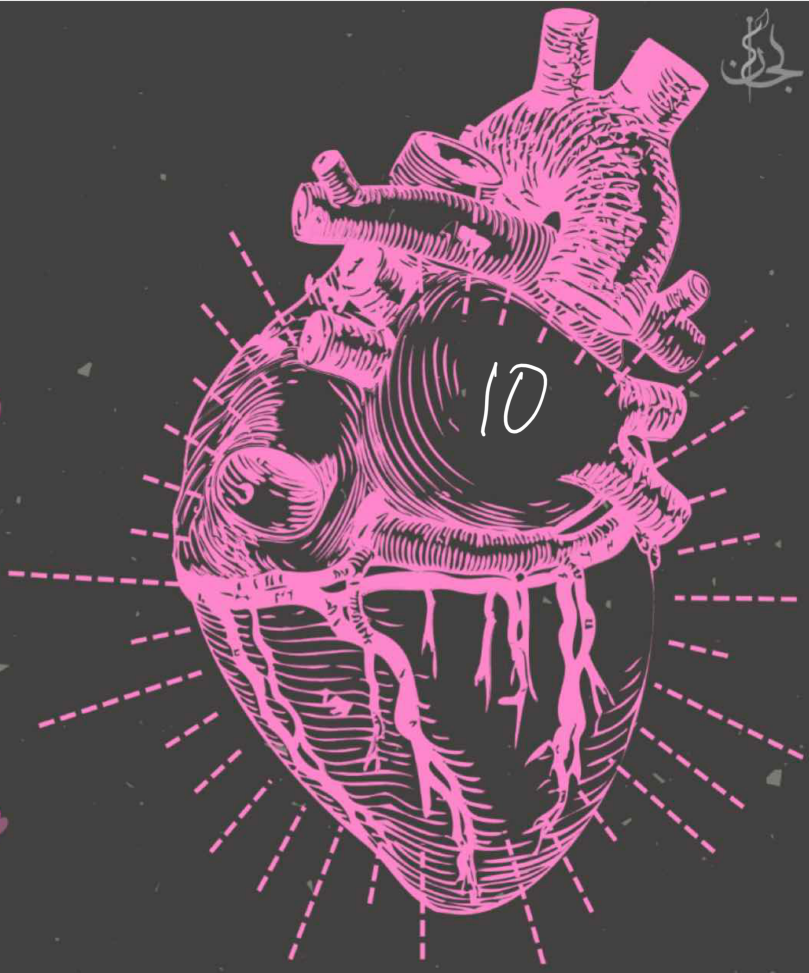
CVS



PHYSIOLOGY

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Electrocardiography-Abnormalities (Arrhythmias)

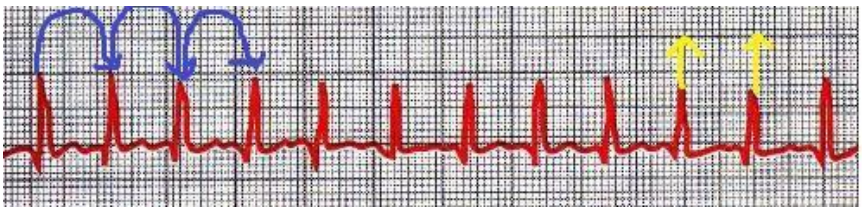
In the previous lecture, we talked about the normal electrocardiograph, in this lecture we are going to talk about the abnormalities in the electrocardiograph (arrhythmias).

❖ 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 =$0.4sec.
    - 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.

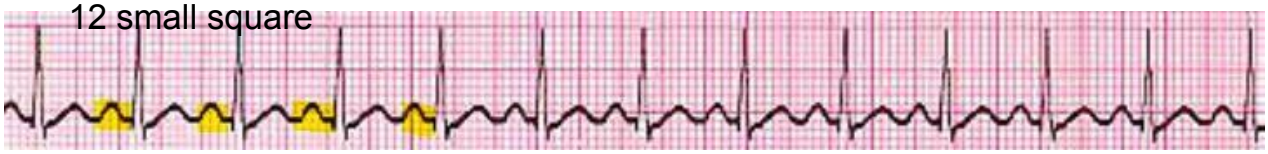


Increase in T3 -4 causes anabolism of receptors mainly beta

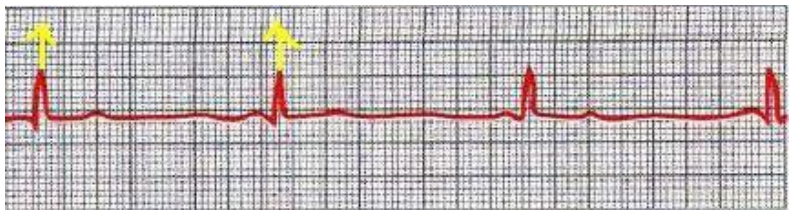
- *Sinus tachycardia:* tachycardia happens due to faster depolarization of SA node, the impulse is conducted normally.
  - On the electrocardiograph we will notice the presence of **P** wave, followed by QRS, then T wave.

Sinus tachycardia is a response to physical or psychological stress, not primary arrhythmia or sympathetic stimulation.

12 small square

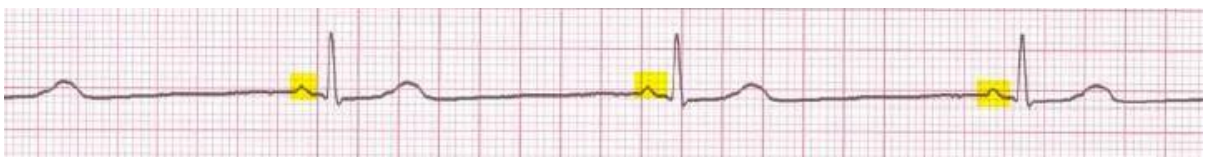


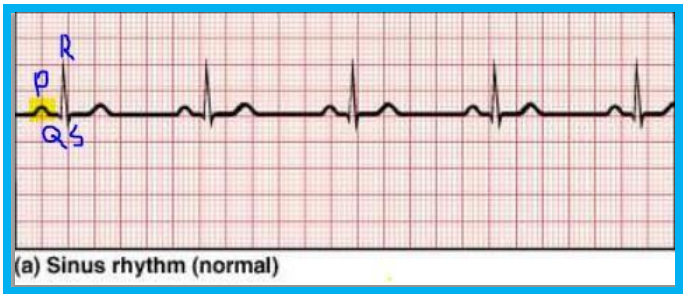
- *Bradycardia:* slow heart rate, usually less than 60bpm.
- The etiology of bradycardia:
  1. Sometimes can be presented normally in athletes, in those who have stronger hearts, so higher ability to pump blood out, thus, do not need more heart rate to have a normal cardiac output, they have *high stroke volume.* $cardiac\ output = stroke\ volume(\uparrow) \times heart\ rate(\downarrow)$.
  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= $35 \times 0.04 = 1.4sec$.
  - The heart rate= $\frac{60}{1.4} \approx 43bpm$, which is very low.



Less than 40 the heart rate

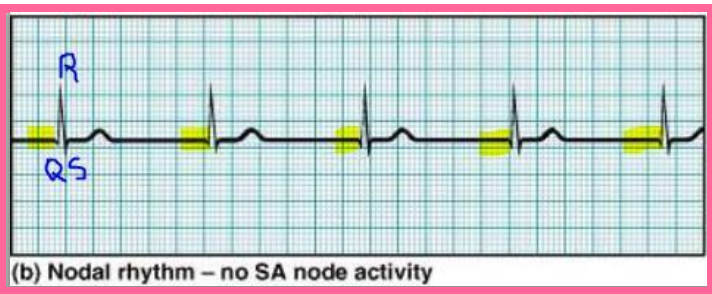
- *Sinus bradycardia:* bradycardia happens due to slower depolarization in the SA node, impulses are conducted normally, heart rate is lower than 60bpm.
  - On the electrocardiograph we will notice the presence of **P** wave, followed by QRS, then T wave.
  - Normal PR interval, which means normal conduction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



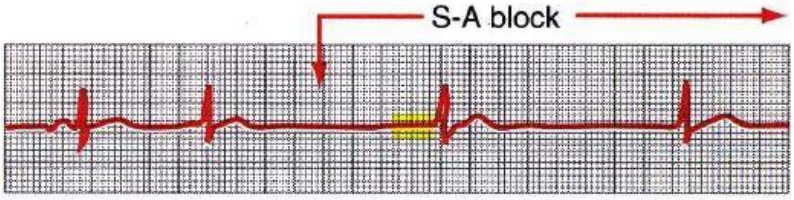
(b) Nodal rhythm – no SA node activity

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

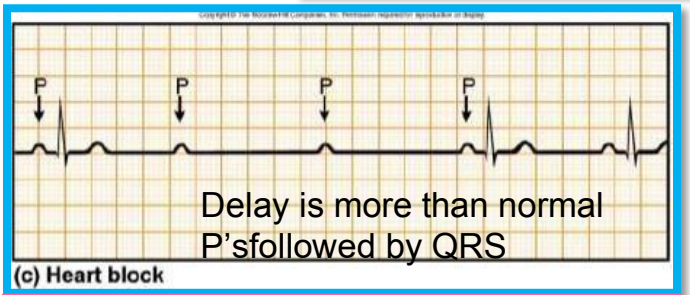
2) Blocks in the points of transmission:

- *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



Note: no P waves and slow rate

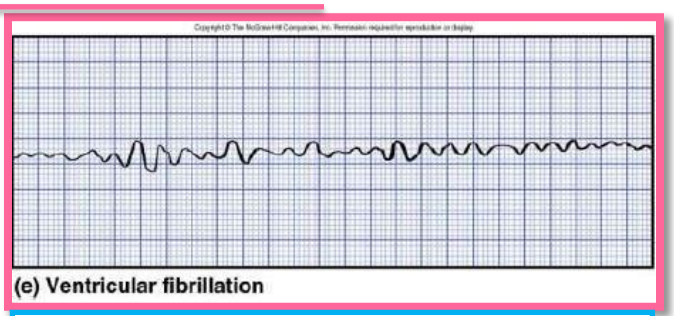


(c) Heart block

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:

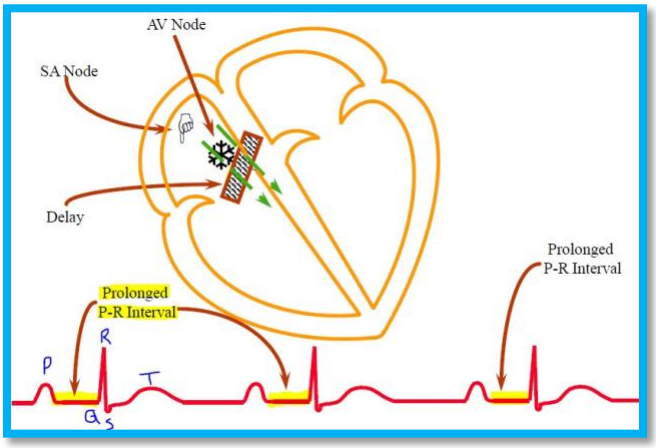
1. Ischemia of AV nodal or AV bundle fibers (coronary ischemia or pressure).
2. Compression of AV bundle (by scar tissue or calcified tissue).
3. AV nodal or AV bundle inflammation.
4. Excessive vagal stimulation (carotid sinus syndrome, strokes-Adams syndrome).

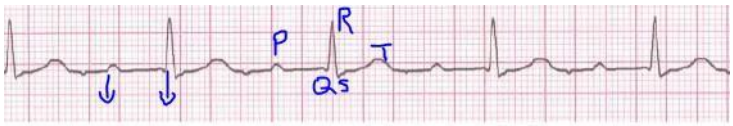
- There's 3 degrees of the atrioventricular block:

Normal P-R interval= (0.16-0.2sec)

➢ 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.

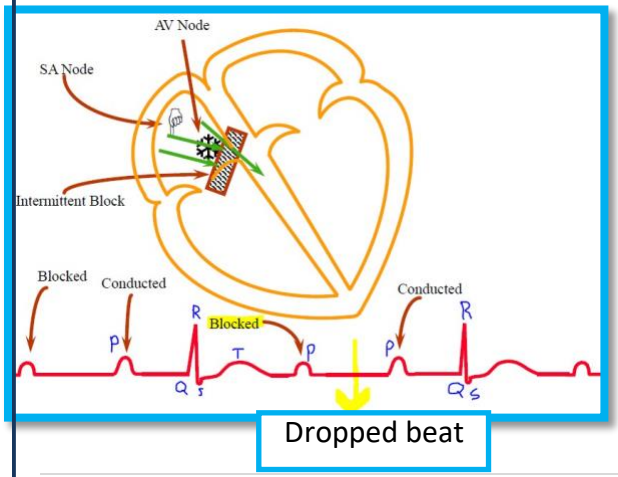




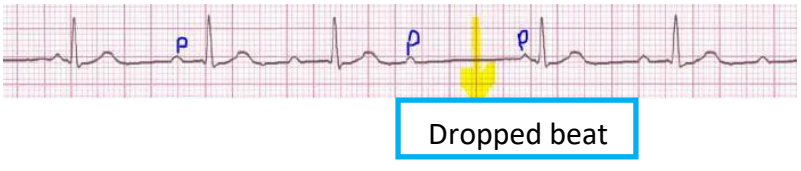
P-R interval $=10\ small\ square \times 0.04 = 0.4sec$ (prolonged), but followed by QRS, T waves. More than 0.2

➢ 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 3rd or 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.

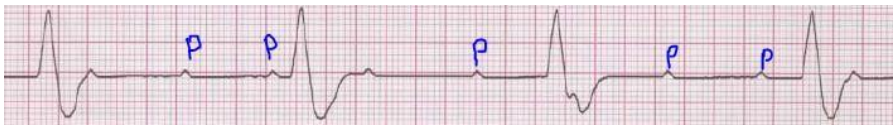


About point 3: This is what the doctor said in the video, however, it's written in the slides "ventricles escape AV nodal rhythm ensues"

without relating it to the vagus nerve.

➢ 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).



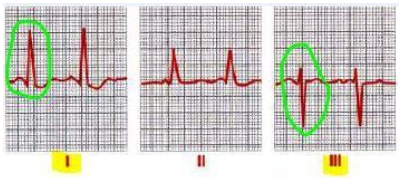
HR=37

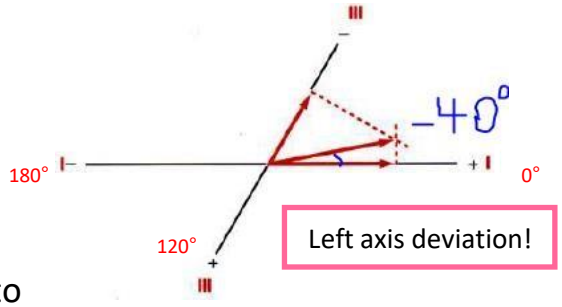
- *Stokes-Adams syndrome:*
  - The patient have pressure from the carotid sinus on the vagus nerve, so the vagus nerve is extremely stimulated causing a decrease in the heart rate, because the SA node & AV node might be suppressed completely, so that the heart stops beating for (15-30 sec), then the 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.

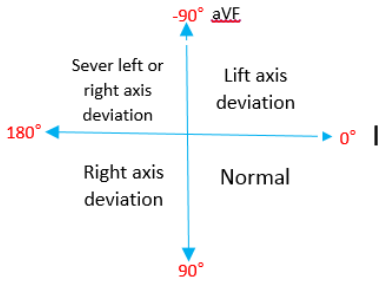
3) Factors causing electrical axis deviation:

- 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.





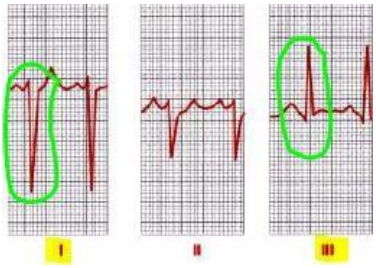
- *Left axis deviation:* could happen due to change in heart position caused by expiration, lying down, excess abdominal fat or short & obese people.
  - Note that we could have left axis deviation normally in some cases but deviation remains in the normal range (-30° to +110°).
  - Also it could be 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".

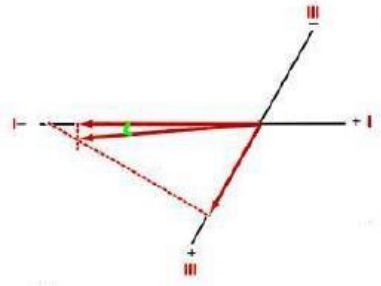


Increase in kinetic energy

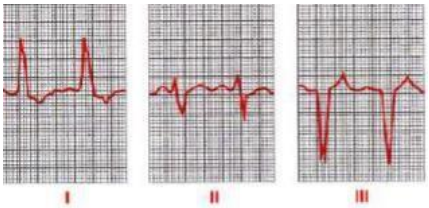
- The previous 3 conditions cause a slightly prolonged QRS (because of hypertension- lot of muscles) and high voltage.

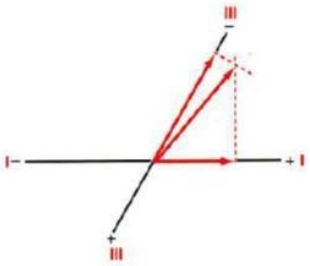
- *Right axis deviation:* could be seen in thin & tall people.
  - Also, It could be due to hypertrophy of the right ventricle caused by:
    - Pulmonary hypertension.
    - Pulmonary valve stenosis.
    - Interventricular septal defect (VSD): not completely closed, so that, blood at higher pressure 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.





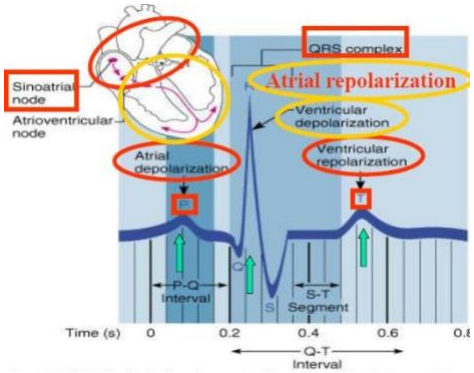
- *Left & right bundle branch block:*
  - Left bundle branch block causes left axis deviation: when there is left bundle branch block, it means that the right ventricle depolarizes first and completes its depolarization before the left ventricle , and since current goes from depolarized to still polarized 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.

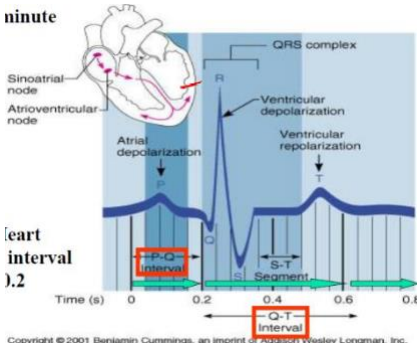




ECG deflection:

- SA node is the pacemaker, it denotes P waves (atrial depolarization).
- Then, we have QRS complex (ventricular depolarization) and within that wave we have atrial repolarization that does not show up since it is masked by the ventricular depolarization that have much electricity.
- After that we have the T wave (ventricular repolarization).
- P-Q interval: should be <0.2sec.
- Q-T interval: represents almost half of the R-R interval (half of the cardiac cycle).
- The cardiac cycle should be 0.8sec so that, the heart rate is 75bpm.
- The normal range for heart rate: (60-100bpm).
- 1st degree heart block: P-Q interval is longer than 0.2sec
- 2nd degree heart block: prolonged P-Q interval, but some P waves are followed by QRS, and others are not (regular irregularity).
- 3rd degree heart block (complete heart block): complete dissociation between P waves & QRS and heart rate is (15-40bpm).

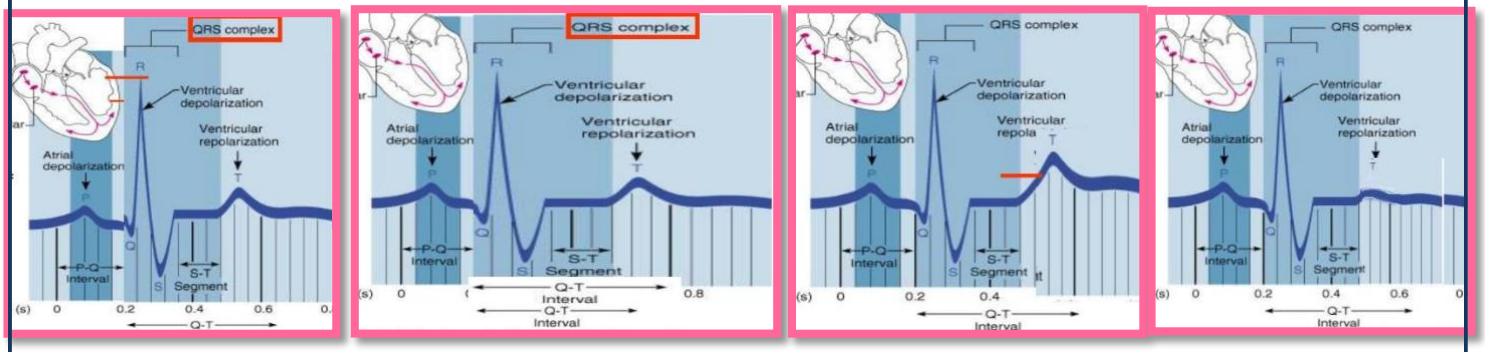




4) ECG deflection wave irregularities:

- *Enlarged QRS (high voltage):* could be due to hypertension, hypertrophy of the left ventricle.
- *Prolonged QT- interval:* repolarization abnormalities, increases chances of ventricular arrhythmias.
- *Elevated T wave:* caused by hyperkalemia. Normal = 5 / 6 ⇒ 4 By 20% so it is very dangerous.
- *Flat T wave:* caused by hypokalemia or ischemia.

- Note: ischemia causes positive or negative deflection of the S-T segment, so T-P segment will be the isoelectric line.



Enlarged QRS	Prolonged Q-T interval	Elevated T wave	Flat T wave

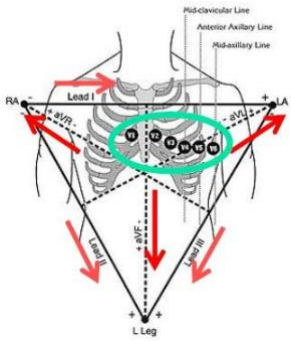
5) voltage abnormalities in standard bipolar limb leads:

- *Increased voltages in standard bipolar limb leads:* If the sum of voltages of leads I-III is greater than 4 mV, this is considered to be high voltage EKG.
- Most of the cases are caused by increased ventricular muscle mass (hypertension, marathon runner).
- *Decreased voltages in standard bipolar limb leads:* caused by cardiac muscle abnormalities (old infarcts causing decreased muscle mass, low voltage EKG, and prolonged QRS).
- Conditions surrounding the heart including fluid in pericardium (cardiac tamponade), pleural effusion, emphysema (enlarged lungs & chest mostly in smokers) in these cases the heart is far from the exploring electrode because there is “more layers” between the heart & the electrode.

Remember

We use 12 leads in electrocardiography:

- 3 limb leads.
- 3 augmented leads.
- 6 precordial (chest) leads.



Good luck