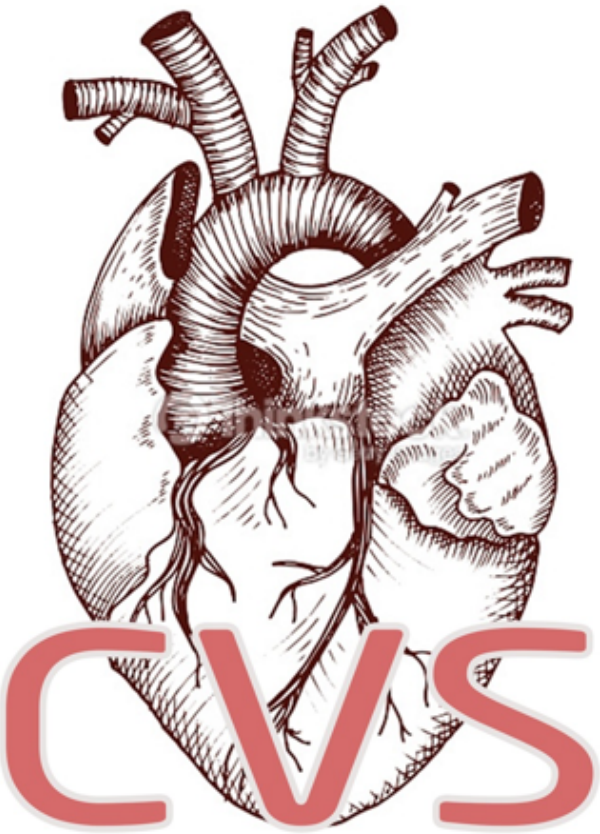


CVS

Physiology

♡ Slide Sheet ♥

Number:	10
Doctor:	Faisel Mohammed
Done by:	Abdel Mu’ez Siyam
Corrected by:	Ziad Fraij

This sheet was written based on section two's recording.

The lecture was started by questions; please don't skip.

Q1/ We said that ventricular volume started as 100 ml, atrial contraction made it 125 ml, ejection lowered it to 55 ml, how to return to 100 ml?

Answer: Just after Isovolumic relaxation the AV valves open, leading to the rapid filling phase (what is collected in the atria during ventricular systole will pour into the ventricles), followed by the diastasis phase (slow filling). These processes increase ventricular volume to re-reach 100ml just before the next atrial systole. So basically the ventricles fill during diastole.

What is the significance of this?

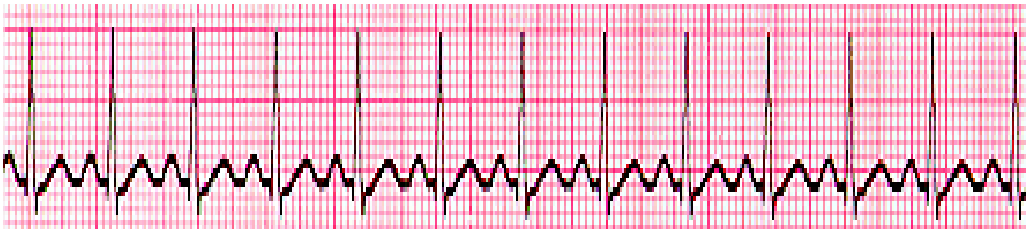
When the heart rate increases substantially, (like if it's on the higher side of normal limit of 100 beats/min, this means that each cardiac cycle will take around 0.6 sec), this shortening mainly affects the diastole (both systole and diastole are reduced, but diastole is markedly decreased). In this new situation (cycle = 0.6 sec), the time of systole becomes 0.28 sec (instead of 0.3 sec in a 0.8 cardiac cycle), while diastole duration becomes 0.32 sec instead of the original 0.5 sec (so markedly decreased relative to systole). This means that the time for filling is shortened, so the ventricles are filled with less blood, and consequently eject less blood. So basically, increased heart rate → compromise ventricular filling time → compromise the stroke volume.

****

We talked previously about the ECG, and we mentioned some cardiac arrhythmias, such as

1- Sinus arrhythmias: sinus tachycardia and sinus bradycardia.

Sinus→ each time you'll have P followed by QRS followed by T (no missing waves, just the firing rate from the SA node is different; higher (tachy) or lower (brady) than normal.

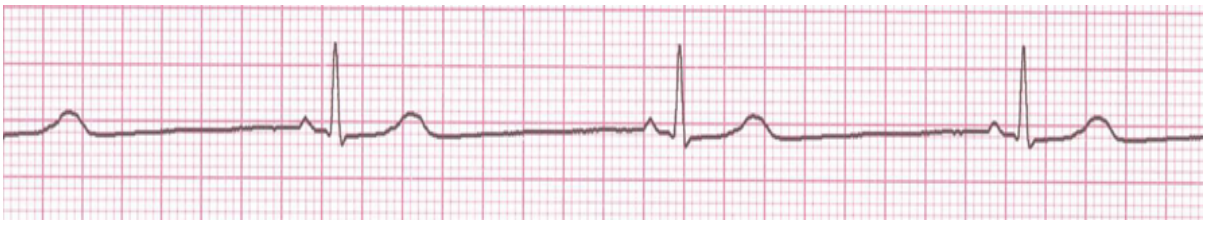


⇒This ECG represents ***sinus tachycardia,*** note that the heart rate is markedly increased (Tachycardia = heart rate > 100 beats/min) while the conduction rate does not change. Actually there are many ways by which you can calculate the heart rate if it is regular:

1- Since each small square represents 0.04 sec, and each large square represents 5 small squares (0.04 x 5 = 0.2 sec), so you can count the number of small or large squares to see the time for one cardiac cycle, then divide by 60. So if between one R and the next R (one cardiac cycle) takes 1 large square (5 small), then the heart rate equals 60/ (time of one cycle) = 60/0.2 = 300 beat/min and so on.

2- 1 Large square = 0.2 sec. so a minute contains 5 large squares (to reach one second) x 60 (to reach one minute) = 300 large square. So see how many large squares are there in a cardiac cycle and divide: 300/number of large squares in one cardiac cycle. For example: R→R takes 2 large squares. The heart rate equals 300/2 = 150 beat/min. (OR 60/ (0.2x2) = 60/0.4 = 150 beat/min).

3- Similarly, one minute contains 5 small squares (to reach one large square) x 5 (to reach one second) x 60 (to reach one minute) = 1500 small squares. So the heart rate = 1500/number of small squares. If P→P takes 15 small squares for example, then the heart rate = 1500/15 = 100 beat/min.
OR 300/(15/5) = 300/3 = 100 beat/min.
OR 15 x0.04 = 0.6, so the rate = 60/0.6 = 100 beat/min.



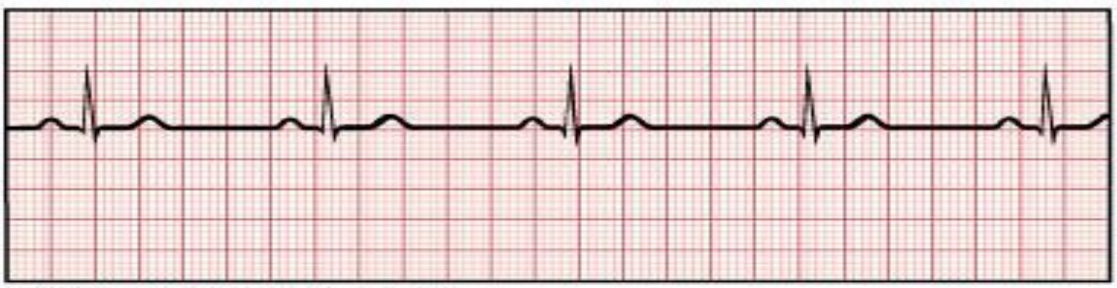
⇒This is sinus bradycardia (bradycardia: heart rate < 60 beats/min).

*SA Block:

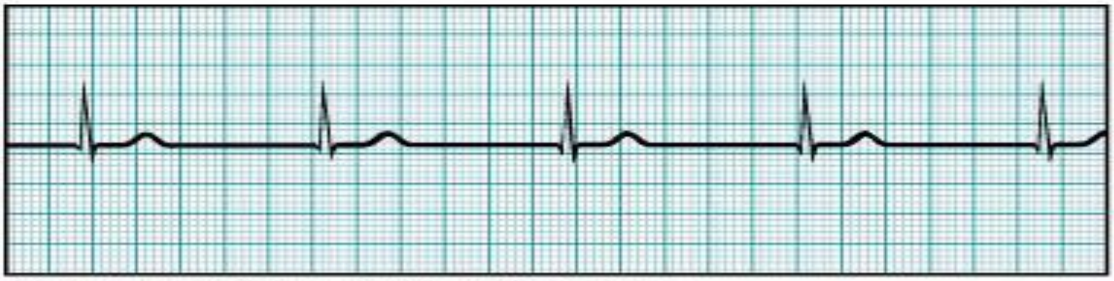
Sometimes the impulses from the SA node are blocked, so no impulses are generated (SA dysfunction/block) from the SA node and this will lead to:

- 1- No P waves (which are due to spread of the impulse generated by the SA node throughout the atria, so no impulse → nothing to spread! → no Ps).
- 2- The ventricles will work by the rate of the AV node (assuming that it's normal), so the rate will be lower but we'll see QRS complexes always followed by T waves.

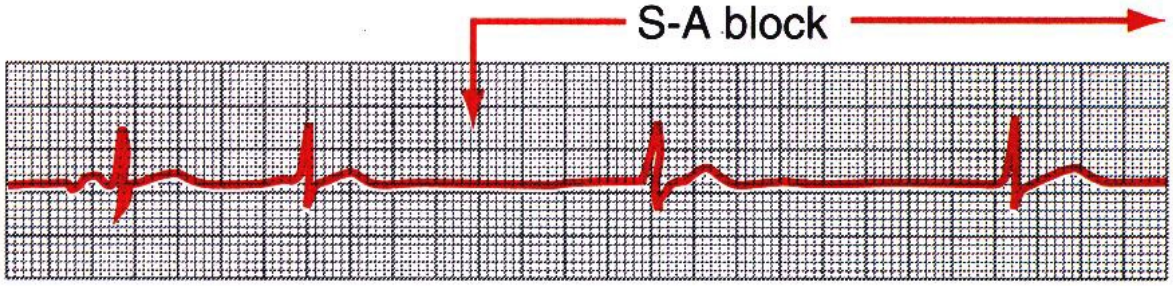
- The figure below (B and the one below) shows SA block, notice the absence of P waves and the lower rate.



(a) Sinus rhythm (normal)



(b) Nodal rhythm – no SA node activity



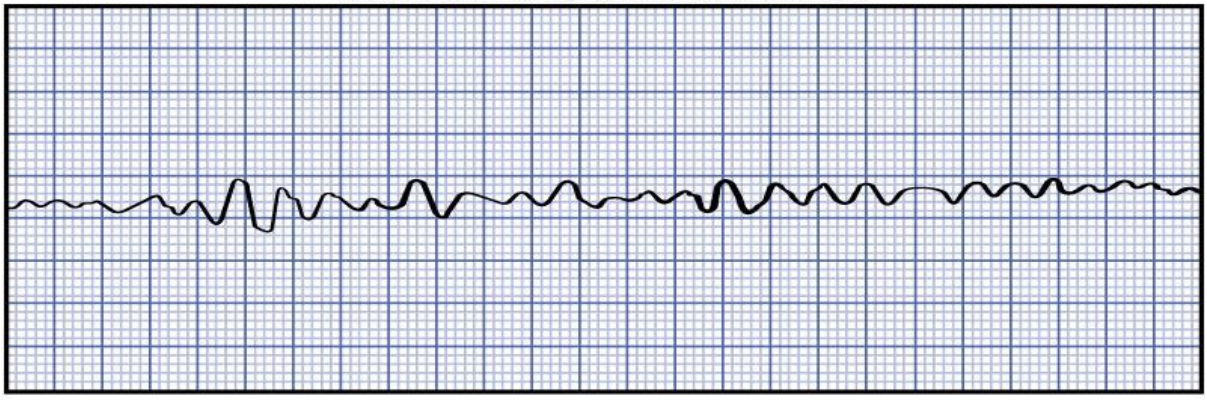
*Ventricular fibrillation:

A condition where each ventricular fibre depolarizes (and thus contracts) by its own regardless of other myocytes (so the ventricle is no longer a syncytium). This manifests as *loss of the QRS complexes*. The ECG is bizarre and shows no regularity.

- No synchronized contraction → loss of pumping action, and this is why ventricular fibrillation is **lethal** (no blood is ejected because single myocytes' contraction cannot generate enough force to eject blood). So the cardiac output is zero, which will lead to infarctions and cerebrovascular accidents eventually causing death.

- Many cells depolarizing independently → each will send its own signal on the ECG (each fibre will have some contribution to a wave in the ECG, but these waves are haphazard, and no regularity or synchronization can be found between them).

Figure of Ventricular fibrillation ECG is shown below:



(e) Ventricular fibrillation

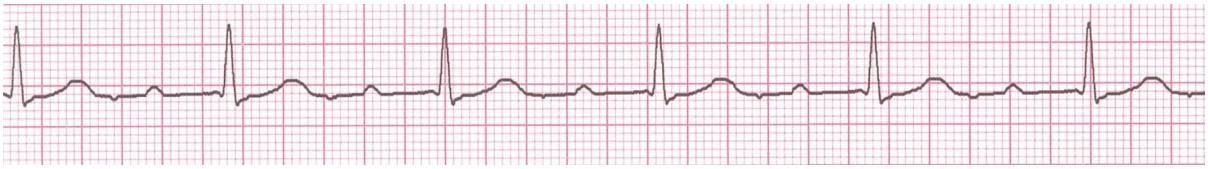
*AV block:

There are three degrees:
1st degree block: prolongation of the PR interval, but each P wave is followed by a QRS complex (so the AV node delayed the conduction of the impulse from the atria to the ventricles more than usual, but it conducts every impulse it receives).

2nd degree block: some P waves are followed by QRS and some are not (not every impulse the AV receives is being conducted), the ones that are not conducted cause dropped or skipped beats (palpitation) –One knows how the heart beats and can imagine how the beats rhythm occur, so you would expect a beat to take place but nothing happens, and it feels as if the heart fell down, this is the skipped beat.

3rd degree block (complete heart block): complete damage of the AV node, so no impulses are conducted to the ventricles, which start to depolarize with the rate of the purkinji fibres. So what you'll notice is that: the rate (QRS to the next QRS) is markedly **decreased** (less than 40) because the rate of firing of the purkinji fibres is slow (15 – 40/min). In addition, there is complete dissociation between the atria and the ventricles, so there will be no rules where the P waves will appear.
Note that the Atria → work by the rate of the SA node and their P waves are regular with each other.
While the ventricles → work by the rate of the purkinji fibres and the QRS complexes are regular together.
But there will be no regularity/synchronization between the atria and the ventricles.

The following figures are examples:

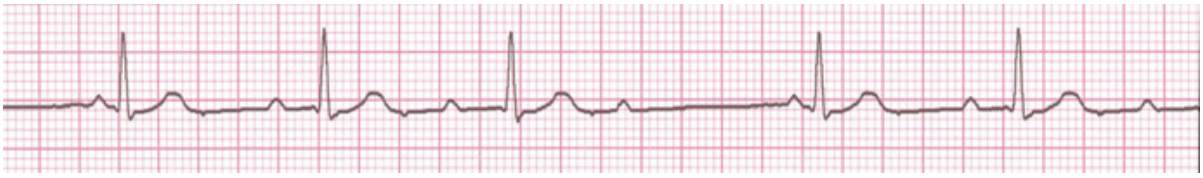


⇒This is 1st degree block.

1- Always P→QRS→T. (no missing P or QRS or T).

2- Regular rhythm (all cardiac cycles take the same time).

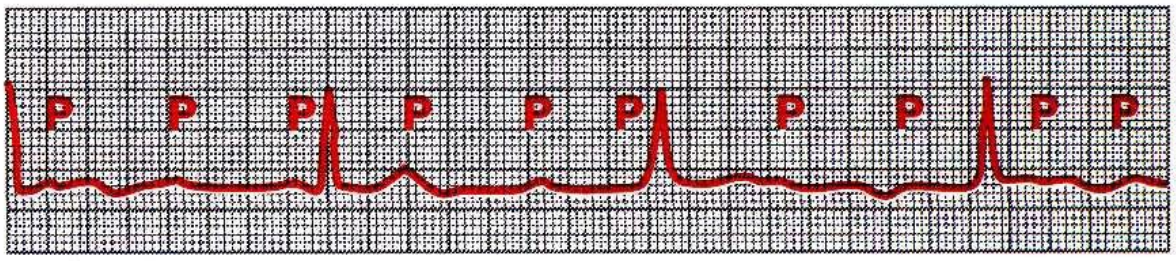
3- Prolongation of the PR interval, which normally should not exceed 0.2 sec, while here it is much higher (around 0.36 sec).

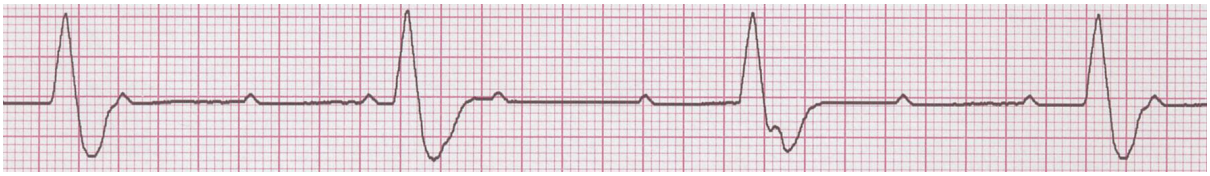


⇒This is 2nd degree block.

1- The number of P waves > number of QRSs

2- Regular rhythm (when the impulse is conducted).





⇒Both of the above figures represent 3rd degree block.

1- Complete dissociation of atrial and ventricular rates (between Ps and QRSs).

2-**VERY IMPORTANT**: for the exam, in order to answer 3rd degree block, please calculate the ventricular rate before (by R→next R), it MUST be less than 40/min.

❖ Stokes Adams Syndrome: (important concepts are in bold)

Excessive vagal stimulation (parasympathetic - maybe caused by carotid enlargement which compresses the vagi) will keep having negative chronotropic and dromotropic effects on the SA node and the AV node respectively, up to a point that causes severe suppression of the SA and AV nodes to the extent that

the heart will stop, so **complete heart block occurs**. For the ventricles which are not supplied by the vagi, it takes them some time (5 – 30 sec) to start working by the purkinji fibres' rate (15-40 beats/min). This is because the purkinji fibres are normally suppressed (**overdrive suppression**) by the higher rate that is coming from the SA node, so after realising that nothing is suppressing it (this requires 5 – 30 sec), the purkinji fibres then start working and firing on their own but with a lower rate.

-**Ventricular escape**: the ventricles escape the effect of the vagus nerve, so the SA and the AV are influenced by the vagus, but if the ventricles are working with the purkinji rate, no matter how much you stimulate the vagus they will not stop.

❖ Axis deviation:

- Right axis deviation (RAD) vs. left axis deviation (LAD)
- RAD → may be normal in tall and thin people (Max. normal is up to +110°).
- LAD → may be normal in obese and short people (Max. normal is up to -30°).
- Dextrocardia (the heart is on the right): everything is reversed. The normal mean electrical axis is to the right, so if this axis shifted to the left (this view in other people is normal) but in him it is a left axis deviation; because the start point is different, so if the normal is 60° and a LAD turned it 100° to the left, the new degree is -40°, while in dextrocardia, let's say the normal is 150°, and a LAD of 100° would make the angle 50°. An angle of 50° is normal in normal patients but in this particular case it represents a LAD (not normal).

LAD may be caused by:

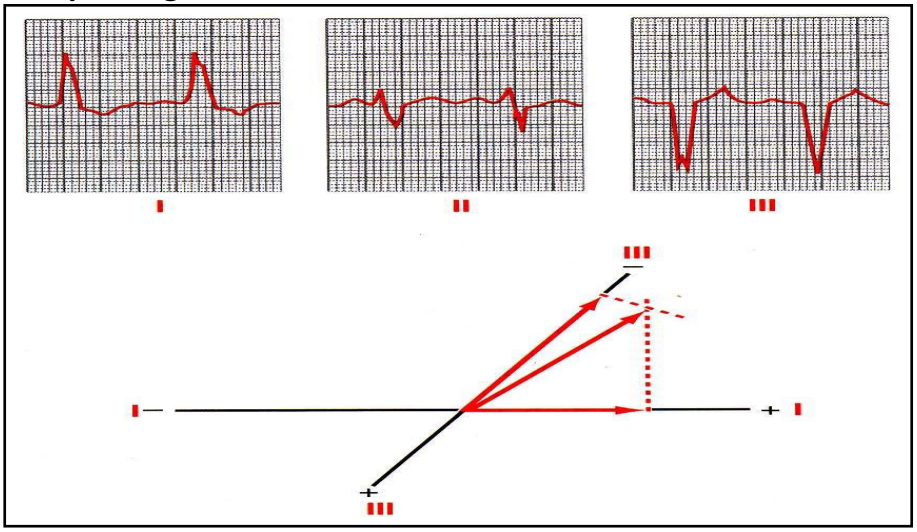
1- Hypertrophy of the left ventricle caused by hypertension, aortic stenosis or aortic regurgitation (aortic incompetence). These cases hinder the pumping of blood from the ventricle causing it to require a higher effort, and this is done by hypertrophy.

2- **Left** bundle branch block causes **left** axis shift because **right** ventricle depolarizes much faster than **left** ventricle, which depolarizes not by the bundle branch (it is blocked!) but by the muscle cells themselves (which conduct the depolarization much slower than the bundle branch, so the duration until all the left ventricle is depolarized is increased) and we know that vectors are directed from the depolarized to the still polarized areas, so new vectors arise from the right ventricle towards (facing) the left ventricle thus increasing the number of vectors facing the left and causing LAD. Note that the QRS complex is prolonged because it appears due to ventricular depolarization, so since the duration of depolarization is prolonged (due to slower depolarization of the left ventricle), the QRS is prolonged.
This prolongation is what differentiates between LBB block and Lt.Vt.hypertrophy.

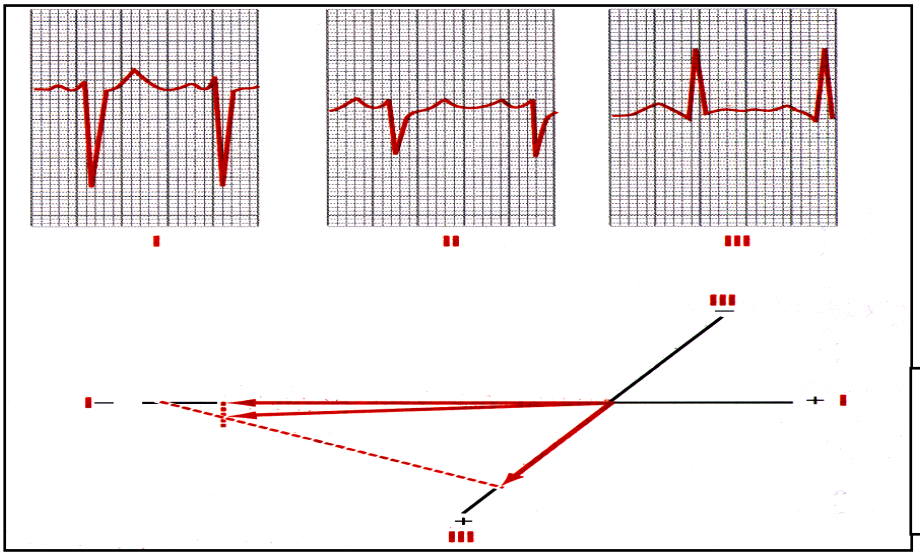
RAD may be caused by:

1- Hypertrophy of the right ventricle caused by pulmonary hypertension, pulmonary valve stenosis or interventricular septal defect. These cases hinder the pumping of blood from the ventricle causing it to require a higher effort, and this is done by hypertrophy.

2- **Right** bundle branch block causes **right** axis shift because **left** ventricle depolarizes much faster than **right** ventricle, which depolarizes not by the bundle branch (it is blocked!) but by the muscle cells themselves (which conduct the depolarization much slower than the bundle branch, so the duration until all the right ventricle is depolarized is increased) and we know that vectors are directed from the depolarized to the still polarized areas, so new vectors arise from the left ventricle towards (facing) the right ventricle thus increasing the number of vectors facing the right and causing RAD. Note that the QRS complex is prolonged because it appears due to ventricular depolarization, so since the duration of depolarization is prolonged (due to slower depolarization of the right ventricle), the QRS is prolonged. **This prolongation is what differentiates between RBB block and Rt.Vt.hypertrophy.**



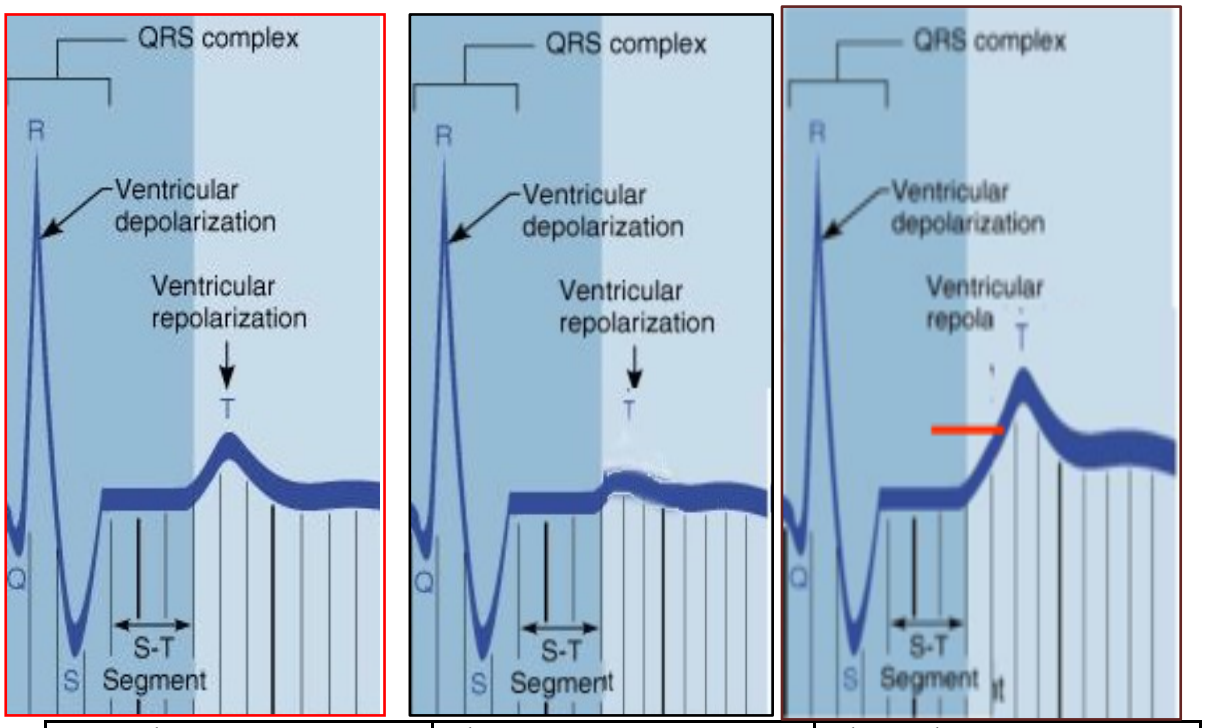
Left axis deviation caused by left bundle branch block. Notice the prolonged QRS. NOTE: in lead III here, the 1st QRS displays 2 peaks (like 1 S and another S, so it resembles letter M directed downward). The presence of 2 peaks occurs in bundle branch block. This is due to great difference of depolarization speed between the two ventricles, so each ventricular depolarization is recorded as a peak (as if the ventricles are separated).



Right axis deviation caused by Right ventricular hypertrophy.

❖ ECG waves abnormalities:

1- Enlarged QRS (inc. voltage): due to hypertrophy of the ventricles.
2- Prolonged QT interval: Repolarization abnormalities increase chances of ventricular arrhythmias.
3- T wave: elevation in hyperkalemia (↑K affects the membrane potential).
Flat T wave: hypokalemia or ischemia
so the ECG is also important in detecting electrolyte imbalances.



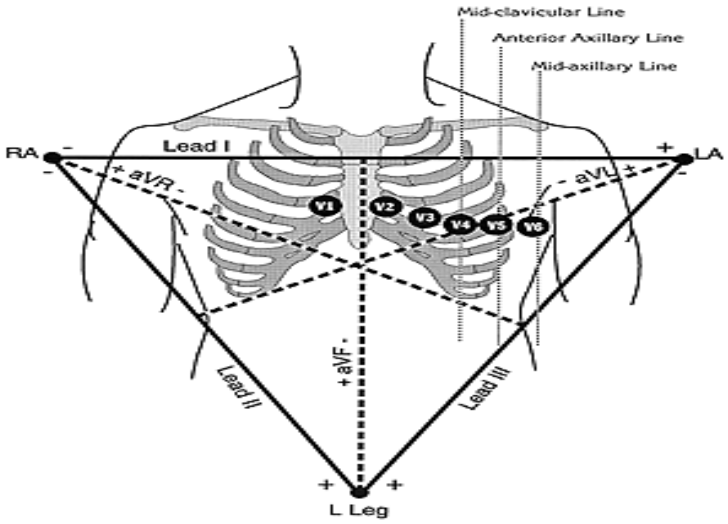
Normal T	Flat T	Elevated T

❖ Increased voltage in bipolar limb leads:

- If sum of voltages of Leads I-III (all the QRS complexes in the standard limb leads) is greater than 4 mV, this is considered to be a high voltage ECG. Note that the summation is with the signs –the positive is positive and the negative is negative- (it is not as if we're saying: anything above 40 small squares is high voltage, rather, what is meant is that the sum of Q + R +S with their signs is higher than 4 mV).
- Most often caused by increased ventricular muscle mass (hypertension, marathon runner, who have physiologic –normal- hypertrophy), because the increased quantity of the muscle generates increased electricity around the heart.

❖ ***Decreased voltage in bipolar limb leads:***

- It is caused by decreased heart mass (infarcts), valve abnormalities, or that the heart is too far from the chest, which occurs in conditions such as emphysema, pleural or pericardial effusion (specifically cardiac tamponade).
- Cardiac muscle abnormalities (old infarcts causing decreased muscle mass, low voltage EKG, and prolonged QRS).
- Conditions surrounding heart (fluid in pericardium, pleural effusions, emphysema).
- (Not mentioned) Why does cardiac tamponade cause decreased voltage? Because extracellular fluid conducts electrical currents with great ease, a large portion of the electricity flowing out of the heart is conducted from one part of the heart to another through the pericardial fluid. Thus, this effusion effectively ''short-circuits'' the electrical potentials generated by the heart, decreasing the voltages that will reach the surface and be recorded on ECG.



These are the 12 limb leads:
3 bipolar limb leads (I,II,III)

3 Unipolar limb leads: aVR, aVL, aVF

6 chest leads (V1-V6)

We finished ECG completely…

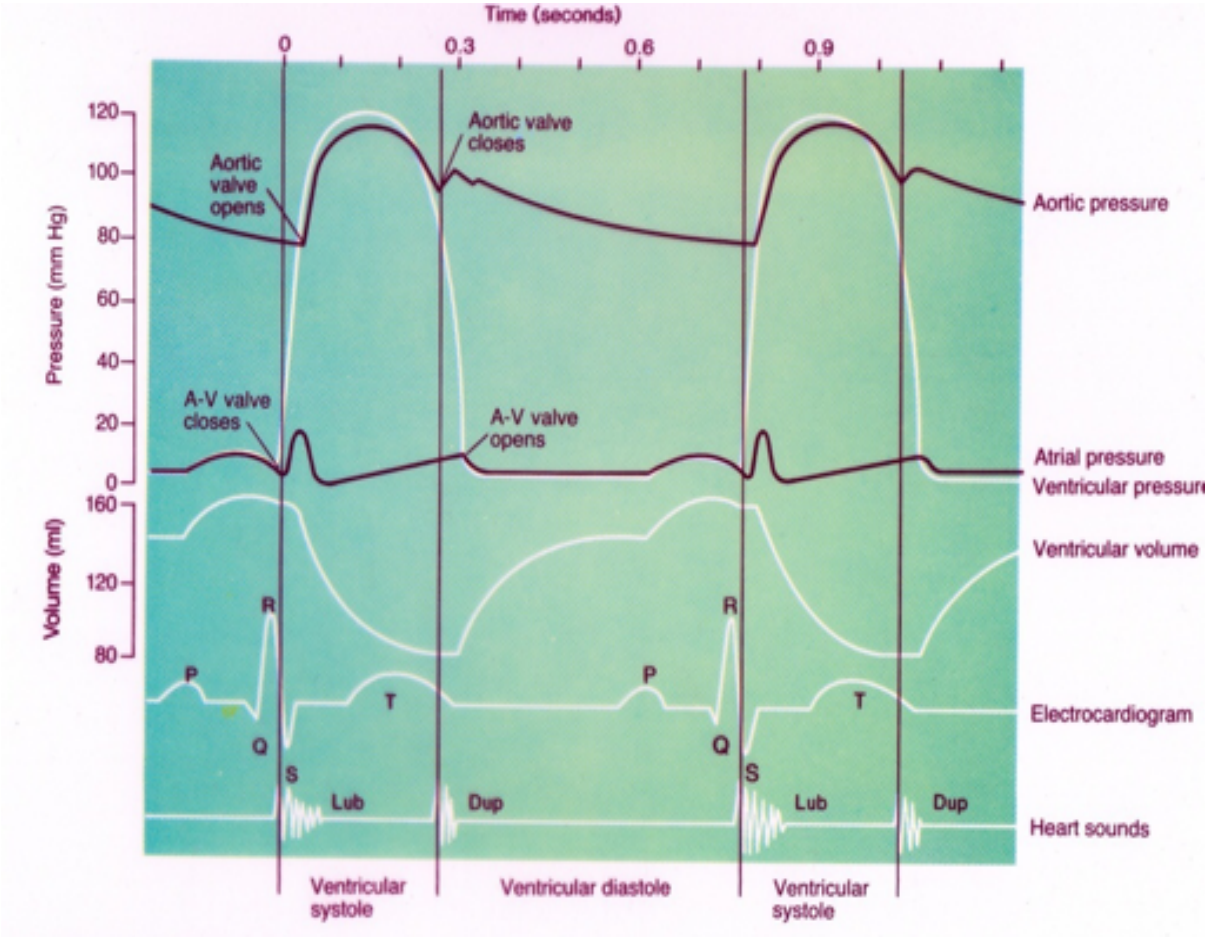
****

❖ ***The cardiac cycle:***

Please keep an eye on the figure below while we revise.

- ***The volume changes***: (the numbers mentioned in the last sheet were rough estimates, so it is OK to find some differences here). The white line in the below figure represents volume changes as follows:
  - Filling of ventricles (fast + slow).
  - Atrial systole, which increases the volume, but the contribution is low (Max. 25%). At the end of atrial systole and just before ventricular systole starts, the volume of blood in the ventricles is called End Diastolic Volume (EDV)

- Vt.contraction starts, which will increase the pressure inside it; the first increase in pressure is higher than the pressure in the atria (zero), so the AV valve closes. This coincides with the 1st heart sound (S1, dup).
- The pressure keeps rising (fast) in the ventricles; the AV valves are closed as well as the semilunar valves, so there is no change in volume + rising pressure = Isovolumic contraction. Note that the semilunar valves only open when the pressure in the ventricle is higher than the diastolic pressure in the aorta (left) which equals 80 mmHg or the diastolic pressure in the pulmonary artery (right) which equals 8 mmHg.
- Ventricular pressure > 80 mmHg → semilunar opens (left) at the end of Isovolumic contraction, then the volume sharply decreases due to rapid ejection. This is followed by a slow (slower) ejection until you reach the end of systole. The volume of blood that is present in the ventricle after finishing systole is called End systolic Volume (ESV).
- Let us say EDV = 125 ml, ESV = 55 ml, so what is ejected <u>*per beat*</u> = 125-55 = 70 ml. This is the stroke volume.



➢ ***Ventricular pressure: (also a white line in the figure, must follow it)***

- At diastole, it is zero. Then it rises up (more than zero) to cause AV closure. Some might ask: why didn't the AV valves close from the beginning of atrial systole? After all, the fast flow of blood during that period increases ventricular pressure to about +5, which is higher than zero?! The answer is that during atrial systole (<u>CONTRACTION</u> of the atria) the pressure in the atria also increases to about +7, so the AV valve remains open. At the end, this wave ends; pressure is back to zero, Vt.systole starts, pressure in ventricles rises above zero → AV closure→ S1.
- Lt. Vt. Pressure rises until it is > 80 → semilunar opening→ejection. Most of the time, ventricular pressure is higher than the aortic pressure except at the end, but there is no backflow because of blood momentum. When aortic pressure overcomes the forward force of blood flow, blood tries to flow back to the ventricle, which causes semilunar closure. This gives us the 2nd heart sound (S2, dub).
- *The time between S1 and S2 is the period of ventricular systole (around 0.3 sec), while the time between S2 and the next S1 represents ventricular diastole (0.5 sec).*
- When the blood tries to flow back, the semilunar closes. This blood now faces a sudden stop, and it can't go forward, so it presses a little bit on the aortic wall (it tries to flow around) causing a brief rise in aortic pressure (Just after ventricular systole ends). Then it re-flows back in the aorta normally (to go to periphery). So this slight increase in aortic pressure just after the closure of the semilunar valves is called the *<u>incisura, or dicrotic notch</u>*. (by inc. pressure around the aortic wall).
- The 1st step of ventricular diastole is the Isovolumic relaxation (semilunar and AV are closed, no change in volume, very fast drop in pressure). When atrial pressure is higher than ventricular pressure, the AV valve opens, and all the collected blood (ever since the AV first closed, the atria were actually collecting blood) pours into the ventricles rapidly (Rapid filling), followed by diastasis (slow filling), then atrial systole (which also causes a rapid filling) re-occurs.
- Rapid filling may give a sound which is called S3. The rapid filling during atrial systole may give a sound which is S4.

➢ ***The electrical changes:***

- They <u>precede</u> mechanical changes:
  - Atrial depolarization (P wave) precedes atrial systole.
  - Ventricular depolarization (QRS) precedes ventricular systole.

- Ventricular repolarization (T wave) precedes ventricular diastole.

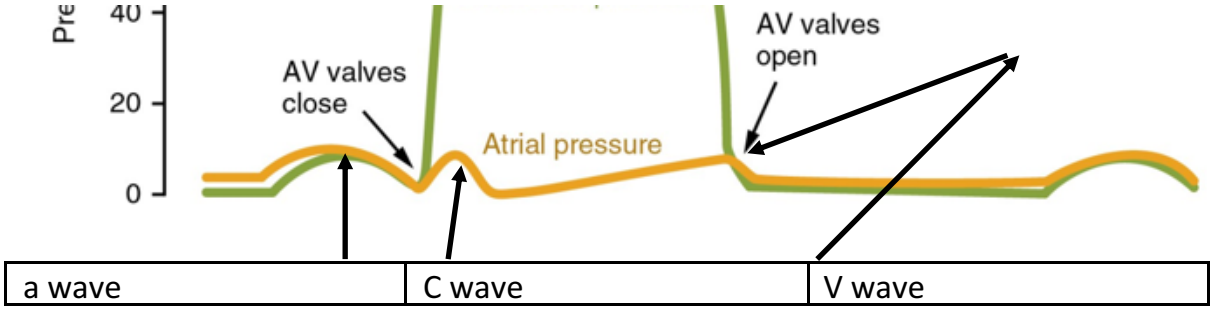
- ***Heart sounds:***
  - S1 → movement of blood around a closed AV valve.
  - S2 → movement of blood around a closed semilunar valve.
  - S3 → Rapid filling of ventricles (the movement of blood generates S3).
  - S4 → Rapid filling of ventricles during atrial systole.
  - Normally you can hear S1 and S2. If you have musical ears (very sensitive) you can hear S3 and S4. Nowadays. A phonocardiogram records all the sounds and tells you if there are any abnormal heart sounds. Such sounds are murmurs. If they are present between S1 and S2 they are systolic, if between S2 and the next S1 they are diastolic.
  - **An example of a murmur**: if there is a mitral valve regurgitation; when the ventricles contract, the high pressure will push the blood to the atria, and this movement of blood gives a sound, but it's abnormal (systolic murmur). In addition, in case of aortic incompetence, when blood tries to flow back to the ventricle it succeeds, this movement gives a sound (diastolic murmur).
  - S1 coincides with the QRS of the ECG (S4 is just behind S1, but it is more accurate to say S1 → QRS), while S2 coincides with the T wave.
  - Where to hear the heart sounds? (the Dr didn't concentrate on details)
    S1 → at the apex of the heart (from the left side) to hear the mitral, while from the right side to hear the tricuspid valve.
    S2 → aortic valve on the right side while pulmonary from the left side.

- ***Atrial pressure curve: (follow the figure bellow then the one above)***
  - During Atrial diastole, atrial pressure is zero. Then it rises due to atrial contraction. This rise in atrial pressure gives a wave on the atrial pressure curve which is called the **a** wave.
  - When the ventricles contract, they try to push the blood to the atria, the AV valve closes; but because of the very high pressure in the ventricles it pushes the AV valve towards the atria (pushes but doesn't open the cusps apart, if they are opened →incompetence/prolapse). This pushed AV valve increases the atrial pressure a little bit, which gives another wave which is called the **c** wave. The c wave appears due to ventricular systole (during Isovolumic contraction).
  - At the end of Isovolumic contraction, the semilunar valve opens, so ventricular blood finds an opening to go through, it will no longer press

on the AV valve, rather, it will go towards the opened semilunar to be ejected.

- During systole (of ventricles), the AV valves are closed, and the atria fill with blood. The atria are compliant (can accommodate good amounts of blood by stretching its wall, being flexible/expandable). During atrial filling, the pressure inside them increases but slowly, and this continues until the end of Isovolumic relaxation (when the AV valve opens) and blood flows from the atria to the ventricle. This gives us another wave which is called the **v** wave.
- So basically, a → atrial systole// c → Vt.systole// v → Vt.diastole.

This represents the atrial pressure curve:



The cardiac cycle:

**Atrial systole 0.1 second
**Atrial diastole 0.7 second
**Ventricular systole 0.3 second:

I. Isovolumic contraction 0.01 seconds
II. Rapid ejection period
III. Slow ejection period

**Ventricular diastole 0.5 seconds

I. Isovolumic relaxation 0.02 sec (longer than Isovolumic contraction)
II. Rapid filling
III. Slow filling (Diastasis)
IV. Atrial contraction

- EDV – ESV = SV. (SV = stroke volume)
  SV x HR = cardiac output.
  So if cardiac cycle time is 0.8 sec, EDV = 130 ml and ESV = 60 ml, CO??→

CO=SV x HR= ((130-60)(ml/beat)) x ((60/0.8)(beat/min))=5.25 L/min | Or 5250 ml/min

- Ejection fraction (EF) = (SV/EDV) x 100%.
  It is proportional to how much is ejected out of how much is there in the Vt. Normally, it should be > 55%. It is a measure of the efficiency (effectiveness) of the heart.
- Contractility of the heart: when increased (<u>positive inotropic effect</u>), this means that the EDV is the same (fixed, doesn't change) while the SV increases. This means that when the contractility increases, for the same EDV, instead of ejecting 55%, the heart ejects 70% (for example), and this is due to increase in the contraction power of the muscle, which is manifested as: fixed EDV, increased SV, and decreased ESV (because EDV = ESV + SV), increased EF.
- Positive chronotropic effect = increased heart rate.
- Left Ventricular pressure varies between 0 and 120 mmHg. The aortic pressure varies between 80 and 120 mmHg. The right ventricular pressure varies between 0 and 25 mmHg. The pulmonary artery pressure varies between 8 and 25 mmHg.

****

- If the volume of blood before systole increases (<u>EDV increases</u>), the amount ejected is increased (<u>SV increases</u>). This is **the Frank – Starling law**. This is different from increased contractility (where EDV is fixed and SV increases, here EDV is not fixed), but rather, due to increased stretching of the muscle by more blood present in the Vt, when it contracts, it ejects more amount of blood to return to normal.

Frank – Starling law: Within physiological limits, an increase in the length of the muscle increases the force of contraction. (**↑EDV → ↑SV**).

Increased EDV = more stretching = increased length (in the law).

⇒''Within physiological ranges'' has a limit, so the stretch can't go forever. Intrinsically (without any autonomic stimulation/sympathetic activity/drugs; just by the Frank-Starling law), this upper limit equals 15 L/min (as a cardiac output). Note that the normal cardiac output is around 5 L/min. So normally we can increase the cardiac output from 5 L/min at rest to 15 L/min only by the effect of Frank-Starling law. The difference between the two (15 – 5 =10 L/min) is called the <u>*cardiac reserve*</u>.

-So the cardiac reserve is the difference between the resting CO and the maximum

CO. If autonomic stimulation occurs (like if one is running, his SV is influenced by both Frank-Starling and autonomic stimulation), this upper limit may reach around 35 L/min. so the cardiac reserve is around 30 L/min (35 – 5 = 30). So athletes have a great cardiac reserve.

From this we can deduce that the regulation of the stroke volume may be

1- Intrinsic regulation (Frank-Starling): increase in blood volume before contraction increases SV. This volume (EDV) is called the **preload**. (Preload = EDV).
Note: Anesthesiologists call the End diastolic pressure (instead of volume) as preload.

So Frank Starling in another way: **↑Preload → ↑SV**.

Another concept is the **Afterload**; *which is the aortic pressure during diastole* (which we previously determined as 80 mmHg). This is in relation to the left ventricle, while the afterload in the right side is the *pulmonary* pressure during diastole.
If the Afterload is increased, much of the pressure made by the ventricle is just to have the semilunar valve opened (much of the work made by the heart is consumed just to have the ventricular pressure > afterload thus opening the semilunar, for example if the afterload is 90 instead of 80), so the ventricle must work harder to have the same SV. On the other hand, if the afterload decreases, the opening of the valve is easier, now the ventricle's force and work are used to pump more, so the SV increases. All in all, **↑ Afterload → ↓ SV and ↓ Afterload → ↑SV.**

This is why we treat hypertension; because hypertensive patients' left ventricle has to work harder (so more oxygen demand) just to open the valve and eject blood. So any coronary disease would immediately cause ischemia in such heart. **So we decrease the pressure in such patients to decrease the afterload.**

2- Extrinsic regulation (next lecture).

Best of luck