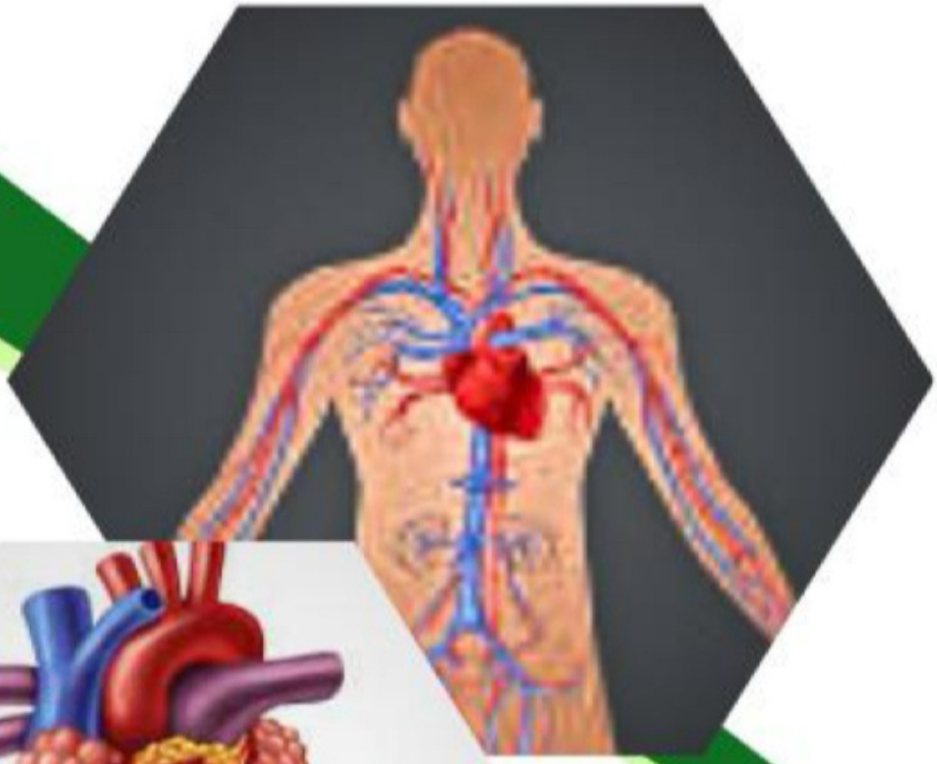
Doctor.021

no. 10

# CVS PHYSIOLOGY

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The doctor covered a lot of different topics in this lecture, so we'll provide you with what he said, as well as extra info to aid in your understanding. Good luck!

# Cardiac Cycle Recap

The cardiac cycle consists of:

- ➢ Diastole, which takes 0.5s total

  Early diastole:

  - The first part of diastole is isovolumic relaxation (0.15s), where the ventricles have repolarized, stopped contracting, and are relaxing. The left ventricular pressure $P_{LV}$ is decreasing and has fallen below the aortic pressure $P_{aortic}$ (so the aortic semilunar valve closes, and this marks onset of diastole), but $P_{LV}$ is still greater than the pressure in the left atrium $P_{LA}$, so the mitral valve/AV valve is closed too. The LV is a closed chamber, so there's no change in volume (isovolumic.)

  Mid-late diastole:

  - Second is the rapid filling phase. The $P_{LV}$ decreased to less than the $P_{LA}$ at the end of isovolumic relaxation, so the AV/mitral valve is open. Blood is flowing into the ventricle from the atrium. (Recall also that atrial pressure is slightly higher than ventricular pressure because the atrium is filled with blood entering from the veins.)
  - Third is reduced filling (slower) and it's called diastasis.
  - Fourth and last is atrial systole (0.1s). Near the end of diastole, atrial depolarization (P wave) occurs. The atria contract and pump blood into the ventricles. The volume of the ventricles at this point is the end diastolic volume EDV.

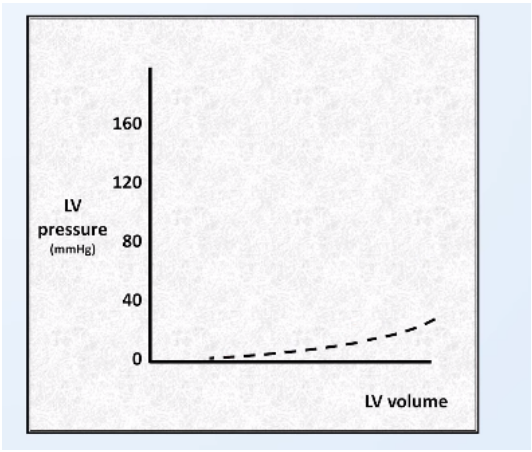
- ➢ and systole, which takes 0.3s in total
  - First is isovolumic contraction (0.05s.) The ventricles depolarize during atrial systole, and once the atria are done contracting, the ventricles contract immediately. This increases $P_{LV}$ above $P_{LA}$ (5 mmHg) so the AV/mitral valve shuts (here we hear the first heart sound S1), but for some time $P_{LV}$ is still less than $P_{aortic}$ (80 mmHg) so the aortic

semilunar valve is still closed. So once again, the LV is a closed chamber (isovolumic.)

- Contraction causes $P_{LV}$ to increase; when we reach a point where $P_{LV}$ exceeds $P_{aortic}$ (80 mmHg), the aortic semilunar valve opens. Now we have the ventricular ejection phase (0.25s) which we split into rapid ejection followed by slow ejection.
- $P_{LV}$ starts to decrease towards the end of systole because the strength of ventricular contraction diminishes here (it still contracts but weaker.) So $P_{LV}$ will decrease until it drops below $P_{aortic}$ and the semilunar valve closes (second heart sound S2). As previously mentioned, this marks onset of diastole. The amount of blood remaining in the ventricle after ejection is called the end-systolic volume (ESV).
- For some time even after $P_{LV}$ drops below $P_{aortic}$, blood continues to move into the aorta via momentum of the blood. There is a small peak in aortic pressure due to backflow of aortic blood against the valve (represented by the dicrotic notch/incisura on an aortic pressure graph), and this helps with coronary perfusion. (Remember, the openings of the coronary arteries are found in coronary sinuses right above the cusps of the aortic semilunar valve.)
- The ventricles stop contracting and start to repolarize as we said before, and diastole starts again.

# Tension vs. Ventricular Volume

➢ Let's say we have a heart that is completely **relaxed** (in diastole). We fill the left ventricle with increasing volumes of blood and measure the tension in the heart wall (without contracting the heart itself/no stimulation). Initially, there will be a very small change in the heart wall's tension/$P_{LV}$ per added volume until a certain volume (the EDV). After this, there will be a larger increase in $P_{LV}$ per added volume until we can add no more blood. Why? At EDV, the heart is full, like a balloon blown full of air. If we want to add any more blood to the heart, we will need to exert a greater pressure than




before to force it in, more greatly increasing the pressure of the heart. If we plot this on a $P_{LV}$ vs. LV volume graph, it will give us the **passive function curve or diastolic curve**:

EXTRA: This curve is also known as the EDPVR (end diastolic pressure-volume relationship.)

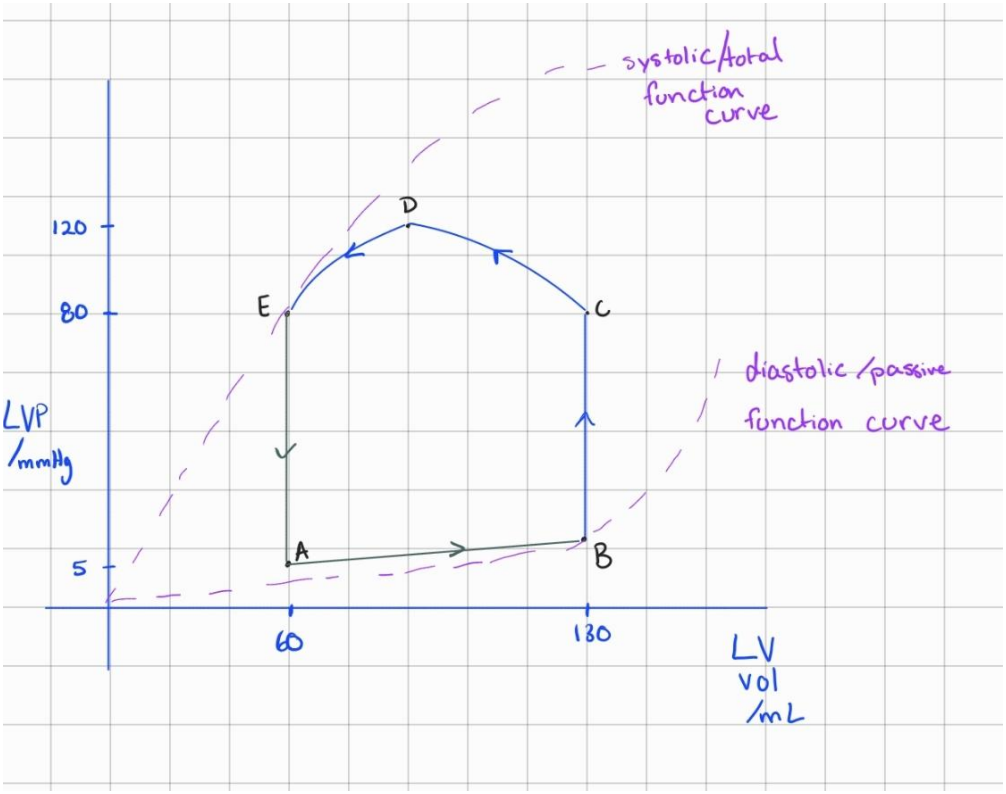
- If we repeat this same experiment, but for each volume of blood added we completely **contract** the heart (like systole) and measure $P_{LV}$ and tension of the heart wall muscle, this will give us the **systolic function curve or total function curve**. So here, $P_{LV}$ has 2 components: one due to diastole (the volume of blood that fills the LV will provide some tension/pressure) and the other due to contraction. The sum of these two effects gives the total $P_{LV}$ or tension.

EXTRA: This curve is also known as the ESPVR (end systolic pressure volume relationship.) When contractility of the heart increases, the force of contraction increases, so pressure caused by contraction increases, so the slope of the curve will increase.

- If we plot these two curves on a graph, the difference between them will give us the active tension of the heart muscle (aka tension due to contraction.) This is the same as the tension vs. sarcomere length curve in the previous lecture.
- Summary of this topic: During diastolic ventricular filling, the progressive increase in ventricular pressure causes a corresponding increase in muscle tension, which passively stretches the resting cardiac muscle to greater lengths along its resting length-tension curve. During cardiac ejection, the cardiac muscle is simultaneously generating active tension and shortening.

# PV Loop

Why did we describe these two curves? They provide the limits upon which the PV loop, our next topic, is found within. Take a look at this diagram:




This is a PV loop, a time-independent representation of the cardiac cycle (which is why we can go backwards). One loop represents one heartbeat (one complete counterclockwise cycle around the loop.) The blue segments represent systole, and the green diastole. Keep referring back to this diagram. Let's take this apart point by point, and relate it to our knowledge of the cardiac cycle:

- **Point A:** Look closely at the values for LV volume and pressure here, and think back to the cardiac cycle. The pressure is very low, and the volume is approx 60 mL. This point represents the **end systolic volume ESV** (the leftover blood in the ventricles after ejection is over.)

- **Point B:** This point is the **end diastolic volume EDV.** The volume of blood in the LV has increased to approx 130 mL (we moved forward along the x-axis), and the pressure has slightly increased due to this additional blood. Also, the QRS complex/ ventricular depolarization occurs right before B and then ventricular contraction. (Excitation always precedes contraction.) So at this point the $P_{LV}$ becomes greater than $P_{LA}$, and the mitral valve closes. The first heart sound "lub" is heard (S1.)

- **Segment AB:** This line segment represents **diastole/filling** of the heart. The duration of this line is 0.35s. (diastole without isovolumic relaxation) There

is an increase in volume (60 to 130 mL) but, since the ventricles are relaxed/relaxing during the filling phase, there is no drastic change in pressure. Towards the end of this line, the P wave (atrial depolarization) occurs, so we'll have pumping of blood from the atrium to the ventricle and see a slightly steeper increase in volume of the ventricles before B (not drawn for simplicity). The QRS complex occurs after this, as we mentioned above. After ventricular contraction and AV valve closure, we will enter the vertical line, which we'll describe in a bit.

- **Point C:** This point represents the **diastolic aortic pressure $P_d$**, aka the pressure the ventricle must overcome to open the semilunar valve and force blood into the aorta = 80 mmHg. (Don't get mixed up, this is a graph representing ventricular pressure, but here the ventricular pressure will be equal to this aortic pressure.) So after this point, the $P_{LV}$ will increase above the $P_{aortic}$, so the **semilunar valve opens.**

- **Segment BC:** This represents **isovolumic contraction phase, the first part of systole**. The pressure has increased greatly, from approx 6 mmHg to 80 mmHg without any change in volume (it's a vertical line, so the volume is the same along it.) Remember at B the mitral valve is closed, and since at C the semilunar opens, between these two points the LV is a closed chamber. It contracts, increasing the pressure, but there is still no ejection (closed chamber) and so it is a **vertical line with no change in volume.** The duration of this segment is 0.05s.

- **Point D:** This represents the **systolic pressure $P_s$**, the maximum pressure reached by the ventricle due to contraction and thus also by the blood entering the aorta.

- **Point E:** The strength of the ventricular contraction diminishes to the point where the $P_{LV}$ falls below the $P_{aortic}$ (reaches 80 mmHg again) and so the **semilunar valve shuts** and we hear our second heart sound "dub." (S2) Here

we have reached the end of systole. (Point E represents end-systolic volume and pressure.)

- **Segment DE:** At Point D, the strength of ventricular muscle contraction starts to decrease. Muscle shortening and ejection continue, but at a reduced rate. During this period of **decreased ejection**, ventricular pressure falls from 120 to 80 mm Hg. Even so, blood continues to leave the ventricle, and ventricular volume falls(from approx 75 mL at point D) to 60 mL at point E.

- **Segment CE:** This line represents **ejection/emptying of the ventricle**. After the semilunar valve opens at C, blood is pumped out of the ventricles and so **volume falls** (the line moves to the left along CE) from 130 to 60 mL. The pressure increases to $P_s$ due to ventricular contraction and then starts to decrease afterwards due to repolarization of the ventricles (blood still moves out so volume keeps decreasing.) The duration of this line is 0.25s.

- **EA line:** This line represents **isovolumic relaxation**. The semilunar valve closes at E and the mitral valve reopens at A as mentioned before. So between these 2 points the LV is **again a closed chamber while relaxing**. Pressure decreases from 80 to 5 mmHg due to this relaxation. without any change in volume (blood still didn't flow in from the atrium because of the closed AV valve, so it is a vertical line.) At A, the AV valve opens ($P_{LV} < P_{LA}$) and filling occurs. Then we repeat the loop all over again. The duration of this segment is 0.15s.

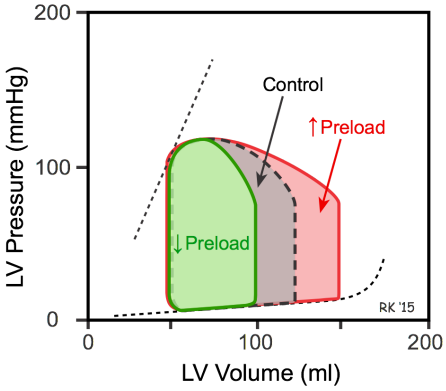
Why is this representation of the cardiac cycle useful? We can extract a lot of information from it with a single glance:

- ESV (point A)
- EDV (point B)
- Stroke volume (EDV-ESV)
- Diastolic blood pressure $P_d$ (point C)
- Systolic blood pressure $P_s$ (point D)

- Pulse pressure ( $P_s$ - $P_d$ )
- Mean pressure (⅔ $P_d$ + ⅓ $P_s$ )
- Ejection fraction (SV/EDV)

When do the PV loops change position? When we have pathological changes in the heart and blood pressure. The doctor mentioned two:

- Increased preload: (eg. Occurs in a shock patient following fluid infusion which results in increased venous return )

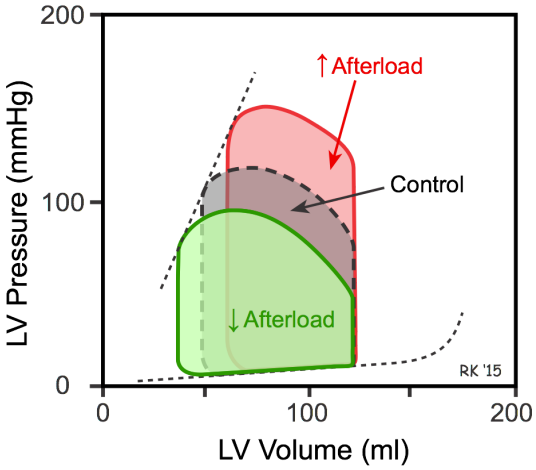



Recall that preload is the EDV, or how much blood fills the ventricle before it contracts. A greater EDV than normal will:

- Stretch the ventricles
- Increase sarcomere length
- Increase force of contraction and **cardiac output** (frank starling)
- Increase stroke volume represented by the width of P-V loop (we filled up excess blood and now we want to get rid of it)
- Slight increase in ejection fraction (both SV and EDV increased)

Afterload is the same and so:

- **Point C on the AV loop is the same** (the aortic pressure that the ventricles must overcome is unchanged.)
- The ESV is the same. (It should be noted from the figure that increasing preload increases initial muscle length without significantly changing the final length to which the muscle shortens (ESV) )




- Increased afterload: ( occurs in many pathological situations such as hypertension and aortic valve obstruction)

Recall that afterload is the aortic blood pressure, or the load the ventricle must overcome after contraction to eject blood.

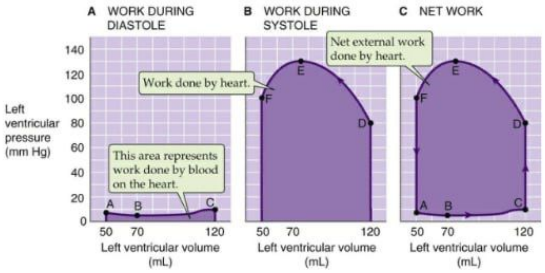
If the aortic pressure is higher:

- ➢ The ventricle must overcome this higher pressure with stronger contraction, and so the isovolumic contraction reaches higher pressure. **(higher $P_d$ )**
- ➢ **Decreased stroke volume** and thus reduced ejection fraction (remember that SV = EDV-ESV, so increasing ESV will decrease SV)
- ➢ Same EDV (preload unaffected)
- ➢ Increased end systolic volume and **higher $P_s$**

(An increase in ESV occurs as a result of reduced SV , since less blood is ejected from the ventricle >> more blood remains at the end of systole )
(Extra very important note: When describing the curves of increased preload/afterload we assume that Contractility is constant !! Otherwise, there will be changes in the graphs shifting!)

# External Work of the Heart

- ➢ Work done is the energy produced by the heart applied to the blood. The external work refers to the work done to eject the blood, and it is equal to P times V.
- ➢ This is also represented by the area within the PV loop.
- ➢ External work of the heart gives us an idea of **how much oxygen** is consumed by the heart.
- ➢ The concentration of oxygen in arterial blood is approx 20mL/dL




Let's compare the heart and the kidneys:

- ➢ The kidneys are reconditioning organs (changes composition of the blood by removing waste) so it receives more blood than it needs.
  - ○ Also, despite being very active, the kidneys extract a low percentage of oxygen from the arterial blood it receives (e.g. the conc of oxygen in the renal veins may be about 18.5 mL/dL (low arteriovenous oxygen difference))

- Should the blood flow to the kidney be halved (eg. During exercise or sympathetic stimulation), it won't experience hypoxia or ischemia (receives excess blood and doesn't utilize a lot of the oxygen it receives.)

➢ The heart receives exactly how much blood it needs (it is not a reconditioning organ.)

- The heart also extracts a very high percentage of the oxygen is receives in the coronary blood flow (60%, so the concentration of oxygen can go from 20 mL/dL in coronary arterial blood to 8 in its venous blood)
- So any decrease in blood flow or **increase in the work done by the heart (without sufficient oxygen supply to accommodate the increased oxygen consumption)** will instantly lead to hypoxia and ischemia.

# The End

# V2

Changed the sentence (diastole without isovolumetric relaxation represented by the EA line) to (diastole without isovolumetric relaxation) in the point about segment AB in PV loops