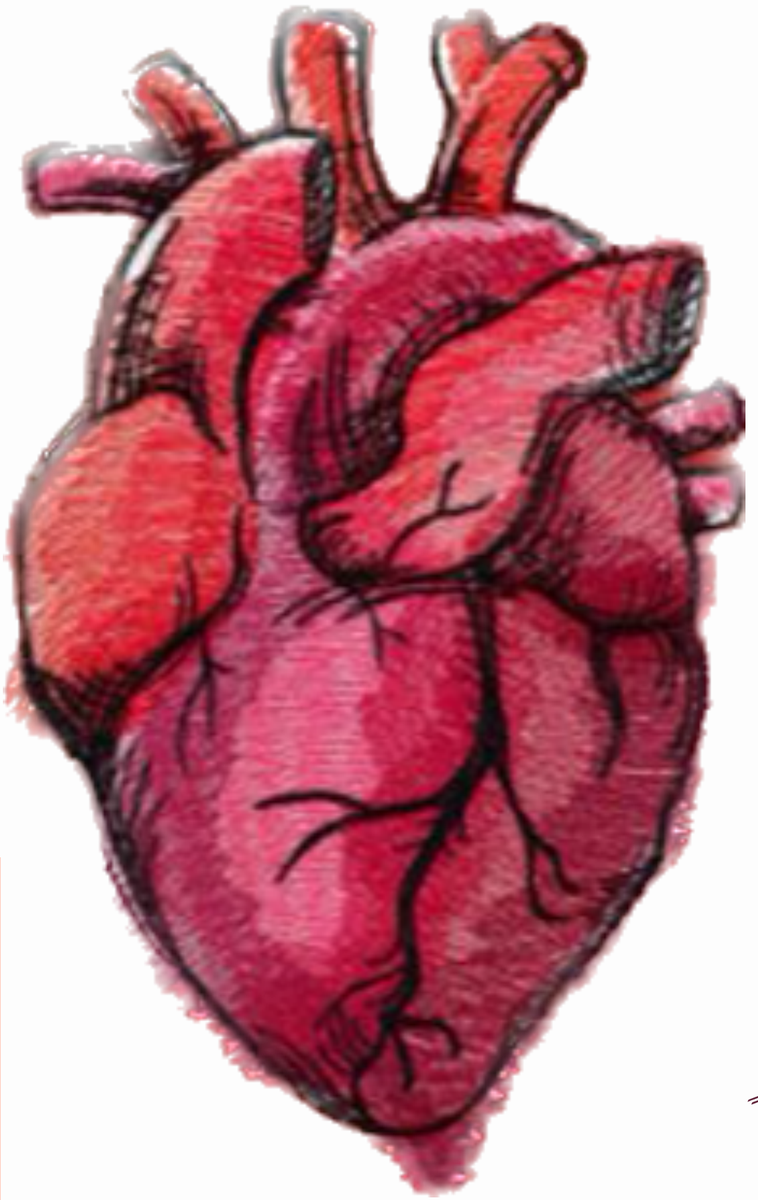

النادي الطبي

CARDIOVASCULAR SYSTEM

SUBJECT : physiology

LEC NO. : 7

DONE BY : Abdullah Bmnj Mustafa

وَقُل رَّبِّ زِدْنِي عِلْمًا

SCAN ME!

The Cardiac Cycle

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Lecture Objectives:

1. Define cardiac cycle.
2. Be familiar with and explain curves for the various events occurring in the heart, inlet veins, and outlet arteries.
3. Recognize systolic and diastolic duration.
4. Understand isometric and relaxation; and the ejection phases.
5. Explain volume – pressure relationship in the left ventricle.
6. Explain the development of first and second heart sounds.
7. Define murmurs and how systolic and diastolic murmurs are produced.

- **Definition:** understand all events that happen in the heart at which time

  The cardiac events that occur from the beginning of one heart beat to the beginning of the next

- **The Cardiac Cycle consists of *Diastole and Systole*** → we talk about ventricles

  During diastole → heart relaxes and fills with blood Longer than systole

  During systole → the heart contracts and eject blood (i.e. emptying)

Note: If heart rate is 72 beats/min, the duration of the cardiac cycle is about 0.8 second per beat. Of which 0.3 second is for systole and 0.5 second is for diastole.

$\frac{\text{time seconds}}{\text{heart beat}}$ = time of the cardiac cycle

secondary role → most of the cardiac cycle is carried by the ventricles

Atrial contraction (= Primer Pump)

Normally 80% of the ventricular filling is achieved even before the onset of atrial contract. that is why atrial fibrillation is not fetal

Atrial contraction usually increases the ventricular pumping effectiveness as much as 20%. However; atrial failure is only noticed in exercise, then signs of heart failure develop, especially dyspnea (shortness of breath) heart failure → start as dyspnea at exercise stress test

air hunger when excercised.

Systole is composed of:

1. Isovolumic (isovolumetric) contraction phase. In this phase cardiac muscle tension is increasing but little or no shortening of the muscle fibers is occurring. Same as skeletal muscle
2. Ejection phase (70% of ejection is completed in the first ⅓ of the phase). Not constant amount of blood is getting out per unit of time

Diastole is composed of:

1. Isovolumic (isovolumetric) relaxation phase
2. Ventricular filling phase – this phase can be further divided into three thirds, namely the rapid filling, reduced filling (diastases), and the contribution of atrial contraction

very important to memorise them !!

The Cardiac Volumes

amount of blood at the maximum amount of filling → not fixed due to its elasticity

- End diastolic volume (110-120 ml) – it is the maximal amount of blood the ventricle contains during the cycle. The maximal value it can reach is 150-180 ml. The EDV is an index of the ***Preload***

directly proportional with EDV

how much is the ventricle stretched before the begning of contraction → sarromen length

- End systolic volume (40-50 ml) – it is the least amount of blood the ventricle contains during the cycle. The minimal value it can reach is 10-20 ml. The higher the myocardial contractility the lower is the ESV.

decrease in heart rate lead to increase in diastole → more filling → volume increases → give us a clue about the contractility function

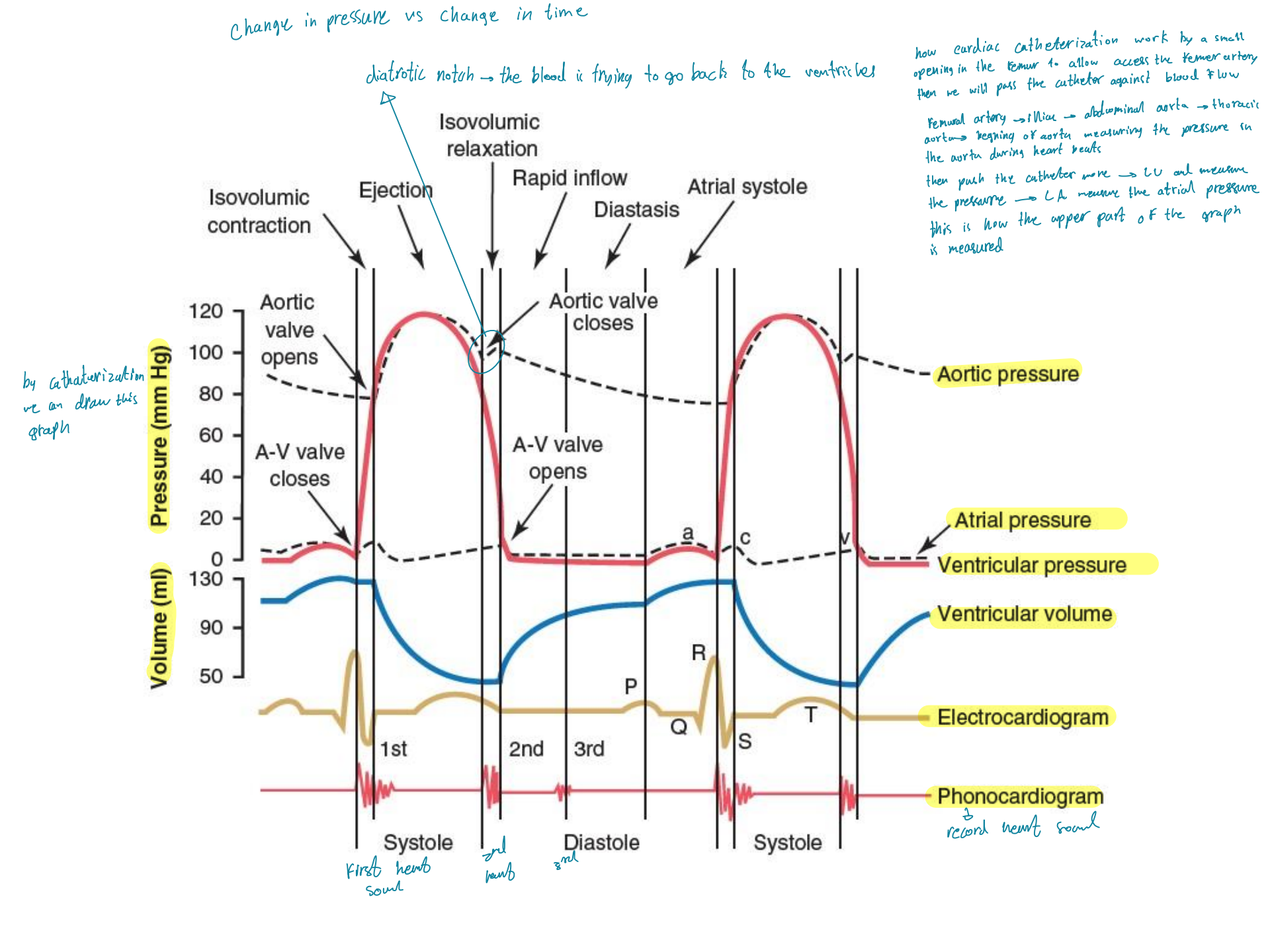
- Stroke volume output (70 ml) – it is the amount of blood pumped out of each ventricle with each contraction. SV= EDV – ESV

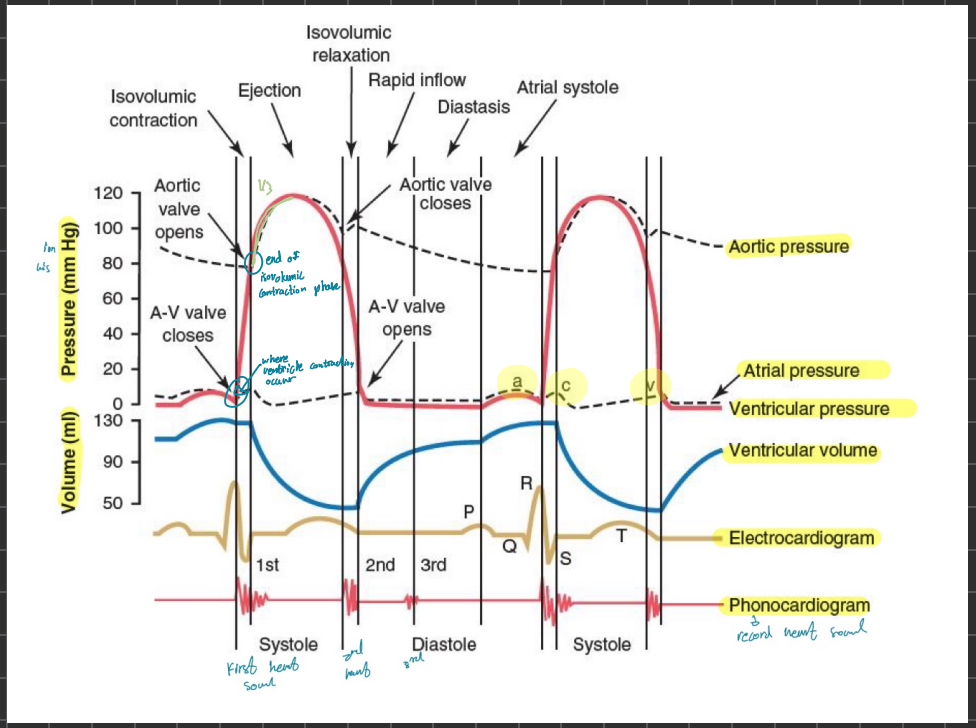
the amount of blood pumped by the heart called stroke volume

- The ejection fraction (EF) is the fraction of the EDV that is ejected. It is usually equal to about 60 percent.

the percentage of how much blood is pumped from the blood in the ventricle

2/3 of what was in the ventricle


Change in pressure vs change in time
diatrotic notch → the blood is trying to go back to the ventricles
how cardiac catheterization work by a small opening in the femur to allow access the femur artery then we will pass the catheter against blood flow
femoral artery → iliac → abdominal aorta → thoracic aorta → begning of aorta measuring the pressure in the aorta during heart beats
then push the catheter more → LV and measure the pressure → LA measure the atrial pressure
this is how the upper part of the graph is measured
by catheterization we can draw this graph
Isovolumic contraction
Ejection
Isovolumic relaxation
Rapid inflow
Diastasis
Atrial systole
Aortic valve opens
Aortic valve closes
A-V valve closes
A-V valve opens
Pressure (mm Hg)
120
100
80
60
40
20
0
Volume (ml)
130
90
50
a
c
v
P
Q
R
S
T
1st
2nd
3rd
Aortic pressure
Atrial pressure
Ventricular pressure
Ventricular volume
Electrocardiogram
Phonocardiogram
record heart sound
Systole
Diastole
Systole
First heart sound
2nd heart
3rd



* opening of aortic valve doesn't cause sound, after the opening of the aortic valve the ejection phase start where blood is being pumped out of ventricles, so the aorta & ventricle cavity is the same one now & they will be equal in pressure.

diastolic pressure :· the pressure in the aorta at it lowest because at this point it is the end of the previous diastole so the aortic pressure ab this point is the aortic pressure at the end of diastole and it's the lowest pressure of aorta so it's diastolic blood pressure

* the highest pressure of the aorta = to the highest pressure of ventricle when the aortic valve open and it's called systolic pressure — the average is 80 → diastolic 120 → systolic

at the first third of ejectio phase ≈ 70% of the blood is out then the ejection decrease → ↓ pressure in ventricle & ↓ pressure in aorta. then the pressure in the ventricle falls rapidly so the blood in the aorta try to get back to the ventricle this it when the aortic valve close again to prevent the blood backflow to the ventricles making the second heart sound → aortic valve is isolated from the ventricle cavity this is when T wave occur after the end of T wave the diastole start

* the second sound is the end of systole

the contraction occur when the QRS end (the heart is completly depolarized) when that happen blood try to go to the atria but the matrial valve close & prevent that from happening also the tricuspid valve closed making the first sound → so when hearing the 1st sound is when diastole start

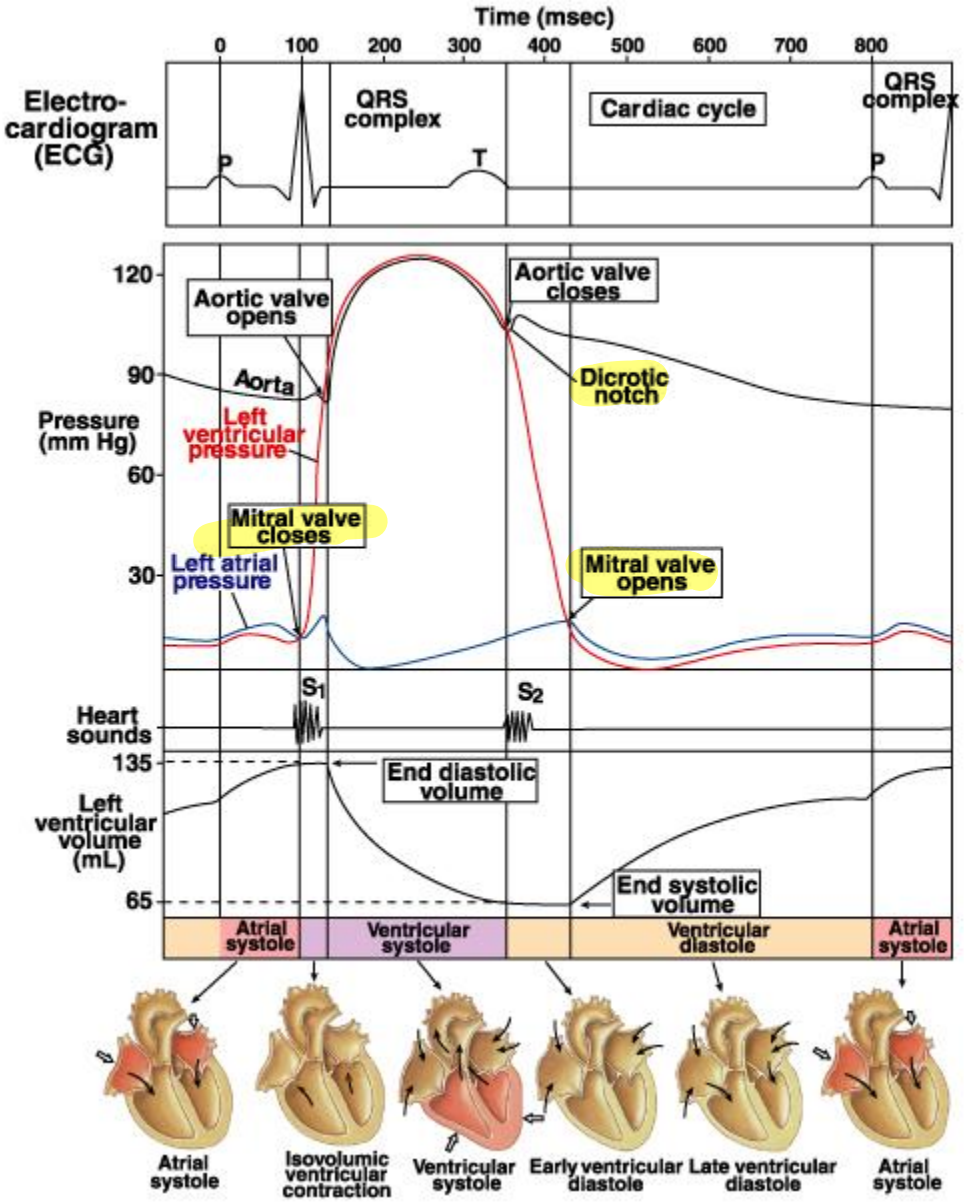
at the begning of contraction the pressure increase but ventricle volume stay the same why? because aortic pressure = 80 while ventricular pressure ≈ 3 so this pressure won't allow the aortic valve to open so when the ventricular pressure is higher than the aortic pressure the aortic valve will open & the aortic valve open then the ventricular volume decrease.

* Note the first part of diastole there is no change in ventricular volume so that is why it's called isovolumic contraction phase ↑ pressure without change in pressure

the short phase of diastole is called isovolumic relaxation phase → when the aortic valve close, the volume in this phase represent the amount of blood that stayed in the ventricle after ejection phase also in this phase the heart volume stay the same because the pressure in the ventricle is higher than the pressure in the atria so the mitral valve stays open, at the end of this pahase the mitral valve open

the second stage of diastole is longer "filling stage" it happen when the AV valves open, it's divided to three stages 1) Rapid inflow 2) diastasis 3) atrial systole (ventricle pressure drops)
the first 2 stages there is no contributing from the atria to fill up the ventricles while in the third stage the atrial contraction contribute to the ventricle filling during the diastole the ventricles & atria is considered the same cavity,
during the ventricular systole the atria were filling up with blood by the IVC, SVC & pulmonary veins, when the AV valves open the rapid filling occur
then the second stage starts known as reduce filling "diastasis" now one cavity from the pulmonary veins to the LV same as right, the third stage cause increase in atrial pressure & increase the filling between 10% to 0%.

why the pressure in the aorta decrease during systole? because the blood is being distributed to the aortic branches causing to in its pressure


Time (msec)
0
100
200
300
400
500
600
700
800
Electro-cardiogram (ECG)
P
QRS complex
T
Cardiac cycle
P
QRS complex
120
90
60
30
Pressure (mm Hg)
Aortic valve opens
Aorta
Left ventricular pressure
Aortic valve closes
Dicrotic notch
Mitral valve closes
Left atrial pressure
Mitral valve opens
S1
S2
Heart sounds
135
65
Left ventricular volume (mL)
End diastolic volume
End systolic volume
Atrial systole
Ventricular systole
Ventricular diastole
Atrial systole
Atrial systole
Isovolumic ventricular contraction
Ventricular systole
Early ventricular diastole
Late ventricular diastole
Atrial systole

Atrial Pressure Changes and the Jugular Pulse

because the pulmonary veins are within the chest we can't visual these waves in the left atrium while in the right atrium we can visualise these waves by the jugular vein specially in children due to their thin skin when the patient in supine view

When the right atrium contracts, a retrograde pressure pulse wave is sent backward into the jugular vein producing three characteristic waves in the record of jugular pressure.

- The **a wave** is due to atrial systole.

  Factors that impede the flow of blood from the atria to the ventricles, such as *tricuspid valve stenosis*, increase the amplitude of the a-wave.

- The **c wave** is produced by the bulging of the tricuspid valve into the atria during isovolumetric ventricular contraction.

  Tricuspid valve incompetence results in a high amplitude c-wave.

- The **v wave** mirrors the rise in atrial pressure before the tricuspid valve opens during diastole.

  Tricuspid valve stenosis results in an attenuation of the descending phase of the v-wave.

it can be seen but can't be felt

so the jugular veinous pulse disapear → if not right side heart failure

The Heart Sounds

1. The First Heart Sound (S_1) (lub) is associated with the closure of the atrioventricular valves. It signifies the start of systole. It is a low-pitched sound.

   The intensity of the first heart sound is proportional to the strength of myocardial contraction, and its evaluation is useful in clinical diagnosis.

2. The Second Heart Sound (S_2) (dub) is caused by the sudden closure of the semilunar valves. Its intensity is proportional to the intensity of the valve closure.

   Clinically, it signifies the end of systole and the start of diastole. However, systole is more correctly considered to be concluded when the T wave ends on the ECG.

   Splitting of S_2, also known as **physiological split**, normally occurs during deep inhalation. A widely split S_2 can be associated with several different cardiovascular conditions, including Right bundle branch block and atrial septal defect.

   more blood in the RA during deep inspiration → when heart contract the RA & RV have more blood than in the left side, so the LA will be empty before the RA so the pulmonary valve closes after the aortic valve

   left side close before the right side

3. The Third Heart Sound is not normally heard in healthy people. This sound is due to abrupt cessation of ventricular distention and the deceleration of blood flow just before diastasis.

   the pressure in the LA is higher than in the RA so blood will move to the RA so MV more blood so the contraction will take longer in the right side

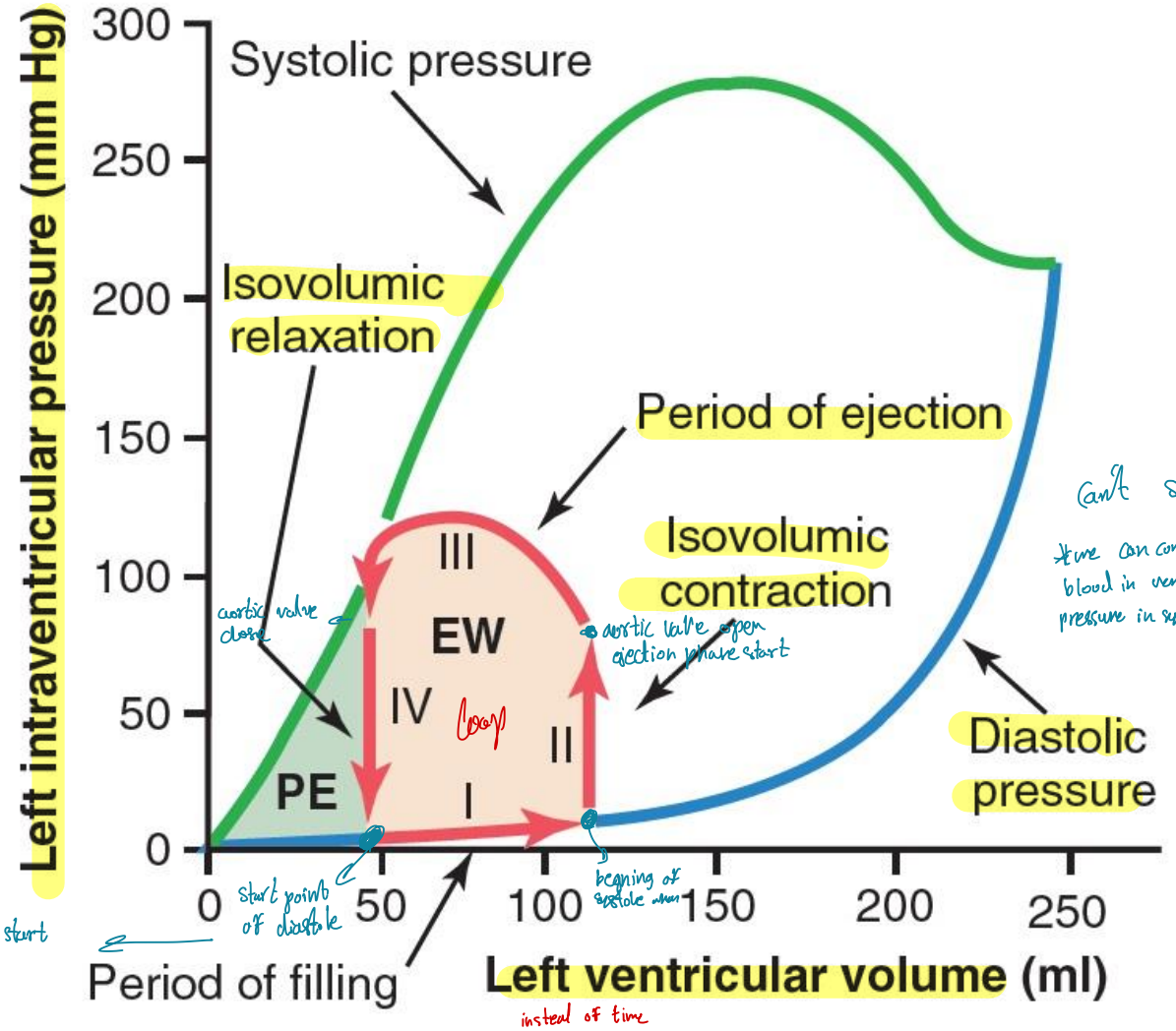
   the rapid flow of blood to the ventricle cause vibration of the ventricles wall creating the 3rd sound which can't be heard by stethoscope, if can be heard is an indication of heart failure

   The third heart sound is amplified in abnormally stiff or distended ventricles, such as that associated with **heart failure**, and its presence over the age of 40 is considered a serious sign of underlying cardiac abnormalities.

The Volume-Pressure Curves

1. The diastolic pressure curve – notice the significant rise in pressure after 150 ml filling.
2. The systolic pressure curve – notice that maximum pressure is reached at 150-170 ml filling then pressure decline starts.
3. The Volume – Pressure loop – it describes the cycle independent of time factor.

Volume pressure loope



when AV valve open & filling start

can't stretch more

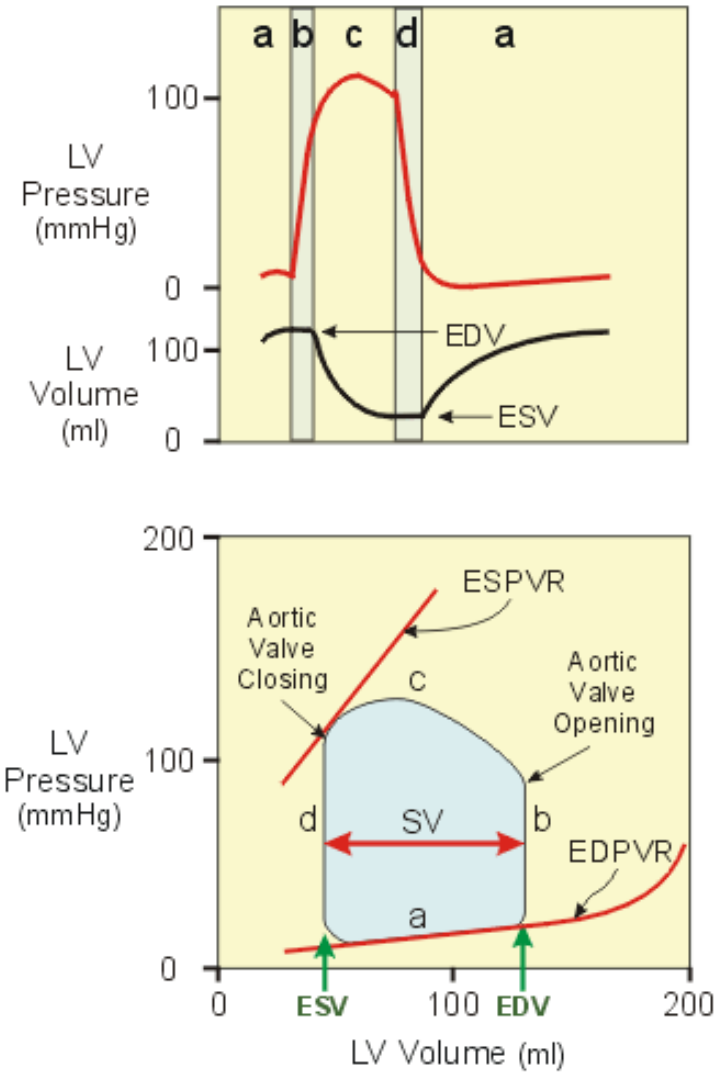
*we can conclude that the more blood in ventricles the more the pressure in systole

the area of the loop represents net external work $cm^3 \times \frac{N}{cm^2} = N \cdot cm$ (work)

the work (energy) that carried by the blood ejected from the W

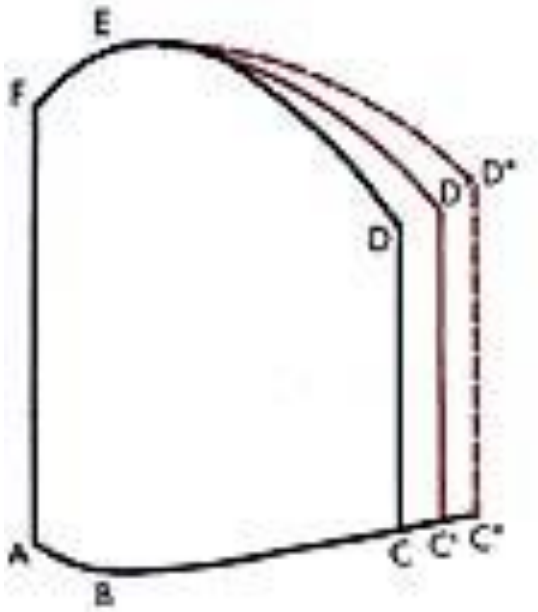
the horizontal diameter = SV amount of pumped blood

Relationship between left ventricular volume and intra-ventricular pressure during diastole and systole. Also shown by the red lines is the "volume-pressure diagram," demonstrating changes in intra-ventricular volume and pressure during the normal cardiac cycle. EW, net external work; PE, potential energy.



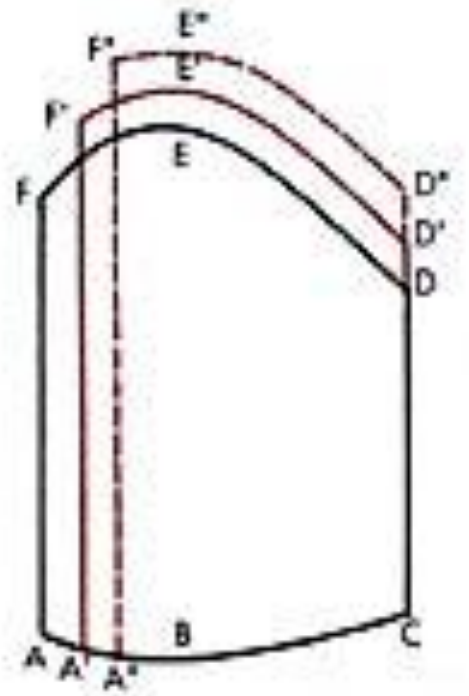
The generation of a left ventricular pressure-volume loop *(bottom panel)* from pressure and volume changes during cardiac cycle *(top panel)*. *Abbreviations*: a, ventricular filling; b, isovolumetric contraction; c, ventricular ejection; d, isovolumetric relaxation; EDV and ESV, left ventricular end-diastolic and end-systolic volumes, respectively; EDPVR, end-diastolic pressure-volume relationship; ESPVR, end-systolic pressure-volume relationship.

* the normal situation is represented in black



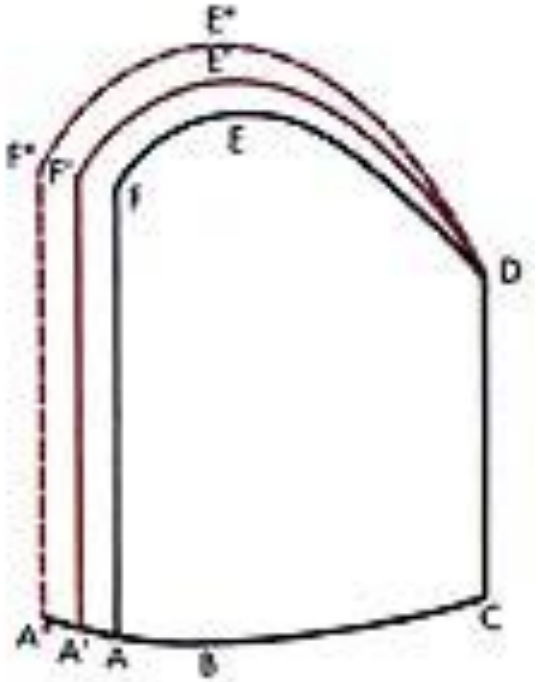
Increased preload

increase blood volume in ventricles before contraction, the pressure in isovolumic period increases before the opening of aorta but it will contract and return to the same end systolic volume



Increased afterload

increase in the force needed to move the blood from the ventricle to the aorta, so the systolic pressure increase and the remaining blood volum in ventricle after contraction increase such as in valve stenosis



Increased contractility

the end of systolic pressure will be higher, and the remaining blood after ventricular contraction will be decreased

Test Question:

Q. The dicrotic notch on the aortic pressure curve is caused by?

A. Closure of the mitral valve.

B. Closure of the tricuspid valve.

C. Closure of the aortic valve.

D. Closure of the pulmonary valve.

E. Rapid filling of the left ventricle.