CVS

Physiology

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SHEET NO. : 7

ATRIOVENTRICULAR BLOCK

Av node is some how damaged. However, it might not be completely damaged, AP sometimes passes [but duration is longer than usual] sometimes it does not.

This block could be due to:::

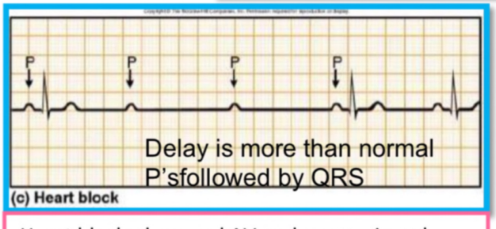
1. Ischemia of the av node, av bundle. Coronary ischemia; myocardial ischemia.

2. Compression of av bundle, by scar tissue (after infarction) or calcified tissue.

3. Av node or av bundle inflammation.

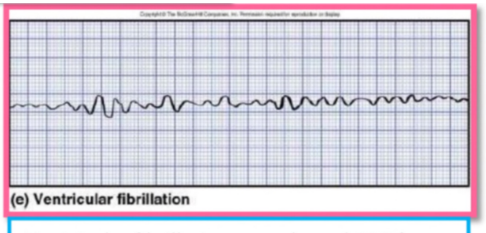
4. Excessive vagal stimulation (negative dromotropic effect).

5. Rarely by tumor, like atrial myxoma.



(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

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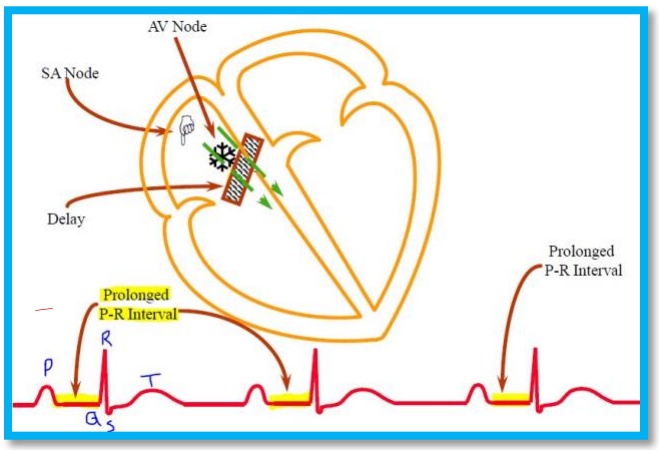
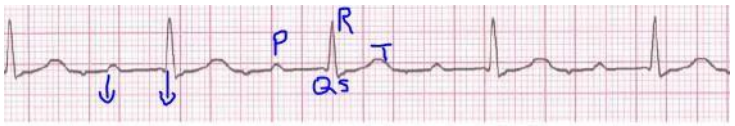
- There's 3 degrees of the atrioventricular block:
  - ➢ Incomplete degree block: first degree block

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

- 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.



Conduction from atria to ventricle is always there



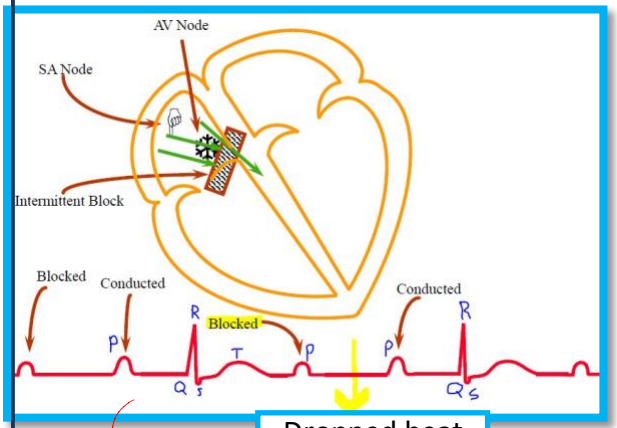
P-R interval $= 10\ small\ square \times 0.04 = 0.4 sec$ (prolonged), but followed by QRS, T waves. More than 0.2

Sinus bradycardia: P-R normal
Non- sinus bradycardia: P-R is not normal

  - ➢ Second degree: incomplete block

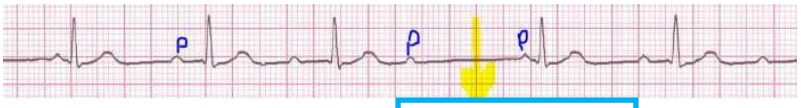
>> Incomplete block (1st & 2nd degree)
>> Complete block (3rd degree)

- 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).* → not always.
- We represent it using a ratio (#P waves: #QRS waves), examples: 3:2, 4:3, 4:2… note that: no.P > no. QRS
- 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.



this can be any lead.

- So there is abnormality in the AV node
  - Sometimes it permits conduction.
  - Sometimes it doesn't conduct → Dropped beat.



Dropped beat

palpitation

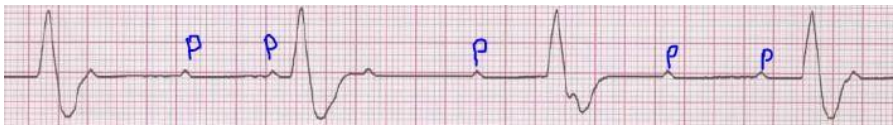
Here, atrial repolarization might show up, but since it's small it normally does not. If it shows up it must be negatively deflected.

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

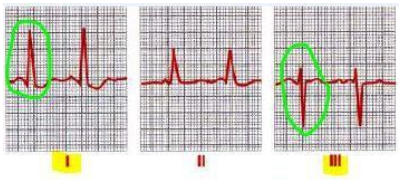
- *Stokes-Adams syndrome:*
  - The patient have pressure from the carotid sinus on the vagus nerve, so the vagus nerve is extremely stimulated causing a decrease in the heart rate, because the SA node & AV node might be suppressed completely, so that the heart stops beating for (15-30 sec), then the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

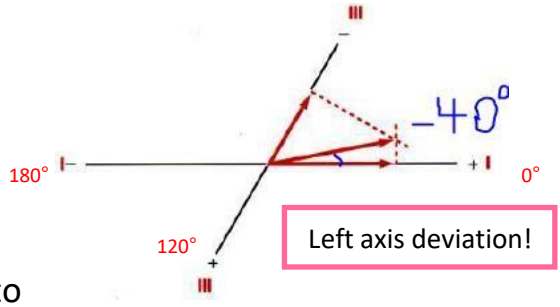
Artificial pacemaker is the treatment of any stage if needed.

- 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.

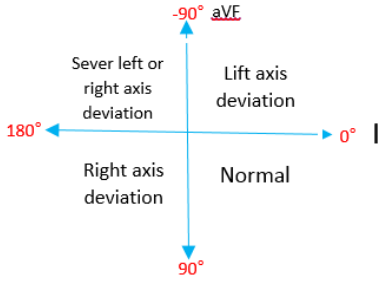
3) Factors causing electrical axis deviation:

- 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. Athletes also
  - ➢ Note that we could have left axis deviation normally in some cases but deviation remains in the normal range (-30° to +110°). on edge
  - ➢ Also it could be due to hypertrophy of the left ventricle caused by:



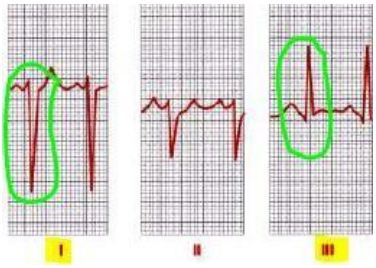
Large mass >> longer dep. time although the right ventricle has finished, that's why mean vector is pointed to the left

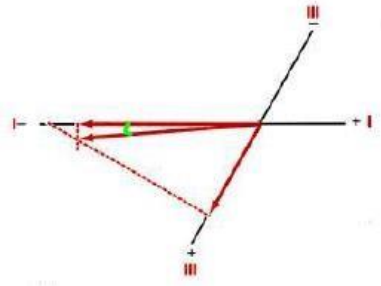
- Hypertension: the heart develops too much pressure against the high pressure of the aorta.
- Aortic stenosis: narrowing of the aortic valve so that the left ventricle needs to develop a lot of pressure to pump the blood into a narrow valve causing ventricular hypertrophy.
- Aortic regurgitation incompetence of the aortic valve, so blood returns to the left ventricle during the ventricular contraction causing ventricular dilation "cardiomegaly".

Increase in kinetic energy

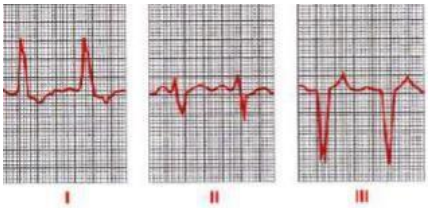
- The previous 3 conditions cause a slightly prolonged QRS (because of hypertension- lot of muscles) and high voltage.

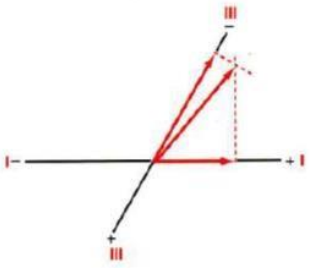
- *Right axis deviation:* could be seen in thin & tall people.
  - Also, It could be due to hypertrophy of the right ventricle caused by:
    - Pulmonary hypertension.
    - Pulmonary valve stenosis.
    - Interventricular septal defect (VSD): not completely closed, so that, blood at higher pressure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





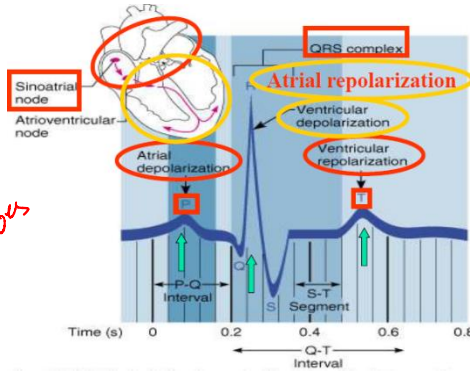
- *Left & right bundle branch block:*
  - Left bundle branch block causes left axis deviation: when there is left bundle branch block, it means that the right ventricle depolarizes first and completes its depolarization before the left ventricle , and since current goes from depolarized to still polarized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



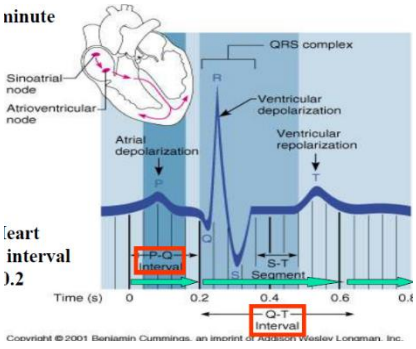


ECG deflection:

- 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).



Q in most of the time does not show.



4) ECG deflection wave irregularities:

- *Enlarged QRS (high voltage):* could be due to hypertension, hypertrophy of the left ventricle. ventricular dep. takes longer time ∴ hypertrophy [mostly left side].
- *Prolonged QT- interval:* repolarization abnormalities, increases chances of ventricular arrhythmias.
- *Elevated T wave:* caused by hyperkalemia.
- *Flat T wave:* caused by hypokalemia or ischemia.

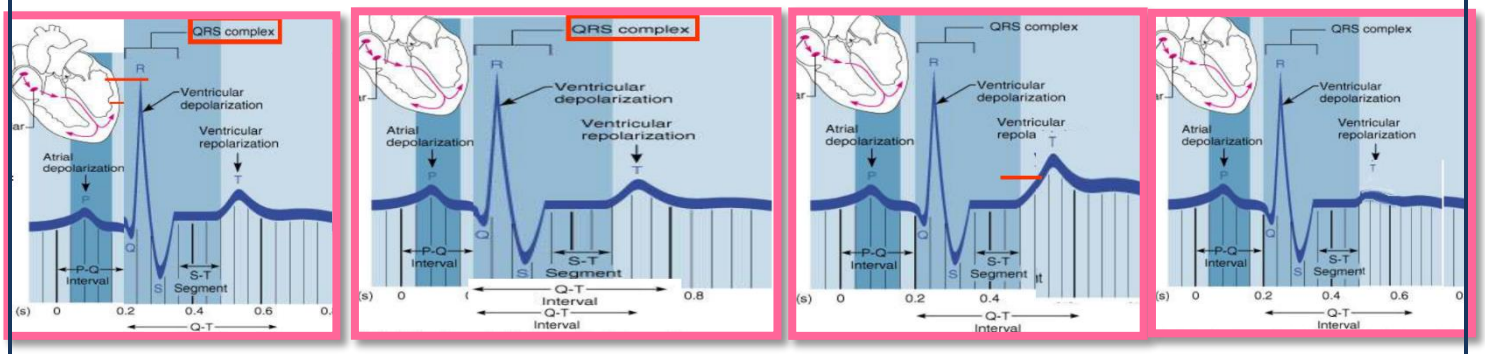
maybe caused by drugs.

T wave ↑ ∴ K^+ ↑ exp cases,

sometimes causes inverted T wave.

•Remember:- ECG → electrical record not mechanical!

- 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

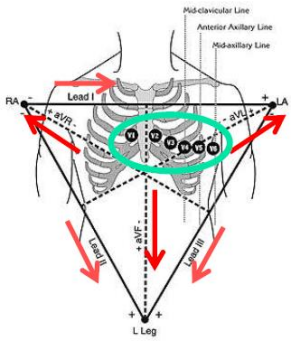
5) voltage abnormalities in standard bipolar limb leads: sum voltages of lead I, II, III

- *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.



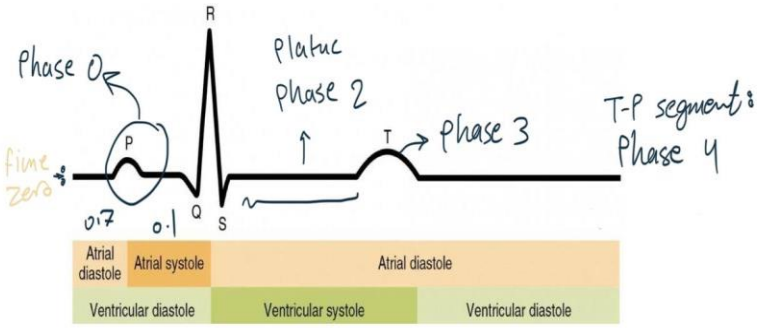
remember:- recorded as - or + without degree.

The cardiac cycle

going to understand:- volume, mechanical, electrical, pressure changes.

- **The cardiac cycle:** is the cardiac events that happens from the beginning of one heartbeat to the beginning of another.
- Each cycle is initiated by spontaneous generation of an action potential in the SA NODE.
- The cardiac cycle consists of a period of relaxation (ventricles do not contract) called diastole, during which the heart fills with blood, followed by a period of contraction called systole.
- One cardiac cycle (one heartbeat) normally takes 0.8 sec (20 small squares on the ECG). So, per minute we have about 75 beats (in other words, the heart rate=75 beats/minute.)
- If your heart rate increases, for any reason, up to 100 beats/minute. In this case, 0.6 sec is required for each cardiac cycle (15 small squares on the ECG) => in 1 minute, we will have more heart beats.
- When a cardiac cycle takes 1 sec (25 small squares on the ECG), the HR is reduced to 60 beats per minute.

❖ The picture below represents the normal ECG



P wave: recorded before atrial systole

QRS: recorded before ventricular systole

T wave: recorded before ventricular diastole

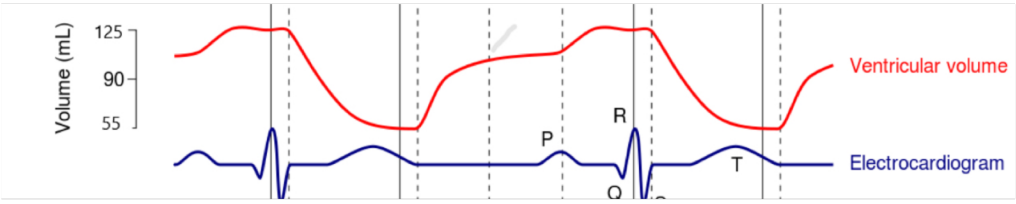
mechanical changes:-

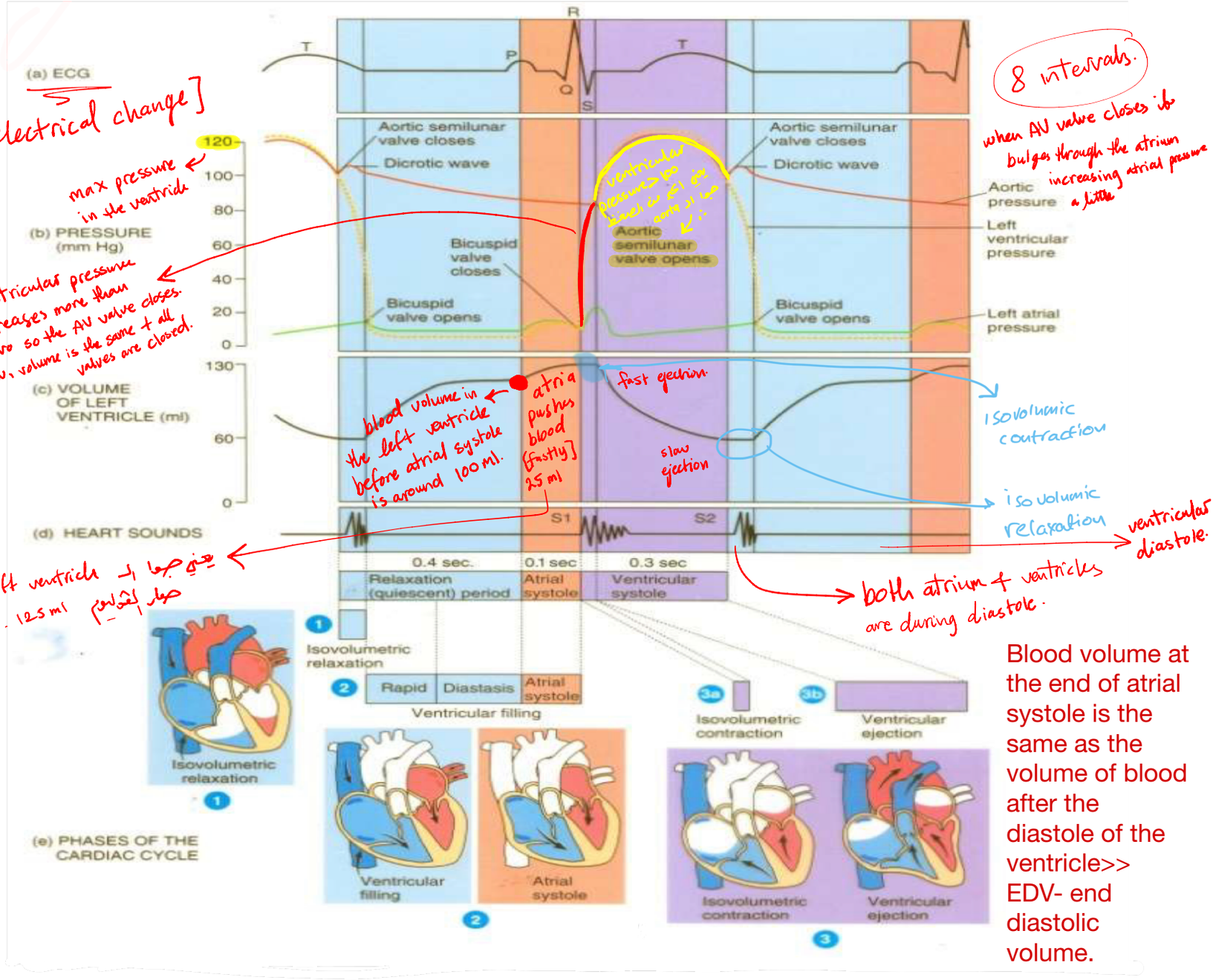
- Recall that the P wave is caused by spread of depolarization through the atria and is followed by atrial contraction (atrial systole), which takes about 0.1 seconds. Then, atria relax (atrial diastole), and that takes about 0.7 seconds. (.8 sec لحد cycle كل تخلص)
- QRS waves, as we already know, appear because of electrical depolarization of the ventricles, which initiates contraction of the ventricles (ventricular systole). This (ventricular contraction) takes about 0.3 seconds. Ventricular Relaxation (ventricular diastole) takes about 0.5 seconds.
- Atria and ventricles could overlap during the diastole (at the normal ECG they overlap for 0.4 sec), but not during systole.
- **Note:** The atrial conductive system is organized so that the cardiac impulse does not travel from the atria into the ventricles too rapidly; this delay allows time for the atria to empty their blood into the ventricles before ventricular contraction begin. AV delay.

Other changes during the cardiac cycle:

- Volume changes:

  Ventricular volume changes:





Blood volume at the end of atrial systole is the same as the volume of blood after the diastole of the ventricle>> EDV- end diastolic volume.

This picture sums up all the cardiac cycle , the doctor recommended trying to draw it

Atrial systole is NOT essential for the heart work and atrial fibrillation is NOT fatal (patient can live a normal life with anticoagulants)

even if the atria didn't contract, still the AV valve is open & the blood will move to the ventricle. here the valve is open because atrial pressure is higher than the ventricular pressure [not due to atrial contraction].

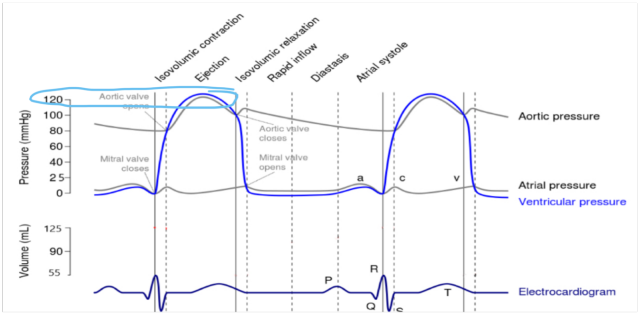
- Volume changes in both, the left and right ventricles, are the same. however, they defer in pressures.
- Before the atrial systole, the ventricular volume of blood is 100 ml. Once the atria contracts, the volume will increase to reach 125 ml (end diastolic volume). So, its contribution to the diastolic volume is around 25 % (maximum) and could even be less.
- When the ventricle contracts, rapid ejection of the blood takes place, because the blood in the ventricle was at a high pressure, after that the blood ejection slows down.
- Once ventricular systole is over, around 55 ml of blood remains in the ventricle => (end systolic volume).
- When the AV valve opens, the blood moves from atria to ventricles in three stages:

1- Rapid filling
2- Slow filling (diastasis)
3- Atrial contraction (last stage of ventricular filling)

AV valve is opened, why?

The atrial pressure during diastole of the ventricle is higher than the ventricular pressure

Pressure changes:



- The Aortic pressure during ventricular diastole is around 80 mm Hg, and the pressure in the ventricle, at the same time, is almost zero mm Hg.
  So, when atria contract, the pressure in ventricles increases a little bit reaching about 5 mmHg. However, and since the semilunar valves are closed, the pressure in the aorta/pulmonary artery remains intact.
- Once the pressure in the atria (about 2mmHg) becomes higher than that in the ventricle (about 0 mmHg), AV valves open, and the blood flows toward the ventricles, and when the atria contract, they push the blood to the ventricles so fast, so the ventricular pressure increases a little bit.
- When the ventricles are about to contract, the pressure inside them increases, so the pressure becomes higher than that in the atria, so the AV valves close.
- At this particular moment, when all 4 valves are closed (2 semilunar, aortic, pulmonary), the first part of ventricular contraction takes place. This is known as **isovolumic contraction**.
  As the name indicates, the volume of blood inside the ventricles doesn't change. However, the pressure increases sharply. Here we are taking the left side of the heart as an example. This happens because the pressure in the left ventricle has not yet exceeded the pressure in the Aorta (semilunar valve is closed). When the ventricular pressure becomes higher than that in the aorta, the semilunar valve opens, and the blood is pumped from the left ventricle to the aorta. It is important to know that the pressure in the ventricle is still going up, and at the same time the pressure at the aorta will go up too but still less than the pressure in the ventricle (around 1 mm Hg less).

What prevent AV valve to be open?

Cordea tendinea

- At the end of systole, the pressure in the ventricle becomes a little bit less than the pressure in the aorta. Yet, the blood keeps going from the ventricle to the aorta because of the momentum of the blood الزخم then the pressure in the ventricle starts to fall as the volume of the blood starts decreasing.

End diastolic volume is 125ml.
End systolic volume is 55ml.
70ml is ejected per beat (stroke volume.
Now, stroke volume multiplied by the heart rate= cardiac output.

- When the pressure at the aorta becomes higher than the pressure in the ventricle, the semilunar valve closes. At this moment the 4 valves are closed, and this very short period is known as **isovolumic relaxation.**

At the end of isovolumic relaxation, the AV valve is open, why?
Because the atrial pressure is higher than the ventricular one

- Then, the pressure drops down until it becomes around zero. When this happens, AV valve opens, so blood starts to flow to the ventricle increasing the pressure and another cycle starts, the pressure in the aorta drops back to 80 mm Hg.

  ✓ The highest pressure in the ventricle during systole is around 120 mm Hg, and the highest-pressure during systole in the aorta is around 118 mm Hg.

  ✓ When measuring the blood pressure from the arm we say that the pressure is 120/80 mm Hg, which means that the pressure during the systole in the aorta is 120 mm Hg, and the pressure during the diastole is 80 mm Hg.

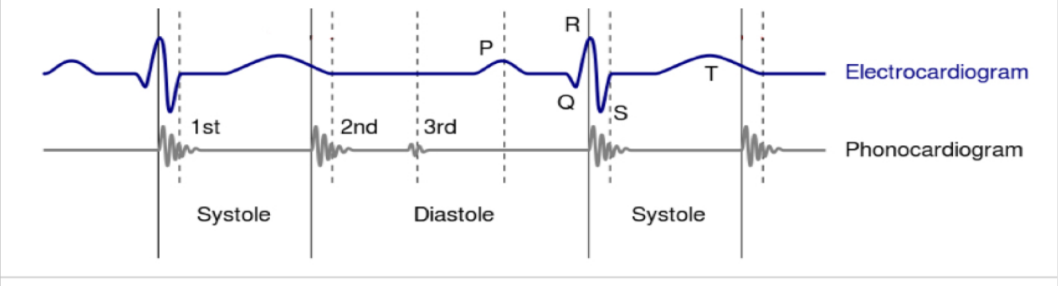
- The pulmonary pressure during the diastole equals 8 mm Hg, and during systole equals 25 mm Hg. Keep in mind that whatever happens in the left side of the heart happens in the right side (the only difference lies in the pressure values in the right and left chambers)
- The pressure of the right ventricle during diastole equals almost zero mm Hg, and during systole it's 25 mm Hg.

• pressure in the ventricle [zero – 125].
• and in the aorta [70 – 110].

NOW,

- When heart rate increases, the duration of each cardiac cycle decreases, including the contraction and relaxation phases. The duration of the action potential and the period of contraction (systole) also decrease, but not by as great a percentage as does the relaxation phase (diastole). Even though the stroke volume may get affected (reduced), the cardiac output will still be increased (due to increased heart rate).
- With each beat, we start with end diastolic volume (EDV) and end with end systolic volume (ESV). Having said that, the difference between EDV (125 ml) and ESV (55 ml) is called the stroke volume (SV), which equals the amount of blood ejected from the left or right ventricle per beat, and we use SV to calculate the cardiac output, which can be calculated by multiplying SV with the heart rate.
- So, if the EDV equals 125 ml, and ESV equals 55 ml, SV = 125 – 55 which equals 70 ml, and when the HR equals 75 bpm, the CO = 70 * 75 = almost 5L/min.

Changes in sounds:

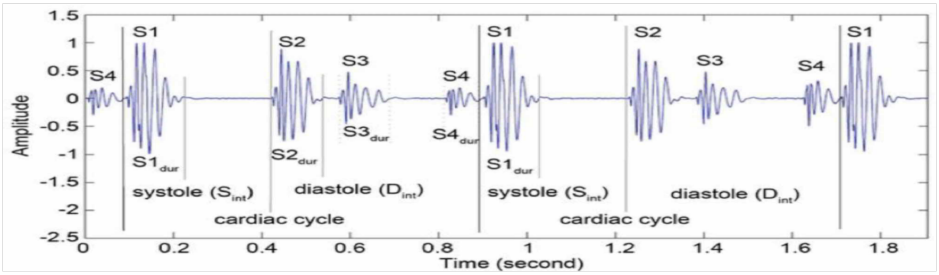


- Auscultation = listening to heart sound using stethoscope
- Once the AV valve closes, the blood tries to go back from the ventricle to the atrium, but because the pressure in the ventricle, now is higher than that in the atrium" during ventricular systole", blood remains sequestered in the ventricle.
- The two major sounds heard in the normal heart sound like "lub dub". The "lub" is the first heart sound, commonly termed S1, and is caused by turbulence caused by the closure of AV valves at the start of systole.
- The second sound," dub" or S2, is caused by the closure of semilunar valves, marking the end of systole.

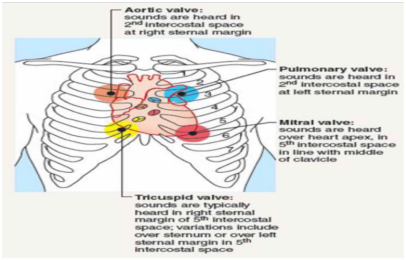
First heart sound is due to movements of blood around the closed av valve. And the second heart sound is due to the movement around the semilunar valve. Time between them both .3sec and the time between the second and the next first sound is .5sec

this page will be explained in the next lecture inshallah.

- The period elapsing between the first heart sound and second sound defines systole (ventricular ejection) and the time between the second sound and the following first sound defines diastole (ventricular filling).



- Incisura or dicrotic notch wave is caused by semilunar valve closure. When the valve closes, the pressure around the valve increases because blood accumulates near the valve as it tries to go back to the ventricle, which increases the aortic pressure.



- When the AV valve opens after the isovolumic relaxation,

  you might hear a third cardiac sound (S3) which is the sound of blood flow from the atrium to ventricle.
  Another sound (S4) is the sound of atrial systole.

General notes
> why do we have Delay in the cardice cyele?
: to make sure that the systoles don't occur toyether
> Prior to
: Just befor, No time between 2 events
> coincide:
Two events synced together [at the same time]
>right and left ventricles have The same variables [volume, ejection time]
-> the cardiac cycle= atrial systole, ventricle systole and then the diastoles with an over all of
0.4 second + 0.1 atrial systole + 0.3 ventricular diastole = 08 cardiac cycle .