Cardio-Vascular System

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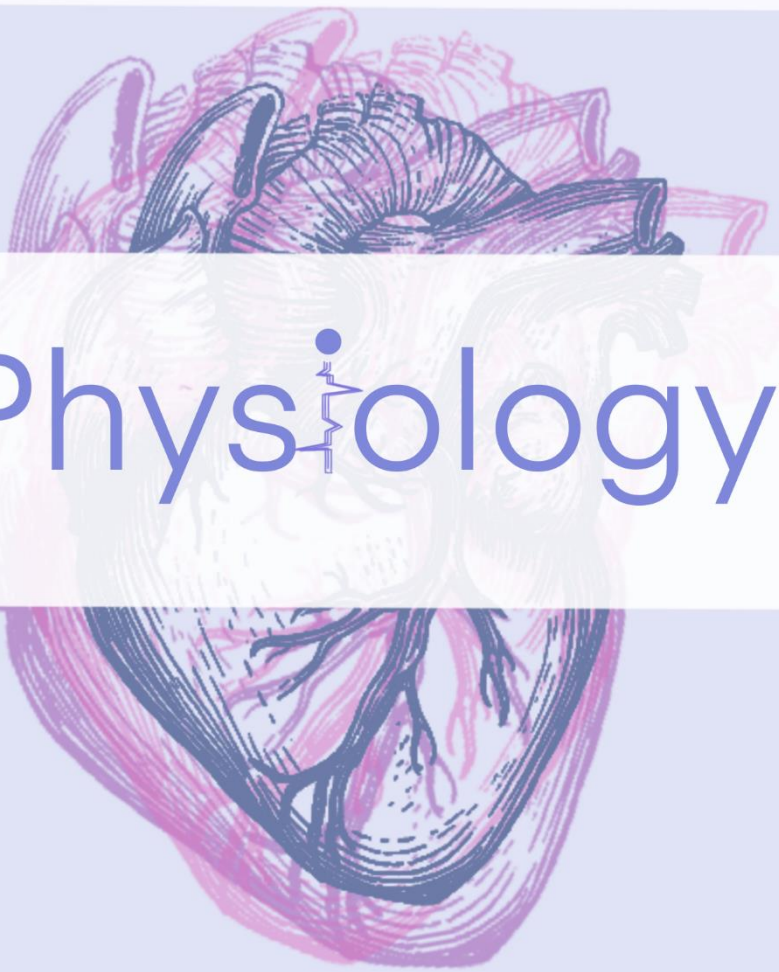

Physiology

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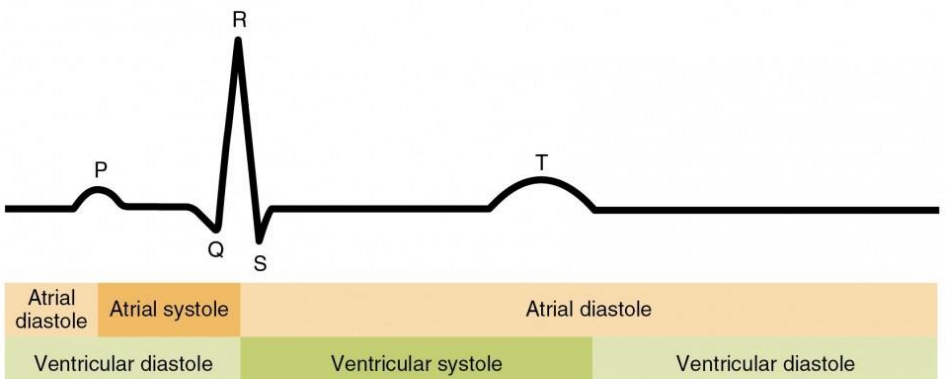
The Cardiac Cycle

- The cardiac events that occur from the beginning of one heartbeat to the beginning of the next are called the **cardiac cycle**.
- Each cycle is initiated by spontaneous generation of an action potential in the sinus 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 heart beat) **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.)
- Assuming that 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



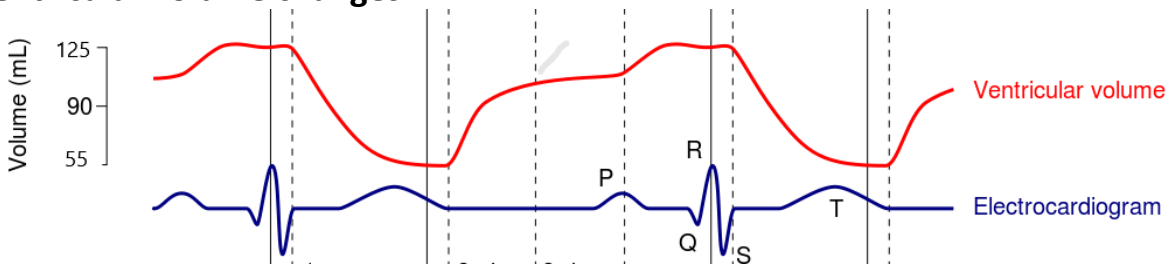
- Recall that the P wave is caused by spread of depolarization through the atria and is followed by atrial contraction (**atrial systole**), which takes about 0.1 seconds. Then, atria relax (**atrial diastole**), and that takes about 0.7 seconds.
- QRS waves, as we already know, appear as a result of 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 begins

Other changes during the cardiac cycle

- **Volume changes**:-

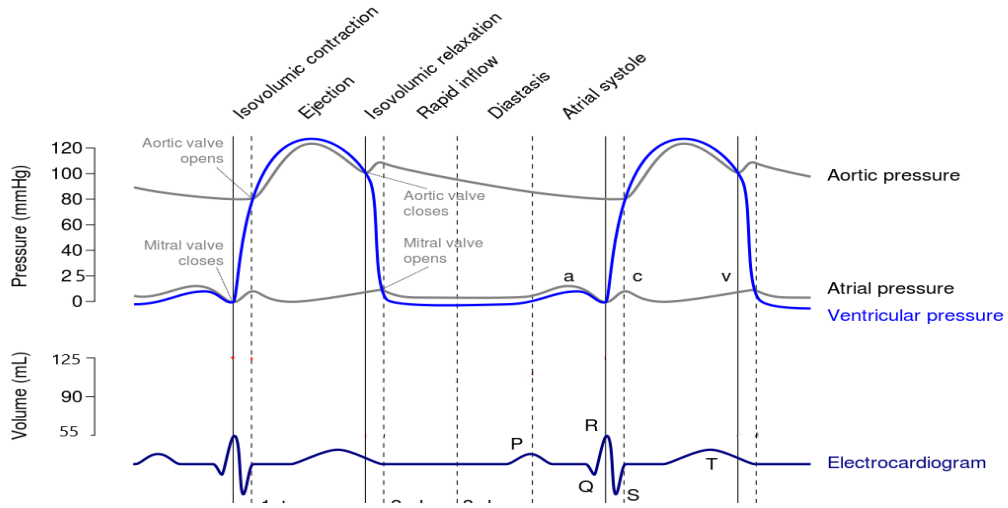
Ventricular volume changes:-



- Volume changes in both, the left and right ventricles, are the same.
- 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)

- 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**.

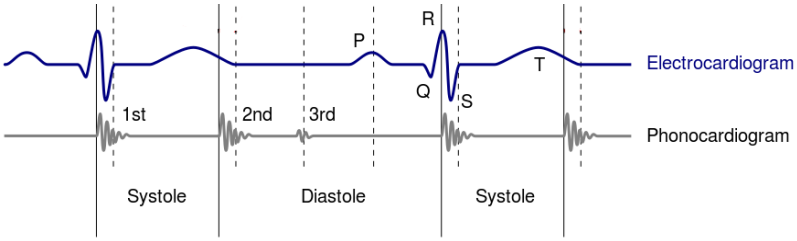
➢ **Pressure changes**:-



- 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).
- 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 down as the volume of the blood starts decreasing.
- 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***.

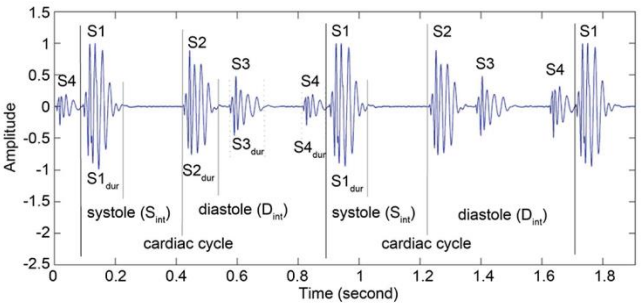
- Then, the pressure drops down until it becomes around **zero**. When this happens, AV valve opens, so blood starts to flow to the ventricle increasing the pressure and another cycle starts, the pressure in the aorta drops back to **80 mm Hg**.
- ✓ The highest pressure in the ventricle during systole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

➢ **Changes in sounds**:-

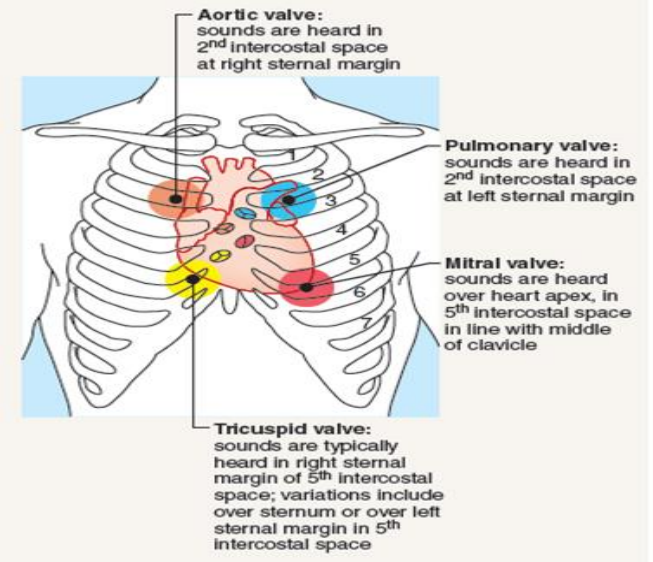


- Auscultation = listening to heart sound using stethoscope (السماعة الطبية).
- Once the AV valve closes, the blood tries to go back from the ventricle to the atrium, but because the pressure in the ventricle, at the moment, is higher than that in the atrium" during ventricular systole", blood remains 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.
- The time 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**.



- 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**.

❖ **The changes in the atria**

➢ **Pressure**: -

- The atrial pressure is almost **zero** during the diastole, while during atrial systole(contraction), an increase in pressure takes place "**A wave**"
- The "**c wave"** occurs when the ventricles begin to contract; it is caused partly by slight back flow of blood into the atria at the onset of ventricular contraction but mainly by bulging of the A-V valves backward toward the atria because of increasing pressure in the ventricles
- The "**v wave**" occurs toward the end of ventricular contraction; it results from slow flow of blood into the atria from the veins while the A-V valves are closed during ventricular contraction. Then, when ventricular contraction is over, the A-V valves open, allowing this stored atrial blood to flow rapidly into the ventricles and causing the v wave to disappear.

- In cases of AV valve stenosis, the atrium pushes certain amounts of blood throw a narrow AV valve, so the "A wave" becomes high and sometimes the blood reflexes to the jugular vein. The "C wave" also peaks, because the pressure in the ventricle pushes the AV valve towards the atrium.

- In AV valve incompetence the AV valve doesn't close properly, so during the ventricular contraction blood goes from the ventricle to the atrium.

What prevents this collapse from happening is the chordae tendineae that is attached to the papillary muscles which are part of the ventricle. When the muscle contracts, it pulls the chordae tendineae down with the valve movement towards the ventricles; preventing the back flow of blood towards the atrium.

- Cardiac output: is the volume of blood ejected from the ventricle per minute.
- Cardiac reserve: is the difference between maximal and resting cardiac output.
  CR=maximum cardiac output – normal cardiac output.
  - Cardiac output might increase to 15 liters instead of 5 liters in normal situations.

  However, in athletes, the maximum value for CO may reach 35 L/min. (Here the CR=35-5=30L/min)
- Ejection fraction : is the fraction of blood ejected from the ventricles of the heart with each heartbeat and it is an inherent volumetric measure of the pumping efficiency of the heart.

- it's the fraction of the end diastolic volume ejected in each stroke volume and normally equals 60%, between 55% -80%.

-It is a measure of contractility, so it will increase in case of hyperactivity.

-Ejection fraction = stroke volume /EDV

❖ **Factors affecting the stroke volume:**

-Preload (Frank -starling Law): it is the end diastolic volume (EDV) that stretches the right or left ventricle of the heart to its greatest dimensions.

-Afterload: (in order for the heart to open the semilunar valve, it has to exert pressure during ventricular contraction which is higher than the aortic pressure(during diastole which is 80mmhg) in case of the left ventricle and higher than the pulmonary pressure (8mmhg)in the case of the right ventricle .

So, the Afterload is the amount of tension that the ventricle has to develop in order to eject blood.

-Contractility: cardiac cell contractile force that is produced due to factors other than EDV.

With a fixed end diastolic volume, there is an increased stroke volume (same as positive Inotropic effect). Positive inotropic effect increases the stroke volume and decreases the end systolic volume.

-increase in contractility ->increase in SV

❖ Frank-starling law: - in physiological limits an increase in the length of the muscle increases the force of contraction. But in cardiac muscles we don't talk about the length instead of it we talk about the volume (end diastolic volume particularly)

✓ In physiological limits an increase in the preload volume increases the stroke volume.
✓ Slow heart beat (more filling time) means an increase in the SV.
✓ Exercise increases the venous return to the heart and so an increase in the SV.
✓ Blood loss and rapid heart rate (shortened filling time) decreases the SV.

Good Luck