The Cardio-VASCULAR System

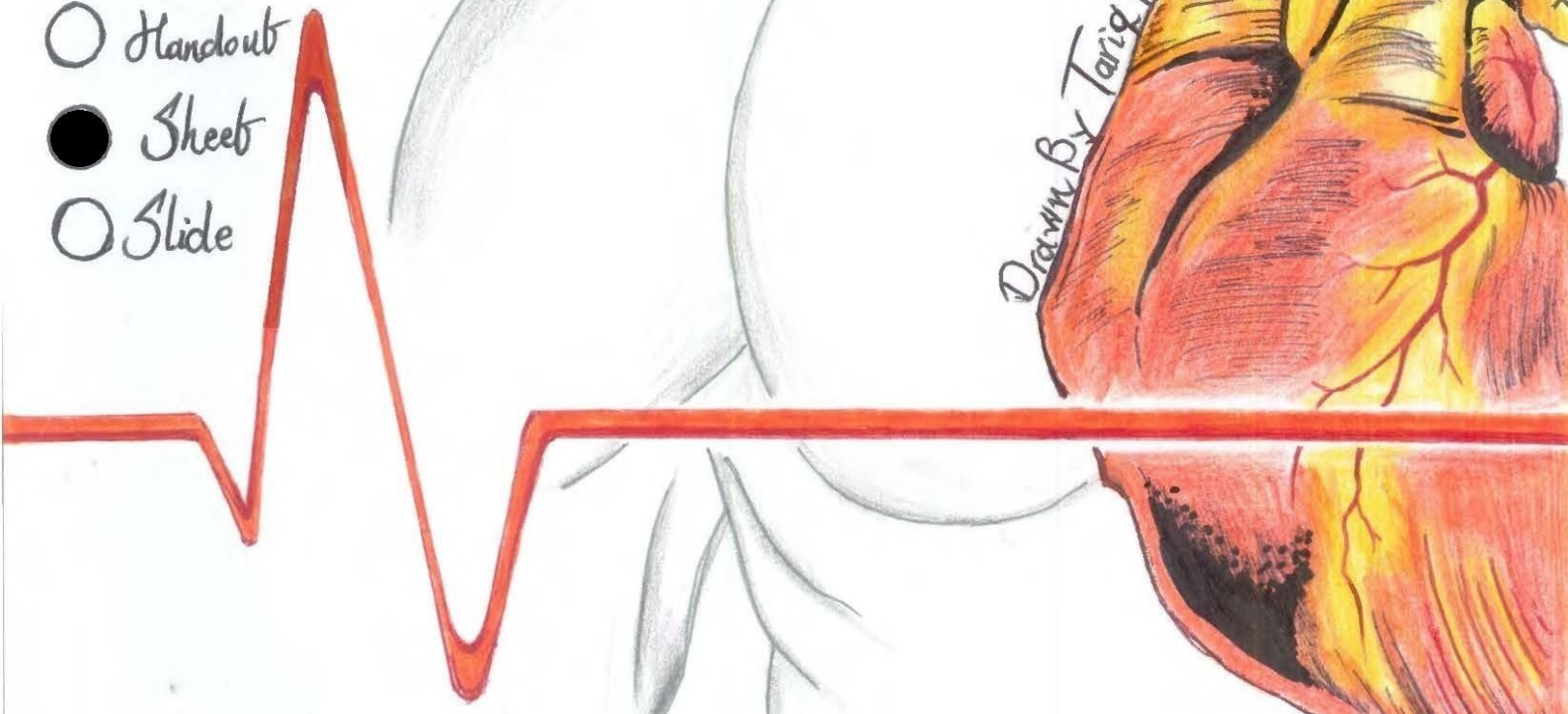
- ○ Anatomy
- ○ Histology
- ○ Pathology
- ○ Pharmacology
- ● Physiology
- ○ Microbiology

Lec #: 8

Dr. Name: Faisal.m

Done By: Omar Rimawi

- ○ Handout
- ● Sheet
- ○ Slide



ECG AND THE CARDIAC CYCLE

Today's lecture is divided into:

1 Revision about cardiac abnormalities and arrhythmias

2 The cardiac cycle

Note: any extra notes mentioned are from Guyton the 12th edition

Abnormal sinus rhythms could be divided into:

1- **Tachycardia**: meaning a fast heart rate, usually above 100 beats / minute. (look at figure 1)
The causes could be increased body temperature, sympathetic stimulation, or toxic conditions of the heart

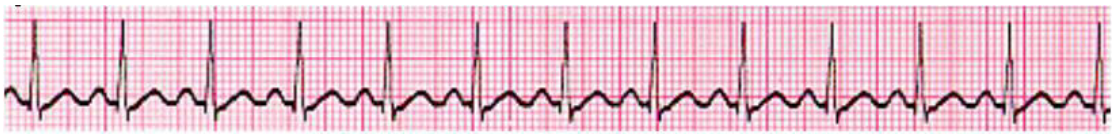


Figure 1: This electrocardiogram is normal except that the heart rate, as determined from the time intervals between QRS complexes, is higher than 100 beats per minute instead of the normal 72 beats per minute.

2- **Bradycardia**: means a slow heart rate usually less than 60 beats /min. (look at figure 2)

It could be Present in athletes who have a large stroke volume thus a lower heart rate. Moreover if it was sinus bradycardia then it might be due to excessive parasympathetic stimulation (like in carotid sinus syndrome).

Extra note about carotid sinus syndrome: In these patients, the pressure receptors (baroreceptors) in the carotid sinus region of the carotid artery walls are excessively sensitive. Therefore, even mild external pressure on the neck elicits a strong baroreceptor reflex, causing intense vagal-acetylcholine (parasympathetic) effects on the heart, including extreme bradycardia.

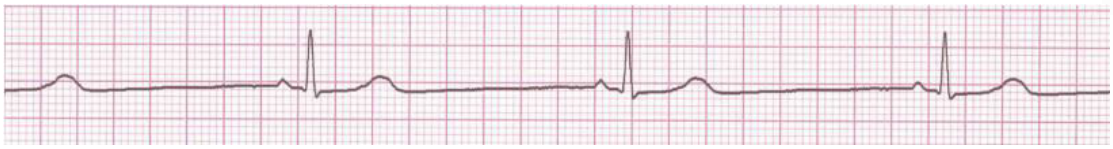


Figure 2: A node is depolarizing slower than normal, impulse is conducted normally (i.e. normal PR and QRS interval) rate is slower than 60/beats per minute

How to calculate the heart rate from the ECG?
You simply count how many small squares from one R to the next, putting in mind that each small square equals to 0.04 seconds. Then you multiply the number of squares by 0.04. Finally you divide the time (one minute) by the value you have obtained. In order for the reading to be considered bradycardiac there should be more than 25 squares (more than 1 second) separating the 2 R'S.

I.e. 30 * 0.04 = 1.2 seconds between 2 cardiac cycles (beats)
60 /1.2 = 50 beat per minute (which is less than normal)

3- **Atrial fibrillation**
In atrial fibrillation there is no activity of the SA node thus no P's will be shown however the QRS-T complex will be normal.(no synchronous atrial systole)

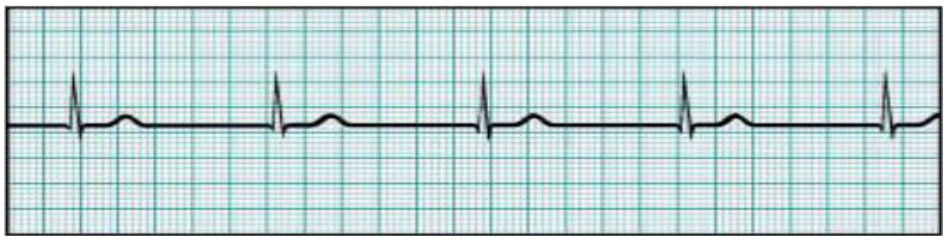


Figure 3

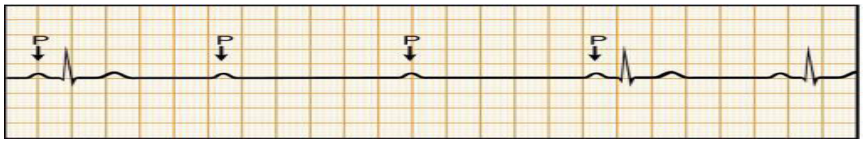
Extra note:
The figure above shows the electrocardiogram during atrial fibrillation. Numerous small depolarization waves spread in all directions through the atria during atrial fibrillation. Because the waves are weak and many of them are of opposite polarity at any given time, they usually almost completely electrically neutralize one another. Therefore, in the electrocardiogram, one cannot see P waves from the atria. Conversely, the QRS-T complexes are normal.

4- AV node conduction failure (AV block)

Causes of AV block are:

a- Ischemia of A-V nodal or A-V bundle fibers(Can be caused by coronary ischemia)
b- Compression of A-V bundle (by scar tissue or calcified tissue)
c- A-V nodal or A-V bundle inflammation
d- Excessive vagal stimulation

Figure 4: When you see an ECG that lacks QRS-T complex following P waves then it is heart block.



There are different degrees for **heart block**.

The normal **P-R interval is 0.16 sec** (from beginning of P till beginning of R).

a- 1st degree heart block:

If P-R interval is > 0.20 sec, and each P is followed by QRS-T complex, then it is considered a first degree block. (Look at figure 5). First degree heat block is due to prolonged conduction delay in the AV node or Bundle of His.

You could measure the P-R interval from the ECG, and if it is more than 5 squares(0.04 second per square) it means the P-R interval is more than 0.20 sec.

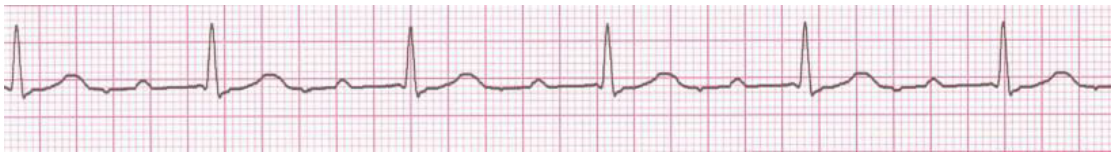


Figure 5: 1st degree heart block

b- 2nd degree heart block:

P-R interval increases to 0.25 - 0.45 sec. Moreover some impulses will pass through the A-V node while others don't thus causing "dropped beats" or palpitations . However there is a rhythm that develops 2:1 or even 3:2…etc. The **heart rate here is between 40 -60.** This is called regular irregularity.

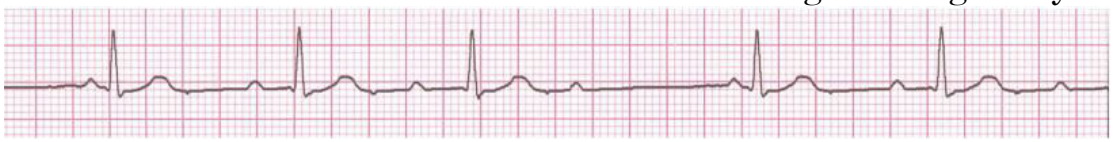
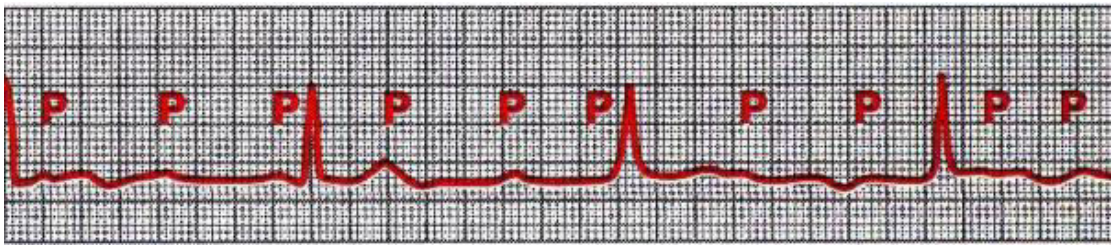


Figure 6: Each successive atrial impulse encounters a longer and longer delay in the AV node until one impulse (usually the 3rd or 4th) fails to make it through the AV node.

c- 3rd degree heart block:

Occurs when there is dissociation (no relation between) the P wave and the QRS-T complex. There is complete block of conduction in the AV junction, so the atria and ventricles form impulses independently of each other. Without impulses from the atria, the ventricles own intrinsic pacemaker **beats at around 15 - 40** beats/minute (this is called ventricular escape).

Figure 6:



5- ventricular fibrillation (fatal)

If you notice in figure 7 there is no actual rhythm at all .QRS complexes appear as saw shape. This occurs in ventricular fibrillation (fatal)

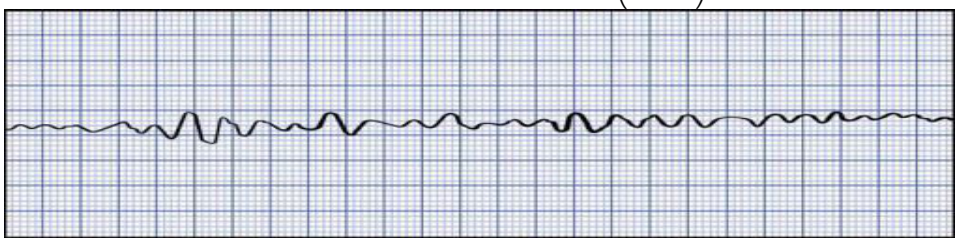


Figure 7: ventricular fibrillation

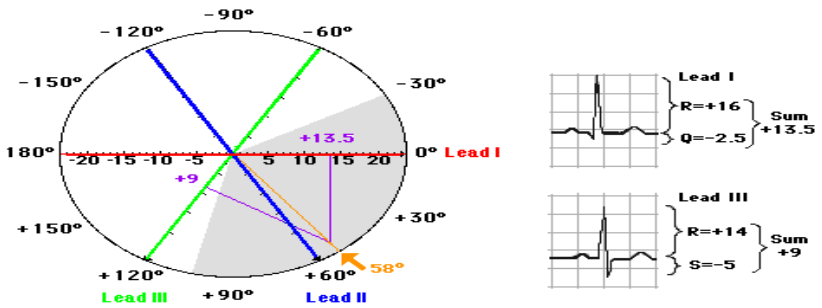
Ventricular fibrillation results from cardiac impulses that have gone berserk(wild) within the ventricular muscle mass, stimulating first one portion of the ventricular muscle, then another portion, then another, and eventually feeding back onto itself to re-excite the same ventricular muscle over and over—never stopping. When this happens, many small portions of the ventricular muscle will be contracting at the same time, while equally as many other portions will be relaxing. Thus, there is never a coordinate contraction of all the ventricular muscle at once, which is required for a pumping cycle of the heart. **There is no pumping of the heart thus there is no heart rate**, causing death.

6- Stoke Adam's syndrome:

Is a complete A-V block due to **excessive vagal stimulation** that comes and goes. In this syndrome the ventricles after the A-V block would stop contracting for 5-30 sec due to overdrive suppression from the atria. Thus the patient faints because of poor cerebral blood flow .Then, a ventricular escape would occur with A-V nodal or A-V bundle rhythm (15-40 beats /min). As a solution an artificial pacemaker connected to right ventricle would be provided for these patients. These artificial pacemakers are battery operated, have a long life, and have to be placed under the skin with electrodes attached to the right ventricle. **NOT in the right atrium** since we need to overcome the AV rhythm.

7- Electrical axis deviation:

Clinically we accept for the electrical axis to be between **zero and 90 degrees**. **Physiologically** we accept for it to be between **-30 and 110.** To calculate the electrical axis you can take any two limp leads (uni- or bi- polar). You then by algebraic summation add the values of QRS of each limb lead separately. Moreover plot the value of each lead on the hexagonal axis. Then extrapolate two lines from each limb lead to find the intersection of the vector. Finally start the vector from middle of the circle toward the intersection point.
I.E(figure 8)

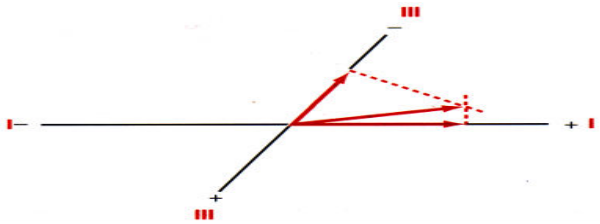


A- Left Axis deviation:

The mean left electrical axis deviation is between 0 and -90. Some people especially the obese and short people naturally will have left axis deviation but it is mild; up to -20 or -30 degrees not more.
Now when we are saying left axis deviation we are meaning that most of the vectors are moving from the right to the left ventricle. This occurs in left ventricular hypertrophy or left bundle branch block. Left ventricular hypertrophy is a product of hypertension, aortic stenosis or aortic regurgitation thus causing a left axis deviation, a **slight prolonged QRS**, and high voltage.

Figure (9)

Bundle branch block
-Left bundle branch block causes left axis shift because right ventricle depolarizes much faster than left ventricle. QRS complex is prolonged. By the same token Right bundle branch block causes right axis deviation.



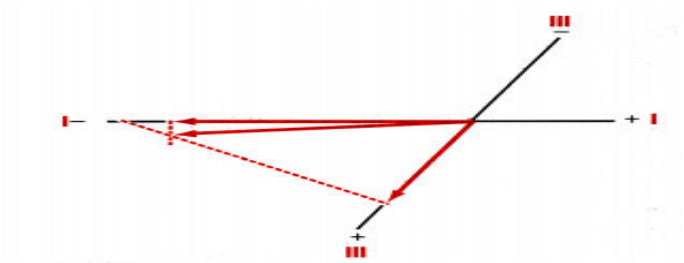
b- Right axis deviation

The mean right electrical axis deviation is between 90 and 180. Here the tall and thin people will have right axis deviation. Also it occurs due to right ventricular hypertrophy or right bundle branch block. Since any of the following problems in the pulmonary system or valve (stenosis of the valve, pulmonary hypertension, tetralology of Fallot, and interventricular septal defect) would cause right ventricular hypertrophy, and a right axis deviation would occur. Also here a **slightly prolonged QRS** and high voltage occur.

Figure (10)

In order to deduce whether the ventricular hypertrophy or bundle branch block caused the axis deviation we look to the QRS complex which is **more delayed (widened) in bundle branch block** than it is in ventricular hypertrophy.

Since the Purkinje system is blocked in the affected side, the impulses would travel through ventricular muscle (0.3m/s) which is considerably slower than the conduction system. Moreover the QRS voltage would be increased (enlarged) in ventricular hypertrophy.



In severe axis deviation AVF and Lead I are always negative.

Remember that in

a- 1st degree heart block the PQ interval is longer than 0.2 seconds
b- **Hypertrophy of ventricles** there would be **enlargement of QRS**(stronger ventricular contractions)
c- In **bundle branch block** there would be **prolonging (widening) of QRS**
d- Elevated T occurs in hyperkalemia
e- Flat T in hypokalemia or ischemia

(Refer to slides number 24-29 for the figures)

Increased Voltages in Standard Bipolar Limb Leads

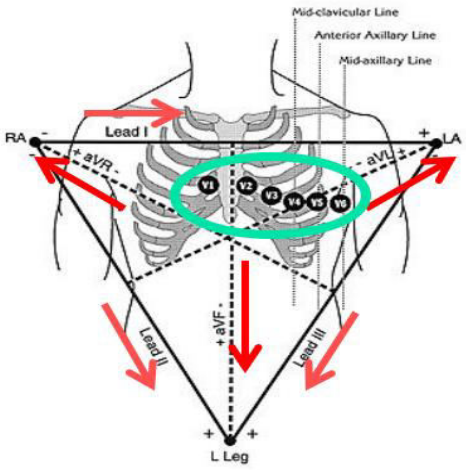
If the sum of voltages of Leads I-III is greater than 4 mV, this is considered to be a high voltage EKG. Most often caused by increased ventricular muscle mass (hypertension, marathon runner).

Decreased Voltages in Standard Bipolar Limb Leads

Cardiac muscle abnormalities (old infarcts causing decreased muscle mass, low voltage EKG, and prolonged QRS). The following Conditions surrounding heart that make it further away from the chest (fluid in pericardium, pleural effusions, emphysema).

This figure (11) illustrates all of the leads required for ECG. You need to be familiar about them by now (you must know the axis, angles, which side is positive and which is negative….etc.)

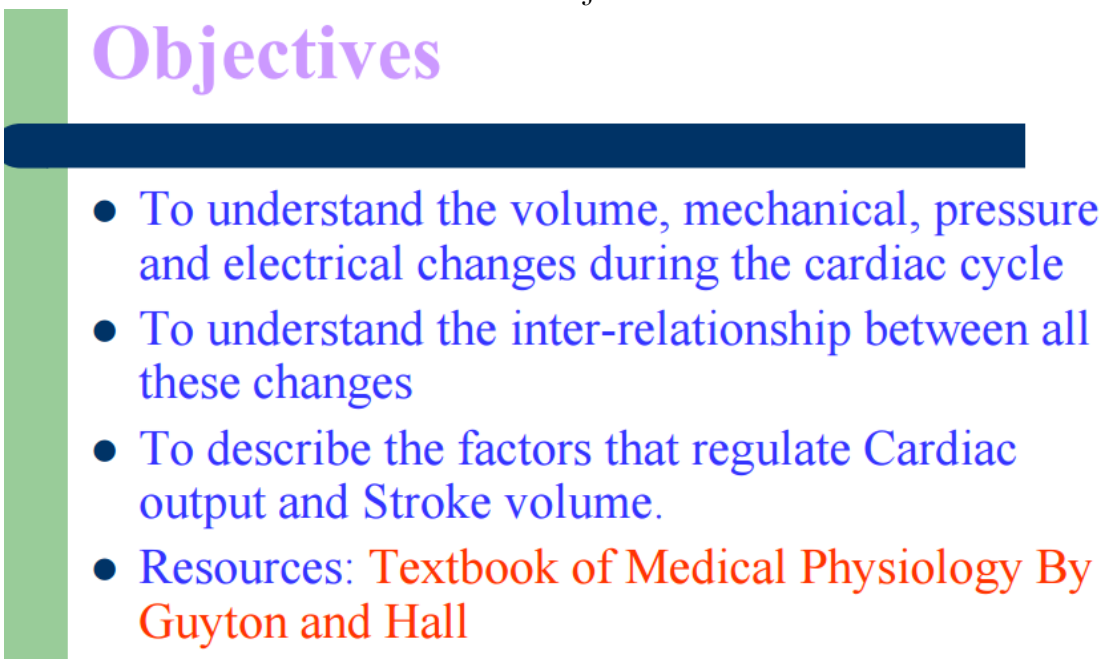
Figure 11



That was the end of ECG lectures. Now we are going to start talking about the cardiac cycle.

The cardiac cycle is the time between one beat and the next one. If we look at the ECG it would be between one R and the next R, or one P and the next P

and so on. The dr. mentioned these objectives.



The intrinsic factor that regulate the cardiac cycle is Frank Starling law of the heart. Which is within physiological limits, an increase in the resting length of the heart muscle (passive tension), increases tension {force} of contraction (Active tension).

The extrinsic factor is the autonomic nervous system.

The cardiac cycle requires 0.8 seconds for all atrial and ventricular systole and diastole. This number is given for teaching purposes and it is not constant from one person to another.

Cardiac Cycle

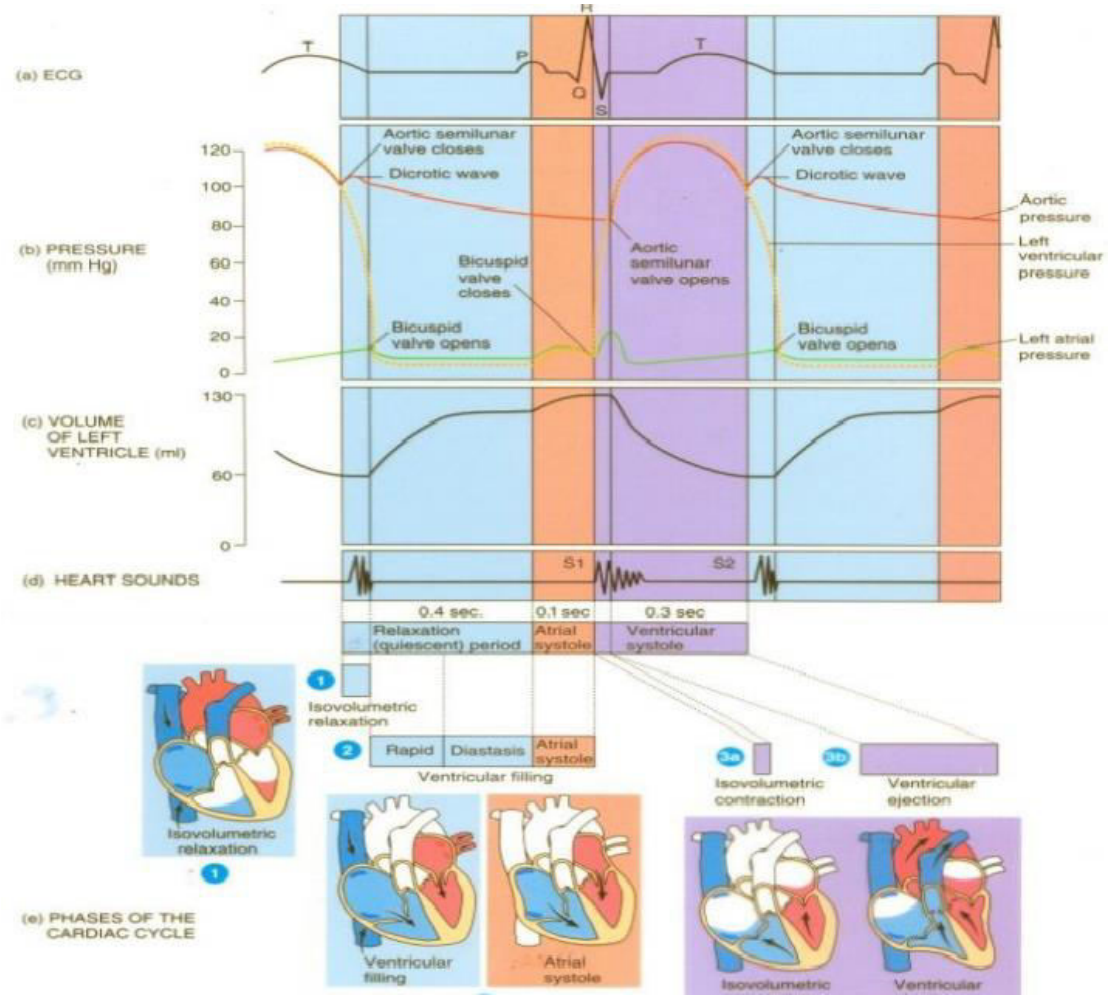


Figure (12)

The time required for Atrial systole is 0.1 second, while atrial diastole is 0.7 second. Normally the atrial systole and ventricular systole never overlap. Following the atrial systole is Ventricular systole which takes around 0.3 seconds. Thus the ventricular diastole would take 0.5 seconds. Although it is written that ventricular diastole is 0.4 seconds, it is actually 0.5 seconds. This is due to the fact that during atrial contraction (0.1second) the ventricle would be filling blood (last part of its diastole).

Any mechanical response should be preceded by an electrical response (ECG). Before atrial systole we have the P wave (which causes atrial depolarization).

Preceding the ventricular systole we would have the QRS complex. Finally the T wave would be before the ventricular diastole.

If you noticed from figure 12 you can record the volume changes in the ventricles during the cardiac cycle. The ventricles have the same volume of blood, and eject the same amount of blood.

Before the atria contract (systole) there is 100 ml of blood in each ventricle, and after contraction there would be around 125 ml in each ventricle. Thus the atrial systole would contribute to 25%.

There is actually no big importance of atrial contraction as are the AV valves are open due to fact that the atrium pressure is higher than ventricular pressure (normally the atrial pressure is zero while the ventricular during diastole is negative 1 or 2 in order to create a pressure gradient allowing blood to move from atria to ventricles). Even if there was no atrial systole in this 0.1 second some blood would pass through the AV valve but in lesser amounts (like 10 ml instead of 25 thus the atrial systole even contribute less than thought). That is why we said that in atrial fibrillation the heart can still function since the systole contribution to the filling of blood in ventricles is minimal (maximum 25%)

All in all there would be 125 ml in the ventricle this volume is called **EDV** (end diastolic volume.

When the ventricles contract the pressure inside the ventricles would be higher than zero (atrium) closing the AV valve. Then an additional 0.01 second is required for the ventricle to build up sufficient pressure to push the semilunar (both the aortic and pulmonary) valves open against the diastolic pressures in the aorta (80mmhg) and pulmonary trunk(8mmhg). Therefore, during this period, contraction is occurring in the ventricles, but there is no emptying. This is called the **period of isovolumic contraction**, meaning that pressure is increasing in the ventricle but little or no emptying of blood. This would cause the ventricle to increase the pressure in it to be higher than the diastolic pressure of the aorta (80 mm hg) and diastolic pressure of pulmonary arteries(8 mm hg) opening the semilunar valves.

Thus at the end of isovolumic contraction the semilunar valves open. When these valves open it would lead to the blood leaving the ventricles into the arteries decreasing the blood volume in the ventricles. This is called **rapid blood ejection**.

During the last part of ventricular systole the pressure inside the ventricles would start to drop suddenly until the arterial diastolic pressure is higher than it; closing the semilunar valves. However during that short period the ventricular pressure is higher than atriums thus the AV valves would still be closed. This short period is called **isovolumic relaxation**. During this period the volume does not change but the ventricular pressure decreases.

When the ventricular pressure becomes lower than the atrium the AV valves would open. During atrial diastole the venous blood would accumulate in the atrium (no valves separating the veins from atrium). And when the AV valves open there would be **rapid filling of blood**, followed by diastasis, and ended by atrial systole contributing the last 25 ml of blood.

note: The period of rapid filling lasts for about the first third of ventricular diastole. During the middle third of diastole, only a small amount of blood normally flows into the ventricles; this is blood that continues to empty into the atria from the veins and passes through the atria directly into the ventricles (diastasis). During the last third of diastole, the atria contract and give an additional thrust to the inflow of blood into the ventricles; this accounts for about 20-25 percent of the filling of the ventricles during each heart cycle.

At the end of atrial systole marks the end of ventricular diastole. This volume again is called **EDV** (end diastolic volume, 125ml). Blood is ejected during ventricular systole. The volume of blood that stays in the ventricle in the end of ventricular systole is called **ESV** (end systolic volume, 55ml).

To calculate the stroke volume you simply find the difference between EDV(125)-ESV(55)= 70 ml (**Stroke volume**)

Ejection fraction = SV / EDV = 65%. It measures the **efficiency of the heart**. It also measures the contractibility of the heat (**inotropic**). As the **contractibility** increases the value of SV increases, which in turn increase the value of ejection fraction. The contractility changes the SV only the EDV stays the same.

If you want to know how much blood is ejected per minute you would multiply the heart rate (beats per minute) against the stroke volume.

Cardiac output = stroke volume * heart rate = 70*70 = 5L/min

Cardiac output is 5L/min.

It is very important that the stroke volume is the **same** in **both ventricles**.

Let's consider as an example that the stroke volume of the right ventricle is 70 while it is 69 on the left. So one ml is the difference only per beat. After a whole minute an extra 70 ml would stay in the left ventricle. After one hour 4,000 ml would stay in the left ventricle! That is illogical (all the blood after one hour would be in the heart). Thus the stroke volume should be equal between the ventricles. It is possible one beat to be different but the next beat would compensate for it.

This sheet is dedicated to Q.