cvs

Physiology

Writer: Doctor 2018/2019

corrected by:

Doctor: Faisal Mohammad

♥ Einthoven's triangle and law:

▪ The three standard limb leads (I, II, III) can be seen to form an equilateral triangle (each angle is 60°), with the heart at the center. This is called the Einthoven's triangle. To facilitate the representation of electrical forces, the three limb leads of the triangle can be drawn in such a way that the three leads bisect each other and pass through a common central point. This produces a *triaxial reference* system with each axis separated from the next by 60°. with the lead polarity (positive and negative poles) and orientation (direction) remaining the same.

Einthoven's Triangle   Axial Reference System
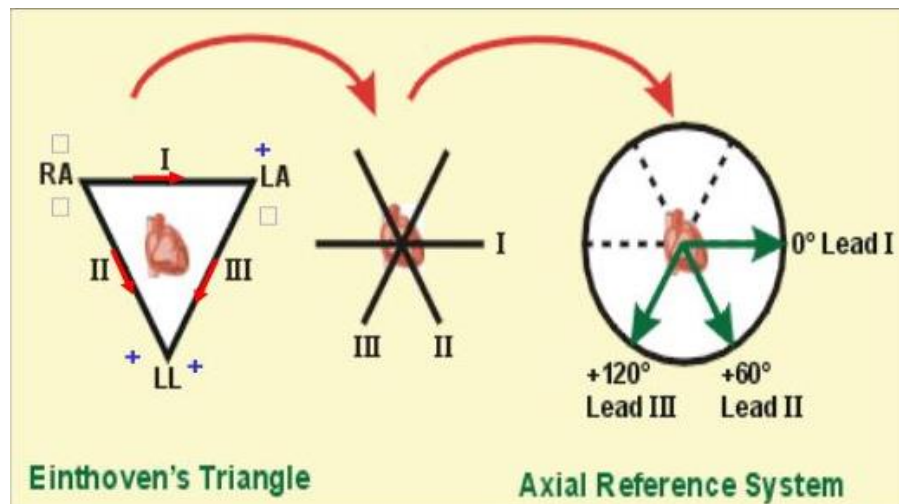
▪ Also, the augmented limb leads are recorded from one limb at a time (unipolar), the limb carrying the positive electrode, and the negative pole being represented by the central point. The three augmented limb leads (aVR, aVL, aVF) form another triaxial reference system, with each axis being separated from the next by 60°. When the triaxial system of the unipolar leads is superimposed on the triaxial system of the bipolar limb leads, we can derive a hexaxial reference system with each axis being separated from the next by 30°.
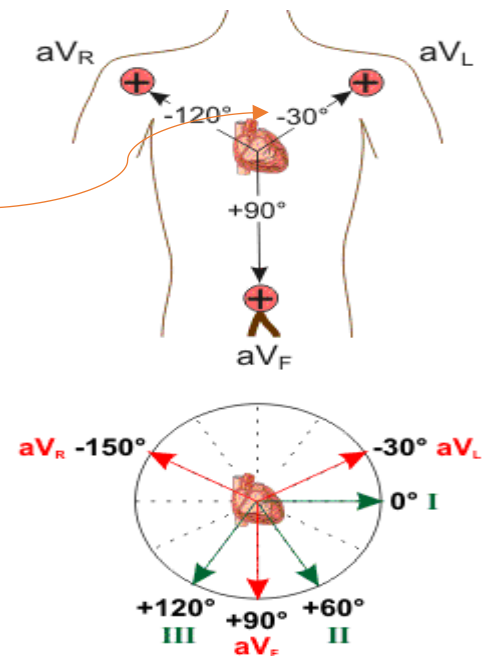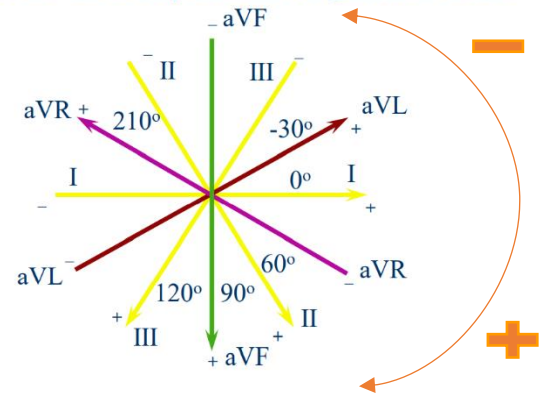
- Note that each of the six leads retains its polarity and orientation. The hexaxial reference system is important in determining the major direction of the heart's electrical forces (the electrical axis of the QRS complex).
- Perpendicular leads:
  1. L I and aVF
 2. L II and aVL
  3. L III and aVR
- Lead I is recorded from two electrodes placed respectively on the two arms. Because the electrodes lie exactly in the horizontal direction, with the positive electrode to the left, the axis of lead I is 0 degrees.
- In recording lead II, electrodes are placed on the right arm and left leg. The right arm connects to the torso in the upper right-hand corner and the left leg connects in the lower left-hand corner. Therefore, the direction of this lead is about +60 degrees.
- By similar analysis, it can be seen that lead III has an axis of about +120 degrees; lead aVR, +210°or -150°; aVF, +90 degrees; and aVL -30 degrees.
- Let's revise the basics of vectorial analysis by this example (extra example to clarify things before digging deeper in the analysis):
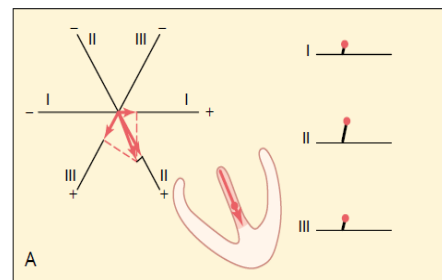- Vector A is the instantaneous electrical potential of a partially depolarized heart. To determine the potential recorded at this instant in the ECG for each one of the three standard bipolar limb leads, perpendicular lines (the dashed lines) are drawn from the tip of vector A to the three lines representing the axes of the three different standard leads, as shown in the figure. The projected vector B represents the potential recorded at that instant in lead I, projected vector C represents the potential in lead II, and projected vector D represents the
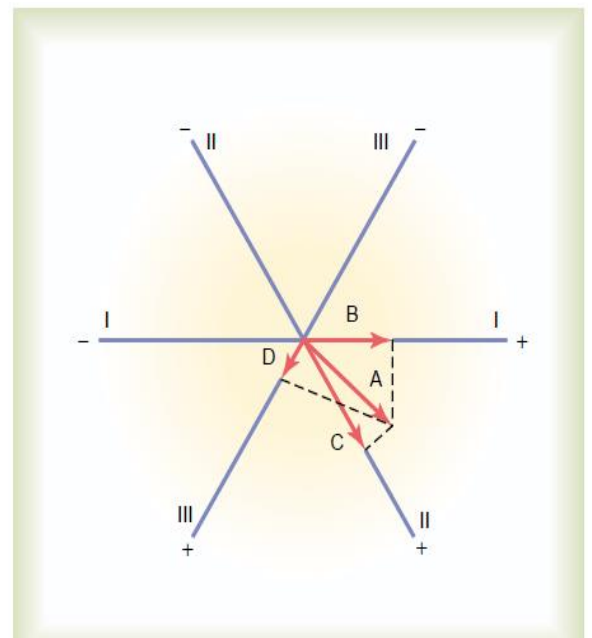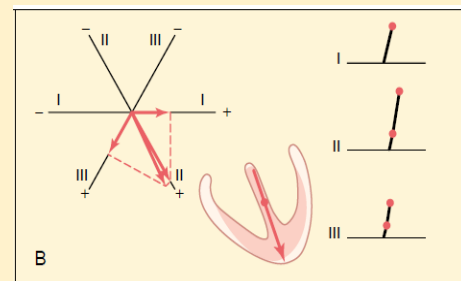
potential in lead III. In each of these, the record in the electrocardiogram is positive that is, above the zero line because the projected vectors point in the positive directions along the axes of all the leads. The potential in lead I (vector B) is about one half that of the actual potential in the heart (vector A); in lead II (vector C), it is almost equal to that in the heart (because almost all of the electrical activity is coming toward the lead in that instant); and in lead III (vector D), it is about one third that in the heart (electrical activity is not heading directly toward this lead, but still detected by the lead).

|	
the ventricular muscle has just begun to be depolarized. At this time, the vector is short because only a small portion of the ventricles—the septum—is depolarized. Therefore, all ECG voltages are low, as recorded. The voltage in lead II is greater than the voltages in leads I and III because the heart vector extends mainly in the same direction as the axis of lead II. • Q wave is present if the left side of the septum depolarizes first.	
The heart vector is long because much of the ventricular muscle mass has become depolarized. Therefore, the voltages in all electrocardiographic leads have increased (especially lead II). the depolarization vector is large because half of the ventricle is depolarized. Lead II should be largest voltage when compared to I and III when the mean vector is 60°.	
the heart vector is becoming shorter and the recorded electrocardiographic voltages are lower. Also, the axis of the vector is beginning to shift toward the left side of the chest because the left ventricle is slightly slower to depolarize than the right.	
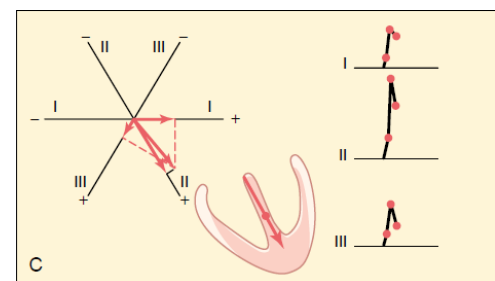
the heart vector points toward the base of the left ventricle, and it is short because only a minute portion of the ventricular muscle is still polarized positive. Because of the direction of the vector at this time, the voltages recorded in leads II and III are both negative that is, below the line (S wave) whereas the voltage of lead I is still positive (sometimes it is also negative).

- the last part to depolarize is near the left base of the heart which gives a negative vector (S wave).


D
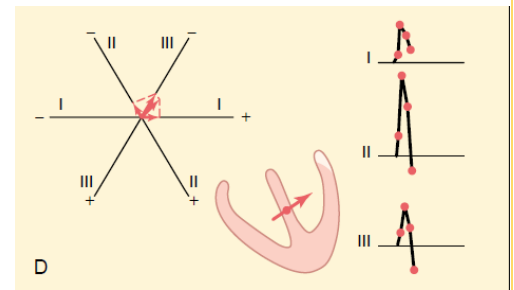
the entire ventricular muscle mass is depolarized, so that no current flows around the heart and no electrical potential is generated. The vector becomes zero, and the voltages in all leads become zero.
EXTRA: The recording represents comparison in voltage detected by the electrodes at two different points not the actual potential. ECG does not record a potential at all when the ventricular muscle is completely depolarized or completely repolarized, because both electrodes are "viewing" the same potential, so no difference in potential between the two electrodes is recorded.

E
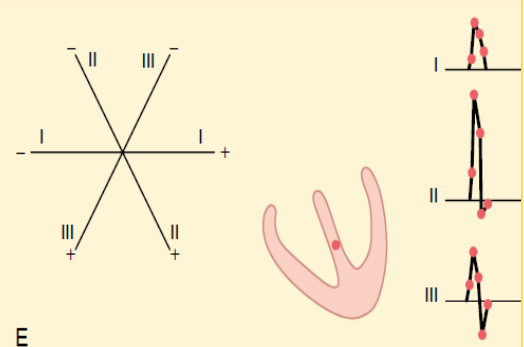
After the ventricular muscle has become depolarized, repolarization begins and proceeds. This repolarization causes the T wave in the electrocardiogram. Because the septum and endocardial areas of the ventricular muscle depolarize first, it seems logical that these areas should repolarize first as well. However, this is not the usual case. The greatest portion of ventricular muscle mass to repolarize first is the entire outer surface of the ventricles. The endocardial areas, conversely, normally repolarize last. Because the outer surfaces of the ventricles repolarize before the inner surfaces, the positive end of the overall ventricular vector during repolarization is toward the apex of the heart. As a result, the normal T
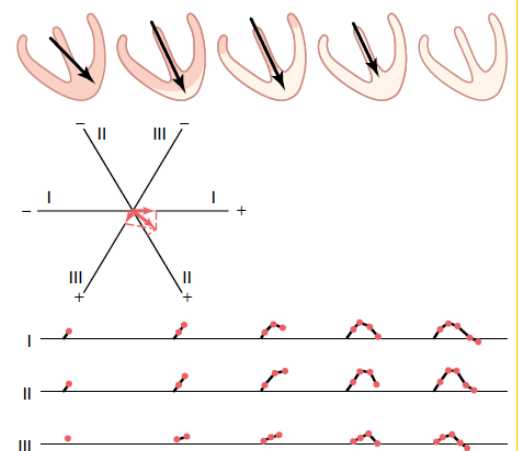
wave in all three bipolar limb leads is positive (upward deflection), which is also the polarity of most of the normal QRS complex.

Repolarization:

Apex→ base

Outer-surface→ inner-surface

- Depolarization of the atria begins in the sinus node and spreads in all directions over the atria. The direction of initial depolarization is denoted by the black vector. The vector remains generally in this direction throughout the process of normal atrial depolarization. Because this direction is generally in the positive directions of the axes of the three standard bipolar limb leads I, II, and III, the ECGs recorded from the atria during depolarization are also usually positive in all three of these leads, this record of atrial depolarization is known as the atrial P wave.

- The area in the atria that becomes repolarized first is the area that had originally become depolarized first. Thus, when repolarization begins, the region around the sinus node becomes positive with respect to the rest of the atria. Therefore, the atrial repolarization vector is backward to the vector of depolarization. (Note that this is opposite to the effect that occurs in the ventricles). atrial T wave follows the atrial P wave, but this T wave is on the opposite side of the zero-reference line from the P wave; that is, it is normally negative rather than positive in the three standard bipolar limb leads.

- In the normal electrocardiogram, the atrial T wave appears at about the same time that the QRS complex of the ventricles appears. Therefore, it is almost always totally masked by the large ventricular QRS complex, although in some very abnormal states, it does appear in the recorded electrocardiogram.
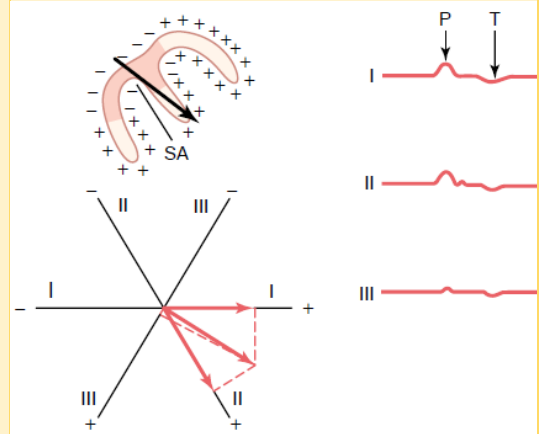
- point 5 is the zero-reference point. While the heart muscle is polarized between heartbeats, the positive end of the vector remains at the zero point because there is no vectorial electrical potential. However, as soon as current begins to flow through the ventricles at the beginning of ventricular depolarization, the positive end of the vector leaves the zero-reference point.
- When half of the ventricle is depolarized, vector-2 has the largest potential difference.
- Vector-4 represents depolarizing of the posterior aspect of the left ventricle (S-wave).
- The recording of the QRS complex is the same on all the leads, even on the unipolar leads and the horizontal chest leads (V1-6).
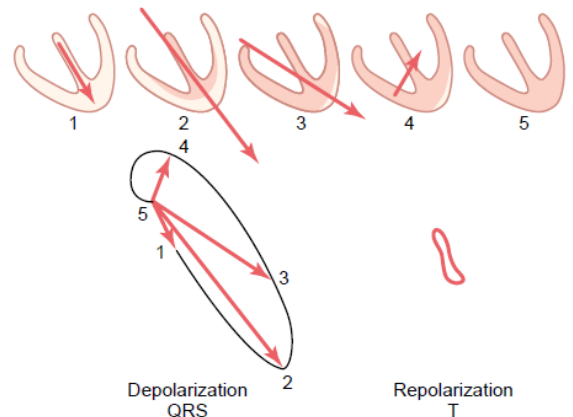
♥ Determining Mean Electrical Axis:
- To calculate the mean electrical axis of the QRS complex in this example, standard leads I and III were used but any combination of two of the six could have been used (we don't use chest leads). The vectorial sum of the deflections of the QRS complex for each lead is calculated in millimeters.

  1. In this example, the Q wave is -2.5 mm deep and the R wave is +16 mm high to give a sum of +13.5 mm for lead I
  2. The point corresponding to this sum is then located on lead I (the positive direction being towards the arrow of each lead) and a perpendicular is dropped from lead I
  3. The same is done for lead III.
  4. A line is then drawn from the center of the grid through the point of intersection of the two perpendicular lines to obtain the mean electrical axis. In this case, the mean
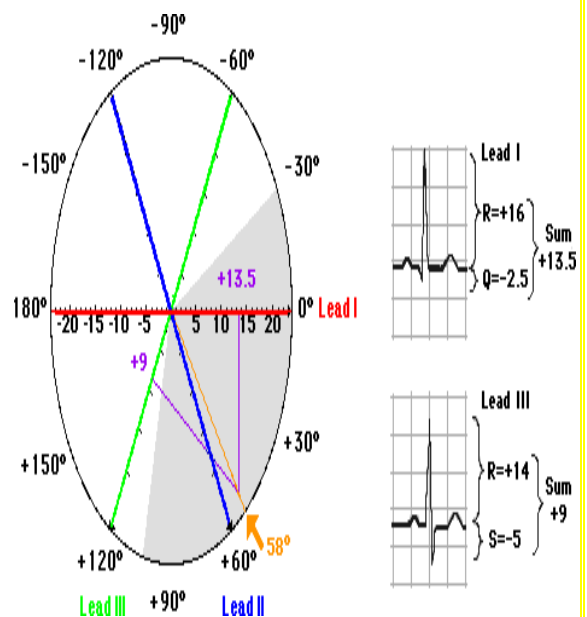
electrical axis of the QRS complex is 58 degrees, which is within the normal range.

♥ In this example, the obtained mean electrical axis of the ORS complex is 60°, which is basically the axis of lead II.
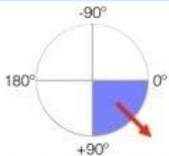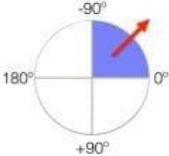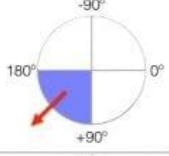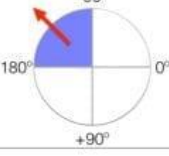
Plot of the Mean Electrical Axis of the Heart from Two Electrocardiographic Leads
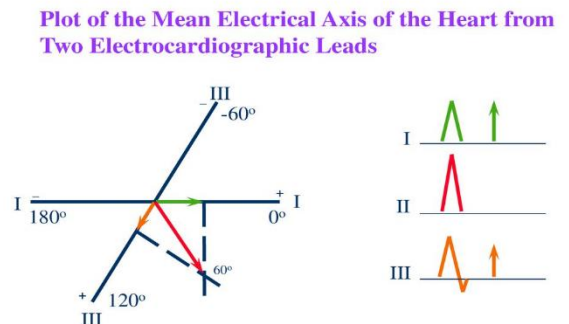
♥ The most efficient way to estimate axis is to look at LEAD I and LEAD aVF.

▪ A positive QRS in Lead I puts the axis in roughly the same direction as lead I.

▪ A positive QRS in Lead aVF similarly aligns the axis with lead aVF.

▪ Combining both coloured areas, the quadrant of overlap determines the axis. So, If Lead I and aVF are both positive, the axis is between 0° and +90° (i.e. normal axis).
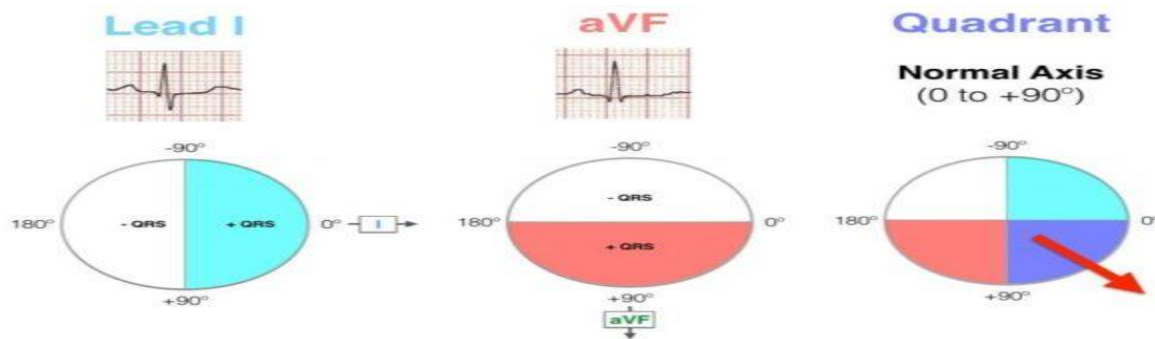
▪ The following table summarizes all the possible cases:

    a. Normal Axis = QRS axis between 0° and +90°.

    b. Left Axis Deviation-LAD = QRS axis between 0° and -90°.

    c. Right Axis Deviation-RAD = QRS axis between +90° to +180°.

    d. Extreme Axis Deviation = QRS axis between -90° (+270) and +180°, to determine whether it is left or right you should trace the patient's history for any clues that suggest right or left deviation, because ECG is not the only tool to reach a diagnosis.

Lead 1	Lead aVF	Quadrant	Axis
POSITIVE	POSITIVE		Normal Axis (0 to +90°)
POSITIVE	NEGATIVE		**Possible LAD (0 to -90°)
NEGATIVE	POSITIVE		RAD (+90° to 180°)
NEGATIVE	NEGATIVE		Extreme Axis (-90° to 180°)

♥ Heart Rate Calculation:

Measure the distance between two successive ECG complexes (as the number of small squares). The number of ECG complexes by 1 min (beats/min) is equal to:

60/ number of small square X 0.04

Or basically **1500/No. of small squares**

Mostly used interval is the R-R interval

R-R interval = 0.83 sec or 20.75 small squares

Heart rate (beat/min) = 60/0.83

= 72 beats/min

- Keep in mind that the R-R interval (period) is the same in every cycle, because the heart rate should be regular. In irregular heart rate, the intervals are not identical.
- P-R interval is measured from the onset of the P wave to the beginning of the QRS complex, it should be less than or equal to 0.2 sec, if it is more than 0.2 it denotes that something is abnormal.
- QRS complex, starts from the beginning of Q to the end of S, and should be less than 0.12 sec.
- Q-T interval is usually about 0.35-0.4 sec
- S-T segment should be isoelectric, you look for deflection, either upward or downward. The same applies for the P-R segment.
- The reference point for the isoelectric line is the T-P segment.

♥ Determine regularity:

☝ The distance between R-R intervals and QRS complexes should be the same, if the distance differs the rhythm is irregular. Several methods can be used to determine rhythm regularity, these include using calipers, marking a paper with a pen. By counting the small squares between the R-R interval (each large square = 5 small squares) and comparing this distance each time (should be the same).

- Patterned irregularity (regular irregularity) is when the irregularity repeats in a cyclic fashion.
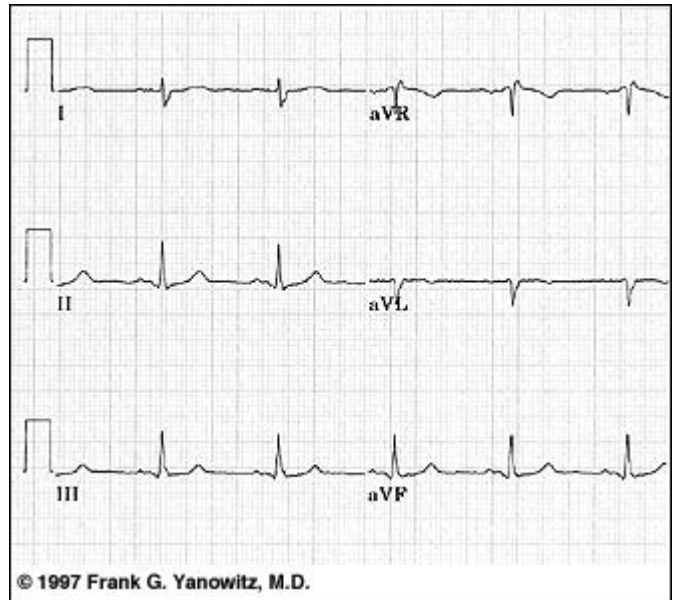

Quiz:

1. **If there were 3 large squares in an R-R interval what would the heart rate be?**

   a. 100 bpm

   b. 90 bpm

   c. 80 bpm

   d. 70 bpm

1. **Determine the QRS axis for this ECG:**
   a. -100 degrees

   b. -30 degrees

   c. +15 degrees

   d. +90 degrees

   e. Indeterminate

© 1997 Frank G. Yanowitz, M.D.

2. **P wave represents:**
   a. Depolarization of right ventricle

   b. Depolarization of both atria

   c. Depolarization of left ventricle

   d. Atria to ventricular conduction time

3. **In normal ECG one of the following waves is not represented, which one is that?**
   a. Depolarization of atria
   b. Repolarization of atria
   c. Depolarization of ventricles
   d. Repolarization of ventricles

♥ Answers:
   1. A
   2. D (Lead I is isoelectric and since aVF is positive, you know the axis is +90 degrees).
   3. B
   4. B

# Electrocardiography-Abnormalities (Arrhythmias)

In the previous lecture, we talked about the normal electrocardiograph, in this lecture we are going to talk about the abnormalities in the electrocardiograph (arrhythmias).

❖ **Causes of cardiac arrhythmias:**
1. Abnormality in the rhythmicity of the pacemaker.
2. Shift of the pacemaker from the sinus node (ectopic pacemaker).
3. Blocks at different points in the transmission of the cardiac impulse.
4. Abnormal pathway of transmission in the heart.
5. Spontaneous generation of abnormal impulses from any part of the heart.

1) Abnormal sinus rhythms:
- The normal heart rate ranges from (60-100 bpm).
- *Tachycardia:* increased (fast) heart rate that is usually greater than 100 bpm.
- The etiology of tachycardia:
  1. Increased body temperature: it increases the permeability of ions.
  2. Sympathetic stimulation: causes positive chronotropic (heart rate) and positive inotropic effect (contractility), which happens after blood loss and the reflex stimulation of the heart.
  3. Toxic conditions of the heart: like hyperthyroidism which causes an increased level of T_3 (thyroxin) and T_4.
- In the electrocardiograph below, calculate the heart rate:
  ➢ We have 10 small squares between the arrows.
  ➢ The duration of a single cardiac cycle= $10 \times 0.04 =$ <mark>0.4sec</mark>.
  ➢ The heart rate= $\frac{60}{0.4} = 150b$ which is more than normal.
  ➢ The heart rate looks regular (blue arrows), but there is increased heart rate.
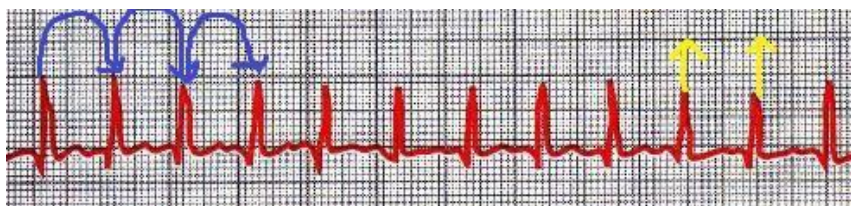
Increase in T3 -4 causes anabolism of receptors mainly beta

- *Sinus tachycardia:* tachycardia happens due to faster depolarization of SA node, the impulse is conducted normally.
  - On the electrocardiograph we will notice the presence of **P** wave, followed by QRS, then T wave.

12 small square

- *Bradycardia:* slow heart rate, usually less than 60bpm.
- The etiology of bradycardia:
  1. Sometimes can be presented normally in athletes, in those who have stronger hearts, so higher ability to pump blood out, thus, do not need more heart rate to have a normal cardiac output, they have *high stroke volume.* $cardiac\ output = stroke\ volume(\text{t}) \times heart\ rate(\downarrow)$.
  2. Can be caused by vagal stimulation (parasympathetic), causing negative chronotropic effect, example: carotid sinus syndrome (pressure on the carotid sinus).
- In the electrocardiograph below, calculate the heart rate:
  - The number of small squares per single cardiac cycle= 35.
  - The duration of single cardiac cycle= $35 \times 0.04 = 1.4 sec$.
  - The heart rate= $\frac{60}{1.4} \approx 43bpm$, which is very low.

Less than 40 the heart rate

- *Sinus bradycardia:* bradycardia happens due to slower depolarization in the SA node, impulses are conducted normally, heart rate is lower than 60bpm.
  - On the electrocardiograph we will notice the presence of **P** wave, followed by QRS, then T wave.
  - Normal PR interval, which means normal conduction between SA node & AV node, and we will notice normal QRS interval.

(a) Sinus rhythm (normal)

Normal rhythm: P, QRS, T waves all are presented, and normal heart rate.

(b) Nodal rhythm – no SA node activity

Nodal rhythm: no SA node, so no P wave, could be due to atrial fibrillation, or the absence of the SA nodal activity.

2) Blocks in the points of transmission:

- *Sinoatrial block:* in rare instances, the impulses from SA node are blocked, causing cessation of P waves, the new pacemaker will be the region of the heart with the fastest discharge rate which is usually the AV node, so the heart rate will become (40-60bpm) which is the intrinsic rate of the AV node.
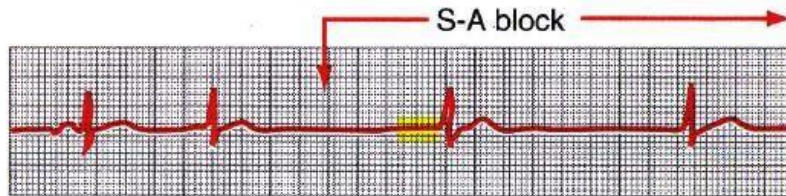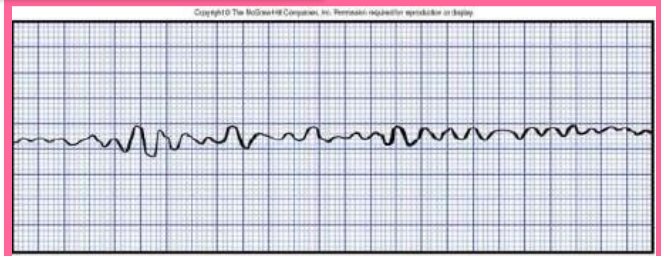
Each cell contract by itself
No synchronous contraction
No p wave

S-A block

Note: no P waves and slow rate
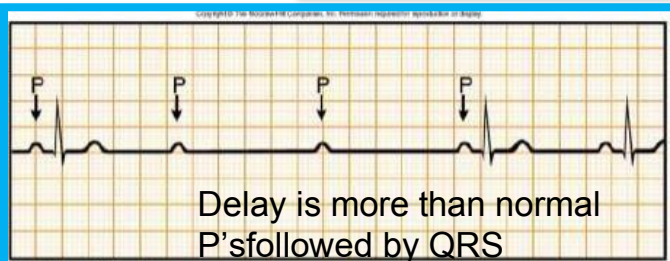
Delay is more than normal
P'sfollowed by QRS
(c) Heart block

Heart block: damaged AV node, so *no* impulse conduction from the atria to the ventricles causing prolonged P-R interval (>0.2 sec).
First degree heart block
Arrhythmia: conduction failure at the AV node.

(e) Ventricular fibrillation

Ventricular fibrillation: saw shaped ECG (no QRS) which is lethal.

No synchronous contraction of both ventricles, so no pumping, cardiac output=0

2nd Degree Heart block: more damage → sometimes it conducts and sometimes it does not regular Irregularities 2:3 P:QRS

- *Atrioventricular block:* impulse through AV node or AV bundle (bundle of His) is blocked, leading to prolonged P-R interval (> 0.2sec).
- The etiology:

1. Ischemia of AV nodal or AV bundle fibers (coronary ischemia or pressure).
2. Compression of AV bundle (by scar tissue or calcified tissue).
3. AV nodal or AV bundle inflammation.
4. Excessive vagal stimulation (carotid sinus syndrome, strokes-Adams syndrome).

- There's 3 degrees of the atrioventricular block:
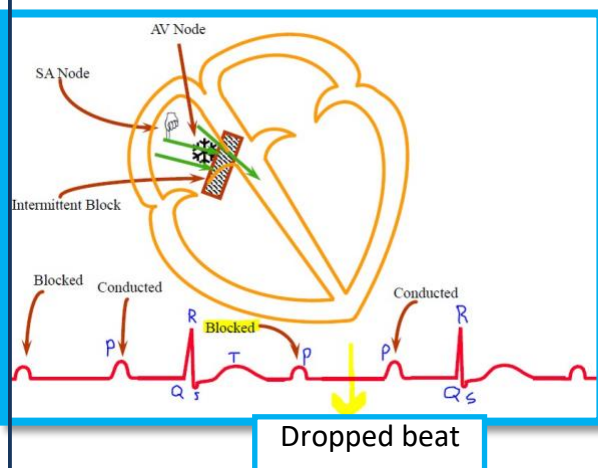
  ➤ Incomplete degree block: first degree block
    - P-R interval is prolonged (> 0.2sec) due to delay in the AV node or the bundle of His, and each P wave is followed by QRS.
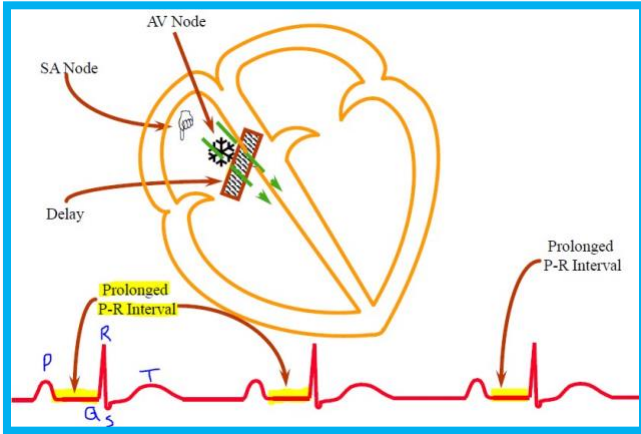
P-R interval $=10\ small\ square \times 0.04 = 0.4sec$ (prolonged), but followed by QRS, T waves.   More than 0.2

 ➤ Second degree: incomplete block
    - P-R interval is prolonged to (0.25-0.45sec), and *some* of the P waves are followed by QRS and others are not *(irregular heart rate).*
    - This irregular heart rate appears in *a regular pattern,* which means that in some cases we may have 3P waves for 2QRS, other cases may have 4P waves for 2QRS (or 3QRS…), so the irregularity repeats itself *(regular irregularity).*
    - We represent it using a ratio (#P waves: #QRS waves), examples: 3:2, 4:3, 4:2…
    - Etiology: some impulses pass through the AV node, and others do not, causing palpitation "dropped beats"
    - Atrial beats are faster than the ventricles.
    - Each successive atrial impulse encounters a longer and longer delay in the AV node until one impulse (usually the 3rdor 4th) fails to make it through the AV node.
      - So there is abnormality in the AV node
        ↳ Sometimes it permits conduction.
   ↳ Sometimes it doesn't conduct→Dropped beat.
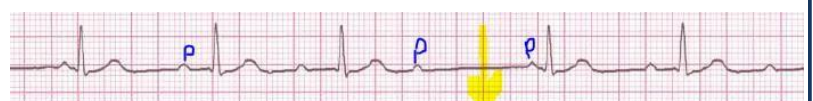
Dropped beat

Dropped beat

➢ Third degree: complete block

- Also P-R interval is >0.2sec but there is NO association between P waves and QRS (completely dissociated, complete block of AV node).
- Heart rate now depends on Purkinje fibers, and its intrinsic rate is between (15-40/min) so heart rate become 40bpm.
- Ventricles escape the AV nodal rhythm because they are not affected by the vagus nerve.
- On the ECG: we notice that P-R intervals are longer than normal, and there is no association between P and QRS.
- Etiology: there is complete block of conduction at the AV junction, so atria and ventricles form impulses independently from each other. Without impulses from the atria, the ventricles own the intrinsic pacemaker (Purkinje fibers) beats around (15-40 bpm).


HR=37
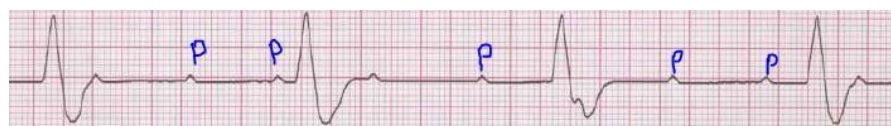
- *Stokes-Adams syndrome:*
  - The patient have pressure from the carotid sinus on the vagus nerve, so the vagus nerve is extremely stimulated causing a decrease in the heart rate, because the SA node & AV node might be suppressed completely, so that the heart stops beating for (15-30 sec), then the Purkinje resumes its own rate (15-40/min).
  - Complete AV block comes and goes (once carotid sinus presses on the nerve, suppression occurs).
  - Why the Purkinje do not resumes its rate right away? Because this is a biological system and it needs some time, normally, the Purkinje receives higher rate than its intrinsic rate (70 impulse per minute by the SA node) so that, the rate of conduction in the Purkinje will be 70/min and the ventricular muscle's rate of contraction will be 70bpm. So Purkinje's own rate (15-40/min) will be suppressed "overdrive suppression" (the ventricles are used to atrial drive).
  - Why does the Purkinje resume its own rate? Because it is not supplied by the vagus nerve and that's why ventricular escape occurs.

- Patients usually faints because of poor cerebral blood flow (no pumping activity, cardiac output), then, after Purkinje resumes its function, the patient gets conscious again.
- The treatment for the complete heart block is to implant an artificial pacemakers connected to the right ventricle.
- These pacemakers are usually battery operated, the battery is usually placed under the skin, nowadays, the battery can live for a longer time (might not be changed for 8-10 years) and people can live on these artificial pacemaker normal lives.
- Some -very advanced- pacemakers changes there rate automatically and others have fixed rate.

3) Factors causing electrical axis deviation:

- Remember: when we want to measure the mean electrical axis, we take any 2 limb leads (whether bipolar or unipolar)

  → Calculate algebraic summation of QRS waves on both limbs

  → Draw the vectors that represents QRS on both limbs→draw perpendicular lines starting from the point where the vectors end until both lines meet→draw a line starting from the center and ends at the site of meeting→this vector represents the mean electrical axis.
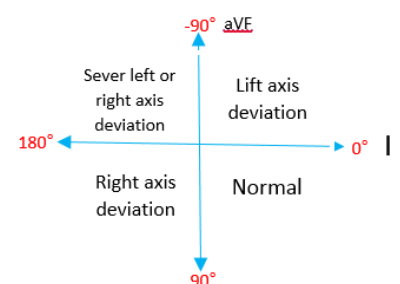

Left axis deviation!
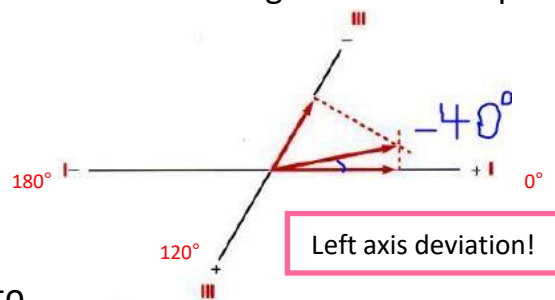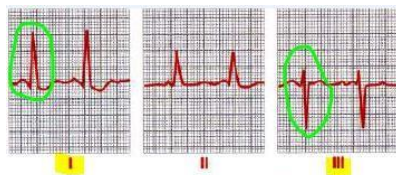
- *Left axis deviation:* could happen due to change in heart position caused by expiration, lying down, excess abdominal fat or short & obese people.
 - ➢ Note that we could have left axis deviation normally in some cases but deviation remains in the normal range (-30° to +110°).
  - ➢ Also it could be due to hypertrophy of the left ventricle caused by:
    - Hypertension: the heart develops too much pressure against the high pressure of the aorta.
    - Aortic stenosis: narrowing of the aortic valve so that the left ventricle needs to develop a lot of pressure to pump the blood into a narrow valve causing ventricular hypertrophy.
    - Aortic regurgitation incompetence of the aortic valve, so blood returns to the left ventricle during the ventricular contraction causing ventricular dilation "cardiomegaly".

Increase in kinetic energy

- The previous 3 conditions cause a slightly prolonged QRS (because of hypertension- lot of muscles) and high voltage.
- *Right axis deviation:* could be seen in thin & tall people.
  ➢ Also, It could be due to hypertrophy of the right ventricle caused by:
    - Pulmonary hypertension.
    - Pulmonary valve stenosis.
    - Interventricular septal defect (VSD): not completely closed, so that, blood at higher pressure in the left ventricles is pumped to the right ventricle which become hypertrophied and dilated "cardiomegaly".
    - The previous 3 conditions cause a slightly prolonged QRS and high voltage on the leads that are denoting the right side of the heart.
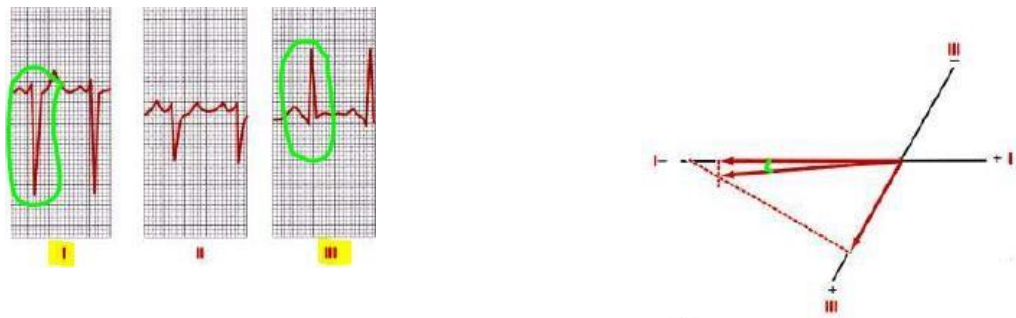
- *Left & right bundle branch block:*
  ➢ Left bundle branch block causes left axis deviation: when there is left bundle branch block, it means that the right ventricle depolarizes first and completes its depolarization before the left ventricle , and since current goes from depolarized to still polarized area (from right to the left ventricle) *most of the time there is current going from the right to the left*, and since the bundle branch is blocked, there is no way for the passage of the current except through the ventricular syncytium which conducts current in much slower rate (0.5m/s) than Purkinje fibers (4-5m/s). So left axis deviation occurs due to the movement of current from the right to the left, also the left bundle branch block causes prolonged QRS (depolarization takes longer time).
  ➢ Right bundle branch block causes the right axis deviation: left ventricle depolarizes first & right ventricle takes longer time so that, there will be current moving from the left to the right causing right axis deviation & prolonged QRS.