CVS

Physiology

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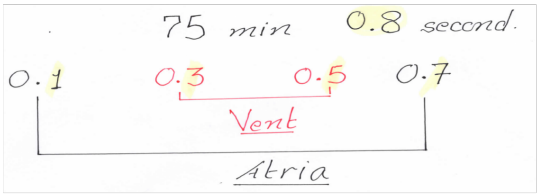
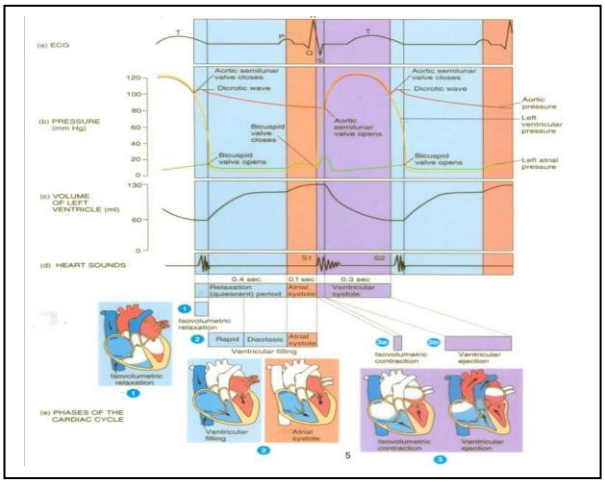
Review of the cardiac cycle:

Cardiac cycle: it's a series of changes that happens in one heartbeat, theses changes are: volume, pressure, mechanical and sound changes.



Pressure changes:

- Atrial pressure (almost zero) is higher than the ventricular one (negative) regarding to reference pressure (760 mmHg) → AV valve is opened.
- After the first contraction of the ventricle, pressure inside it increased and become higher than the atrial pressure → AV valve closes.
- For short period of time, the ventricle still contracting (isovolumic contraction) and all valves are closed.
- With the fast increasing of the ventricular pressure, it becomes higher than aortic pressure → semilunar valve opens.
- At the end of the ventricular systole the semilunar valve opens because the aortic pressure becomes higher than the ventricular pressure.
- Because of the momentum, blood tries to go back to the ventricle so the semilunar valve closes (isovolumic relaxation).



Dicrotic notch:

Happened because of the closure of the semilunar valve in front of the blood that coming from the aorta to the ventricle.

Pressure in the aorta during systole is 118-120 mmHg , Pressure in the aorta during diastole is 80 mmHg
Pressure in the left ventricle during diastole is zero , Pressure in the left ventricle during systole is 120 mmHg

Pressure changes:

- By the opening of the semilunar valve the volume suddenly increased (rapid ejection) at the end diastolic volume
- At the end of systole there are about 55 ml left in the ventricle
- So, each beat ejects 70 ml which called stroke volume
  Stroke volume = EDV – ESV
- Cardiac output: the amount of blood ejected per one minute = SV*HR

- When the AV valve opened (rapid filling) and then the slow filling because of the opened AV valve we call it diastasis

Heart sounds:

- First sound: due to the closure of the AV valve (lub)
- Second sound: due to the closure of semilunar valve (dub)
- Third sound: due to the rapid filling of the ventricle
- Fourth sound: due to the atrial contraction

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 papillary muscles, when the muscle contracts it pulls the chordae tendineae down with the valve towards the ventricle preventing the back flow of blood to the atrium.

Small equations summary:	
SV = EDV – ESV	EJECTION FRACTION = STROKE VOLUME\ EDV
C.O = SV * HR	55-80% normal, if it increased it indicates hyperactivity

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.

FACTORS affecting 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 must 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 must develop 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 heartbeat (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.

The left figure indicates the **preload**, the amount of blood found in the ventricle before it contracts. The ventricles are dilated and full of blood.

The right figure indicates the **afterload**, the force that the ventricle has to exert to open the semilunar valves. To do so, the force that has to develop in the right and left ventricles should be higher than the diastolic pressure in the pulmonary artery and aorta, respectively.