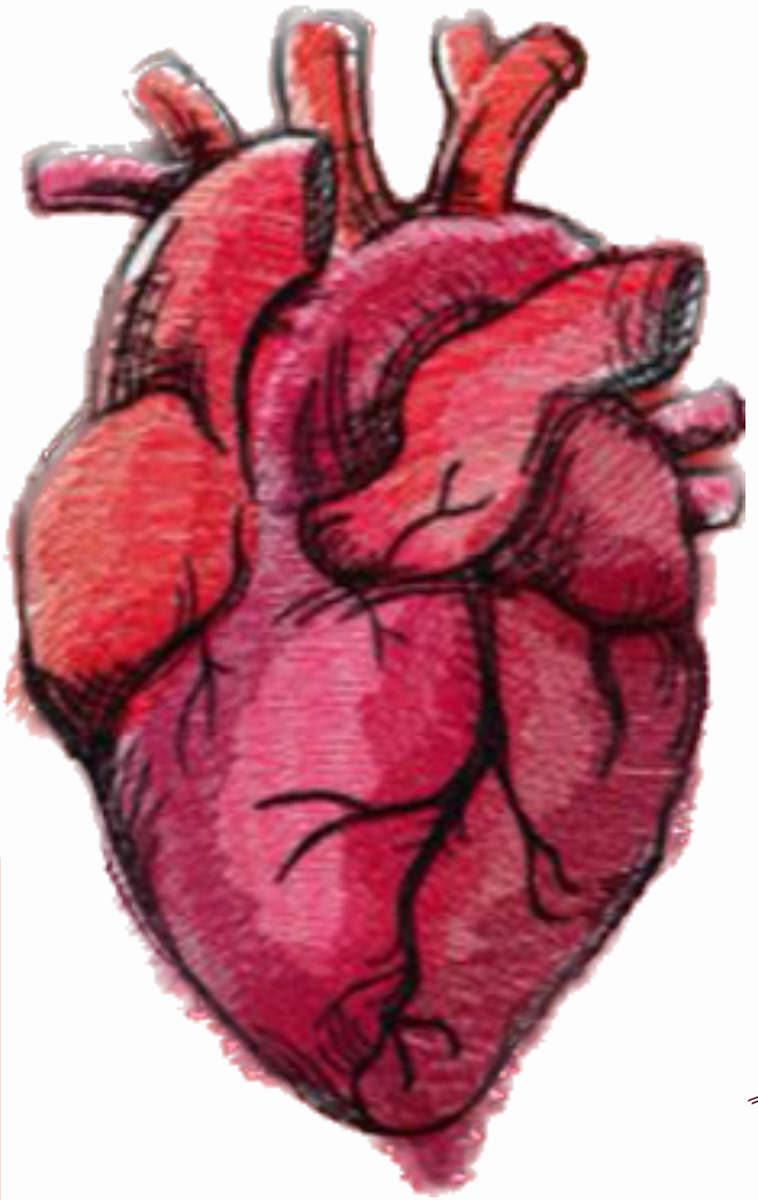


النادي الطبي

CARDIOVASCULAR SYSTEM

SUBJECT : The Cardiac Cycle

LEC NO. : 7

DONE BY : Malek Hassan

وَقُل رَّبِّ زِدْنِي عِلْمًا

SCAN ME!

The Cardiac Cycle

Dr. Waleed R. Ezzat

Lecture Objectives:

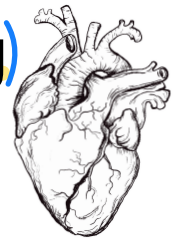
1. Define cardiac cycle.
2. Be familiar with and explain curves for the various events occurring in the heart, inlet veins, and outlet arteries.
3. Recognize systolic and diastolic duration.
4. Understand isometric and relaxation; and the ejection phases.
5. Explain volume – pressure relationship in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

Today we will start talking about the mechanical properties of the heart

Definition:

Understanding the cardiac cycle involves grasping the series of changes occurring in the heart, including variations in pressure, volume, and the sounds produced. Crucially, linking electrical activity to mechanical activity hinges on comprehending the timing and genesis of each cardiac event. The heart operates through a rhythmic succession of beats akin to a repetitive muscle twitch, where contraction alternates with relaxation due to the absence of tetanization in the heart muscle.

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*

The relaxation period of the heart allow blood to go inside thus filling it until a pump (contraction) happens

- During diastole → heart relaxes and fills with blood

Better filling = Better contraction

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

Systole is the period where contraction happens leading to pumping blood different parts of the body

That mean in every 0.8 second we have a heart beat occur

$$\frac{60\ \text{sec}}{\text{Heart Rate (HR)}} = \text{One Cardiac Cycle}$$

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.

Note that diastolic longer than systolic period this indicates that when a person is at rest & the heart is lowest the diastole is longer than systole

So, is the duration of the cardiac cycle fixed?

In reality, it's not; it actually varies depending on the heart rate. For instance, if the heart beats at a rate of 72 beats per minute, using a simple mathematical equation, we can calculate the time taken for a single heartbeat, which in this case is 0.8 seconds. By accounting for this 0.8-second interval, we can discern the durations of diastole and systole. In this scenario, diastole occupies 62.5% of the 0.8-second period, which equates to 0.5 seconds, while systole comprises 37.5% of the 0.8-second period, amounting to 0.3 seconds.

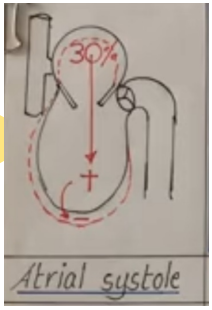
But the real question are the diastolic and systolic periods also fixed?

Again NO, the more the heart rate The shorter the diastole period, at the expense of the decrease in systole meaning both are going to decrease but the percentage will NOT be similar to the previous example So, the more the Heart Rate the bigger the cut will be between the diastolic periods so if we increase the heart rate a lot the period of systole will be equal to that of diastole

الشرح بالفيديوات اللي تحت

Atrial contraction (= Primer Pump)

اللي بالاحمر المراحل

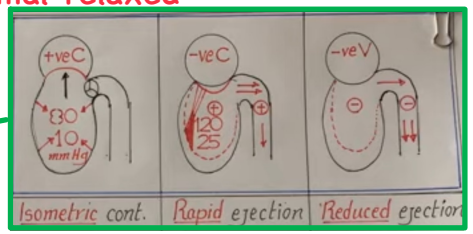


Normally 80% of the ventricular filling is achieved even before the onset of atrial contract.

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)

Very important clinical feature of The atria

This is mainly because the rule of atria is secondary so in normal relaxed position the atria is not, under the stares if in during exercise



Systole is composed of:

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1. Isovolumic (isovolumetric) contraction phase. In this phase cardiac muscle tension is increasing but little or no shortening of the muscle fibers is occurring.

It mean the hight of the Cardiac muscle doesn't change during contraction

2. Ejection phase (70% of ejection is completed in the first ⅓ of the phase).

The Atrial Contraction

In the context of the cardiac cycle, both the atria and ventricles undergo periods of contraction (systole) and relaxation (diastole). While the ventricles are the primary drivers of blood circulation, the atria play a supportive yet significant role. Physiology texts often emphasize the ventricular function due to its primary role in pumping blood; however, the atrial contribution, though secondary, is crucial for efficient cardiac function.

Atrial and Ventricular Dynamics in the Cardiac Cycle

The cardiac cycle encompasses a series of events that occur from the beginning of one heartbeat to the start of the next. During this cycle, the heart chambers—atria and ventricles—experience alternating phases of contraction and relaxation, facilitating blood flow throughout the body.

Atrial Function: The atria contract slightly ahead of the ventricles, aiding in the latter's filling. Atrial contraction, or atrial systole, contributes to approximately 20% of the ventricular filling. This active phase of filling complements the passive filling that occurs during ventricular diastole, where about 80% of the ventricular volume is acquired without atrial contraction.

Ventricular Function: The ventricles, being the main pumps of the heart, undergo diastole and systole. During diastole, they fill with blood, primarily through passive flow. Subsequently, during systole, they contract to propel blood into the circulation.

Systole is composed of:

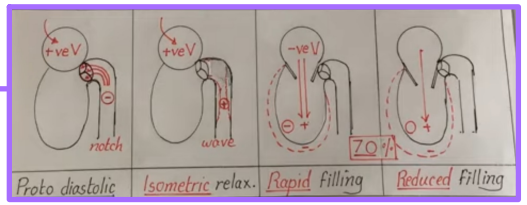
1. **Isovolumic contraction phase :** In the heart, a similar event occurs of that in skeletal muscle during the initial phase of systole known as isovolumetric contraction. During this phase, the ventricles contract without changing volume because all heart valves are closed, which increases the pressure inside the ventricles but does not change their size. This is different from skeletal muscle isometric contraction because it involves pressure changes rather than just force generation

In skeletal muscles, isometric contractions result in increased power output without a change in muscle length, maintaining the same "height" or length of the muscle. In cardiac physiology, the equivalent phase is the isovolumetric contraction, occurring at the onset of systole. During this phase, the ventricles contract, increasing pressure without changing volume, as all heart valves are closed, ensuring no blood is ejected during this period. This phase is crucial for building the pressure necessary to open the semilunar valves for the subsequent ejection of blood.

2. Ejection Phase:

During the ejection phase, the ventricles contract, propelling blood into the circulation. The ejection fraction, which measures this phase, typically ranges from 55% to 70% for a healthy heart in the first third of this phase. This means that with each heartbeat, a normal heart will pump out 55-70% of the blood contained in the left ventricle.

Diastole is composed of:

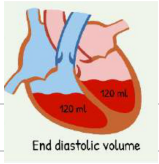


1. Isovolumic (isovolumetric) relaxation phase
2. Ventricular filling phase – this phase can be further divided into three thirds, namely the rapid filling, reduced filling (diastases), and the contribution of atrial contraction

The Cardiac Volumes

- 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***
- 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.
- Stroke volume output (70 ml) – it is the amount of blood pumped out of each ventricle with each contraction. SV= EDV – ESV
- The ejection fraction (EF) is the fraction of the EDV that is ejected. It is usually equal to about 60 percent.

Cardiac Volumes:



1. End-Diastolic Volume (EDV):

EDV is the volume of blood in the ventricles at the end of diastole, representing the maximum filling of the heart at that moment. Typically, the left ventricle fills with about 110-120 milliliters (ml) of blood before contraction. However, this number is NOT fixed and can reach up to 150-180 ml under certain conditions.

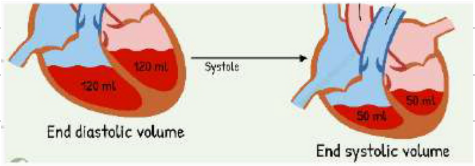
The myocardium, the heart muscle, possesses elasticity and stretchability superior to that of skeletal muscle. This allows the ventricles to stretch and accommodate varying volumes of blood. The more blood that returns to the heart, the more the ventricles will stretch, potentially increasing the EDV. Additionally, a higher heart rate can reduce the time available for ventricular filling (lowering the EDV), and reduce the preload due to decreased stretching of myocardial cells

Preload : how much the ventricle is stretched before the beginning of contraction or length of sarcoma

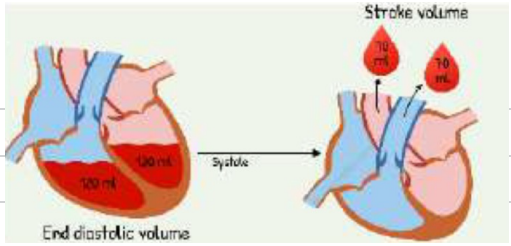
In a state of homeostasis, with a normal heart rate typically ranging from 60-70 beats per minute, the ventricles maintain a standard filling volume of 110-120 ml1. It's important to note that myocardial elasticity and heart rate are interrelated; changes in one can affect the other, impacting the heart's efficiency and function.

2. End Systolic Volume (ESV)

Is the remaining amount of blood in the ventricle after systole (contraction) so the harder the heart contract the less the amount of ESV indicating a stronger contractility of the heart (normal 40-50ml) and (minimal= 10-20ml)



Cardiac Volumes (Cont.):

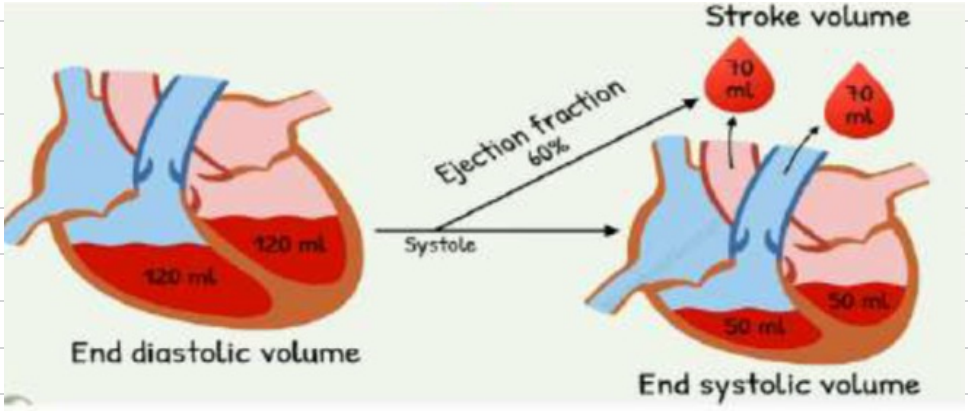


3. Stroke Volume (SV) :

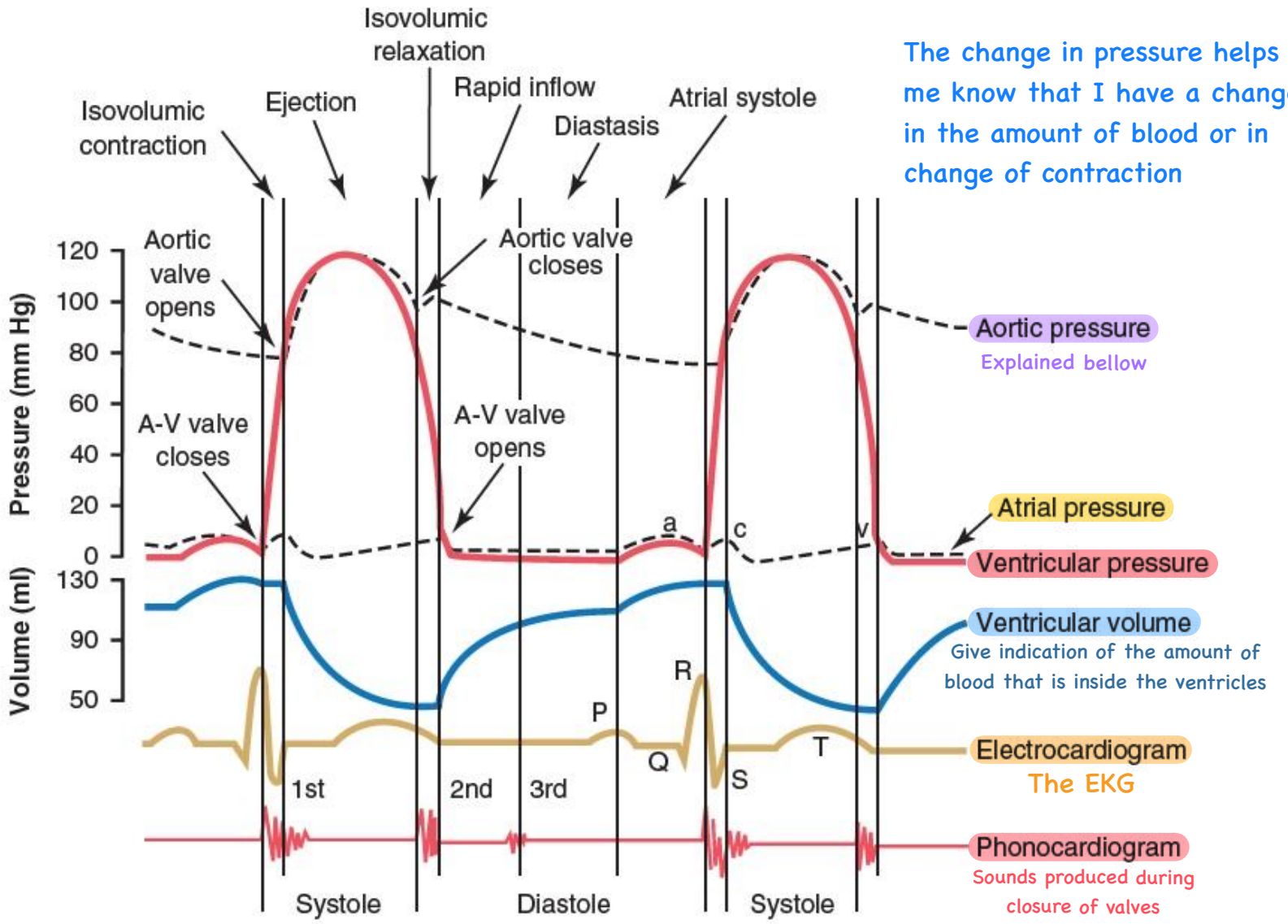
The amount of blood that will be pumped out of the ventricle in one contraction and is about 70ml in normal condition and could be calculated via : SV=EDV-ESV

4. Ejection Fraction (EF):

EF quantifies the proportion of blood the heart pumps out during each contraction. Specifically, it measures the volume of blood ejected from the left ventricle with each heartbeat as a percentage of the total End-Diastolic Volume (EDV). A normal EF typically is about %60, indicating efficient heart function and the ability to meet the body's circulatory demands. This parameter is crucial in diagnosing and managing various cardiac conditions, including heart failure.



This is a diagram that show the change in pressure inside the heart vs the change in time



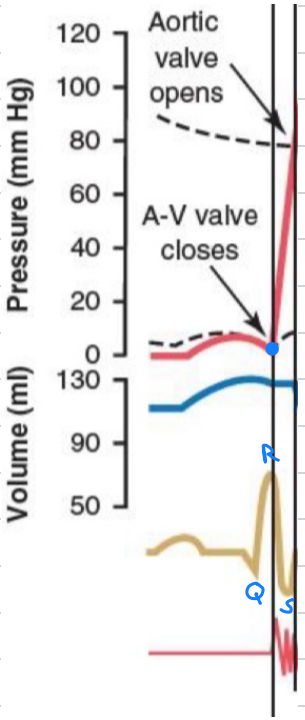
The change in pressure helps me know that I have a change in the amount of blood or in change of contraction

Cardiac Cycle - Systole

The Onset of Systole and the QRS Complex The cardiac cycle begins with systole, the phase where the heart Ventricle contracts (blue dot) to pump blood out. The QRS complex, a distinctive pattern on the electrocardiogram (ECG), marks the start of this phase. It represents the rapid electrical depolarization of the ventricles, which triggers their contraction.

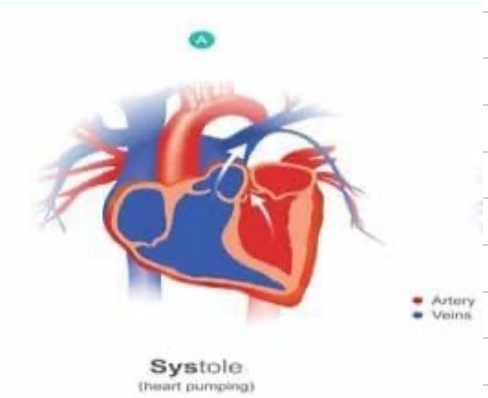
Electrical Conduction and Ventricular Contraction As the heart transitions into systole, a wave of electrical impulses, or action potentials, originates from the bundle of His. These impulses swiftly travel through the ventricular myocardium, the heart's muscular layer, via the Purkinje fibers. This network ensures the efficient spread of electrical activity, leading to a coordinated contraction of the ventricles.

Depolarization and Mechanical Contraction Depolarization is the process by which the heart's muscle cells become activated and ready to contract. Once the entire ventricular myocardium has depolarized, a mechanical contraction follows immediately.



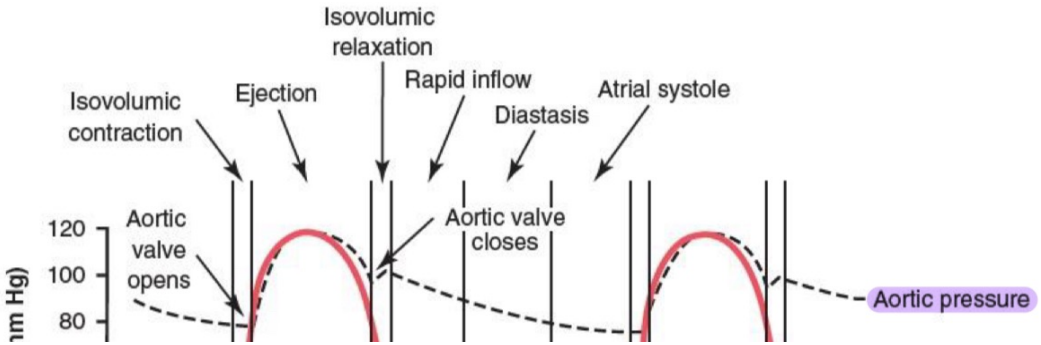
During the systolic phase of the cardiac cycle, the ventricles contract, causing the blood pressure within them to rise. This increased pressure could potentially push blood back into the atrium. However, the mitral valve closes firmly to prevent this backward flow. The closure of the mitral valve, along with the tricuspid valve, produces the first heart sound—often described as 'lub.' This sound signifies the beginning of systole.

Very important the first heart sound indicates beginning of Systole



Before we delve into the details, it's important to grasp what normal blood pressure means. The standard measure is 120/80 mm Hg. The '120' represents the systolic pressure, which is the force of blood in the arteries when the heart beats. It's the higher number because it's when the heart is exerting the most force to circulate blood. On the other hand, the '80' is the diastolic pressure, which is the force of blood when the heart is resting between beats — this is the lower number.

In our aorta, the body's main artery, there are special sensors called baroreceptors. These receptors are constantly monitoring the pressure of the blood flowing through. If there's any change from the normal pressure, these receptors send signals to the brain to take action to bring it back to normal.



So what affects blood pressure in general?

1. Amount of blood. (Directly related)
2. Contraction (Size of Cavity). (Inversely related)

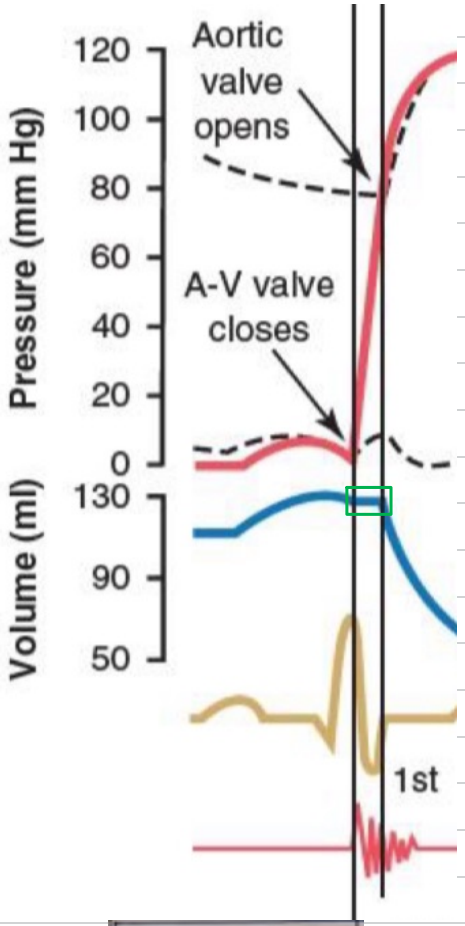
The Cardiac Cycle - Isovolumic Contraction Phase

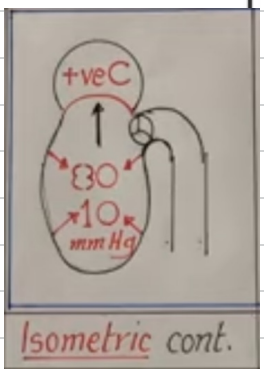
Understanding Isovolumic Contraction: During the cardiac cycle, there is a phase where the ventricles contract but the volume of blood within them remains unchanged. This phase is known as the isovolumic contraction phase. It occurs after the atria have filled the ventricles with blood and the heart is ready to pump it out to the body.

Ventricular Pressure Dynamics: As the ventricles begin to contract, the pressure inside them starts to rise. However, this increase in pressure does not immediately lead to the ejection of blood. Under normal conditions, the resting pressure in the ventricles is between 5 to 8 mmHg. During isovolumic contraction, the pressure increases significantly but the semilunar valves (aortic and pulmonary valves) remain closed until the pressure within the ventricles exceeds the pressure in the aorta and pulmonary artery.

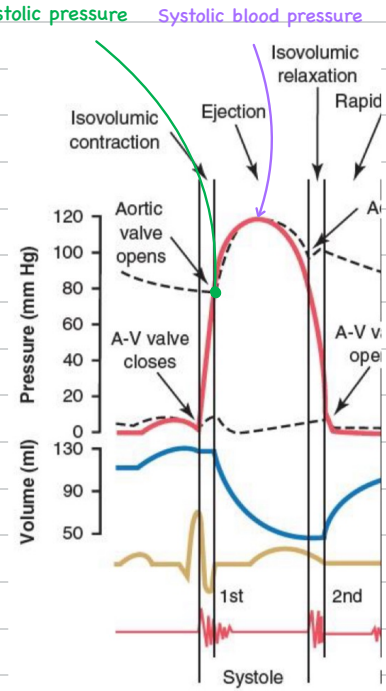
Role of Semilunar Valves: The semilunar valves, which include the aortic valve, serve a critical function by preventing the backflow of blood from the arteries into the ventricles during the heart's relaxation phase (diastole). The aortic pressure during diastole is approximately 80 mmHg. These valves remain closed during the isovolumic contraction phase, ensuring that the ventricular volume does not change despite the increasing pressure.

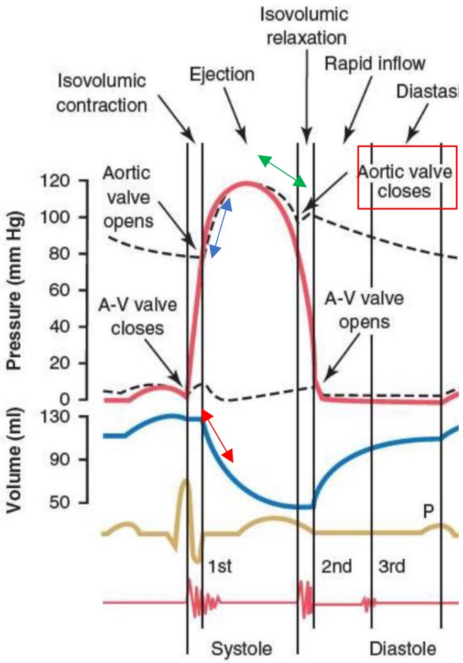
Isovolumic Contraction Explained: The term 'isovolumic' or 'isometric' refers to the constant volume within the ventricles during this phase. Despite the powerful contraction of the ventricular muscles, the volume of blood does not change because the semilunar valves are closed. This phase is crucial as it builds the pressure necessary to open the semilunar valves and propel blood into the aorta and pulmonary artery, marking the transition to the ejection phase of the cardiac cycle.





The opening of the aortic valve (green dot) during the cardiac cycle is indeed a silent event, not typically associated with an audible sound. It is the closure of the valves, specifically the mitral and tricuspid valves (S1) and the aortic and pulmonary valves (S2), that produces the characteristic "lub-dub" sounds of the heartbeat. During the ejection phase, after the isovolumetric contraction, the ventricles contract more forcefully, increasing the pressure until it exceeds that in the aorta. This rise in pressure causes the aortic valve to open, allowing blood to flow from the ventricles into the aorta. As the ventricles continue to contract, the pressure within them and the aorta increases, which can be visualized on a diagram as a rising line starting from the aorta's baseline diastolic pressure, typically around 80 mmHg.





In the ejection phase of the cardiac cycle, the heart begins to pump blood, propelling approximately two-thirds of the stroke volume—around 70%—in just the initial third of this phase (red & blue arrow). As the ventricles contract, they expel blood, leading to a reduction in their size (green arrow). This contraction continues until the ventricular pressure starts to fall below the aortic pressure. During this phase, the ventricular and aortic cavities function as a unified chamber, with pressures equalizing between them. This unity persists until the ventricular pressure diminishes, falling below the aorta's diastolic pressure. At this juncture, the aortic valve closes (red box). This closure is swift and results in a brief reversal of blood flow from the aorta back towards the ventricle, due to the pressure gradient. However, this retrograde flow is halted by the closed aortic valve, creating a small dip in the aortic pressure curve known as the dicrotic notch.

Furthermore, valve closures are associated with specific heart sounds. The closure of the aortic valve generates the second heart sound (S2), signaling the end of systole. Thus, the interval between the first heart sound (S1) and the second heart sound (S2) encompasses the systolic period of the cardiac cycle.

1-After the aortic valve closes, the pressure in the aorta indeed begins to decrease. This event is marked by the dicrotic notch on the pressure curve, which occurs due to a brief backflow of blood that closes the valve and causes a slight increase in aortic pressure before it continues to fall.

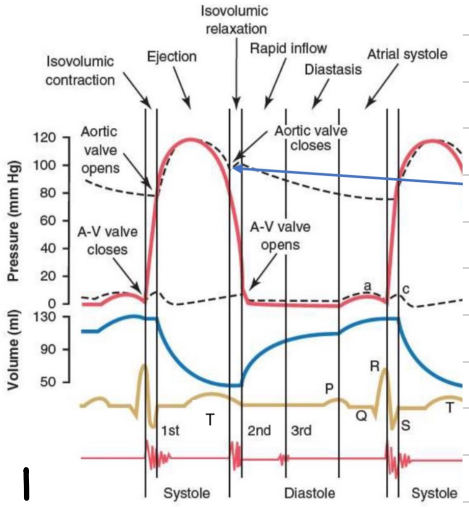
Diastole starts after the second heart sound (S2), which is associated with the closure of the aortic valve. The T wave on the ECG corresponds to ventricular repolarization, indicating the relaxation of the ventricles and the onset of diastole.

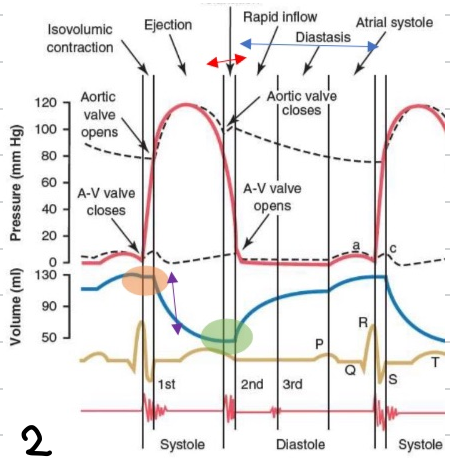
2-Diastole is composed of several phases (red and blue arrows), starting with the isovolumic relaxation phase. During this phase, the ventricles relax, the pressure inside them decreases, but the volume of blood remains unchanged because all the heart valves are closed. Notice the (orange highlight) indicating The end-diastolic volume (EDV) is the amount of blood in the ventricle at the end of diastole and is typically about 110-120 mL, (notice the green highlight) while the end-systolic volume (ESV) is the amount of blood remaining in the ventricle at the end of systole, usually around 40-50 mL. (notice purple arrow) The difference between EDV and ESV is the stroke volume, which is the amount of blood ejected from the ventricle during systole.

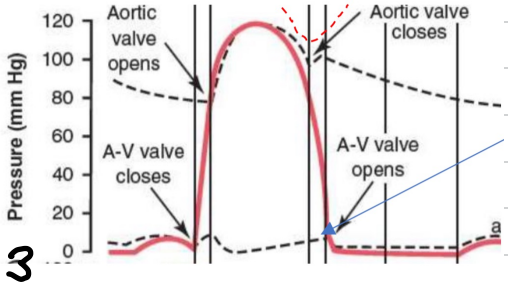
3-Blood volume does not change during the isovolumic relaxation phase because the atrioventricular (AV) valves between the atria and ventricles are still closed. Atrial pressure during this time is low, not exceeding 5 to 6 mmHg, which is insufficient to open the AV valves against the higher ventricular pressure. Diastole begins when ventricular pressure falls below the aortic pressure, not necessarily below 100 mmHg as stated.

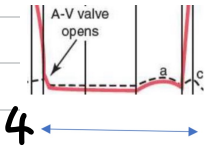
4-The AV valves will open when the ventricular pressure drops below the atrial pressure, allowing blood to flow from the atria into the ventricles, marking the end of the isovolumic relaxation phase and the beginning of the ventricular filling phase.

5-The dicrotic notch on the aortic pressure curve, occurring after the closure of the aortic valve, signifies the transition from the ejection phase to the isovolumic relaxation phase and is not directly associated with the second heart sound or the beginning of the second diastole phase. The second diastole phase, or the ventricular filling phase, begins after the opening of the AV valves, allowing blood to enter the relaxed ventricles.







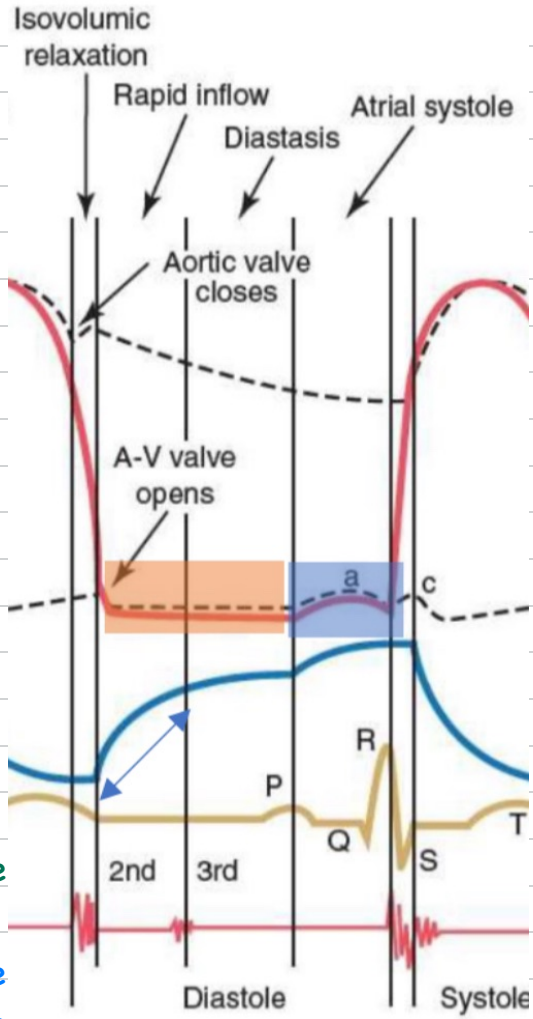


The ventricular filling period is segmented into three distinct phases. Initially, Rapid Filling occurs, marked by a swift influx of blood into the ventricles. This is followed by Reduced Filling (Diastasis), the intermediate phase where filling decelerates. The final phase, Atrial Systole or Atrial Contribution, precedes ventricular contraction and is characterized by the atria contracting to augment ventricular filling.

In the accompanying diagram, atrial contribution is denoted in Blue highlight, signifying the active participation of the atria in topping off ventricular volume. The rapid filling and diastasis phases, indicated in orange highlight, represent the ventricles' passive filling, which accounts for the majority 80%-85% of their capacity.

The AV valve opens as a result of the ventricles' relaxation, which reduces their pressure to approximately 5 mmHg, lower than that within the atria. This pressure gradient prompts the valve to open, merging the atrial and ventricular chambers.

The AV valve opens due to the ventricles' relaxation, which lowers their pressure to below that of the atria, creating a pressure gradient that allows the valve to open and the chambers to merge. During ventricular ejection, the right ventricle contracts to send deoxygenated blood to the lungs, while oxygen-rich blood from the lungs fills the left atrium, ready to enter the ventricle during ventricular systole. As the AV valve opens, blood flows from the atria into the ventricles, initiating the rapid filling phase. The diagram's Blue Arrow represents the significant quick and huge increase in ventricular blood volume that occurs with the opening of the AV valve.



Cont.

During the cardiac cycle, after the ventricles have been filled with blood, the Reduced Filling (Diastasis) phase commences. This phase is characterized by a reduced rate of filling and is considered a critical part of diastole. During diastasis, blood flows in a steady stream from the lungs via the pulmonary veins into the left atrium and subsequently through the open atrioventricular (AV) valve into the left ventricle.

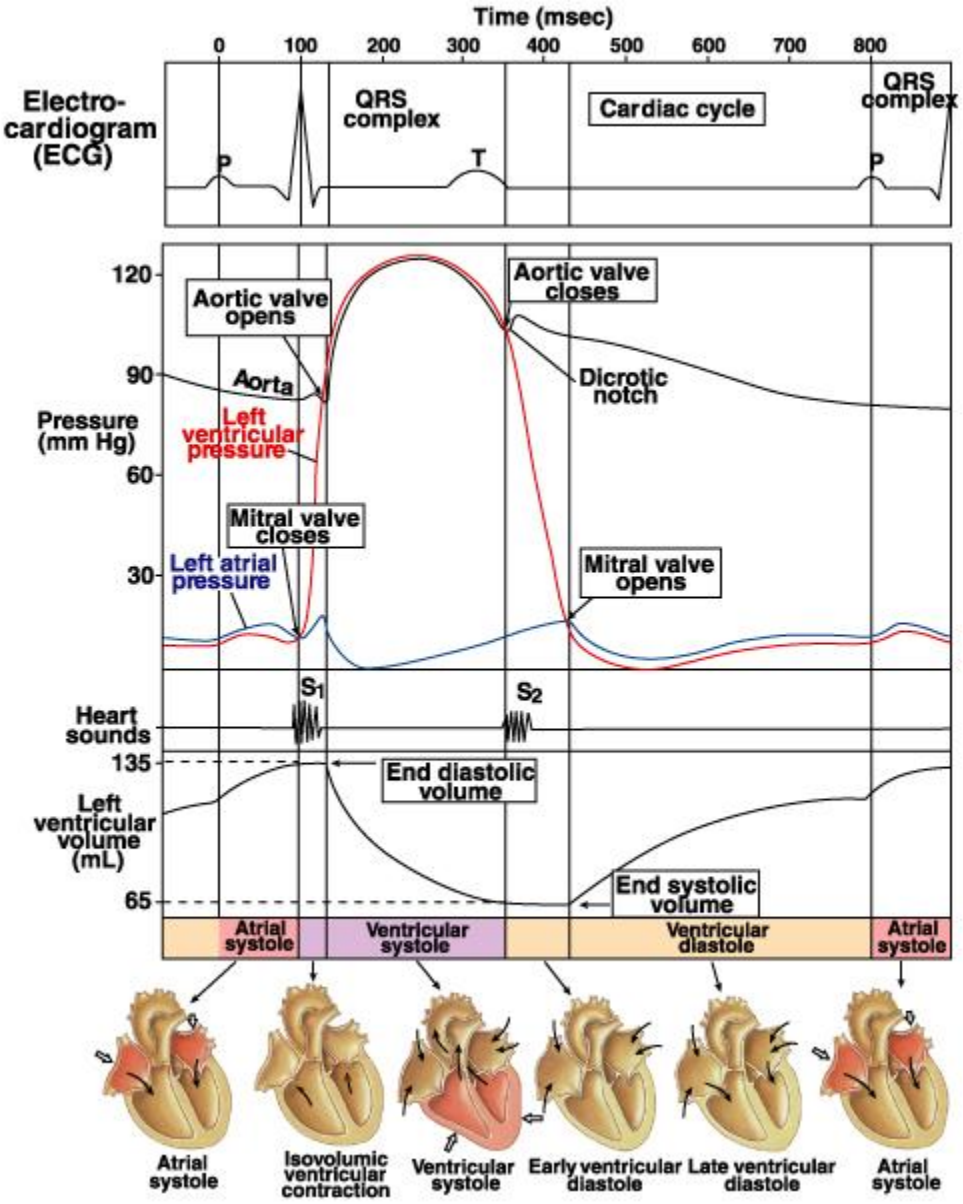
This phase represents a period of relative equilibrium where the inflow of blood from the pulmonary circulation matches the slower outflow into the relaxed ventricle, resulting in a gradual but consistent increase in ventricular volume. The rate of increase during diastasis is less pronounced than during the rapid filling phase, which is the initial surge that occurs when the AV valves first open.

Diastasis is the longest phase of the cardiac cycle and occurs in the middle of diastole, serving as a transitional period that bridges the rapid filling phase and the subsequent atrial systole. It is during this phase that the heart prepares for the atrial contraction that will complete the ventricular filling process.

The diagram illustrates this phase with a graphical representation of the ventricular volume, showing a steady but less steep incline, indicating the slower pace of blood accumulation in the ventricle during diastasis compared to the initial rapid filling phase.

The cardiac cycle culminates with atrial systole, a phase that precedes the contraction of the ventricles. During atrial systole, the atria contract, effectively boosting the volume and pressure of blood within the ventricles. This active contribution by the atria accounts for approximately 15-20% of the ventricular filling. The remaining 80-85% of ventricular filling occurs passively during the ventricular diastole, without the need for atrial contraction. This distinction is clinically significant, as it explains why conditions like atrial fibrillation, though serious, do not result in immediate fatality. Atrial fibrillation disrupts the rhythmic contractions of the atria but does not halt ventricular filling entirely.

Electrocardiography (EKG) provides a visual representation of the cardiac cycle's electrical activity. The P-wave on an EKG is indicative of atrial depolarization and typically precedes the mechanical contraction of the atria, known as atrial systole. Following atrial systole, the heart transitions from diastole to the next phase of systole. During diastole, aortic pressure diminishes as blood is distributed throughout the body's circulatory system. The aortic pressure eventually stabilizes at a normal diastolic value of approximately 80 mmHg, setting the stage for the cardiac cycle to commence a new cycle.


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

Before going to the three waves read the next slide then come back here 😊

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.

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

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

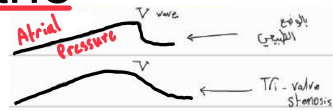
2. The c **wave** is produced by the bulging of the tricuspid valve into the atria during isovolumetric ventricular contraction.

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

Be careful of those two

3. 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.



Atrial pressure changes are indicative of atrial contraction and can be observed in both the left and right atria. These pressure changes are transmitted to the great veins, producing characteristic waves in the record of jugular pressure. The Left Atrium, directly connected to the pulmonary veins, experiences these pressure changes, but they are less apparent due to the intrathoracic location of the pulmonary veins and the left atrium's posterior position relative to the sternum.

Conversely, the Right Atrium's pressure changes have a more visible impact on the jugular veins. The jugular venous pulse (JVP) provides an indirect measure of central venous pressure and is observable because the internal jugular vein connects to the right atrium without any intervening valves, creating a continuous column of blood that reflects changes in right atrial pressure. The visibility of these waves is enhanced when a person is lying down. This is because the angle of the neck veins to the right atrium changes, making the transmission of the venous pulse waves more apparent. The absence of a valve between the right atrium and the superior vena cava allows for the direct transmission of these waves, which can be observed moving towards the jugular vein.

The presence of jugular pulse waves while a person is standing or at a 45-degree angle can indicate elevated central venous pressure, which is often associated with right-sided heart failure. Right-sided heart failure leads to a backup of blood in the systemic circulation, causing distension of the jugular veins. Therefore, the observation of these waves, especially in an upright position, can be a significant clinical finding suggestive of right-sided heart failure.

These jugular pulse waves are typically observed rather than felt, as the venous system does not have the palpable pulsation characteristic of arteries.

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.

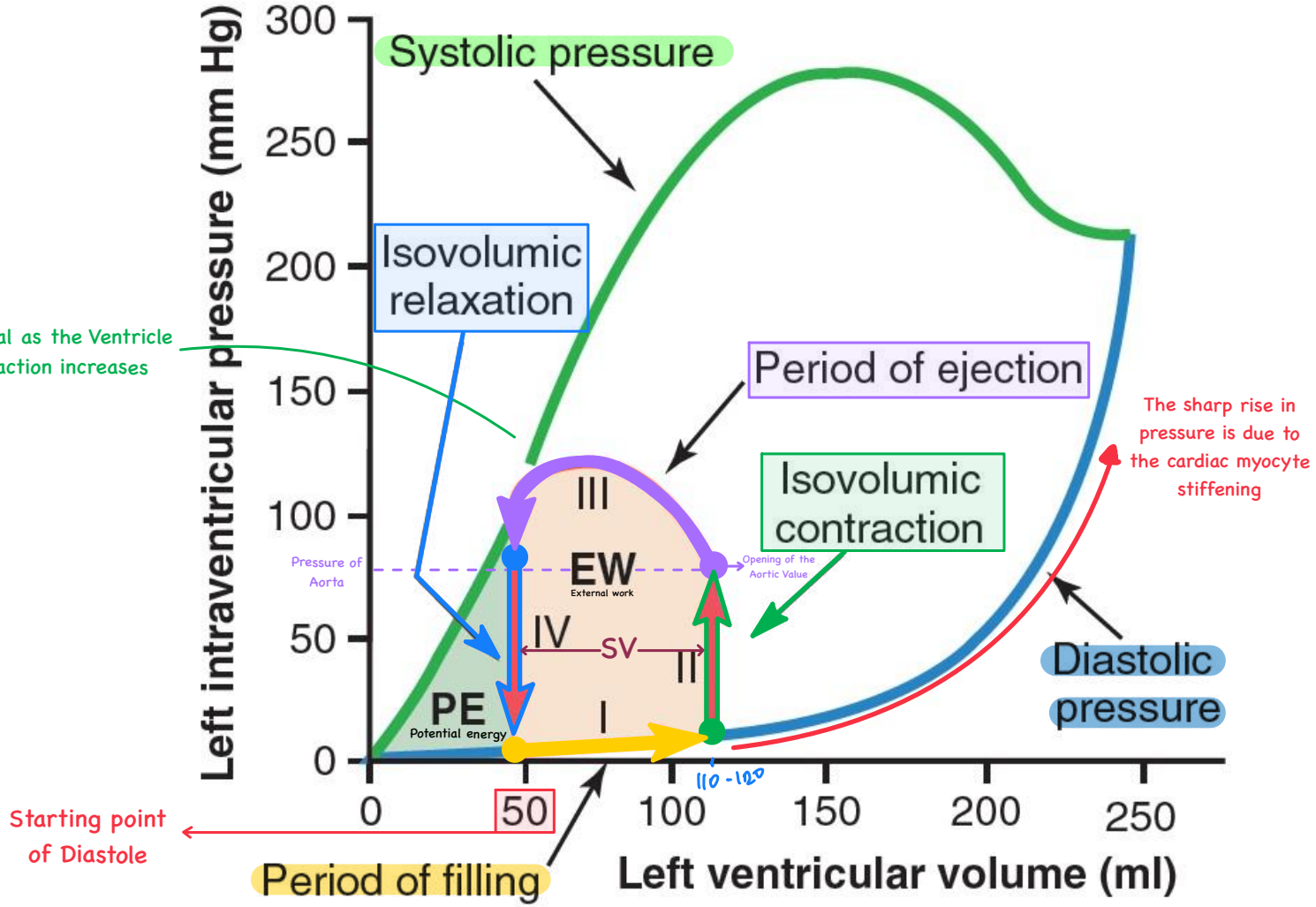
   Splitting of S_2, also known as **physiological split**, normally occurs during deep inhalation. A widely split S_2 can be associated with several different cardiovascular conditions, including Right bundle branch block and atrial septal defect.

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 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.



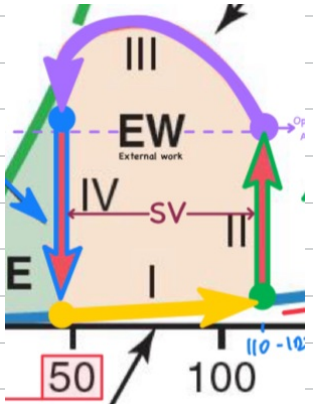
Relationship between left ventricular volume and intra-ventricular pressure during diastole and systole. Also shown by the red lines is the "volume-pressure diagram," demonstrating changes in intra-ventricular volume and pressure during the normal cardiac cycle. EW, net external work; PE, potential energy.

Work of ventricle to push blood
الجهد الذي يبذله القلب لضخ الدم

هي الطاقة الكامنة في الدم

The volume-pressure curves represent a graphical depiction of the fluctuations in volume and pressure within the left ventricle throughout the cardiac cycle. The red loop on the diagram encapsulates the entire cycle, providing a visual summary of the heart's mechanical activity.

We commence at the yellow circle, which marks the onset of diastole—the phase where the heart muscle relaxes. The opening of the mitral valve at this point permits blood to flow into the left ventricle, initiating the filling phase. During this phase, the ventricular volume increases, while the pressure rises only slightly, reflecting the compliance of the relaxed ventricular walls.



Progressing to the green circle, we reach the juncture between diastole and systole. This point also signifies the closure of the mitral valve, preventing backflow of blood into the atrium. From the green circle to the purple circle, the heart undergoes isovolumic contraction. During this phase, the ventricular volume remains unchanged, but the pressure within the ventricle surges as the heart muscle contracts, preparing to eject blood into the aorta.

The purple circle indicates the termination of isovolumic contraction and the commencement of the ejection phase. With the opening of the aortic valve, the left ventricle propels blood into the systemic circulation. The ejection continues until the blue circle, where the aortic valve closes, marking the end of systole and the beginning of isovolumic relaxation.

During isovolumic relaxation, the ventricle begins to relax, and the pressure falls while the volume remains constant. This phase transitions the heart back into diastole, ready for the next cycle of filling and ejection.

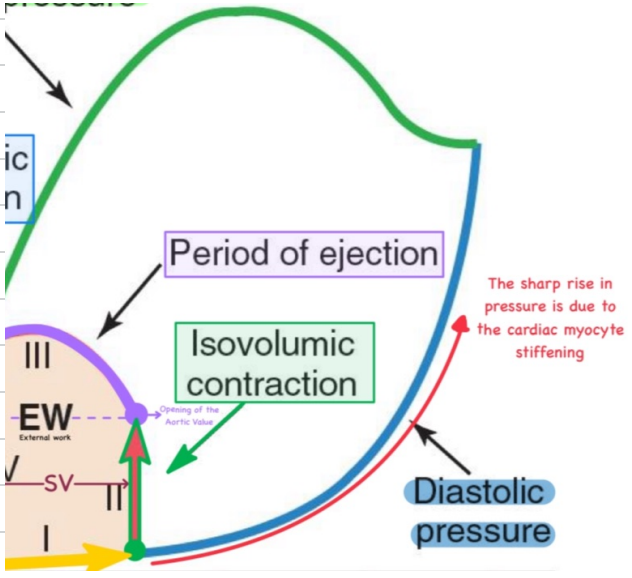
This cyclical process, represented by the red loop, is repeated with each heartbeat, ensuring the continuous circulation of blood throughout the body.

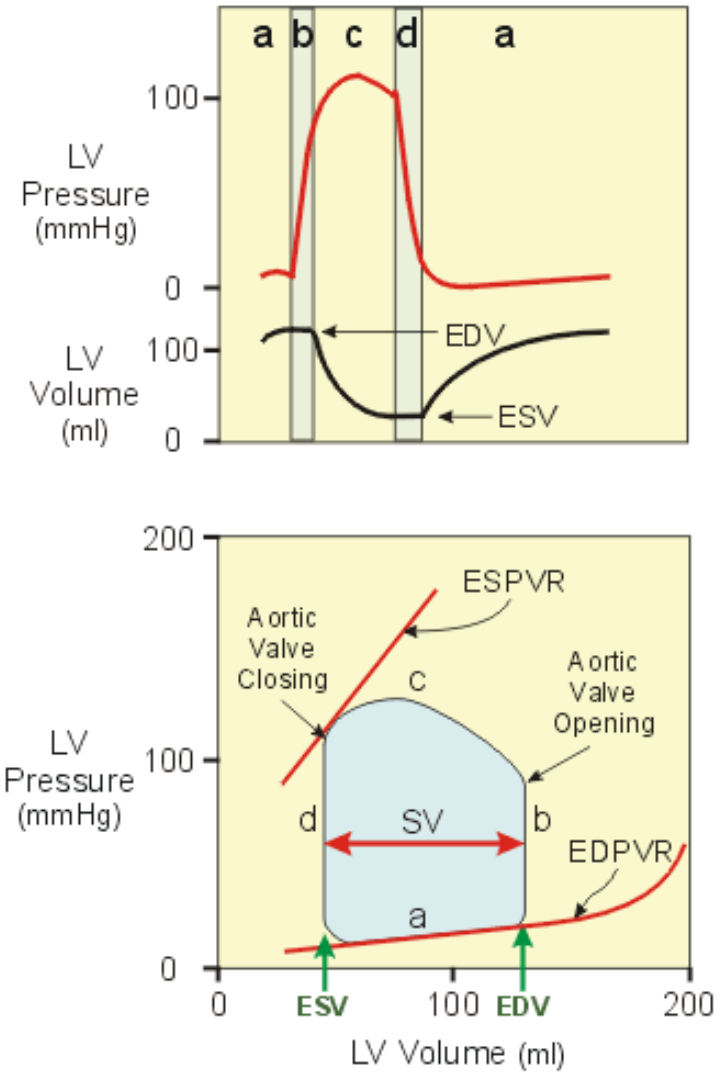
The blue curve represents the End-Systolic Pressure-Volume Relationship (ESPVR), which reflects the maximum pressure the left ventricle can generate at any given volume before contraction. It's a measure of the ventricle's contractility, indicating that with an increase in end-systolic volume, the systolic pressure rises due to stronger myocardial contractions.

The green curve is the End-Diastolic Pressure-Volume Relationship (EDPVR), showing how diastolic pressure responds to changes in volume during diastole, when the ventricle is filling with blood. This curve illustrates the compliance or stiffness of the ventricular walls; a steeper slope indicates less compliance or greater stiffness, resulting in higher pressures during filling at a given volume.

These curves do not show a point where "no further increase in volume occurs," but rather how pressures change with varying volumes within physiological limits. The area between these curves represents the stroke work done by the heart during the ejection of blood.

The volume-pressure curves provide insights into the heart's ability to contract and relax under different loading conditions, which is essential for diagnosing and managing various cardiac conditions. The relationship between diastolic filling and systolic ejection is complex, and while it's true that an increase in preload (end-diastolic volume) can lead to a stronger contraction (Frank-Starling mechanism), this is within the physiological limits of the heart's functioning.

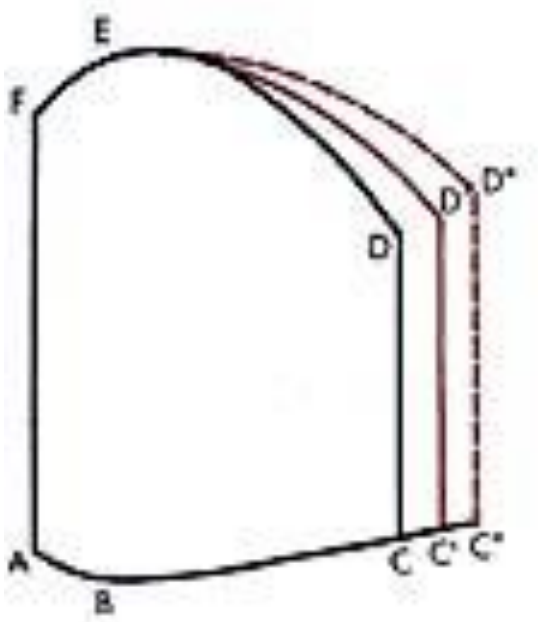




Here it shows the difference between the two diagrams

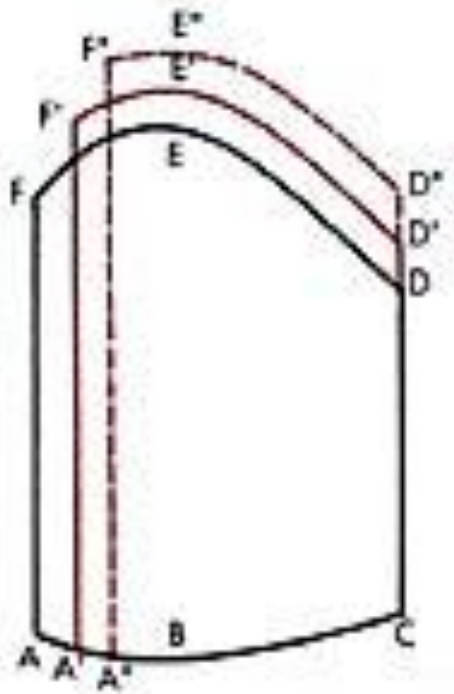
The generation of a left ventricular pressure-volume loop *(bottom panel)* from pressure and volume changes during cardiac cycle *(top panel)*. *Abbreviations*: a, ventricular filling; b, isovolumetric contraction; c, ventricular ejection; d, isovolumetric relaxation; EDV and ESV, left ventricular end-diastolic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

Very important slides



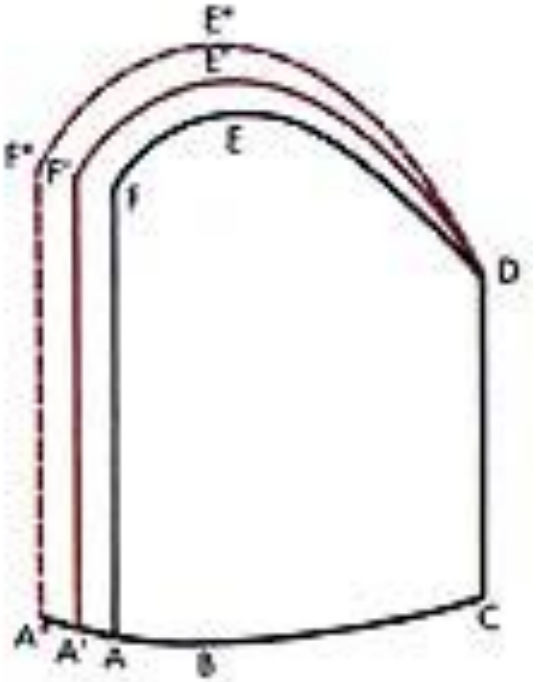
Increased preload

More blood volume remains trapped



Increased afterload

The resistant that the heart faces



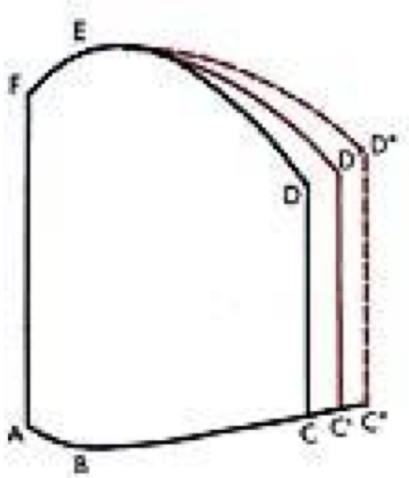
Increased contractility

Sympathetic stimulation

- The heart pumps it what receive

What will be effected here?

- increase in EDV
- Increase in the Isovolumic Contraction
- Increase in the Aortic pressure



Increased preload

What will remain fixed?

- As much as the preload increase it will go back to the same ESV

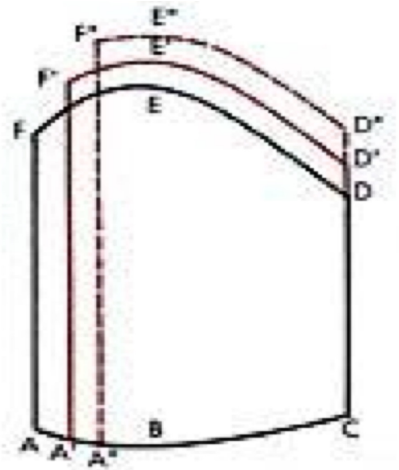
Here we have an increase in the resistance (preload) meaning the ventricle will have a difficulty in getting the blood out similar to when we have an Aortic valve stenosis

What will be effected here?

- Increase in the isovolumic contraction so the power that pushes blood increases
- Increase in ESV, because some blood will remain after systole
- Increase in the Aortic pressure

What will remain fixed?

EDV



Increased afterload

The resistant that the heart faces

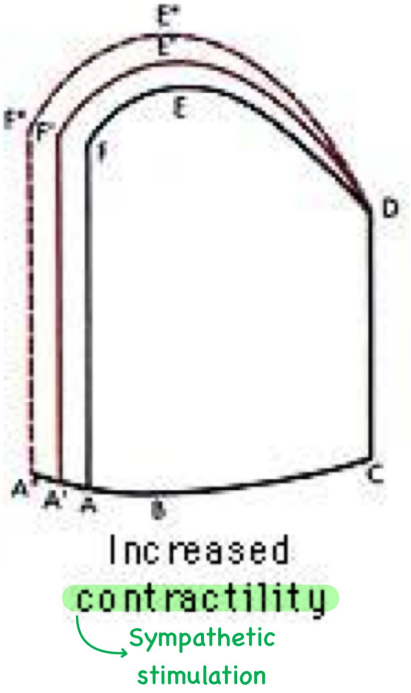
Here we have an increase in the contractility of the heart as if there is more sympathetic stimulation

What will be effected here?

- Decrease in ESV

What will remain fixed?

EDV



تم بحمد الله

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.

Answer : C