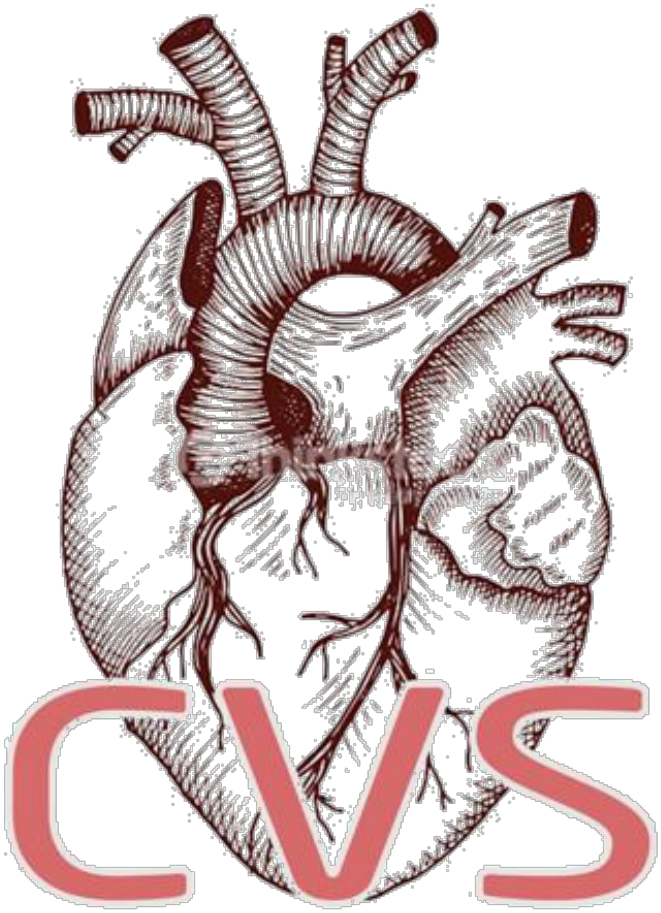


CVS

Physiology

Slide Sheet

Number:	11
Doctor:	Faisal Mohammad
Done by:	Obada Zalat
Corrected by:	Enas Ajarma

Cardiac cycle

◊ Ejection fraction

◊ Is the ratio between the stroke volume and the EDV, this fraction should be more than 55%

◊This fraction is a measure of contractility but what is the contractility?

•The change in the stroke volume with a fixed EDV, but if the EDV is fixed, how the stroke volume will change to get a positive inotropic effect ?

By decreasing the ESV, and that will cause increasing in the heart efficiency (ejection fraction)

** Remember : Stroke volume = EDV – ESV

◊ Atrial pressure

◊ Can be measured by a transducer that can record 3 waves:

1-A wave: record the pressure during the atrial systole.

2- C wave: record the pressure during ventricular systole

3- V wave: record the pressure during ventricular diastole

◊These atrial waves are very important, that they can be seen in the patient who suffer from congestion in the right ventricle (too much volume), as these pressure waves can appears as deflection in the jugular vein .

◊Sometimes these waves could be obvious, in these cases we can have for example third degree heart block; in this case the atrial will contract when the AV valve is closed, that will case backward pressure, which can be seen in the jugular vein pressure.

◊ Sounds of the heart

☞S1 (lup)……appears after AV valve closure (mitral & tricuspid)

☞S2 (dup)……appears after Semi-lunar valve closure (aortic & pulmonary)

•The time between S1 and S2 is 0.3 sec (the duration of ventricular systole), as well as the time between S2 and the next S1 is 0.5 sec (the time of ventricular diastole).

☞S3…..appears in the phase of rapid filling of the ventricles

☞S4…..appears at the atrial systole

◊From where you can hear the heart sounds?

•the first heart sound S1 can be heard at the 5th intercostals space

•the second sound S2 can be heard either from aortic part (at the right side) or from the pulmonary part (from the left side), and the sound can be heard at the both sides at the same time.

• When there is deference in the time (the time of the sound), there will be a split in the sound that called **split heart sounds**. This split is pathological, but sometimes it could be normal, such as in children.

◊The ventricles volume

◊the left and the right ventricle differ in the pressure NOT in the volume. Actually, if they were different in the volume, for example; the right ventricle eject 70ml/beat, and the left eject 69ml/beat, and the heart rate was 70beat/min, every minute there will be 70 ml accumulate in the left ventricle, so after one hour, there will be 4200ml of blood stay in the left ventricle in that one hour, and this shouldn't happen at the normal situation.

◊There is lit bit deference in the stroke volume between the two ventricles; the left ventricle has a higher stroke volume than the right ventricle but why?

•because of **the bronchial veins** that drains the lung tissues , these veins will drains into the pulmonary veins, that will drains into the left atrium and consequently to the left ventricle , and this will make more EDV in the left ventricle, so more stroke volume (But this deference is very small, and not sufficient to produce the case mentioned before) .

◊Cardiac output

◊the cardiac output is the amount of blood pumped per minute, and it's almost the same for the two ventricles (remember that the left ventricle is a higher bit than the right ventricle; due to the fact that the bronchial veins drains to the left ventricle).

◊ CO= heart rate *stroke volume, but if we increase the stroke volume is the CO will increase?

•Actually, I can't know; because of the presence of another factor (HR), that I should take into consideration.

◊according to the frank-starling law, an increase in the passive tension (length of the cardiac muscles) within the physiological limits, the active tension (contraction force) will increase, but in the heart, I'm not able to measure the length of the muscles, instead I would measure the volume, which is proportional to length (once we talk about the volume we mean the EDV).

◊So, within the physiological limits, an increase in the EDV will cause an increase in the stroke volume as well as increase in the cardiac output (if the heart rate is constant) .

◊Cardiac output and cardiac reserve

◊**Intrinsically**, we can regulate the cardiac cycle by frank-starling law. So, we can increase the EDV, which can lead to more cardiac output until 15 L/min, but the working cardiac output (resting CO) is 5L/min, but if the normal blood volume is 5L, from where the extra 10 L came?

-

•Actually , we don't have an extra volume, the heart only eject those 5 L in less time than in normal situation, that in one minute , we will have 15L pumped.

•The deference between the maximum cardiac output and the resting cardiac output (working cardiac output) is called **cardiac reserve**.

•In athletes, the CO might reach 35L/m; so they have a high cardiac reserve.

◊Also we can regulate the cardiac output **extrinsically** by the autonomic nerves system

---

Regulation of the cardiac cycle

◊Regulation of the stroke volume

◊Factor that affect the stroke volume:

•Preload

•Afterload

•contractility

◊**Preload** is the amount of load that is found in the heart before it contracts, so it caused by the passive tension (Which is proportional to the EDV), so we can call the preload, the EDP (end diastolic pressure)

•An increase in the preload within the physiological limits, cause an increase in the stroke volume according to frank-starling law.

◊**Afterload** is the pressure in the aorta during diastole of aorta

•the left ventricle has to develop a pressure higher than diastolic pressure in the aorta; to open the semi-lunar valve, but if the afterload increases, the left ventricle has to develop a higher pressure to open the valve. This increase in pressure needs energy, and energy needs more oxygen, that can come from more blood.

•So, an increase in the afterload will decrease the stroke volume, until the ventricle spends more energy, and this is the serious side of hypertension, so if the patient doesn't have this extra energy to overcome the hypertension (increase in the afterload), he might develop MI.

◊**Contractility** means that we have a fixed EDV and increase stroke volume, by decreasing the ESV

◊Regulation of the cardiac output

◊we can change the CO by changing the SV or the heart rate.

•we can change the stroke volume by what mentioned before:

1-change the preload, according to frank-starling law

2-change in the afterload, decrease in the afterload will cause increase in the SV

3-Cotractility, if you increase contractility, you increase SV by decreasing the ESV, but if you decrease the contractility, you will decrease the SV, by increasing the ESV

•We call the change in contractility; inotropic effect, but how we can change it?

☞Positive inotropic, increase in the contractility that can be achieved by activation of the sympathetic nerves system. Also, drugs such as glucagon , increase in the Ca+ ions and hypokalemia can have positive effect.

☞Negative inotropic, decrease in contractility, that can be caused by Ca+ channels blockers, hyperkalemia and acidosis (decrease in the Ph) that can fix the enzymes activity

•Also, we can change the heart rate, and we call the change in the heart rate; chronotropic effect

☞Positive chronotropic, increase heart rate by sympathetic stimulation, increase in Ca+ ions, catecholamines, thyroid hormones and extreme temperature

☞Negative chronotropic, decrease heart rate by parasympathetic stimulation

◊We know that the sympathetic nervous system cause positive inotropic and positive chronotropic effects, but how?

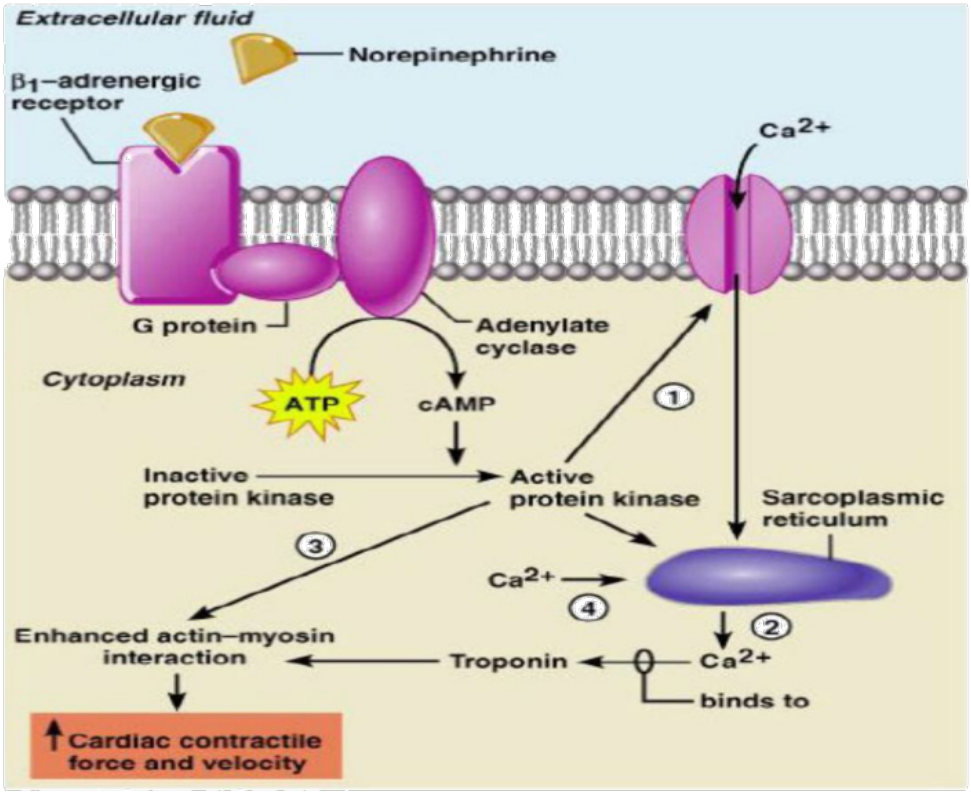
•Norepinephrne is released from sympathetic nerve ending, then it will bind to B1-adrenergic receptor, this will cause the activation of G protein

•The activated G-protein will activate adynelate cyclase, which will produce cAMP, that will activate Protien kinase A

•Protein kinase A will activate (via phosphorylation) :

☞Phospholambin, which will activate the Ca+-pump of the SR, so we will have more Ca+ stored in the SR (sacroplasmic reticulum). In the next beat, more Ca+ will be released, so more contractility (positive inotropic).

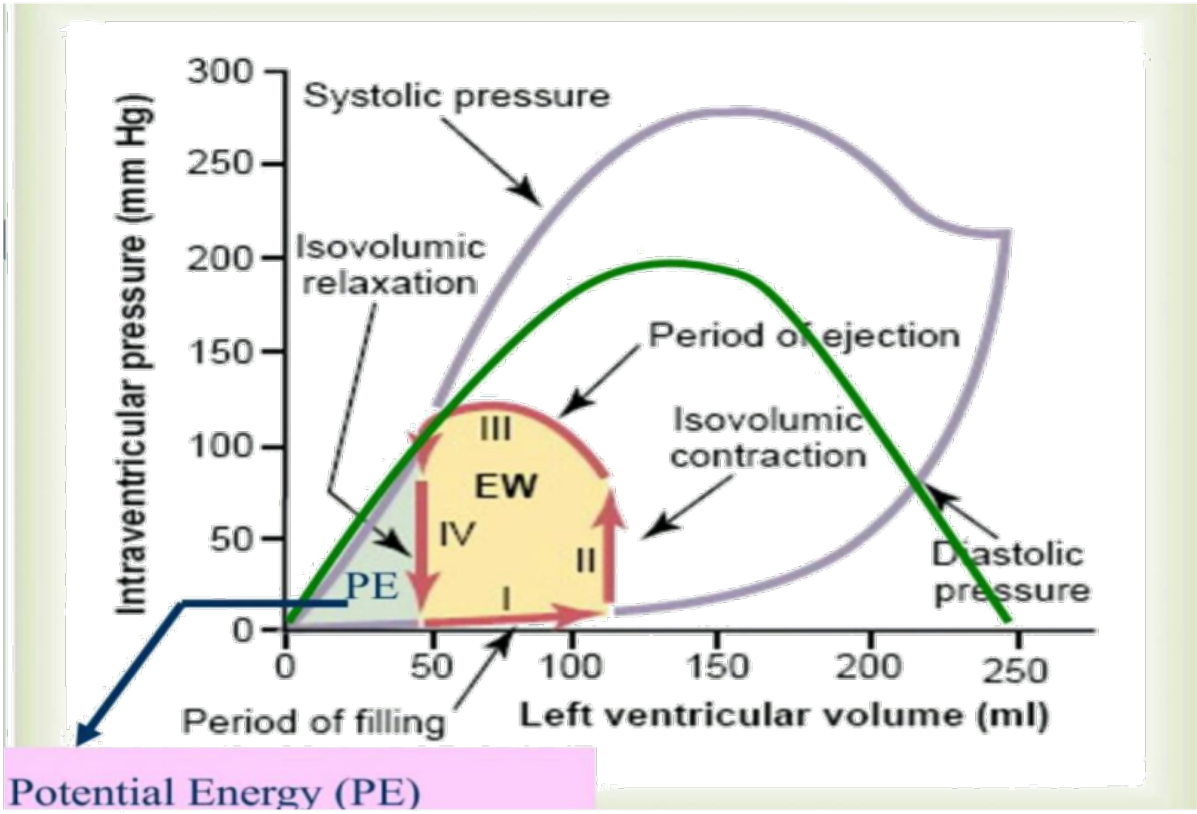
Also, this activated Ca+-pump will retain the Ca+ to the SR faster, so the time of diastole will be decreased, and once you decrease the diastole time, you will decrease the heart beat time, and eventually the heart rate will increase (positive chronotropic)



◊mathematical representation of the cardiac cycle

◊In the case of frank-starling law curve, we represent the X-axis by the length of the muscles, and the Y-axis by the tension

◊But if we want to present these values (length and tension) in the term of cardiac cycle, we can represent the x-axis by the left ventricular volume and the y-axis by the pressure



•The cardiac cycle starts from the ESV, and then the AV value opened, that will cause filling in the ventricle

•during filling, the pressure will increase, but very little amount; because the ventricle is in the relaxed phase. We call this ventricular compliance (line I).

•After that, systolic phase starts, at the beginning of this phase the AV valve will close, and the isovolumic-contraction occurs, where the volume is fixed, but the pressure still rising (line II).

•Until, the pressure of the ventricle exceeds the aortic pressure, and this will cause the open of semi-lunar valve, so the blood volume starts to decrease.

•During the systole after the semi-lunar valve is opened, the volume will decrease as well as the pressure will still increasing until 120 mm Hg, then systole ends, and semi-lunar valve closes (line III)

•After that, the diastolic phase starts by isovolumic-relaxation, until the pressure reaches Zero mm Hg (less than the atrial pressure), then AV valve opens, and the cycle starts again (line IV).

◊We can increase this cycle; by increasing the volume, which will cause the curve to be shifted, until we reach the optimal length. This range (that we can increase the volume of the blood in it); is due to the fact that the heart works in a less length than its optimal length making a range to increase the volume, opposite to the skeletal muscles that works at its optimal length.

◊In this cardiac cycle, we have three types of energy (work) spent by the heart during the cycle, and they can be calculated from the curve:

1) External work, it is the energy spent by the heart to move the blood inside the circulation, and it is equal to the area under the rectangular curve, but how to find the value of this area?

•By multiplying the mean change in pressure (pressure at systole – pressure at diastole) by the stroke volume (EDV-ESV)

2) Potential energy, it is the energy that stored in the system to use it, and equal to area under the triangle.

•How we can use this energy?

For example, if we shift the curve to the left (but with fixed EDV) , then the stroke volume will increase, that mean we have an increase in the contractility(positive inotropic effect) , and this effect needs more energy, that can be obtained from the potential energy.

3) Kinetic energy, it is the amount of energy that spent to move the blood inside the orifice that guarded by the semi-lunar valve.

•Kinetic energy =1/2*Mass*V^2, normally when the semi-lunar opens, the blood will move easily, so normally the kinetic energy is very small (less than 1%)

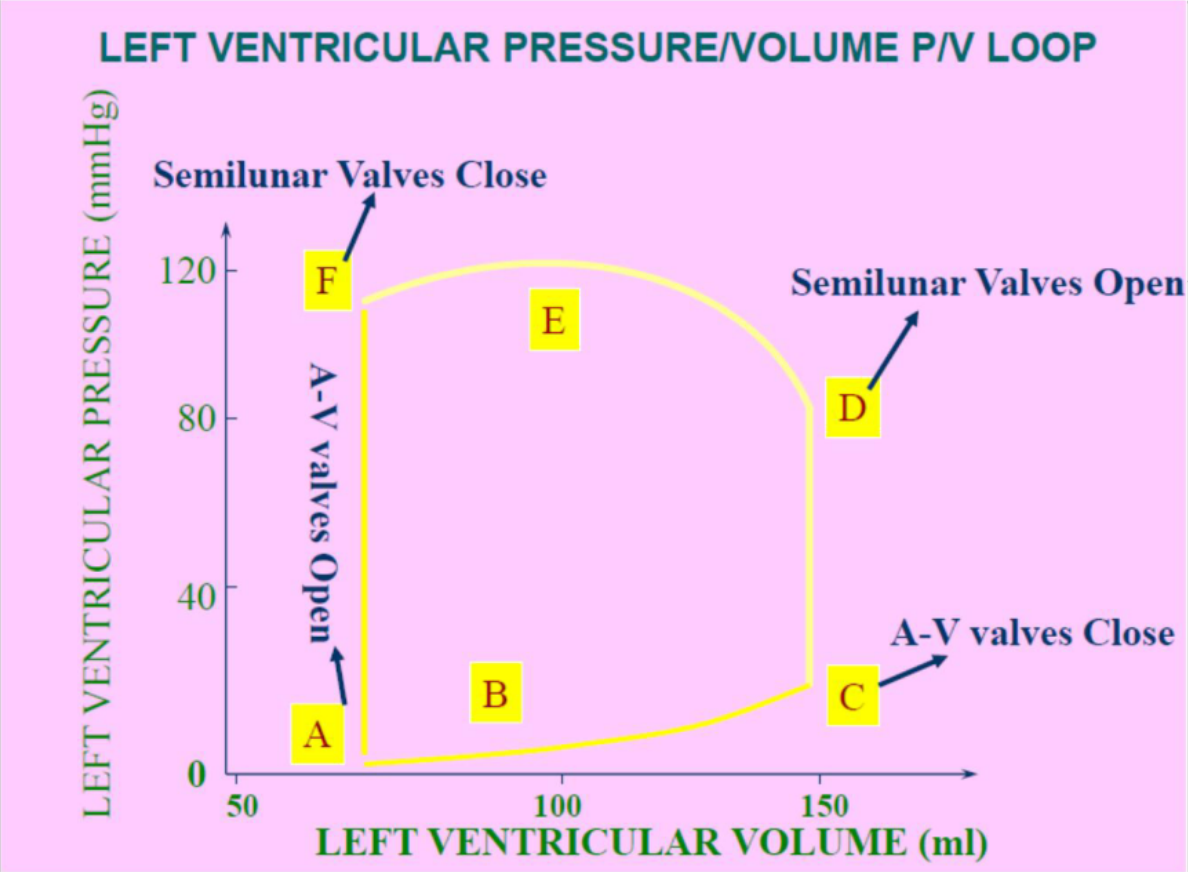
•In some cases, the kinetic energy might increase, such as in aortic stenosis, when the stenosis happened, we need more kinetic energy to

move the blood across this narrow orifice. In this case, it might increase to 50%.

•In order to bring this high energy, the heart need more oxygen, which could be attained by increase blood flow, but if the patient hasn't the enough blood supply, the patient will ends up by infarction.

◊The most work done by the heart is the External

work ◊ let's have a quick review on this curve



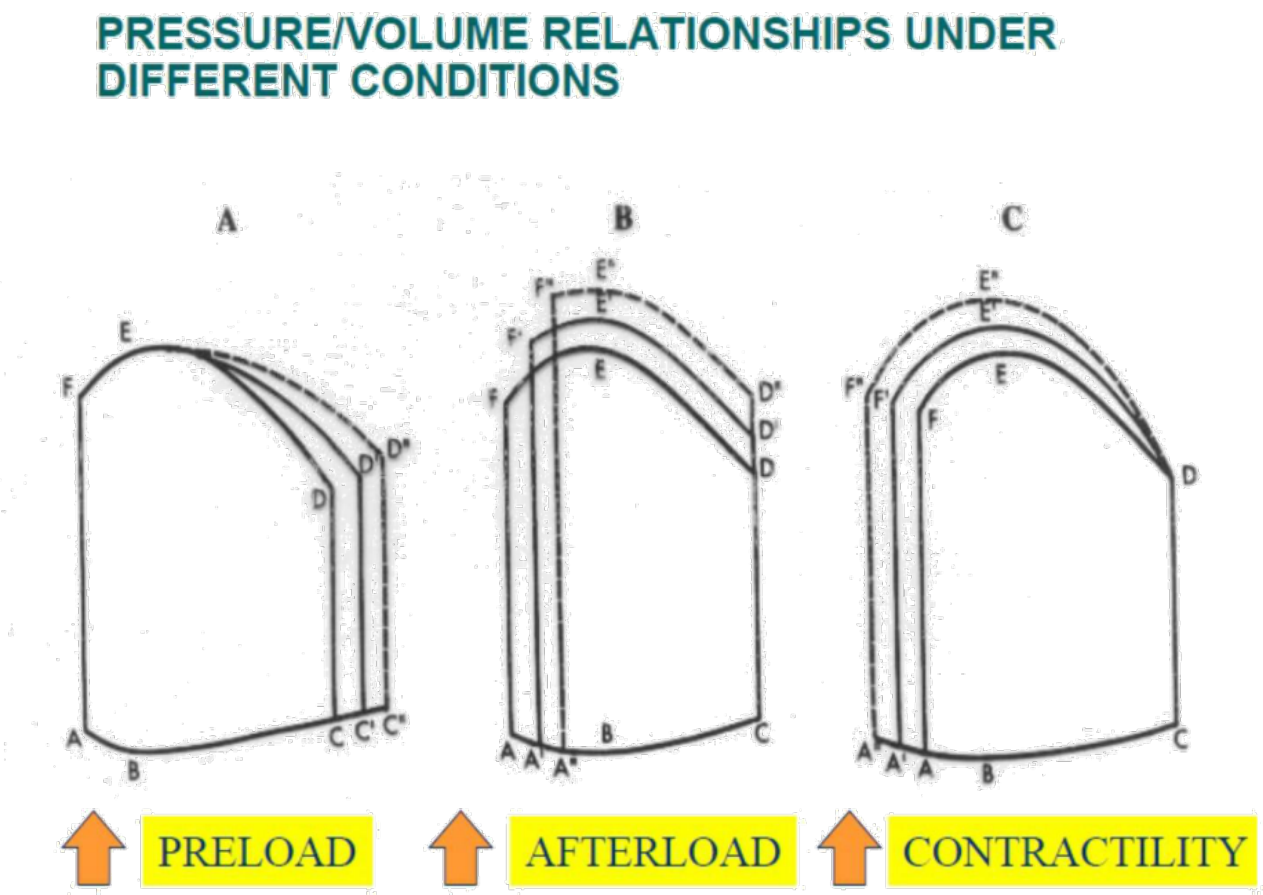
• B is the filling phase, C-D interval is the iso-volumic contraction period, E is the ejection phase, and F is the start point of the iso-volumic relaxation phase.

•the first heart sound (S1) appears at phase C, while the second heart sound (S2) appears at phase F.

•The time between F and C is represent the time of diastole, but the time of C,D,E,F (from C to F) is the time of systole. But which one is bigger?

You may think that is C-F depending on the curve, but be careful that this curve didn't represent the time. So, the F-C time is much greater than C-F, because it represent the diastole.

◊Lets have a look on the curve in different conditions:



•In the first curve from the left, we have an increase in the preload (increase in the EDV), and this mean more the stroke volume, according to frank-starling law (the curve shifted to the right due to the increase in the EDV)

•In the middle one we have increase in the Afterload that will cause decrease in the stroke volume (shift to the right due to the increase in the ESV)

•At the last one, we have increase in the contractility, which will cause increase in the stroke volume, by decreasing the ESV (shift to the left due to the decrease in the ESV)

GOOD LUCK