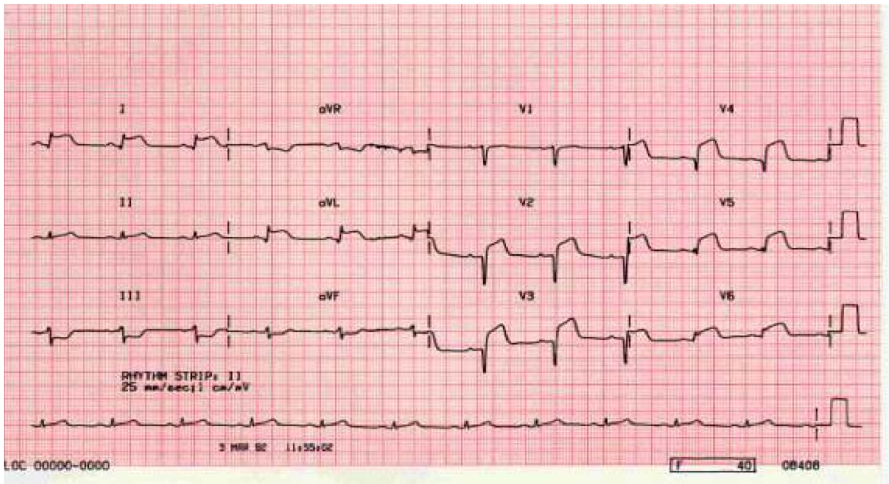
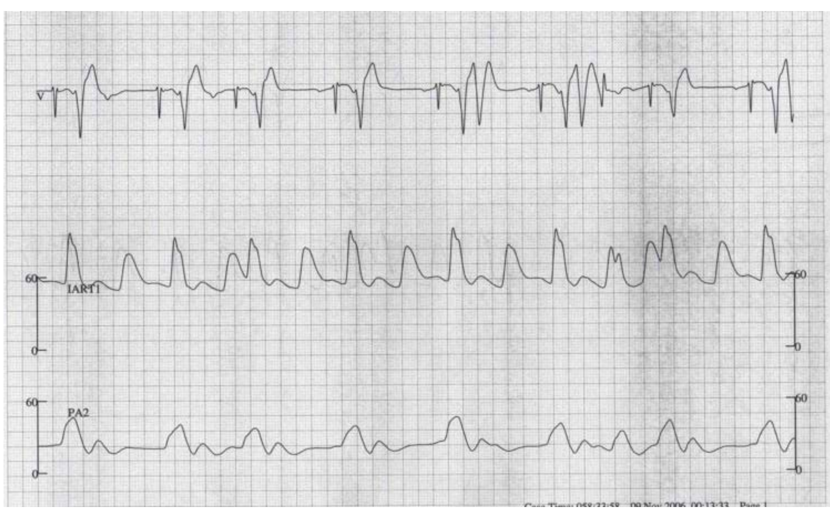


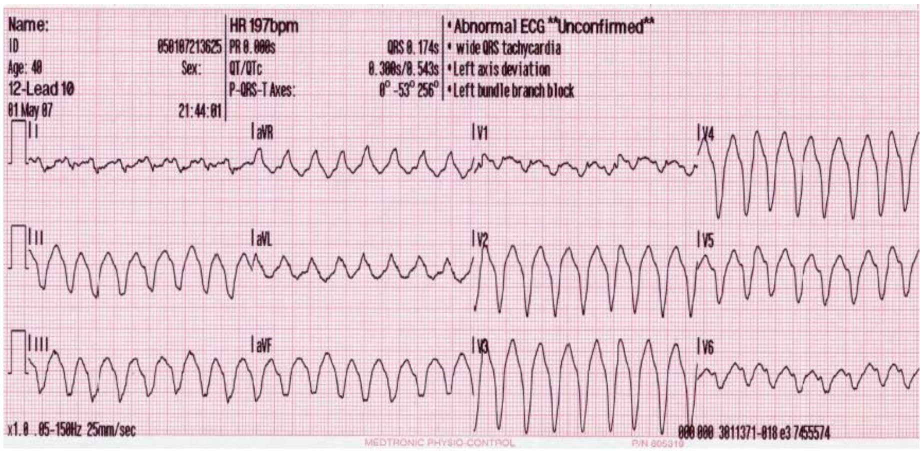
Bifasc--EKG showing second-degree heartblock(bifascicular) with inverted T-wave



Mi1--EKG showing ST-segment elevation myocardial infarction(STEMI) in leads I,II, V2-V6.



VT--EKG showing ventricular tachycardia with widened QRS complex.



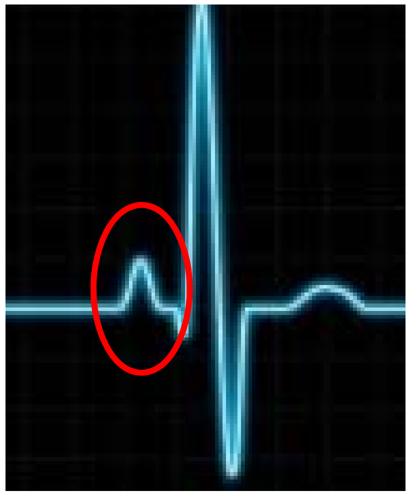
ib--EKG showing ventricular fibrillation in leads II through IV

Calculations

- Millimeters to Millivolts (10mm=1mv)
  - Suppose that we are looking to calculate the amplitude of the "P"wave.
    - So you would measure the "P" wave, lets say that it is 2mm.
    - Then plug it into the equation →

Amplitude of the P wave = $2\cancel{mm} \times \frac{1mV}{10\cancel{mm}} = 0.2$ mV

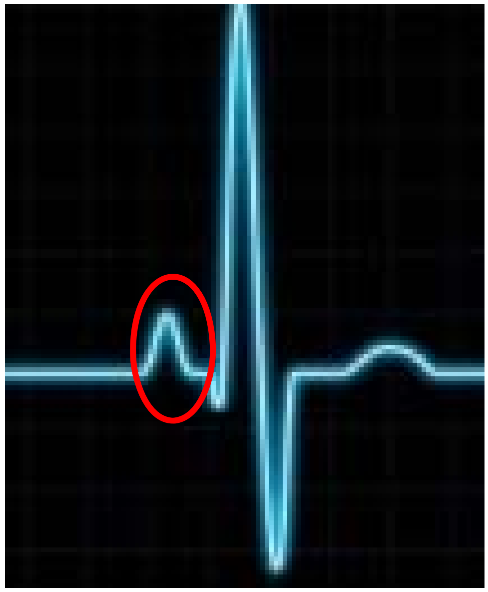
- This equation can be used to calculate the Millivolts of any wave on an ECG.



Calculations

- Millimeters to Seconds (25mm = 1 sec)
  – Suppose that you wanted to measure the duration of an interval.
    - So you would measure the width of the wave, and the PR wave is 4mm.
    - Then you would plug it into this equation→

Duration of the PR wave = $4\ \cancel{mm} \times \frac{1\ sec}{25\ \cancel{mm}} = 0.16$ sec.

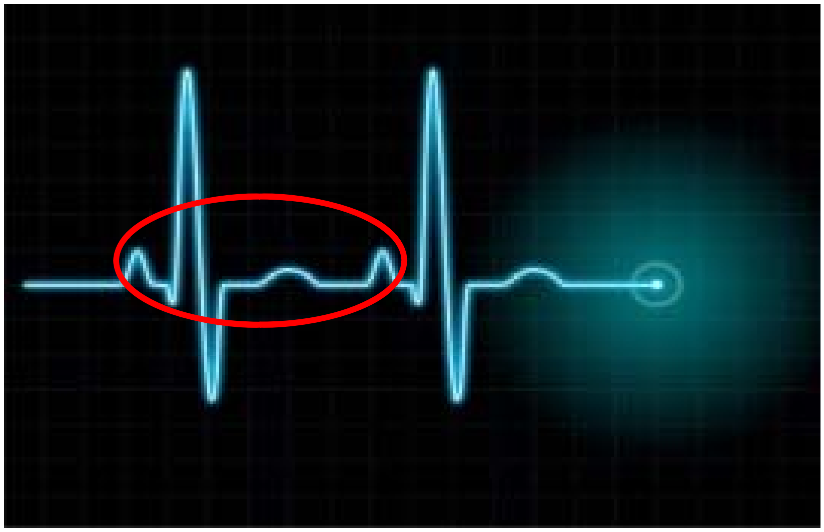


Calculations

- **Artial Rate-** is the number of atrial contractions per minute.
  - This is determined by measuring the PP interval distance. (Suppose 30 mm)
  - Then plug it into this equation:

$$\frac{1\ \cancel{\text{P wave}}}{30\ \cancel{\text{mm}}} \times \frac{1\ \text{atrial contraction}}{1\ \cancel{\text{P wave}}} \times \frac{25\ \cancel{\text{mm}}}{1\ \cancel{\text{sec}}} \times \frac{60\ \cancel{\text{sec}}}{1\ \text{min}} = \frac{50\ \text{Contraction}}{1\ \text{minute}}$$

  - Now to calculate the ventricular rate… assume that the atrial and ventricular contraction rates are the same.

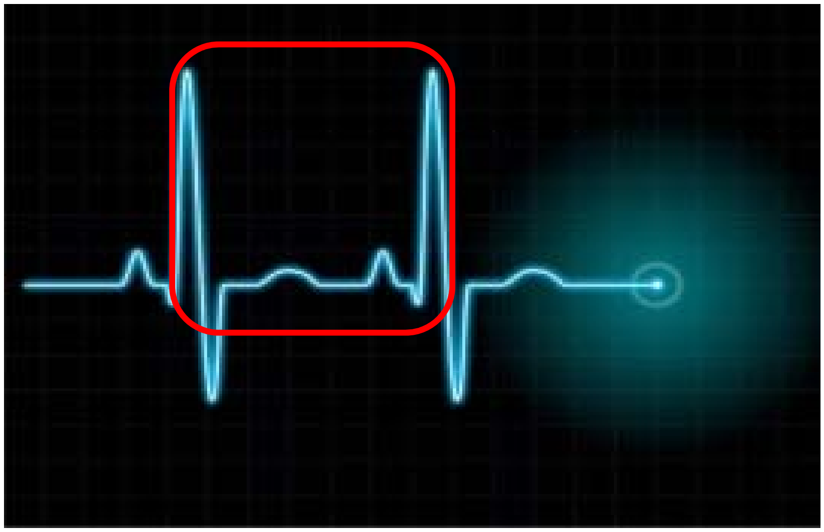


Calculations

- **<u>Ventricular rate-</u>** is the number of ventricular contractions per minute.
  - So in order to figure the ventricular rate out you need to measure the RR interval. (30 mm)
  - Then plug that number into this equation→

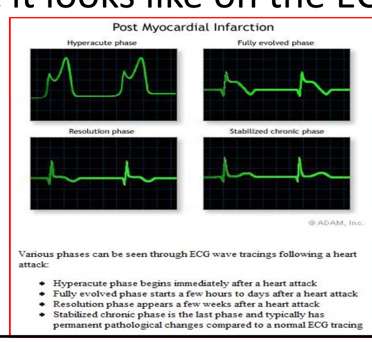
$$\frac{1\ \cancel{\text{QRS}}}{30\ \cancel{\text{mm}}} \times \frac{1\ \text{ventricular contraction}}{1\ \cancel{\text{QRS}}} \times \frac{25\ \cancel{\text{mm}}}{1\ \cancel{\text{sec}}} \times \frac{60\ \cancel{\text{sec}}}{1\ \text{min}} = \frac{50\ \text{Contraction}}{1\ \text{minute}}$$

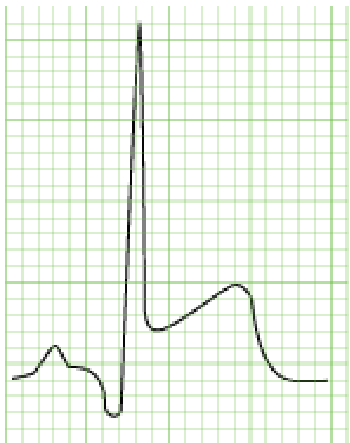
  - If the numbers from the atrial and the ventricular rate come out as the same number… You have calculated a correct heart rate

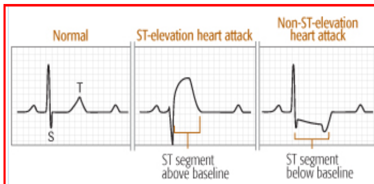


Acute Myocardial Infarction

- Is also known as a Heart Attack.
- Is when the blood supply to part of the heart is interrupted; most commonly because of an occlusion (blockage) of the coronary artery.
- This is what it looks like on the ECG:



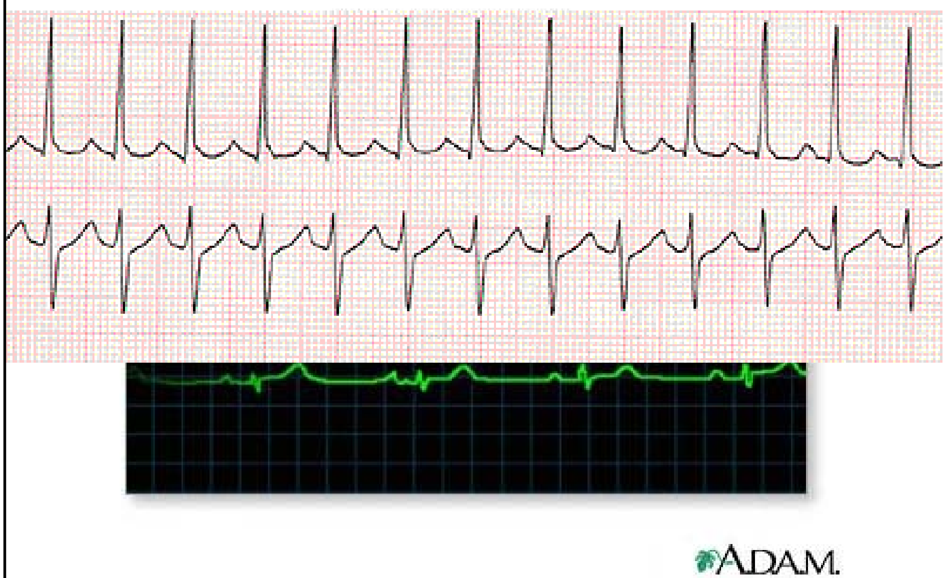




Doctors analyze EKG patterns to help determine what type of heart attack you are having. In an ST-elevation heart attack, which requires the most aggressive treatment, the ST segment is usually above the baseline (middle). In a non-ST-elevation heart attack, the ST segment is below baseline (right). However, EKG patterns are seldom so clear, and blood tests and other tests will confirm a diagnosis.

Cardiac Arrhythmia

- Literally meaning “abnormal electrical activity”
  - Whether the pulse is bradycardic (slow) or tachycardic (fast)
    - Bradycardic- can be caused by a slowed sinus node or a blockage of electrical activity to the atria
    - Tachycardic- can be caused by physical activity or stress



Phases of the Heart During ECG

Events of Contraction

	Skeletal Muscle	***Cardiac Muscle***
"Means of Stimulation" (What makes it contract?)	each sm fiber must be stimulated by a nerve ending	**some cm cells are self excitable and initiate their own depolarization (*Automaticity* or *Autorhythmicity*)**
Type of Contraction	Motor Unit: all cells of a motor unit are stimulated and contract at the same time (impulses do not spread from cell to cell)	**Heart: contracts as a unit or does not contract at all** → **depolarization wave across the heart (from cell to cell via *gap junctions*) ties all cm cells together** → **singe contractile unit**
Absolute refractory period (inexcitable period when Na+ channels are still open/inactivated)	Lasts 1-2ms (for contractions lasting 15-100ms)	**Lasts about 250ms (almost length of contraction)** → **prevents *tetanic contractions* (which would stop the heart's pumping action)**

Cardiac Muscle Contraction

~Pumping activity of the heart by contractile muscle fibers
~Triggered by action potential across cell membranes

1. Depolarization
   - -Na+ channels in sarcolemma open
   - -Extracellular Na+ enters into cell which starts positive feedback cycle
   - -Rise of membrane potential increases form -90mV to +30 mV
   - -Na+ enters cell and threshold is reached
   - -Na+ permeability decreases to resting levels and repolarization begins

   ***BUT this change in membrane permeability across the sarcolemma opens *slow Ca2+ channels* (delayed) in cardiac muscle at the threshold, which prolongs depolarization, creating a *plateau* in the action potential, and a delay in repolarization (K+ permeability)
2. Contraction
   - -influx Ca2+ across the sarcolemma into the cell triggers Ca2+ channels in the Sarcoplasmic Reticulum (SR) tubules to open
   - -Bursts of Ca2+ is released from the cell "Calcium Sparks" and contraction occurs
   - -The action potential lasts 250ms (is so long due to the plateau caused by influx of Ca2+) which provides the sustained contraction needed to eject blood from the heart
3. Repolarization
   - -Ca2+ channels close
   - -K+ channels open and allows a rapid loss of potassium from the cell to restore resting membrane potential
   - -Ca2+ is pumped back into SR and extracellular space

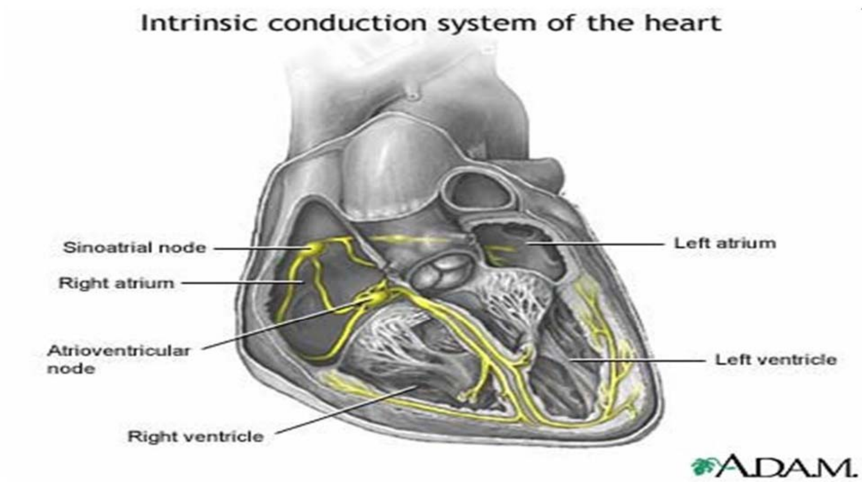
Autorhythmic Cells

- Make up the cardiac intrinsic conduction system
- Self-excitable cardiac cells that generate and distribute electrical impulses throughout the heart (without external stimulation by nerve cells)
- Uses calcium influx (rather than sodium) for rising phase of action potential and serve as the *pacemakers* to initiate the cardiac cycle (pumping cycle of the heart)
- Have unstable resting membrane potentials that continuously depolarize (*pacemaker potentials)*

Pacemakers

- <u>Sinoatrial (SA) Node</u> "Pacemaker"
- <u>Atrioventricular (AV) Node</u>
- <u>Bundle of His</u>
- <u>Bundle Branches</u>
- <u>Purkinje Fibers</u>

Cardiac Intrinsic Conduction



Events During ECG

- P Wave
  - Represents *atrial depolarization*
  - **SA Node** (located in right atrium) generates action potential that spreads through both atria (through gap junctions) and atrial excitation begins
  - **AV Node** (located near interatrial septum) receives the action potential from SA node and delay occurs here
  - allows the atria to contract fully
- QRS Complex
  - Represents *ventricular depolarization*
  - **Bundle of His** (located within the interventricular septum) receive the action potential from the AV Node and carry it to the apex by way of the **Bundle Branches** (also located within the interventricular septum) and ventricular excitation begins

    **Because *Atrial Repolarization* takes place during ventricular excitation, the wave representing atrial repolarization is usually obscured by the large QRS complex being recorded at the same time
  - **Purkinje Fibers** receive the action potential in the ventricles (rely heavily on cell-to-cell transmission through the gap junctions in the ventricular muscle cells)
  - Ventricles contract and ventricular excitation is complete
- T wave
  - Represents *ventricular repolarization*

Events During ECG (cont.)

- PR Interval (sometimes called PQ Interval)
  - Represents the time from the beginning of atrial excitation to the beginning of ventricular excitation
  - About 0.16 seconds
- S-T Segment
  - When action potential is in its plateau phase
  - Entire ventricular myocardium is depolarized
- Q-T Interval
  - Represents the time from the beginning of ventricular depolarization through ventricular repolarization
- See Figure 18.17!!