CVS
PHYSIOLOGY

Modified NO: 11

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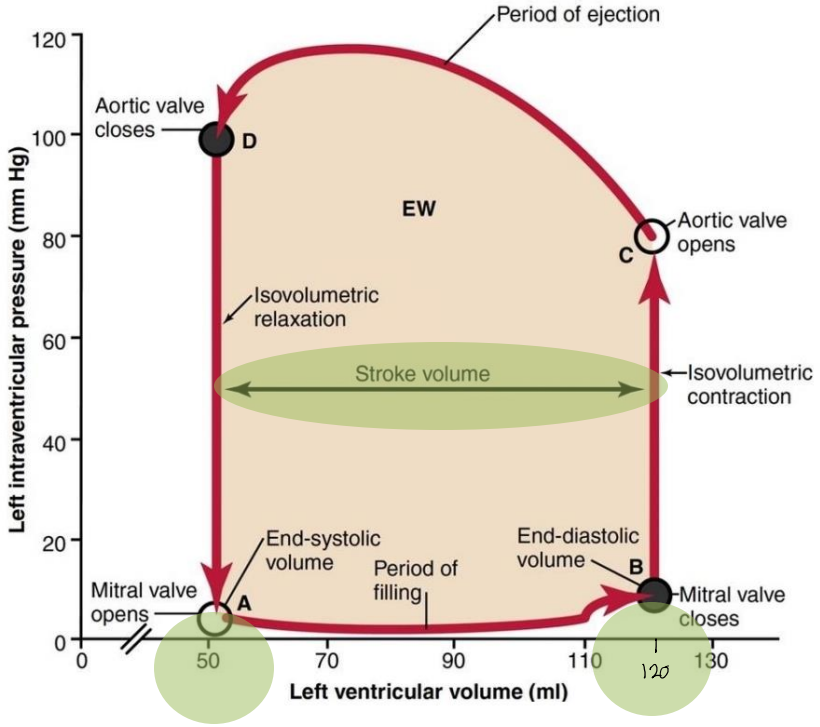
الدكتور: فاطمة الريالات

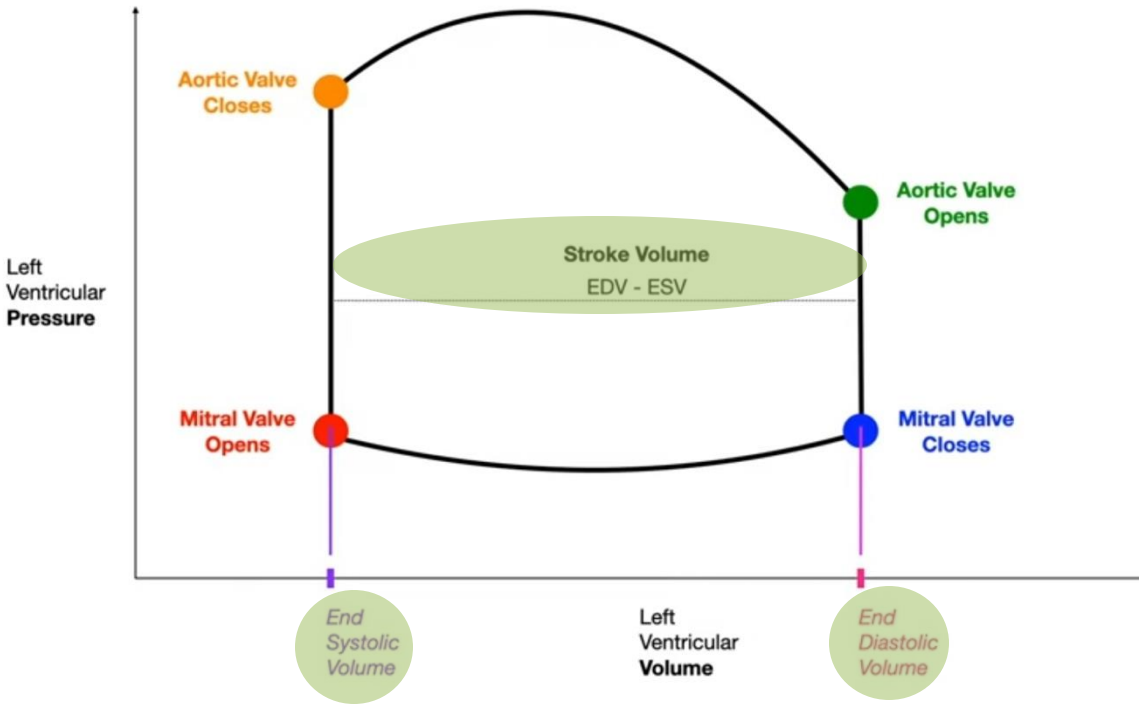
Mechanical events of the cardiac cycle-2

Color code
Slides
Doctor
Additional info
Important

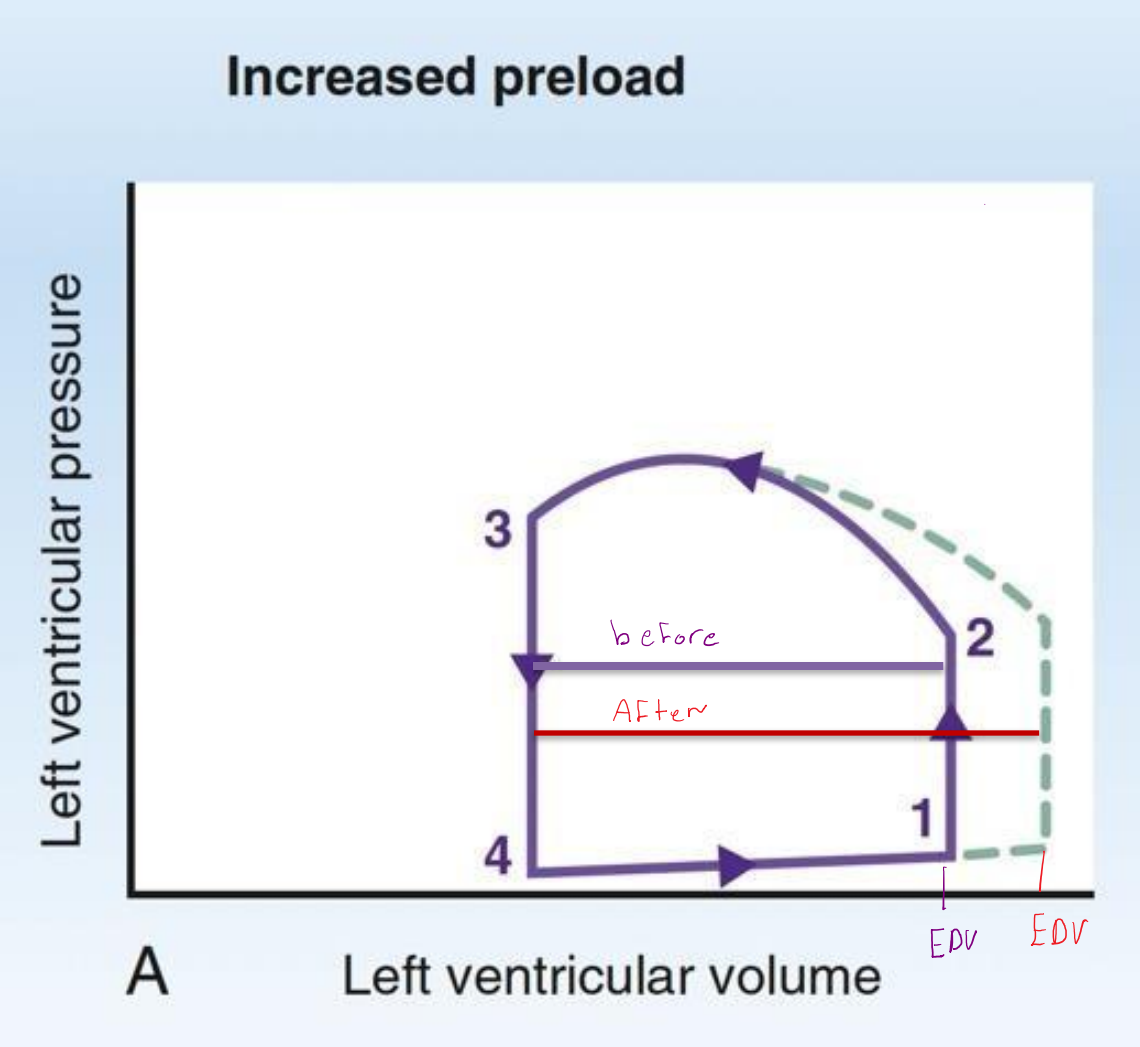
Recall



Additional



Changes in ventricular volume-pressure loop



increased venous return

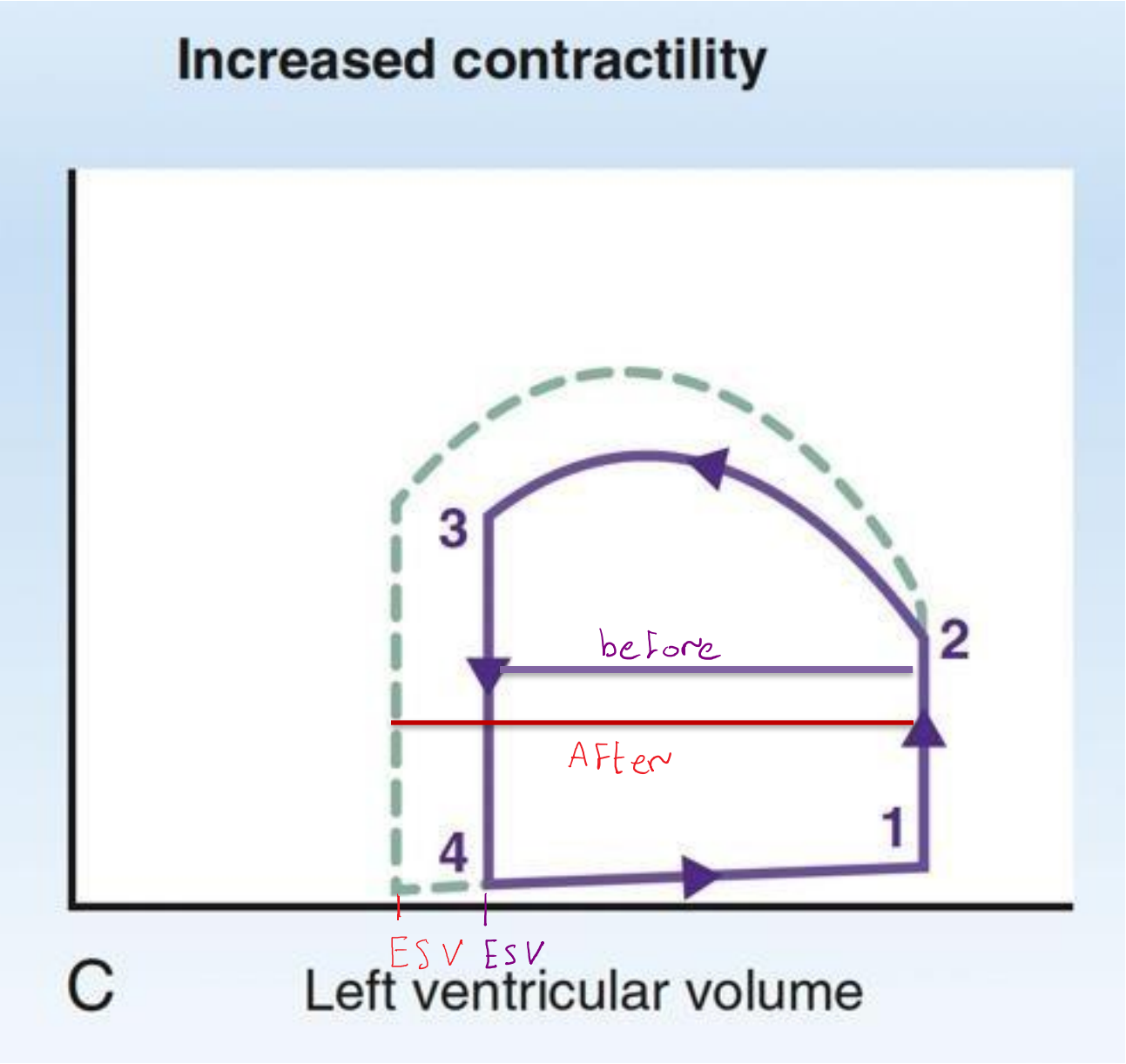
Due to Sympathetic stimulation →Venoconstriction → increasing venous returning

- Increased blood filling
- Leading to Increasing diastolic volume
- **Increased EDV**
- **Increases preload**
- **increased Stroke volume**

Frankin - Starling mechanism

Increasing preload → increasing Diastolic Volume→more stretching→ increased Stroke volume

- We didnt change the contractility nor afterload



Increasing the force of contraction

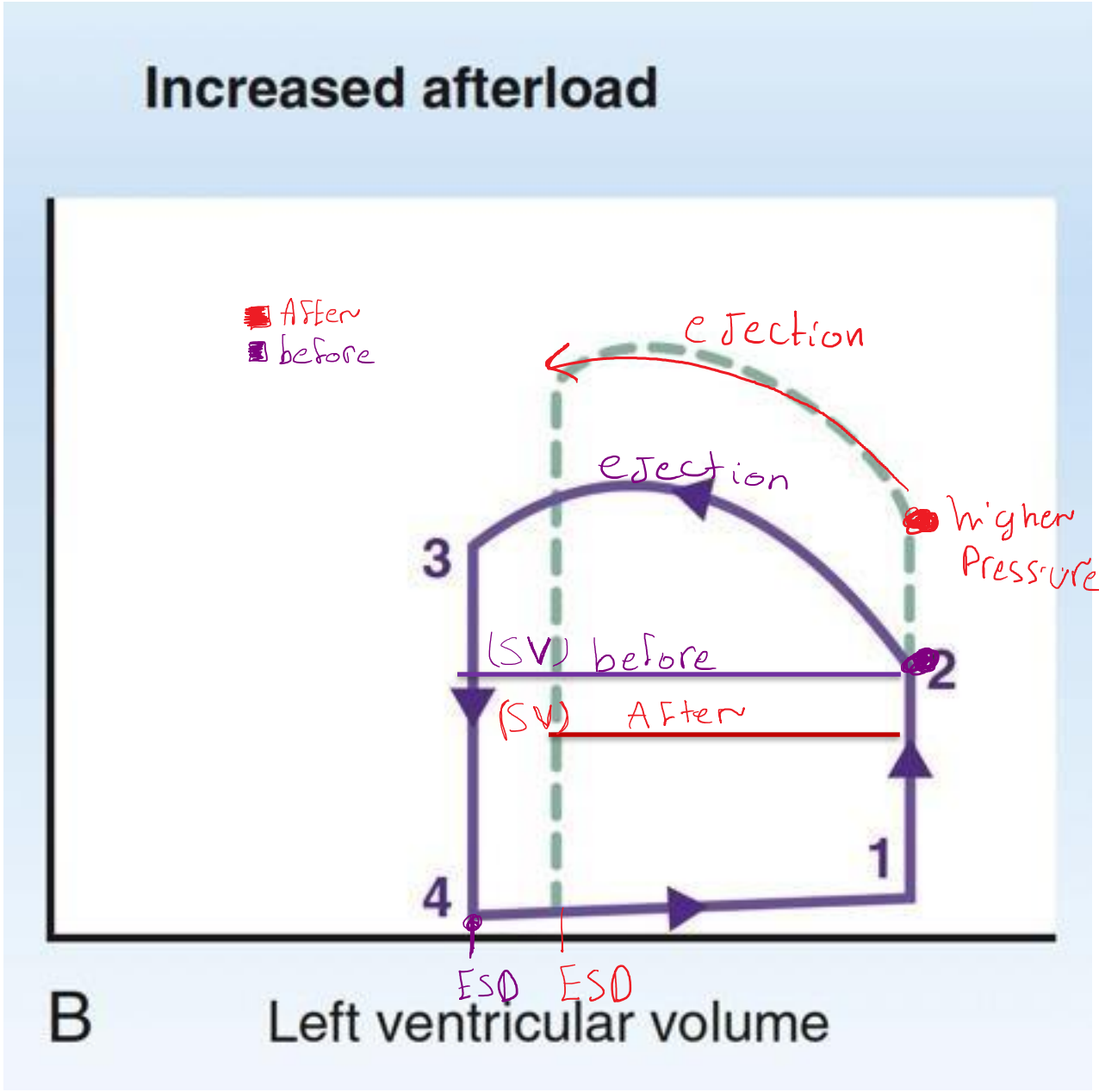
- More blood is ejected from the ventricle
- Less blood is remaining in the Ventricle, lesser than usual
- **Decreased ESV**
- **Increased Stroke volume**

- We didnt change the afterload nor the Preload

Stroke volume

- **Sympathetic stimulation** increases SV not only by [1]strengthening cardiac contractility (decreasing ESV)but also by [2]enhancing venous return.(increasing preload, increasing EDV)

- Sympathetic stimulation constricts the veins, which squeezes more blood forward from the veins to the heart, increasing the EDV and subsequently increasing SV even further.



Increases Afterload

Due to increased arterioler pressure (aortic pressure) or there is a problem with the aortic valve (aortic stenosis , narrowing)

- More resistance against the blood ejection
- **higher pressure** in Ventricle is needed to overcome the higher aortic resistance and eject blood
- So the ventricle is working harder , **taking longer time** to overcome the aortic pressure to eject blood
- The ejection phase is **shorter** than the previous one (purple) , because there is a certain time between depolarisation and repolarisation, **more time is consumed during increasing the pressure to overcome the resistance from the aortic pressure**
- **Increased in ESV , the ventricle cannot be emptied as before**
- **decreased Stroke volume**

- No change in Preload Same EDV

Afterload

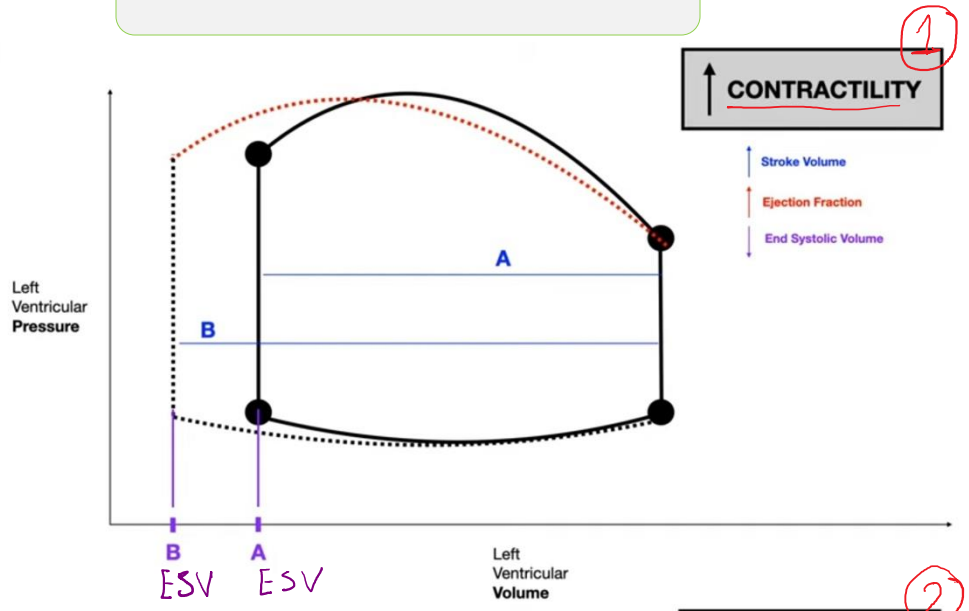
- If arterial blood pressure is chronically elevated (high blood pressure) or if the exit valve is stenotic, the ventricle must generate more pressure to eject blood.

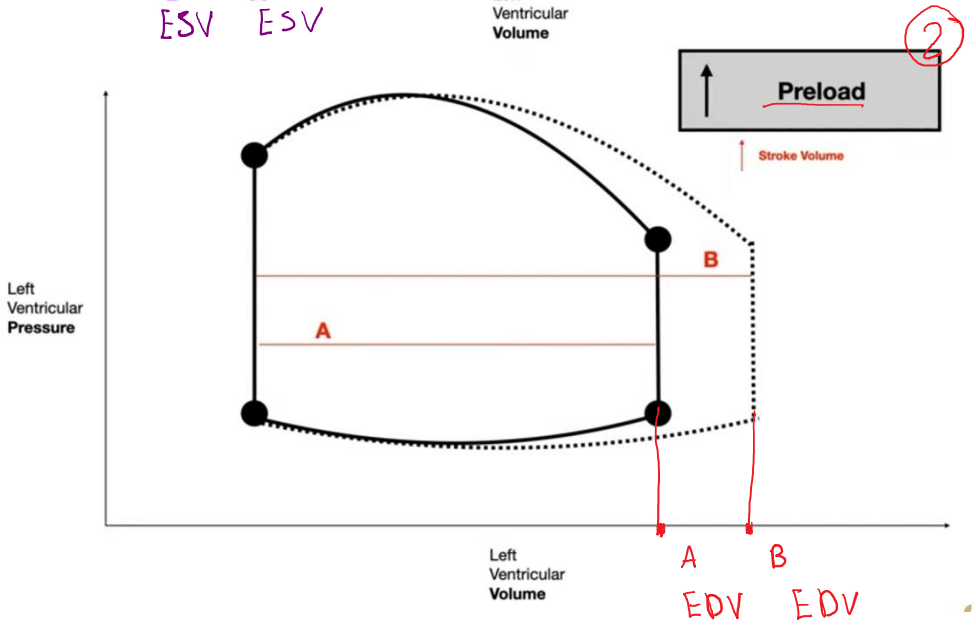
- The heart may be able to compensate for a sustained increase in afterload by hypertrophying, that is, by increasing the thickness of the cardiac muscle fibers.

- This enables it to contract more forcefully and maintain a normal SV despite an abnormal impediment to ejection.
- However, a diseased heart or a heart weakened with age may not be able to compensate completely; in that case, heart failure ensues.

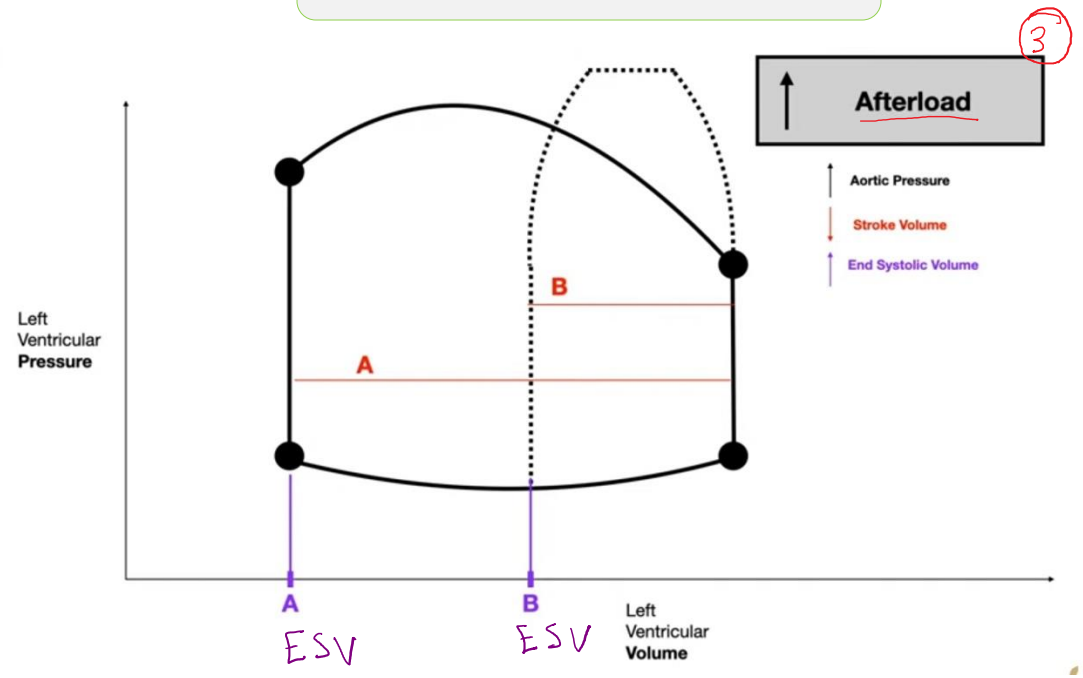
Summary
Factors that affect stroke volume

Increase stroke volume





Decrease stroke volume



A: before the change
B: after the change

Cardiac output

- **Cardiac output (CO)** is the volume of blood pumped by **each** ventricle **per minute** (by one of the ventricles)

(**not** the total amount of blood pumped by the heart)(by the two pumps,ventricles which means you should add the both ejected volumes from the left and right ventricles together)

- During any period, the volume of blood flowing through the pulmonary circulation is the same as the volume flowing through the systemic circulation.

- This is the quantity of blood that flows through the circulation.

Cardiac output

- **The two determinants of cardiac output are:**

1. heart rate (HR) (beats per minute)
2. stroke volume (SV) (volume of blood pumped **per beat or stroke).**

- **CO = HR x SV**

Clinical importance of calculating **cardiac output** is to know how much blood reached tissues **(tissue perfusion)**

- The difference between the cardiac output at rest and the maximum volume of blood the heart can pump per minute is called the cardiac reserve.

Modulation of cardia output by modulating the heart rate and the stroke volume

Increasing CO by

1. **Increasing HR**
- Sympathomimetic agents
- Positive chronotropic agents: increase the heart rate

2. **Increasing Stroke volume**
- increasing the preload

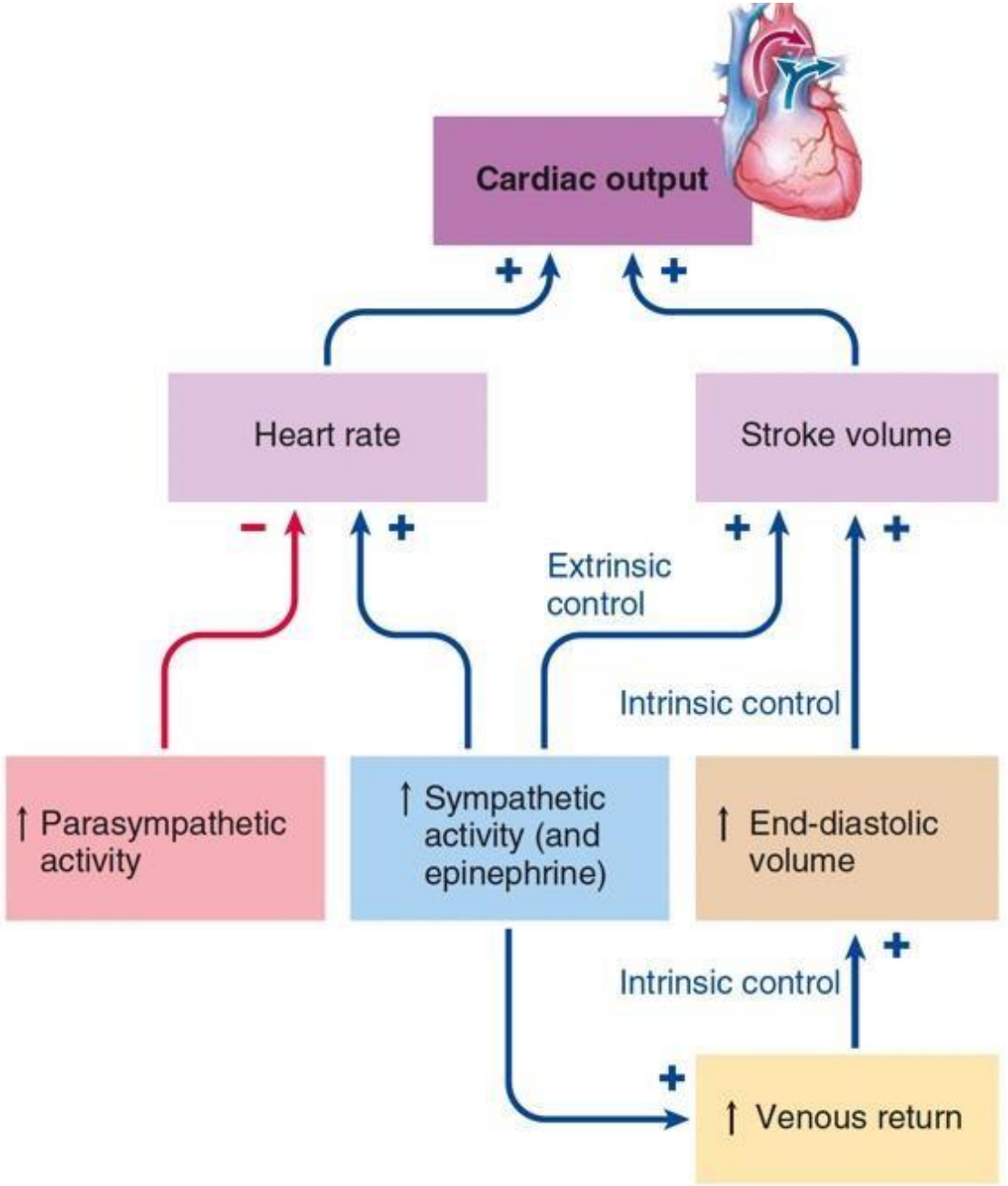
(Sympathomimetic agents positive inotropic agents →venoconstriction →venous returning)
- Increasing the contractility

Sympathomimetic agents→ positive inotropic

Note:

Chronotropic is related to the heart rate

Inotropic is related to the contractility of heart



Cardiac output

- The following factors, among others from person to another, directly affect cardiac output:

- (1) the basic level of body **metabolism**; Young adult and children have higher metabolic rate need more blood supply more cardiac output
- (2) whether the person is **exercising**; Activity of the person
- (3) the person's **age**;
- (4) the **size** of the body. Large size means more tissues need more blood supply

Cardiac index

هون نحنا منثبت عامل ال size

- cardiac output increases approximately in proportion to the surface area of the body.

- Therefore, cardiac output can be stated as **cardiac index,** which is **the cardiac output per square meter** of body surface area.

- declining cardiac index is indicative of declining activity and/or declining muscle mass with age.

CLINICAL APPLICATIONS

- Of **mechanical** function of the heart using **echocardiography** which calculates :HR, SV,ESV,EDV,Ejection fraction
- In previous lectures we discussed the **electrical** function of the heart and its diagnostic tool wich is **ECG**

Heart failure

- Heart failure (HF) is the inability of CO to keep pace with the body's demands for supplies and removal of wastes.
- Heart failing doing its function which is pumping blood, no enough blood supply to tissues

- Heart failure can be of **two types**:

1. systolic HF, in which the heart has difficulty pumping blood out
2. diastolic HF, in which the heart has trouble filling.

Ejection fraction

- The ejection fraction is the ratio of stroke volume to end-diastolic volume **(ejection fraction = SV/EDV)**; that is, it is the proportion of the blood in the ventricle that is pumped out.

- The ejection fraction is often used clinically as an indication of contractility.

- A healthy heart **normally** has an ejection fraction of **50% to 75% under resting** conditions and
- may go as **high** as 90% during **strenuous exercise**,
- but a **failing heart may pump out 30% or less** **mainly the systolic heart failure.**

The heart is unable to pump enough volume blood to accommodate the demand of tissues

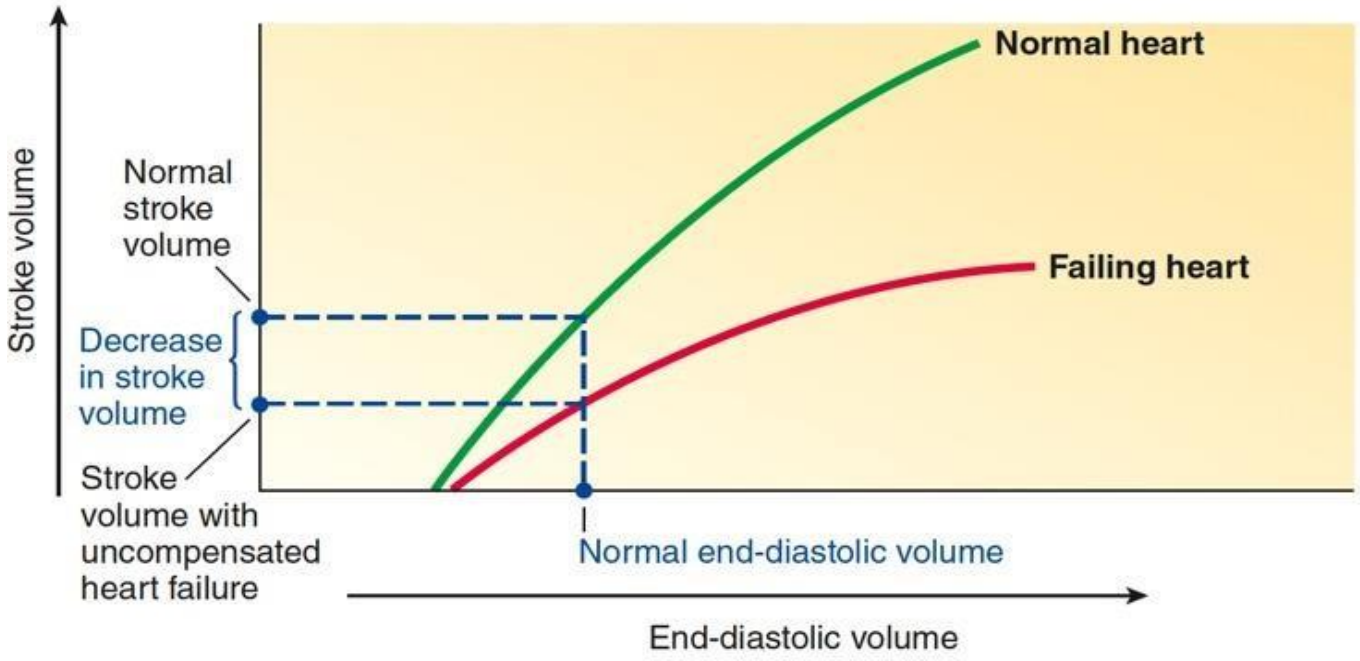
Systolic Heart failure

Causes of systolic heart Failure
- **ischemic** pathologies of myocardiocytes • **aging** decreasing in efficiency of heart pump

- The prime defect in systolic HF is decreased cardiac contractility —that is, weakened cardiac muscle cells contract less effectively, resulting in a **greatly reduced ejection fraction.**

- With systolic HF, the intrinsic ability of the heart to develop pressure and eject a **SV is reduced** so that the heart operates on a lower length–tension curve.

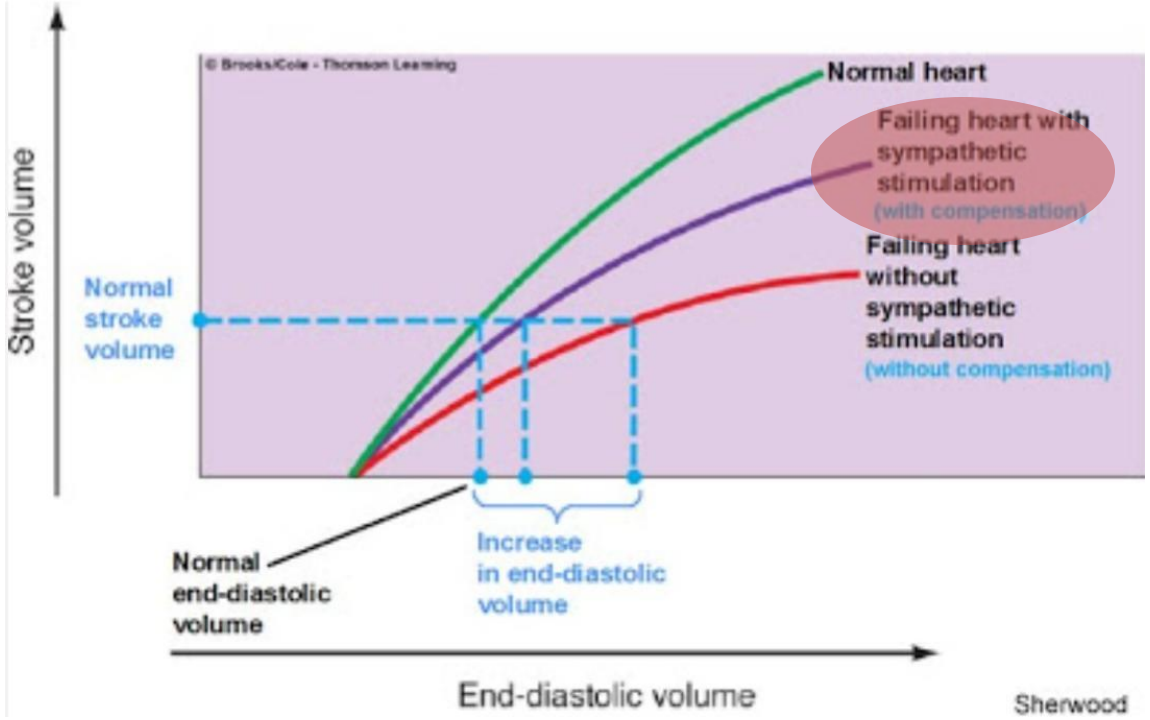
- The Frank–Starling curve shifts downward and to the right such that, for a given EDV, a failing heart pumps out a smaller SV than a normal healthy heart does.



Heart has Normal End Diastolic Volume but pumping less Stroke volume ,
The failing heart curve shift to the right downward

because the decrease of ability of myocardiocytes to contract , leading to decrease in tissue blood supply,which is bad thing so the heart try to compensate

what could **increase the SV** ???? Increasing the preload (venoconstriction), Contractility → sympathetic nervous system



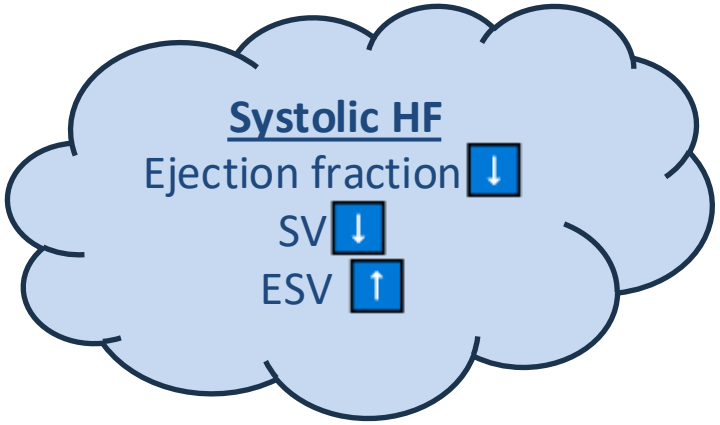
The sympathic stimulation curve is midway between the normal heart curve and the failing heart curve
Sympathetics stimulation tries to compensate for heart failure to address the same stroke volume I need to increase the end diastolic volume however in feeling hot we need much more increase in end diastolic volume

The problem is theat nervous compensation is limited check the next slide →

- Hormonal , Decreasing blood supply to the **kidneys** → expansion blood volume by increasing Na+ and water retention , decreasing urine output → increasing venous return → increasing EDV → increasing the stroke volume

- Limited, the cardio myocytes are pathological can't not contract well, cannot compensate check slide 23

While systolic HF(decreased ejection fraction), the sympathetic system and kidney try to help/compensate enough blood volume by increasing the preload(venous return) or contractility of the ventricle.Until reaches the decompensated systolic heart failure because the myocardial cells become almost nonfunctional/non-responsive with stretching in other words on the force of contraction anymore.



At result, the blood will 1)accumulate in ventricle = ESV ↑
2)Then it will backward to veins, as we know in left ventricle it will backward to polmunary veins to lungs 3) increase extracellular fluid = polmunary edema that effects on gase exchange (less oxygen and more CO2 in our body = acidosis) or 4) congestion in venous system of lower limb = lower limb swelling.

Common clinical symptoms:

- Fatigue
- Shortness of breath
- Edema/swelling of lungs or lower limbs.
- Blood acidosis.

The next 7 slides are repeated explain with little more details.

Compensated Systolic Heart failure

- **In the early stages of systolic HF, two major compensatory measures help restore SV to normal.** **The heart tries to compensate**
- **First**, sympathetic activity to the heart is increased, which increases heart contractility toward normal.
- Sympathetic stimulation can help compensate only for a limited time, however, because [1] the heart becomes less responsive to norepinephrine after prolonged exposure, and furthermore, [2] norepinephrine stores in the heart's sympathetic nerve terminals become depleted.

Compensated Systolic Heart failure

- **Second**, when CO is reduced, the kidneys, in a compensatory attempt to improve their reduced blood flow, retain extra salt and water in the body during urine formation to expand the blood volume.

- The increase in circulating blood volume increases the EDV. The resultant stretching of the cardiac muscle fibers enables the weakened heart to pump out a normal SV. The heart is now pumping out the blood returned to it but is operating at a greater cardiac muscle fiber length.

Decompensated Systolic Heart failure

- As the disease progresses and heart contractility deteriorates further, the heart reaches a point at which it can no longer pump out a normal SV despite compensatory measures. At this point, the heart slips from compensated HF into a state of decompensated HF.

- Now the cardiac muscle fibers are stretched to the point that they are operating in the **descending limb of the length–tension curve.**

- Forward failure occurs as the heart fails to pump an adequate amount of blood forward to the tissues because the **SV becomes progressively smaller.**

Decompensated Systolic Heart failure

- Backward failure occurs simultaneously as the failing heart cannot pump out all of the blood returned to it (SV cannot keep pace with venous return) so that the returning blood continues to dam up in the venous system.

- **Congestion of blood** in the venous system behind a failing ventricle is the reason this condition is sometimes termed congestive heart failure.

- Left-sided failure has more serious consequences than right-sided failure.

Because the venous return will back to lungs through 4 polmunary veins.

Decompensated Systolic Heart failure

- Backward failure of the left side leads to pulmonary edema (excess tissue fluid in the lungs) because blood dams up in the lungs. This fluid accumulation in the lungs reduces exchange of O2 and CO2, reducing arterial oxygenation and elevating levels of acid-forming CO2 in the blood.

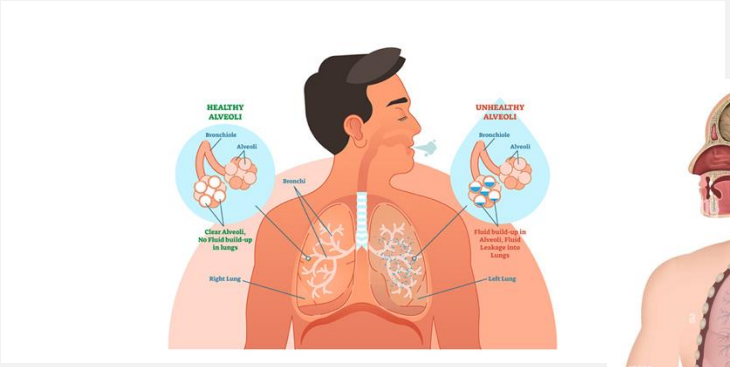
- In addition, one of the more serious consequences of left-sided forward failure is inadequate blood flow to the kidneys, which causes a twofold problem.

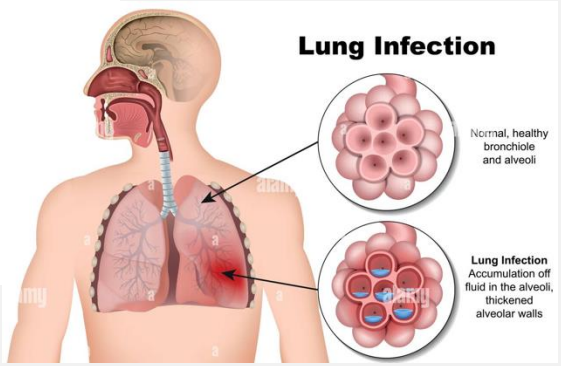
- First, vital kidney function is depressed;

Decompensated Systolic Heart failure

- Second, the kidneys retain even more salt and water in the body during urine formation as they try to expand the blood volume even further to improve their reduced blood flow.
- Excessive fluid retention worsens the already existing problems of venous congestion. احتقان/ركود وريدي

Extra images:





- Treatment of congestive heart failure therefore includes measures that reduce salt and water retention and increase urinary output and drugs that
enhance the contractile ability of the weakened heart—digitalis, for example.
- Digitalis increases cardiac contractility by causing accumulation of cytosolic Ca.

The medical intervention should be before decompensate heart failure stage, but if the patient reach this stage what we can dotdecrease symptoms:

- Diuretics)↓ volume(
- Positive inotropic agent (↑contractility of the heart)

Diastolic Heart failure

Diastolic HF
Ejection fraction(normal)
SV ↓
EDV ↓

HF with Preserved ejection fraction)another name diastolic HF(

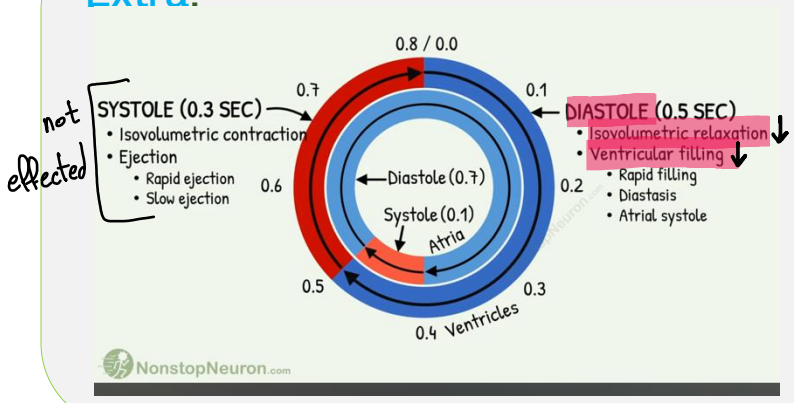
The contractility is normal so, the ECG and ejection fraction appear normal ,but the SV, CO will decreased result of abnormal filling phase

البطاقة التعريفية للمرض:

- **The problem is** dexaler nu ,ssenffirts : .sllec laidracoym
- **The pathophysiology/cause** nwonknU :
- **The treatment** citamotpmys ,egnellahc yrev: .ylno tnemtaert

I can not treat the cause
I don't know the cause!

Extra:



Diastolic Heart failure

- With the more recently recognized diastolic HF, the ventricles do not fill normally either because the heart muscle does not adequately relax between beats or because it stiffens and cannot expand as much as usual.

- The heart can pump properly and the ejection fraction is normal. For this reason, diastolic HF is alternatively called heart failure with preserved ejection fraction.

- However, even though the ejection fraction is normal (that is, the ventricles pump out a normal percentage of the blood present in their chambers), less blood than normal is pumped out by a diastolic failing heart because the ventricles are inadequately filled with blood.

Diastolic Heart failure

- Both forward and backward failure, including congestive heart failure, can result, so symptoms are similar to systolic HF.

- No drugs are available yet that reliably help the heart relax, so treatment of diastolic HF is aimed at relieving symptoms, halting underlying causes, or lessening aggravating factors, such as by controlling high blood pressure.

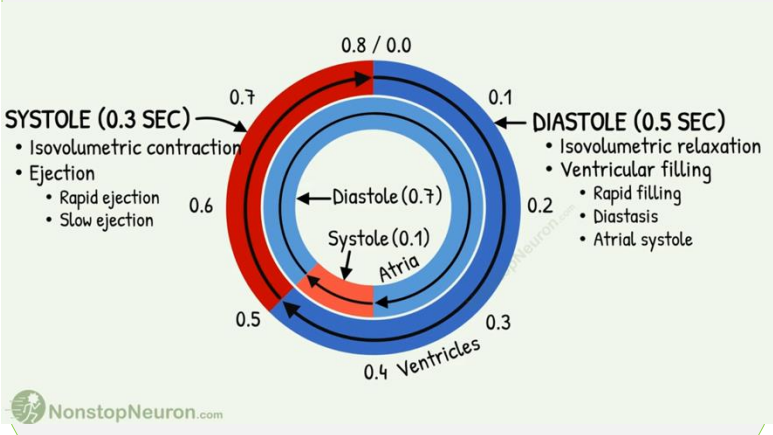
Heart sounds

You have to differentiate between these terms before talking about pathological valve conditions:

S1 AV valves closure, happened at the beginning of ventriclar systole
S2 Semilunar valves closure (aortic and pulmonary), happened at the end of ventricular systole

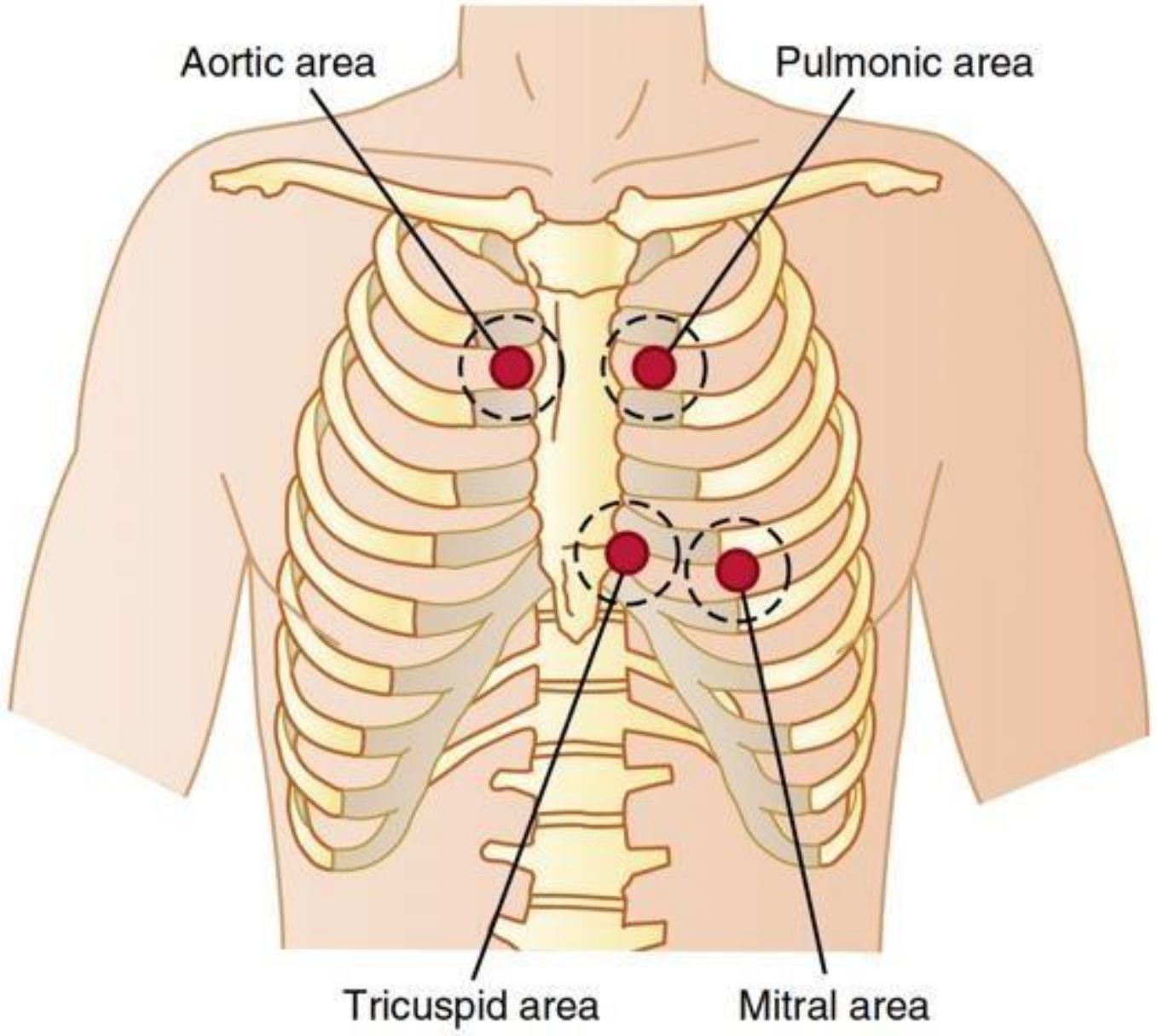
S1 to Sesahp noitcejE + esahp noitcartnoc cimelovosi= esahp cilotsys=2
S2to Sesahp gnillif + esahp noitaxaler cimelovosi = esahp cilotsaiD=1

Extra image:



Heart sounds

- when the valves close, the vanes of the valves and the surrounding fluids vibrate under the influence of sudden pressure changes, giving off sound that travels in all directions through the chest.
- When the ventricles contract, one first hears a sound caused by closure of the A-V valves.
- The vibration pitch is low and relatively long-lasting and is known as the first heart sound (S1).
- When the aortic and pulmonary valves close at the end of systole, one hears a rapid snap because these valves close rapidly, and the surroundings vibrate for a short period.
- This sound is called the second heart sound (S2).



Each point is not an indication of the anatomical site of the valve, it is the most appropriate site of higher vibration sound.

We have to know each point related to any valve for a specific diagnosis.

The pathologies of valves are:

1) **Valve stenosis=** the valve can not open completely /narrowing of opening .

2) **Valve incompetancy/regurgenation/insufficiency**= the valve can not close completely = cospids تلولح كما قال الدكتور مالك

Murmur is abnormal heart sound = turbulance of blood flow

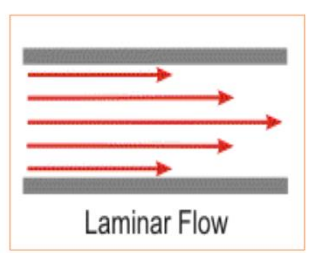
This murmer can happen during

1) Systole S1toS2 and the problem can be in:
- Aortic valve: stenosis
- Mitral valve: incompetency/regurgenation

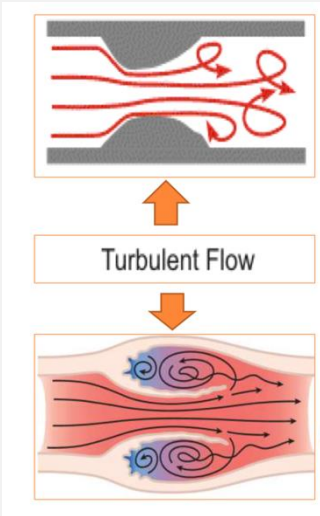
1) Diastole S2toS1 and the problem can be in:
- Aortic valve: incompetency
- Mitral valve: stenosis

In the first pathology lecture the doctor explained turbulence and this image from it:

- Normal blood flow



- Turbulence blood flow:



There are other sound we can notice them are explained in the last slides.
- Check the next slides pls ;-;

Valve stenosis

- Abnormal heart sounds, or murmurs, are usually (but not always) associated with cardiac disease. Result from turbulent blood flow rather than laminar flow.

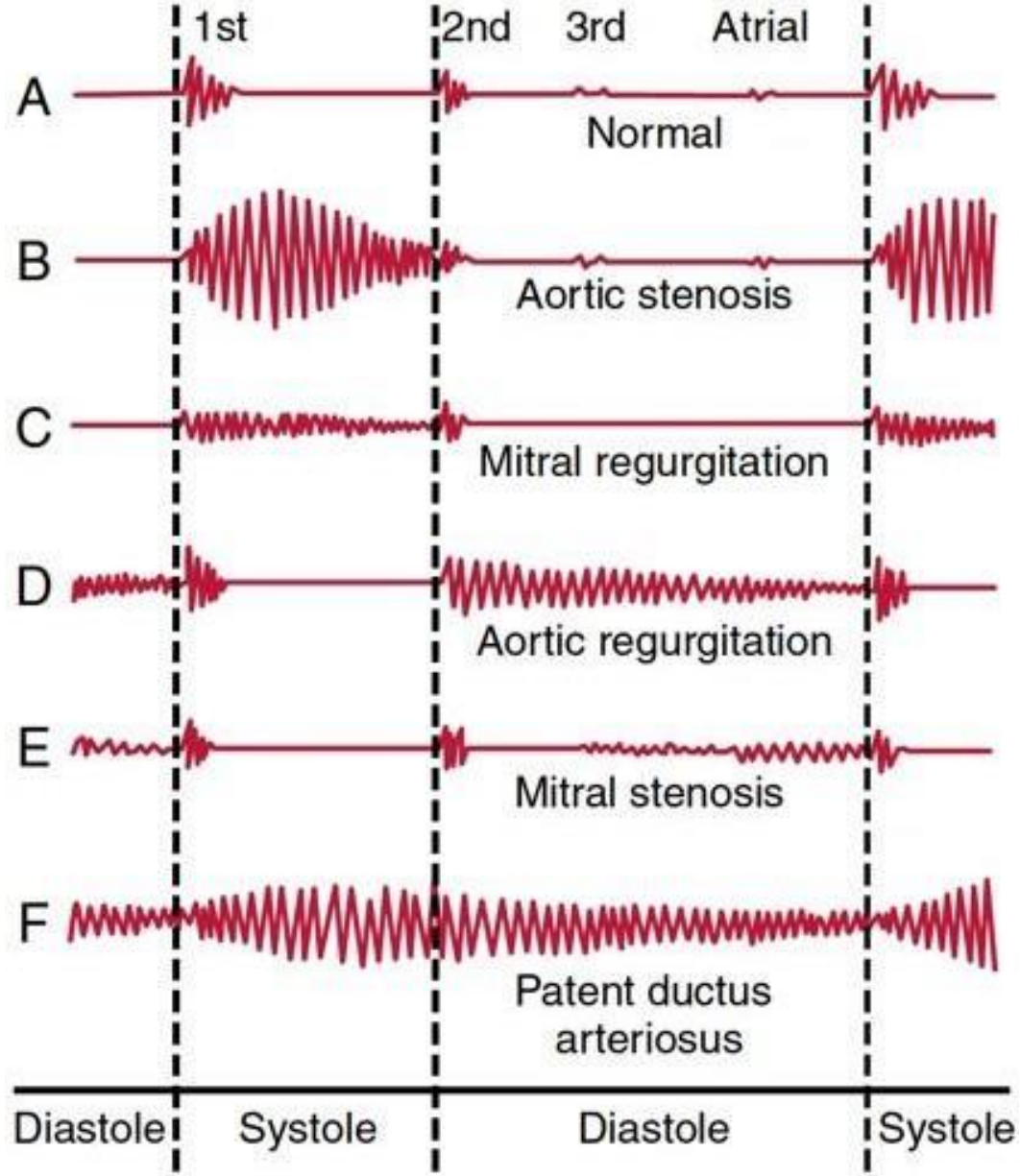
- A stenotic valve is a stiff, narrowed valve that does not open completely.
- Blood must be forced through the constricted opening at tremendous velocity, resulting in turbulence that produces an abnormal whistling sound.

Valve incompetency

- An insufficient, or incompetent, valve is one that cannot close completely because the valve edges do not fit together properly.
- Turbulence is produced when blood flows backward through the insufficient valve and collides with blood moving in the opposite direction, creating a swishing or gurgling murmur.
- Such backflow of blood is known as regurgitation. An insufficient heart valve is often called a leaky valve because it lets blood leak back through when the valve should be closed.
- rheumatic fever

Murmurs

- The valve involved and the type of defect can usually be detected by the timing and location of the murmur.
- The timing of the murmur refers to the part of the cardiac cycle during which the murmur is heard.
- Recall that the first heart sound signals the onset of ventricular systole and the second heart sound signals the onset of ventricular diastole.
- Thus, a murmur between the first and the second heart sounds is a systolic murmur.
- A diastolic murmur occurs between the second and the first heart sounds.
- The sound of the murmur characterizes it as either a stenotic murmur or an insufficient murmur.


1st
2nd
3rd
Atrial
A
Normal
B
Aortic stenosis
C
Mitral regurgitation
D
Aortic regurgitation
E
Mitral stenosis
F
Patent ductus arteriosus
Diastole
Systole
Diastole
Systole

S3

- The rapid flow of blood from the atria to the ventricles produces the third heart sound (S3).

- normal in children.
- but in adults indicates volume overload, as in congestive heart failure or advanced mitral or tricuspid regurgitation.

S4

- The fourth heart sound (S4) is not audible in normal adults, although it may be heard in ventricular hypertrophy, where ventricular compliance is decreased.

- When present, S4 coincides with atrial contraction. The sound is caused by the atrium contracting against, and trying to fill, a stiffened ventricle.

Spilt

- Closure of the AV valves produces the first heart sound (S1), which may be split because the mitral valve closes slightly before the tricuspid valve.
- The aortic valve closes slightly before the pulmonic valve, producing the second heart sound **(S2)**.
- Inspiration delays closure of the pulmonic valve and causes splitting of the second heart sound; because the associated decrease in intrathoracic pressure produces an increase in venous return to the right side of the heart.
- The resulting increase in right ventricular end-diastolic volume causes an increase in right ventricular stroke volume, and prolongs right ventricular ejection time; so delays closure of the pulmonic valve relative to the aortic valve.

Additional sources

Pressure volume loop

https://youtu.be/-0CXs9L9O0A?si=5OV427NLwWDsgF-K

Cardiac output

https://youtu.be/WuGMqezV3eo?si=NE4jurwxIWhqMT0y

Frank starling mechanism

https://youtu.be/UGK7Ni8yQSQ?si=NXf2mHwE7967Jc5M

عن ابن عمر رضي الله عنهما: أنّ النبيّ صلى الله عليه وسلم قلّما كان يقوم من مجلسٍ حتّى يدْعو بهؤلاء الدّعوات لأصحابه:

"اللّهُمَّ اقْسِمْ لَنَا مِنْ خَشْيَتِكَ مَا تَحُولُ بِهِ بَيْنَنَا وَبَيْنَ مَعْصِيَتِكَ . . . واجْعَلْ ثَأْرَنَا عَلَىٰ مَنْ ظَلَمَنَا، وَانصُرْنَا عَلَىٰ مَنْ عَادَانَا"

.(رواه الترمذي).

تذكّر أن الله لا يكلف نفسًا إلا وسعها فلا تيأس مهما اشتدت المحن فبعد كل عسر يسر, فاجعل الثقة بالله نبراس حياتك

واعلم ان من يتوكل على الله فهو حسبه

VERSIONS	SLIDE #	BEFORE CORRECTION	AFTER CORRECTION
V1→ V2			
V2→V3			

امسح الرمز و شاركنا بأفكارك لتحسين أدائنا!!