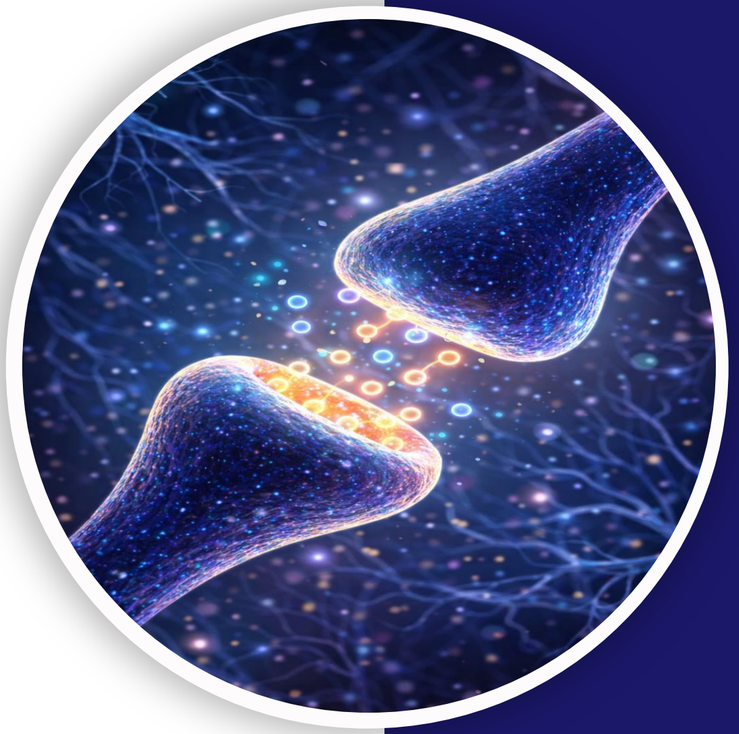
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HLS Physiology | MID 7

Cardiac Cycle pt.2



Written by : NST memberah

Reviewed by : NST

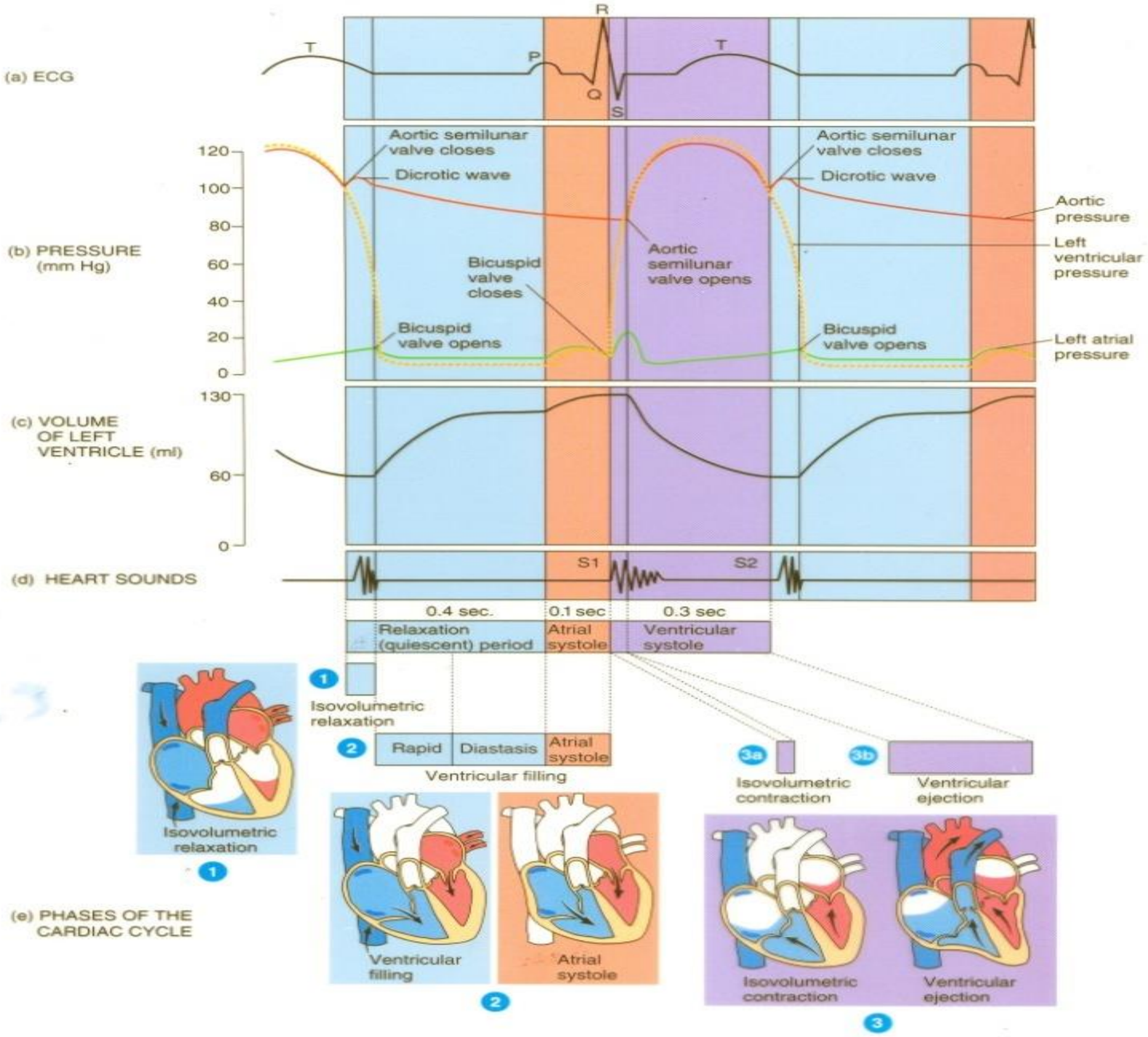
Heart Pump and Cardiac Cycle

Faisal I. Mohammed, MD, PhD
Yanal A. Shafagoj, MD, PhD

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Objectives

- To understand the volume, mechanical, pressure and electrical changes during the cardiac cycle
- To understand the inter-relationship between all these changes
- To describe the factors that regulate Cardiac output and Stroke volume.
- Resources: Textbook of Medical Physiology By Guyton and Hall


(a) ECG
R
T
P
Q
S
T
(b) PRESSURE (mm Hg)
120
100
80
60
40
20
0
Aortic semilunar valve closes
Dicrotic wave
Aortic semilunar valve opens
Bicuspid valve closes
Bicuspid valve opens
Aortic pressure
Left ventricular pressure
Left atrial pressure
(c) VOLUME OF LEFT VENTRICLE (ml)
130
60
0
(d) HEART SOUNDS
S1
S2
0.4 sec.
0.1 sec
0.3 sec
Relaxation (quiescent) period
Atrial systole
Ventricular systole
1
Isovolumetric relaxation
2
Rapid
Diastasis
Atrial systole
Ventricular filling
3a
Isovolumetric contraction
3b
Ventricular ejection
Isovolumetric relaxation
1
Ventricular filling
Atrial systole
2
Isovolumetric contraction
Ventricular ejection
3
(e) PHASES OF THE CARDIAC CYCLE

Diastolic Function Curve

The **diastolic function curve**, also known as the passive curve, can be studied using an isolated cardiac muscle fiber *in vitro*.

During this experiment, the initial length of the cardiac muscle fiber is progressively increased, which corresponds to increasing its **preload**.

In the intact heart, preload can also be discussed in relation to right atrial pressure.

Therefore, increasing muscle length, increasing preload, and increasing right atrial pressure are related concepts used to describe the effects of cardiac filling.

As ventricular volume increases, the heart initially accommodates the additional blood.

However, when left ventricular volume reaches approximately **150 mL**, further increases in volume become limited.

This occurs because the heart cannot continue accommodating increasing amounts of blood indefinitely. The **pericardium** restricts excessive stretching of the heart and limits its ability to expand further.

This relationship is represented by the **diastolic function curve**, which illustrates the passive properties of the cardiac muscle during filling.

Systolic Function Curve and Frank-Starling Relationship

In addition to the diastolic function curve, there is a **systolic function curve**, which represents the tension generated during cardiac muscle contraction.

The total tension developed by cardiac muscle consists of two components:

- **Active or developed tension:** The tension produced by active cardiac muscle contraction.
- **Passive tension:** The tension resulting from stretching the cardiac muscle before contraction.

Therefore:

[Total tension=Active tension + Passive tension]

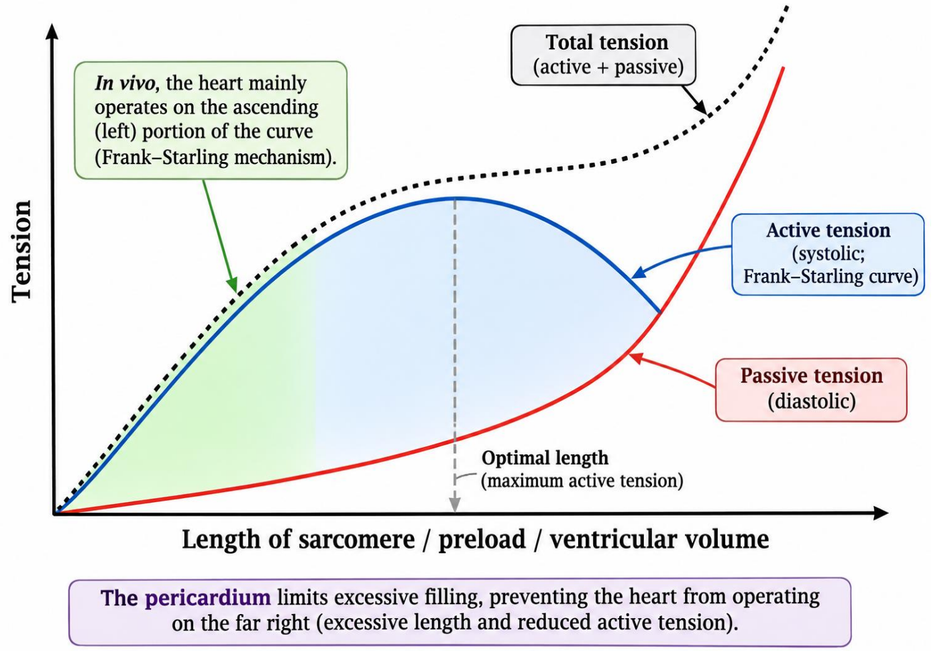
The **Frank-Starling curve** illustrates the relationship between the initial length of the cardiac muscle and the tension it develops.

As muscle length increases, the developed tension initially increases until it reaches a maximum. Beyond this point, further stretching causes a decrease in developed tension, producing the descending portion of the curve.

However, in the intact heart *in vivo*, the descending portion is not normally observed. The heart operates primarily along the ascending portion of the curve.

Thus, within the physiological range, increasing the initial length of the cardiac muscle increases the force generated during contraction.

This relationship is referred to as the **cardiac function curve** and explains the effect of increasing preload on cardiac contraction.



Ventricular Volumes and Stroke Volume

To understand how the heart functions as a pump, consider an example in which the **left ventricle contains 120 mL of blood** at the end of diastole.

During ventricular contraction, 70 mL of blood is ejected, leaving 50 mL inside the ventricle.

These volumes represent three important measurements.

3.1. End-Diastolic Volume (EDV)

End-diastolic volume is the amount of blood present in the ventricle at the end of diastole, immediately before contraction.

In this example:

EDV=120mL

3.2. Stroke Volume (SV)

Stroke volume is the amount of blood ejected by the ventricle during each heartbeat.

SV=70 mL

3.3. End-Systolic Volume (ESV)

End-systolic volume is the amount of blood remaining in the ventricle at the end of systole.

In this example:

ESV=50 mL

The doctor referred to this remaining blood as the *residual volume*, although the physiological term is **end-systolic volume**.

An important point is that the ventricle does not empty completely during each contraction. Instead, a certain volume of blood remains inside it after blood has been ejected.

Therefore:

SV=EDV−ESV

SV=120−50=70mL

Ejection Fraction (EF)

Ejection fraction represents the percentage of end-diastolic ventricular volume that is ejected during systole.

It is calculated using:

$$EF = \frac{SV}{EDV} \times 100$$

the result as approximately **55%**, representing a normal ejection fraction in this example.

Ejection fraction is one of the most important measurements used to evaluate cardiac function.

Clinical Importance of Ejection Fraction

Ejection fraction is particularly important in assessing patients with **heart failure**.

Different ejection fraction values can be used to evaluate the degree of cardiac dysfunction.

For example, the doctor mentioned ranges of 40–50% and 30–40% when discussing the classification of heart failure.

However, he emphasized that the detailed classification is not required for the examination. Students may consult additional references if they wish to understand how these categories are determined.

The important concept is to understand the significance of ejection fraction in evaluating the pumping efficiency of the heart.

The doctor also mentioned that a healthy individual of approximately 20 years of age may have an ejection fraction of around **65%**

Effect of Sympathetic Stimulation on Ejection Fraction

Sympathetic stimulation increases **myocardial contractility**, resulting in a stronger ventricular contraction.

When contractility increases, the cardiac function curve shifts **upward and to the left**.

As a result, the heart ejects a greater proportion of its end-diastolic volume, leading to an increase in ejection fraction.

The doctor mentioned that, during markedly increased sympathetic stimulation, ejection fraction may reach approximately **90%**.

Cardiac function can be examined using the diastolic and systolic function curves.
Depending on the type of graph, the horizontal axis may represent ventricular volume, sarcomere length, or preload-related pressure, such as right atrial pressure.

Preload

Preload is related to the initial stretching of the ventricular muscle before contraction.
For example, suppose the volume of blood inside the left ventricle increases from **50 mL to 120 mL** during diastole.
On the pressure-volume graph, these volumes are represented by two points:

- **Point A:** End-systolic volume = 50 mL.
- **Point B:** End-diastolic volume = 120 mL.

The line from A to B represents ventricular filling along the **diastolic function curve**.
In the ventricular pressure-volume loop, the horizontal axis represents ventricular volume, while the vertical axis represents ventricular pressure.

Afterload

Afterload represents the load imposed on the ventricle during contraction.
For the left ventricle, this load is related to **aortic arterial pressure**.
Suppose the aortic pressure is 80 mmHg.
The ventricle must generate enough pressure to overcome this arterial pressure before the aortic valve can open and blood can be ejected.
The doctor described afterload as the load imposed on the ventricle after contraction begins.
In other words, if the ventricle does not contract, it does not need to overcome the aortic pressure to eject blood. However, once contraction begins, ventricular pressure must exceed aortic pressure to open the aortic valve.
Therefore, as arterial pressure increases, the load against which the ventricle contracts also increases.
This makes ventricular ejection more difficult and tends to **decrease stroke volume**.
If afterload becomes extremely high, the ventricle may be unable to eject blood, causing stroke volume to become zero.

Ventricular Pressure-Volume Loop

The **ventricular pressure-volume loop** represents the relationship between ventricular pressure and ventricular volume during the cardiac cycle.

The doctor used the left ventricle to explain the cardiac cycle, together with the pressure relationships between the left atrium, left ventricle, and aorta.

The same general principles apply to the right side of the heart, although the pressure values are different.

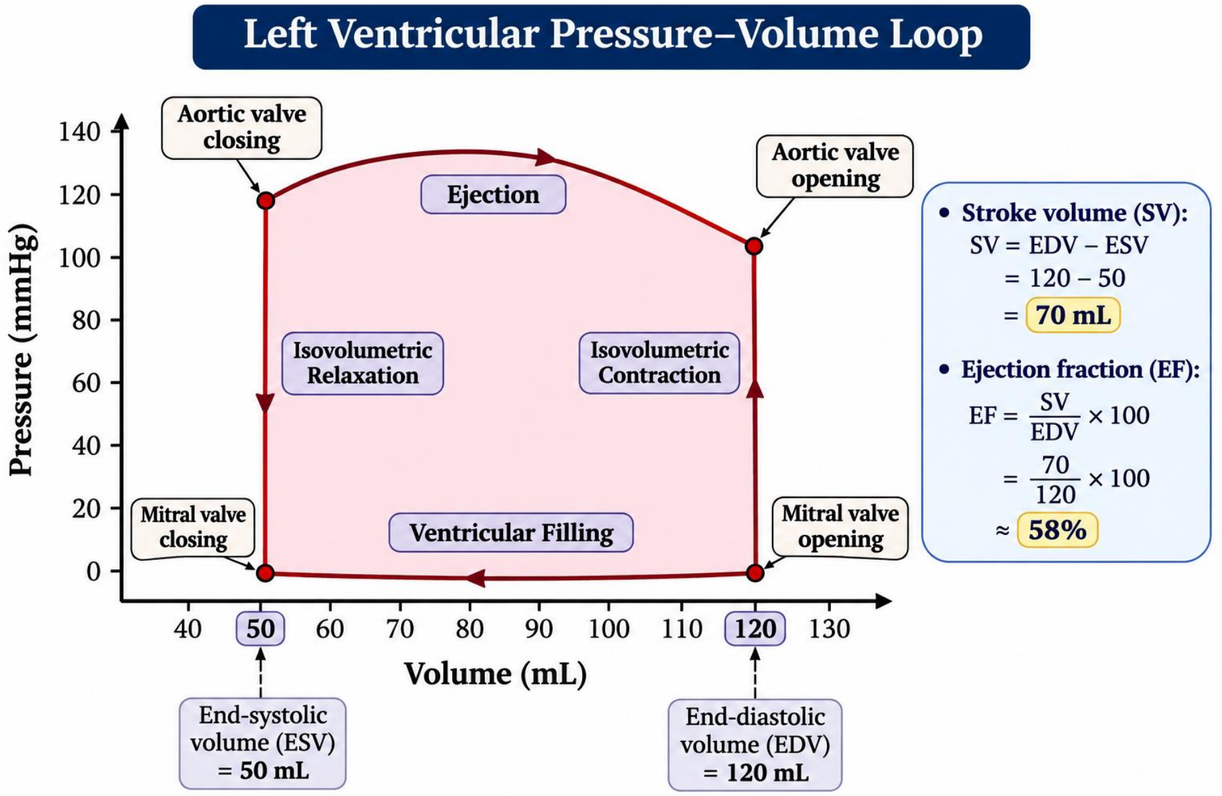
Ventricular Filling

Consider the following pressures:

Structure	Pressure
Left ventricle	0 mmHg
Left atrium	5 mmHg
Aorta	80 mmHg

Because left atrial pressure is greater than left ventricular pressure, the **mitral valve is open**, allowing blood to flow from the left atrium into the left ventricle.

As ventricular filling continues, the volume increases until it reaches the end-diastolic volume of **120 mL**.



Phases of Ventricular Systole

Ventricular systole lasts approximately **0.3 seconds** and consists of three phases:

1. Isovolumetric contraction.
2. Rapid ejection.
3. Reduced ejection.

Isovolumetric Contraction

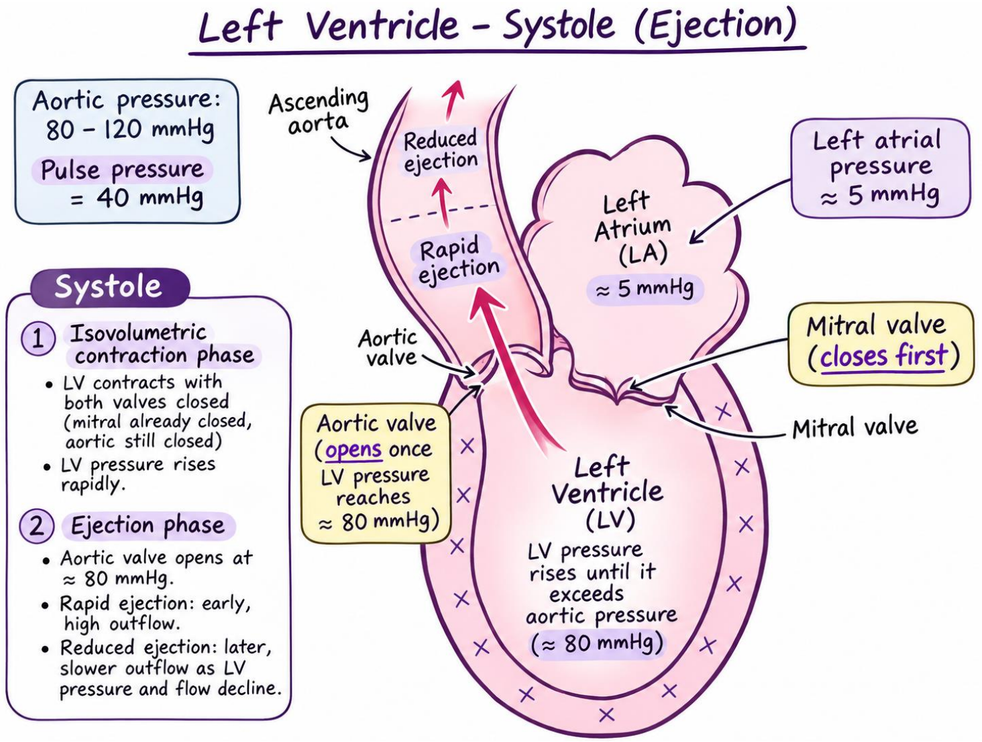
The first phase of ventricular systole is **isovolumetric contraction**, also called isovolumic contraction.
It begins when ventricular pressure becomes greater than atrial pressure, causing the atrioventricular valves to close.
During this phase, both the atrioventricular and semilunar valves are closed.
Consequently, ventricular pressure increases without any change in ventricular volume.
In the previous example, ventricular volume remains at **120 mL**, while ventricular pressure rises progressively toward 80 mmHg.
The doctor mentioned that this initial contraction phase lasts approximately **0.01 second**.
As ventricular contraction continues, the pressure rises until it becomes sufficient to open the aortic valve.
Once the aortic valve opens, blood begins to leave the left ventricle, marking the beginning of the next phase.

Rapid Ejection

The **rapid ejection phase** begins when the aortic valve opens and blood flows from the left ventricle into the aorta.
Most of the stroke volume is ejected during the early part of this phase.
For example, if the total stroke volume is 70 mL, approximately **50 mL** may be ejected during rapid ejection.
The doctor mentioned that most of the blood is ejected during approximately the first third of the ejection period.
He also referred to a duration of approximately **0.1 second** for rapid ejection.

Reduced Ejection

After rapid ejection, blood continues to leave the ventricle, but at a slower rate.
This is called the **reduced ejection phase**.
During this phase, the remaining portion of the stroke volume is expelled.
Together, the three phases constitute ventricular systole, which lasts approximately 0.3 seconds.



Changes in Aortic Pressure During Ventricular Ejection

At the beginning of ventricular ejection, aortic pressure is approximately **80 mmHg**.
As blood is ejected from the left ventricle into the aorta, the amount of blood entering the aorta increases, causing a rise in aortic pressure.
The doctor explained this relationship using the equation:

$$P = \frac{F}{A}$$

The force is produced by blood acting against the wall of the aorta.
During ventricular ejection, the force exerted against the aortic wall increases.
At the same time, the aorta contains **elastic fibers (elastin)** that allow it to dilate as blood enters.
Therefore, both the force exerted by the blood and the area of the aorta increase.
The doctor illustrated this by explaining that if the force increases twice as much as the area, the pressure will still increase.
As a result, aortic pressure rises from approximately **80 mmHg to 120 mmHg** during systole.

Arterial Blood Pressure

The changes in aortic pressure during the cardiac cycle are described using systolic pressure, diastolic pressure, pulse pressure, and mean arterial pressure.

Systolic Pressure

Systolic pressure is the maximum arterial pressure reached during ventricular systole.

In this example:

$$SBP = 120 \text{ mmHg}$$

Diastolic Pressure

Diastolic pressure is the arterial pressure associated with ventricular diastole.

In this example:

$$DBP = 80 \text{ mmHg}$$

Pulse Pressure

Pulse pressure is the difference between systolic and diastolic arterial pressures.

$$\boxed{PP = SBP - DBP}$$

Mean Arterial Pressure

Mean arterial pressure represents the average arterial pressure during the cardiac cycle.

The doctor explained that diastole lasts longer than systole. Therefore, the approximate calculation of mean arterial pressure gives a greater contribution to diastolic pressure.

$$\boxed{MAP \approx \frac{2}{3}DBP + \frac{1}{3}SBP}$$

Thus, mean arterial pressure is approximately two-thirds diastolic pressure plus one-third systolic pressure.

General Organization of the Cardiac Cycle

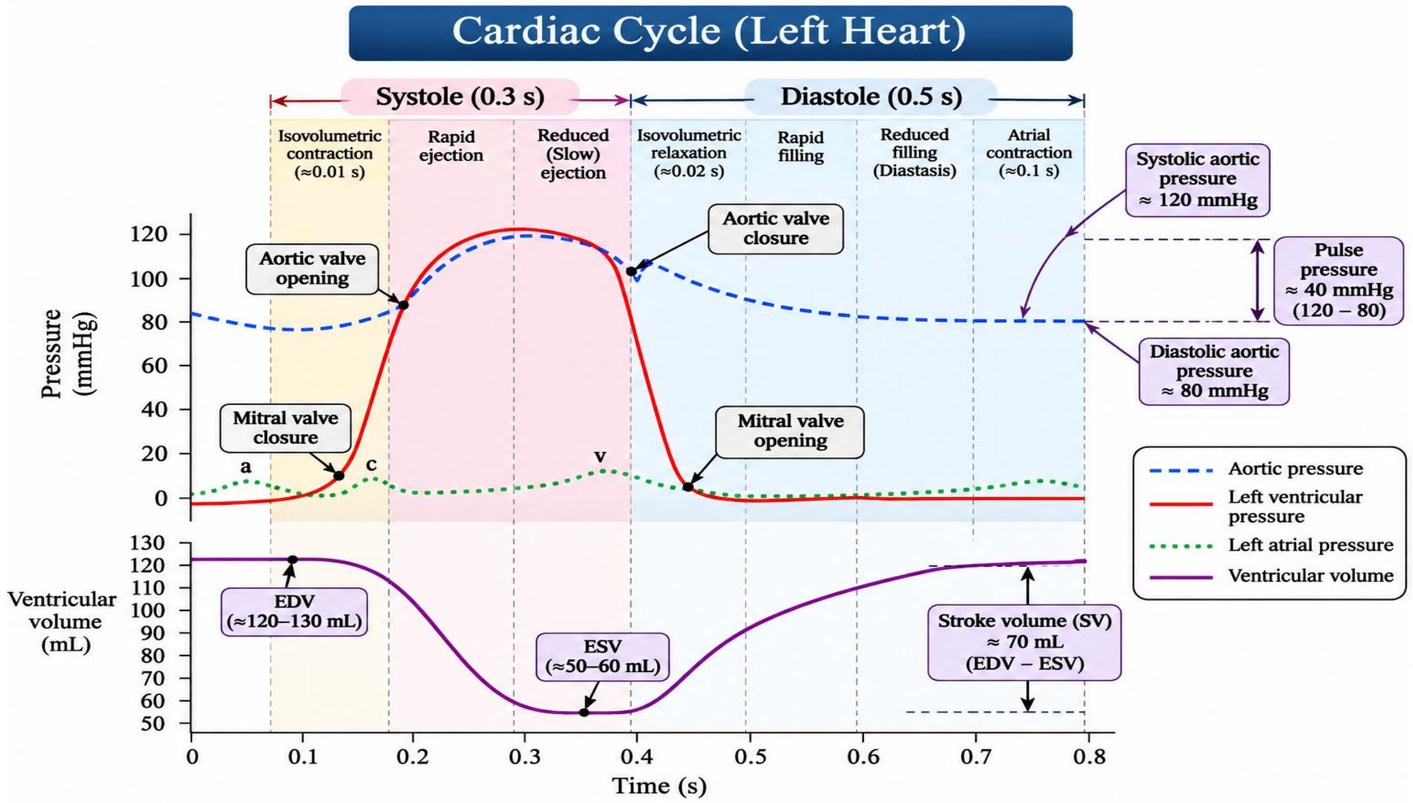
The **cardiac cycle** consists of two major periods: ventricular systole and ventricular diastole.

Ventricular systole is divided into three phases:

1. Isovolumetric contraction.
2. Rapid ejection.
3. Reduced ejection.

Ventricular diastole consists of **four phases**, which will be discussed separately.

Period	Duration
Ventricular systole	0.3 seconds
Ventricular diastole	0.5 seconds



Ventricular Pressure Changes During the Cardiac Cycle

The **left ventricular end-diastolic pressure** may range between approximately 5 and 8 mmHg, whereas the corresponding pressure in the right ventricle ranges between approximately 2 and 6 mmHg. However, for simplicity, the ventricular pressure is represented as approximately zero at the beginning of the diagram.

During ventricular systole, left ventricular pressure rises until it reaches a maximum of approximately **120 mmHg**. It then begins to decrease as the ventricle relaxes.

When aortic pressure becomes greater than left ventricular pressure, the aortic valve eventually closes.

Continued Ejection Despite Higher Aortic Pressure

In the diagram, the **white curve represents ventricular pressure**, while the darker curve represents aortic pressure.

During most of ventricular ejection, ventricular pressure is higher than aortic pressure. However, toward the end of ejection, there is a short period in which aortic pressure becomes greater than ventricular pressure, yet blood continues to leave the ventricle.

Normally, blood flows from a region of higher pressure to a region of lower pressure. Therefore, the question is:

How can blood continue to leave the left ventricle when aortic pressure is higher than ventricular pressure?

The explanation is the **momentum of blood flow**.

Because the blood is already moving from the ventricle into the aorta, its momentum allows a small amount of additional blood to continue flowing forward even after ventricular pressure has fallen below aortic pressure.

Eventually, the aortic valve closes, and ventricular ejection stops

Isovolumetric Relaxation

Following aortic valve closure, the ventricular pressure continues to decrease as the ventricle relaxes.

At this stage:

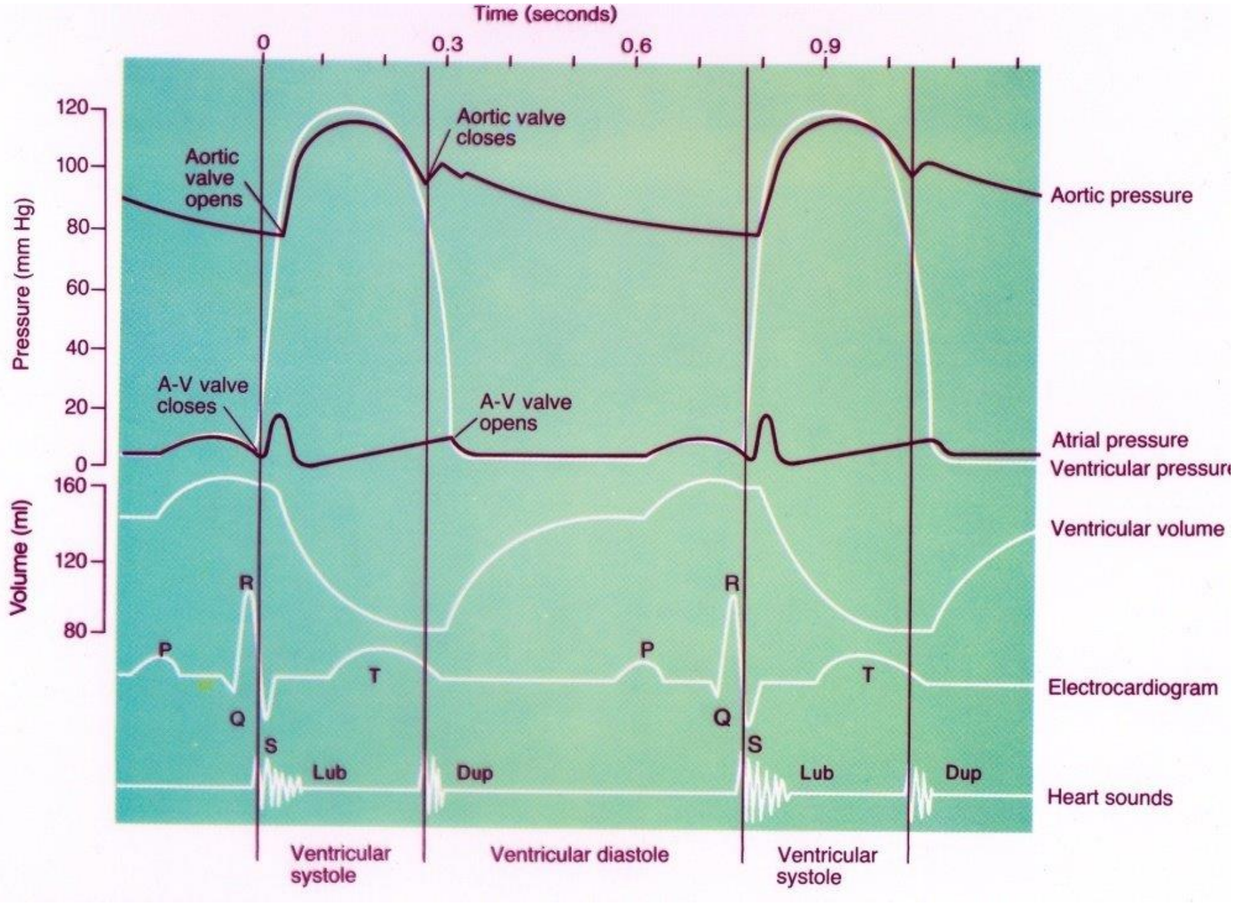
- Aortic pressure is higher than ventricular pressure, so the **aortic valve is closed**.
- Ventricular pressure is still higher than left atrial pressure, so the **mitral valve remains closed**.

Therefore, both valves are closed, and no blood enters or leaves the ventricle.

The ventricular volume remains constant, producing a horizontal segment on the ventricular volume curve.

This phase is called **isovolumetric relaxation**.

It continues until ventricular pressure decreases sufficiently for the atrioventricular valve to open.



The doctor emphasized that an examination question may ask students to calculate **ejection fraction directly from a ventricular volume graph**.

To answer such a question, students must identify the maximum ventricular volume (EDV) and the minimum ventricular volume (ESV), then calculate stroke volume and ejection fraction.

The doctor noted that he is more likely to ask about ejection fraction than stroke volume alone because calculating ejection fraction requires understanding both measurements.

Calculating Stroke Volume and Ejection Fraction from the Graph

The doctor emphasized the importance of interpreting the ventricular volume curve to calculate stroke volume and ejection fraction.

In the second diagram, ventricular volume changes between approximately **160 mL and 80 mL**.

End-Diastolic Volume (EDV)

End-diastolic volume is the maximum ventricular volume, reached at the end of ventricular filling.

EDV=160mL

End-Systolic Volume (ESV)

End-systolic volume is the minimum ventricular volume, remaining after ventricular ejection.

ESV=80mL

Stroke Volume (SV)

Stroke volume is calculated by subtracting end-systolic volume from end-diastolic volume.

SV=EDV-ESV

SV=160-80= 80 mL

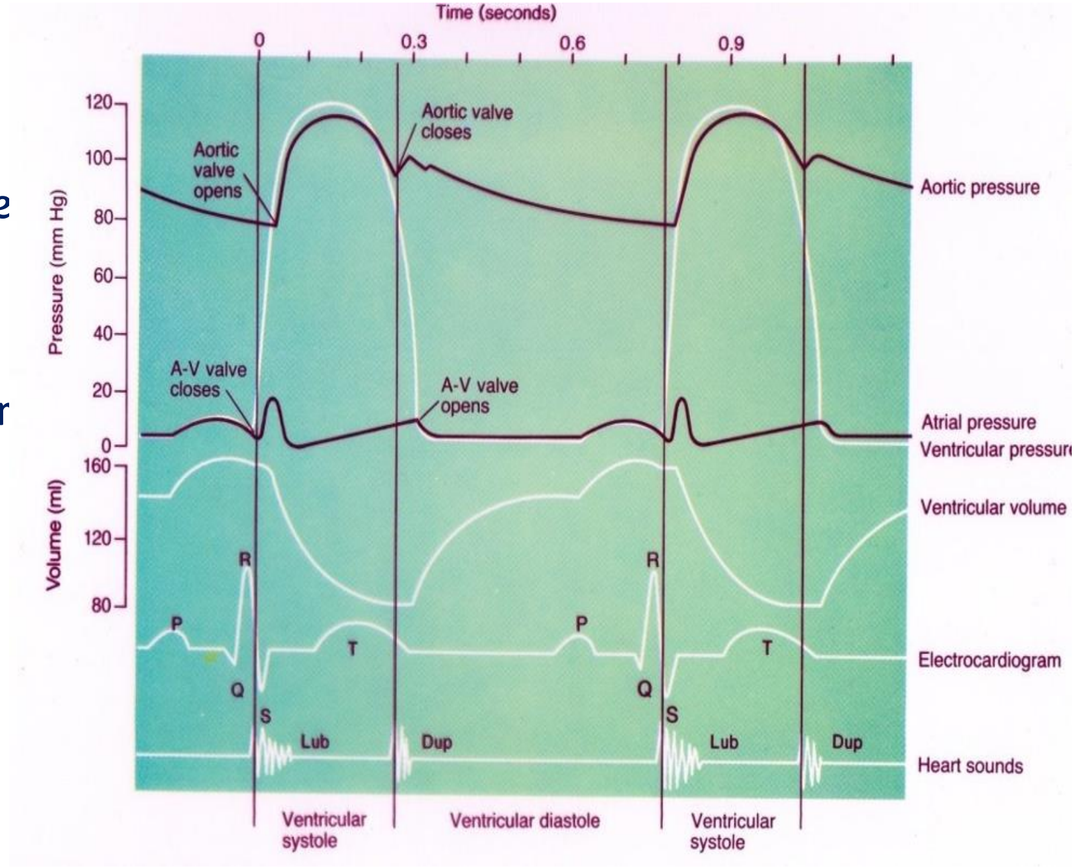
Ejection Fraction (EF)

Ejection fraction is calculated as:

EF=(SV/EDV)*100 %

EF= 80/160*100%

EF=50%



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Pressure Differences and Atrioventricular Valve Closure

The cardiac valves open and close according to pressure differences between the atria, ventricles, and arteries.

During ventricular filling, atrial pressure is higher than ventricular pressure, allowing the atrioventricular valves to remain open.

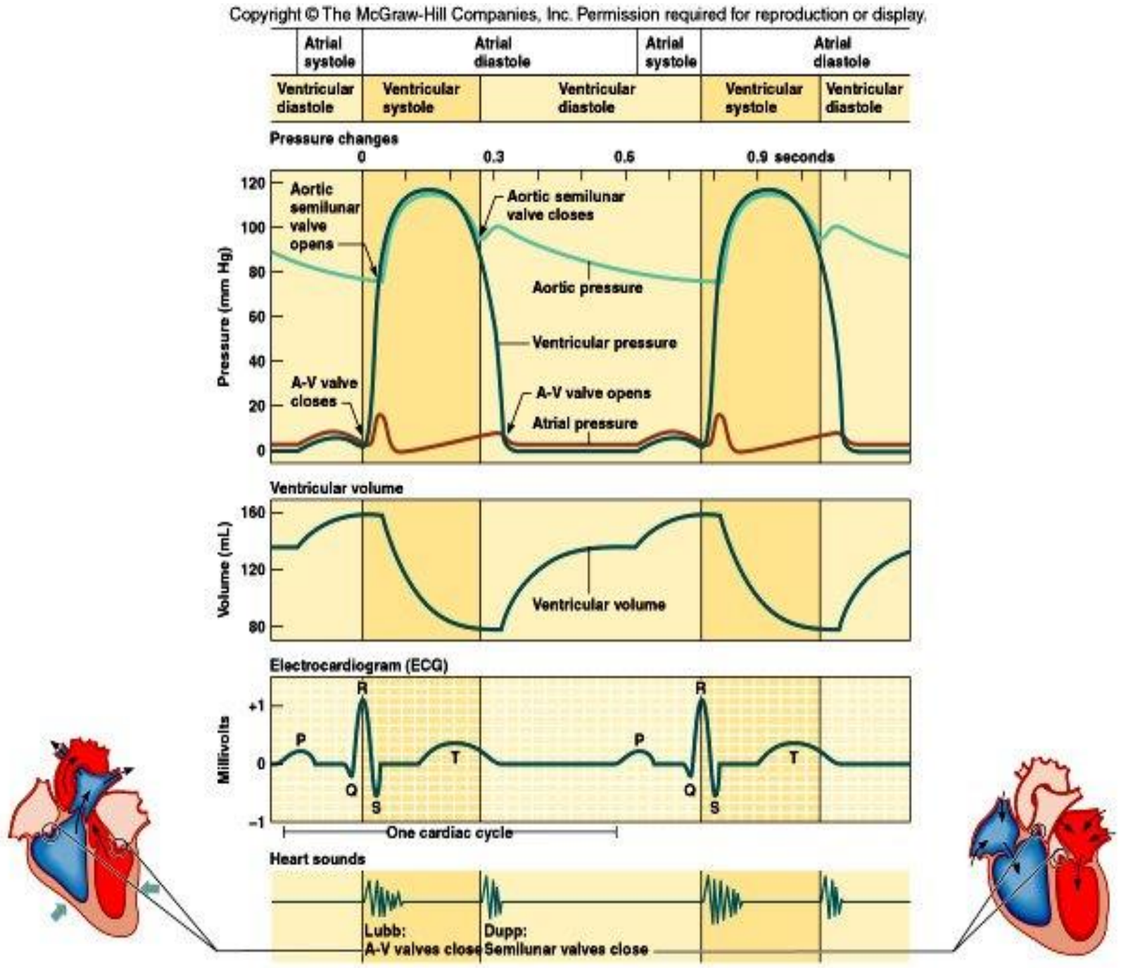
At the beginning of ventricular systole, ventricular pressure increases and becomes greater than atrial pressure.

This causes the **atrioventricular (AV) valves** to close:

- **Mitral valve:** Located between the left atrium and left ventricle.
- **Tricuspid valve:** Located between the right atrium and right ventricle.

Both valves close because ventricular pressure exceeds atrial pressure.

Their closure is associated with vibrations that produce the **first heart sound (S1)**.



First Heart Sound (S1)

The **first heart sound, S1**, is produced by the closure of the atrioventricular valves at the beginning of ventricular systole.

It occurs at approximately the beginning of the **isovolumetric contraction phase**.

Therefore, S1 indicates the beginning of ventricular systole and is associated with closure of both the mitral and tricuspid valves.

At the end of isovolumetric contraction, ventricular pressure becomes greater than the pressure in the corresponding artery, causing the semilunar valves to open.

Unlike valve closure, the normal opening of the semilunar valves is **silent** and does not produce an audible heart sound.

Second Heart Sound (S2)

At the end of ventricular ejection, ventricular pressure decreases as relaxation begins.

When arterial pressure becomes greater than ventricular pressure, the semilunar valves close.

These valves are:

- **Aortic valve.**
- **Pulmonary valve.**

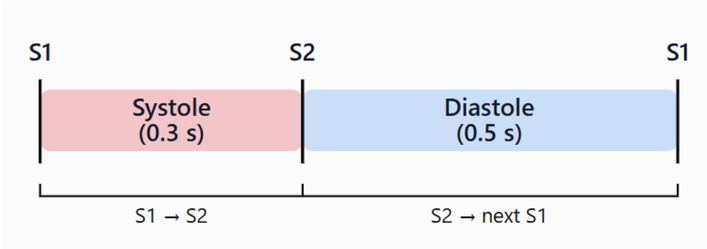
Their closure produces the **second heart sound (S2)**.

S2 marks the end of ventricular systole and the beginning of ventricular diastole, including the isovolumetric relaxation phase.

The doctor emphasized the following relationship:

Normal valve closure produces heart sounds, whereas normal valve opening is silent.

The cardiac cycle consists of ventricular systole and ventricular diastole.
The first and second heart sounds can be used to identify the timing of these two periods.



Interval Between S1 and S2
The interval between S1 and S2 represents **ventricular systole**.
Its approximate duration is: 0.3seconds
Interval Between S2 and the Next S1
The interval between S2 and the next S1 represents **ventricular diastole**.
Its approximate duration is: 0.5seconds
Thus, the first interval corresponds to systole, while the second corresponds to diastole.

Heart Murmurs
The doctor explained that an additional or unusual sound heard during cardiac auscultation may indicate a **heart murmur**.
Heart murmurs can be classified according to their timing in relation to S1 and S2.
Systolic Murmur
A **systolic murmur** occurs between S1 and S2, during ventricular systole.
Diastolic Murmur
A **diastolic murmur** occurs between S2 and the next S1, during ventricular diastole.
Classification According to Timing
Murmurs can also be described according to when they occur within systole or diastole.
For example, a murmur occurring during the early part of systole is called an **early systolic murmur**.
If a murmur extends from the beginning to the end of systole, it is called a **pansystolic murmur**.
The same principle of describing a murmur according to its timing can be applied to diastolic murmurs.
The doctor mentioned that this classification will be discussed in greater detail during the cardiology course in the fourth year.

Duration of the Cardiac Cycle and Heart Rate

In the cardiac cycle diagram, the **X-axis represents time**.

The first heart sound (S1) and second heart sound (S2) occur relatively close together because the interval between them represents ventricular systole, which is shorter than ventricular diastole.

The interval between S2 and the following S1 is longer because it represents ventricular diastole.

One complete cardiac cycle extends from one S1 to the next S1.

If the duration of one cardiac cycle is known, heart rate can be calculated using:

$$HR = \frac{60}{\text{Duration of one cardiac cycle (seconds)}}$$

Relationship Between the ECG and Atrial Contraction

The **P wave** on the electrocardiogram (ECG) represents atrial depolarization, which is followed by atrial contraction.

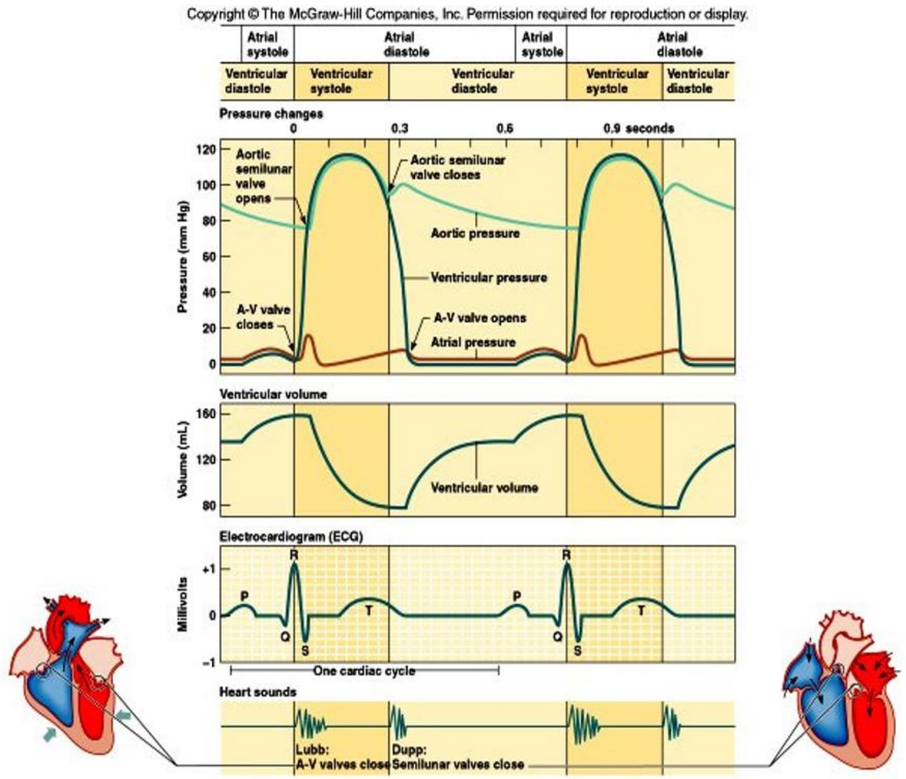
During atrial contraction, atrial pressure increases from approximately **5 mmHg to 8 mmHg**.

This rise in pressure produces the first atrial pressure wave, known as a **wave**.

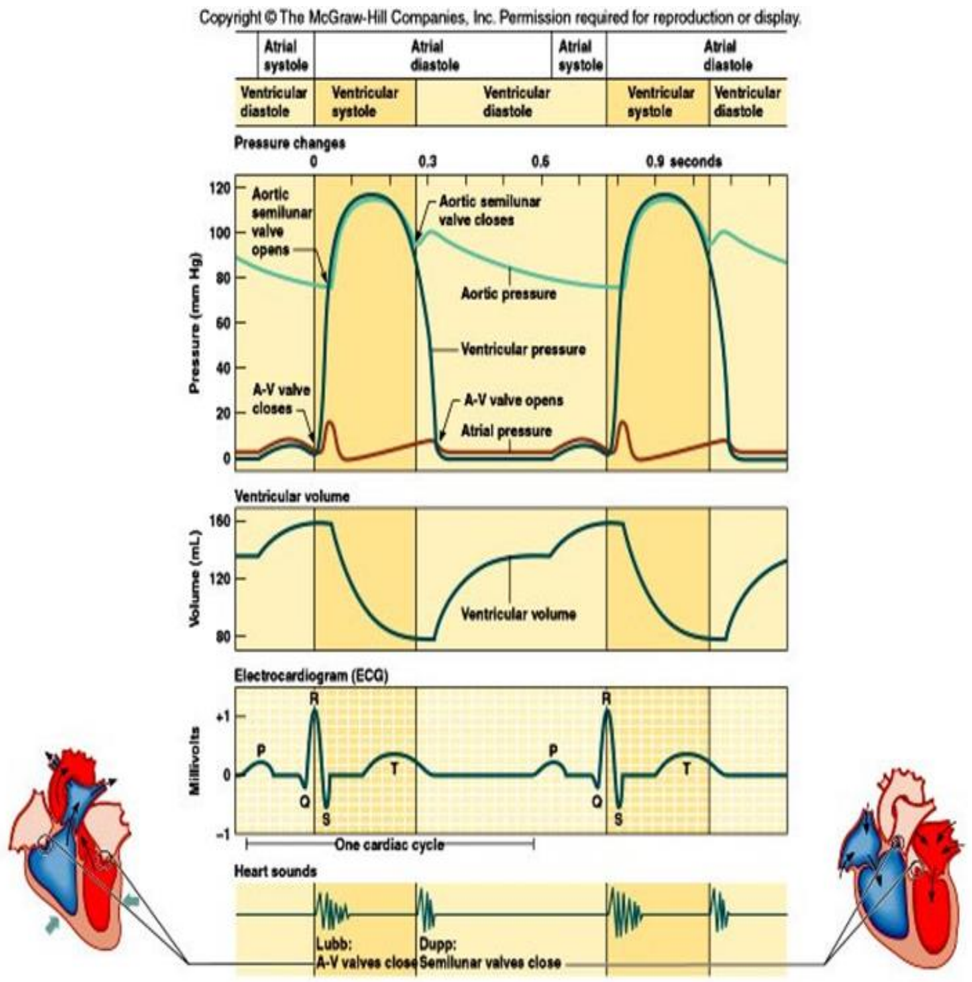
Following the P wave, the **QRS complex** represents ventricular depolarization, which is followed by ventricular contraction.

As ventricular contraction begins, ventricular pressure increases, causing the atrioventricular (AV) valves to close. Their closure produces the **first heart sound (S1)**.

The doctor also mentioned that the relative intensity of heart sounds depends on the location at which they are heard during auscultation.



Contribution of Atrial Contraction to Ventricular Filling
Ventricular filling occurs through both passive blood flow and atrial contraction.
In the example shown in the diagram, ventricular volume increases from approximately **130 mL to 160 mL** during atrial contraction, representing an additional 30 mL of blood.
The doctor explained that atrial contraction contributes approximately **10–30% of ventricular filling**, whereas the remaining **70–90% occurs passively**, without requiring active atrial contraction.
Therefore, atrial contraction contributes to ventricular filling but is not responsible for most of it.
Even in the absence of effective atrial contraction, the ventricles can continue to fill through passive blood flow.



Clinical Significance: Atrial Fibrillation

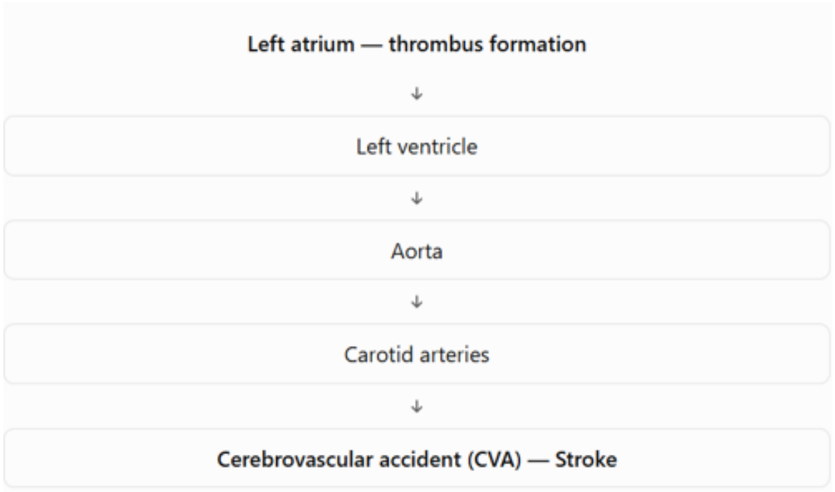
In **atrial fibrillation (AF)**, effective coordinated atrial contraction is lost. However, ventricular filling can still occur because most filling is passive.

Although ventricular filling can continue, atrial fibrillation may be associated with serious complications, including heart failure, ischemia, and stroke.

Mechanism of Stroke in Atrial Fibrillation

The absence of effective atrial contraction can lead to **blood stasis within the left atrium**, increasing the risk of thrombus formation.

If a thrombus forms and subsequently becomes dislodged, it may travel through the following pathway:



This explains how atrial fibrillation may lead to an ischemic stroke through the formation and embolization of a blood clot.

The doctor emphasized that **anticoagulants** are given to patients with atrial fibrillation to reduce the risk of thrombus formation and prevent stroke. He described this treatment as potentially lifelong.

Isovolumetric Contraction and Relaxation

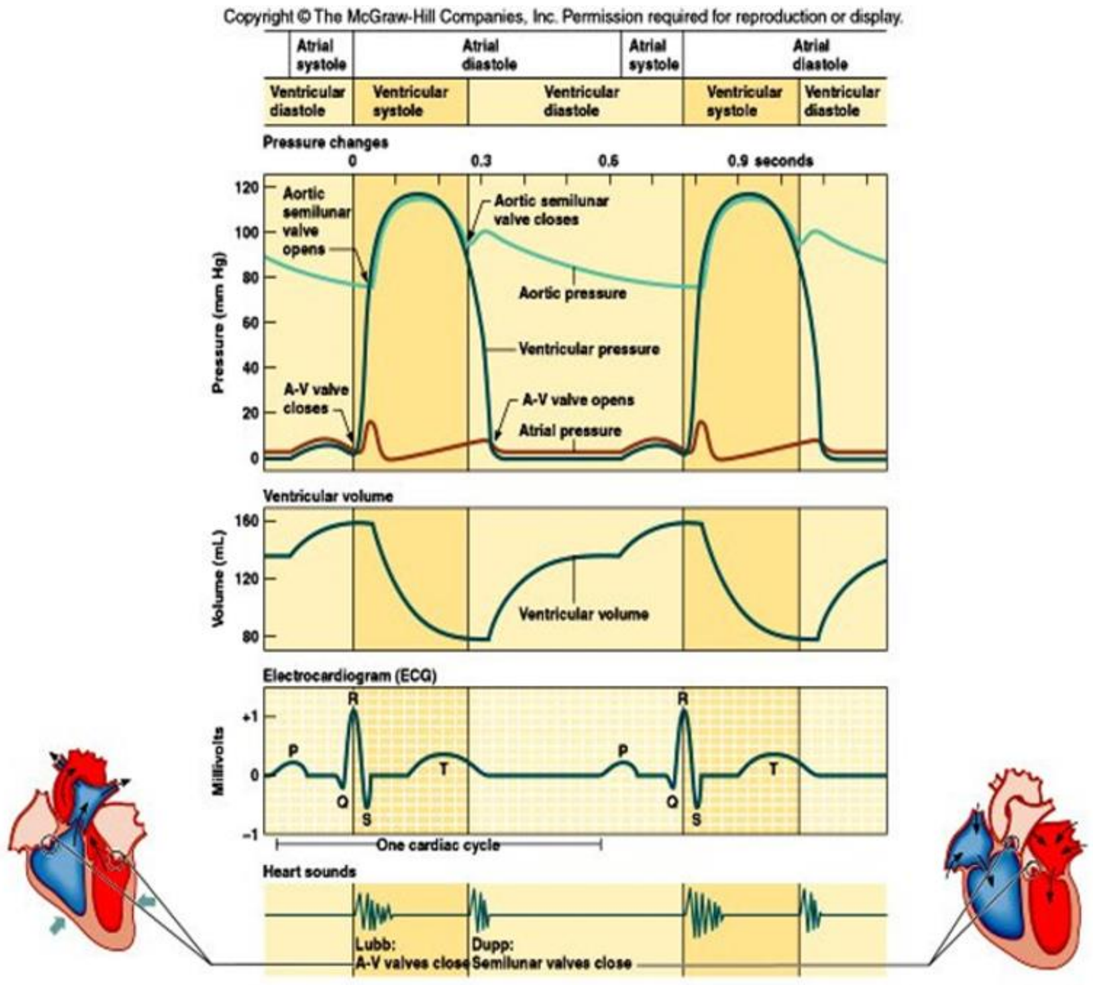
On the ventricular volume curve, the **horizontal segments (plateaus)** represent periods during which ventricular volume remains constant.

These periods correspond to two phases of the cardiac cycle:

- **Isovolumetric contraction:** Occurs at the beginning of ventricular systole.
- **Isovolumetric relaxation:** Occurs at the beginning of ventricular diastole, immediately after ventricular systole.

During isovolumetric relaxation, ventricular pressure progressively decreases as the ventricle relaxes.

This decrease continues until ventricular pressure becomes lower than atrial pressure, allowing the atrioventricular (AV) valve to open.



Atrial Pressure Changes During the Cardiac Cycle

The atrial pressure curve contains three waves: the **a wave, c wave, and v wave**.

1. The a Wave

The **a wave** results from atrial contraction.

2. The c Wave

The **c wave** occurs during ventricular contraction.

As ventricular pressure increases, the closed atrioventricular valve is pushed slightly toward the atrium, producing a rise in atrial pressure.

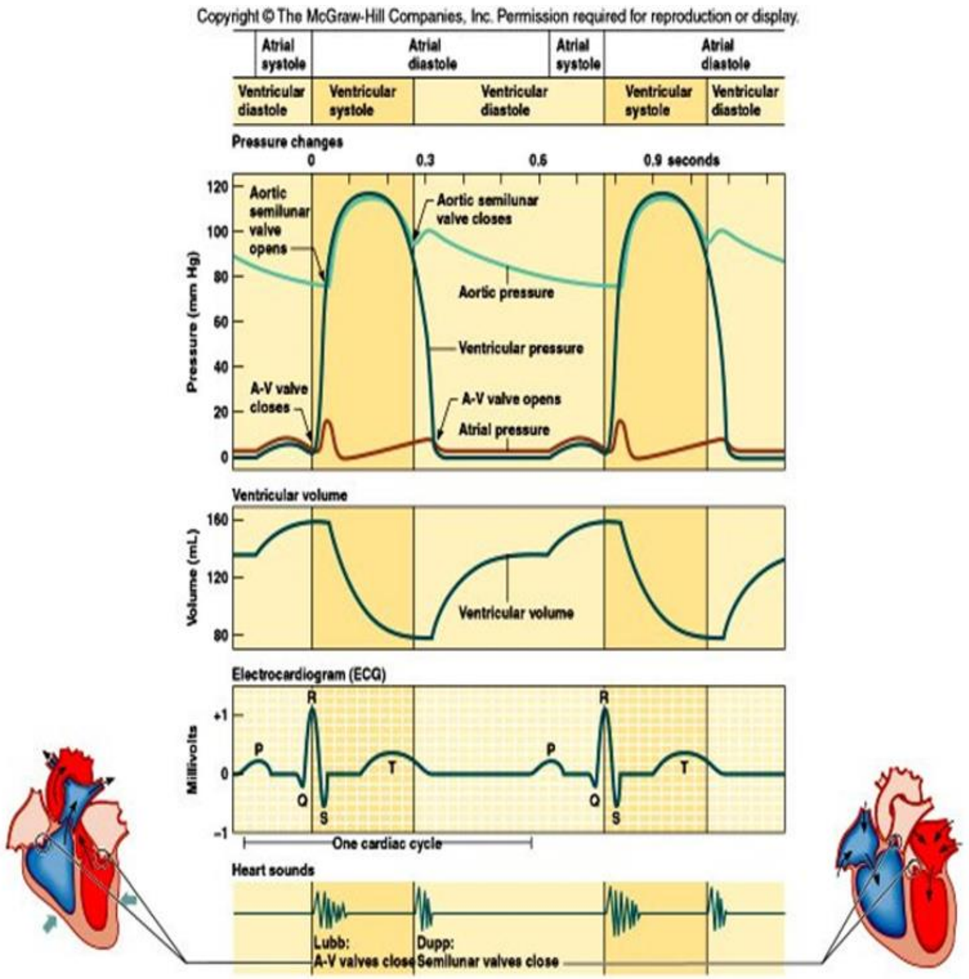
However, the valve is prevented from everting into the atrium by the **chordae tendineae and papillary muscles**, which maintain its position during ventricular contraction.

If the valve were to evert into the atrium, blood could flow backward from the ventricle into the atrium, resulting in **regurgitation**.

3. The v Wave

During ventricular systole, blood continues returning to the left atrium through the **four pulmonary veins**, even though the mitral valve is closed.

As blood accumulates in the left atrium, atrial pressure progressively increases, producing the **v wave**.



When ventricular relaxation causes ventricular pressure to fall below atrial pressure, the atrioventricular valve opens.
The accumulated blood then flows rapidly from the atrium into the ventricle, causing atrial pressure to decrease and the v wave to disappear.
Therefore, the three atrial pressure waves are associated with:

Wave	Associated event
a wave	Atrial contraction
c wave	Ventricular contraction and displacement of the closed AV valve toward the atrium
v wave	Accumulation of blood in the atrium during ventricular systole, followed by a fall in pressure when the AV valve opens

Phases of Ventricular Diastole

Ventricular diastole lasts approximately **0.5 seconds** and includes the following phases.

1. Isovolumetric Relaxation

Following closure of the semilunar valves, the ventricle begins to relax.

During this phase, ventricular pressure decreases while ventricular volume remains constant because the valves are closed.

The doctor mentioned that this phase lasts approximately **0.02 seconds**.

2. Rapid Ventricular Filling

When ventricular pressure falls below atrial pressure, the atrioventricular valve opens.

Blood that has accumulated in the atrium during ventricular systole then flows rapidly into the ventricle.

This produces the **rapid filling phase**, during which the atrium empties a substantial portion of its accumulated blood into the ventricle.

The opening of the AV valve is normally silent and does not produce an audible heart sound.

As blood leaves the atrium and enters the ventricle, atrial pressure decreases. In the doctor's example, the pressure falls from approximately **8 mmHg back to 5 mmHg**.

3. Reduced Ventricular Filling (Diastasis)

After rapid filling, ventricular filling continues at a slower rate.

This phase is known as **diastasis**, or reduced filling.

During diastasis, blood returning through the veins passes through the atria and continues into the ventricles.

The doctor emphasized the importance of this period because **coronary perfusion occurs during ventricular diastole**.

He also described the flow during this phase as decreasing considerably, potentially approaching zero for a short period.

4. Atrial Contraction

The final phase of ventricular diastole is **atrial contraction**, which completes ventricular filling before the next ventricular systole begins.

The doctor described atrial systole as lasting approximately **0.1 second**.

Relationship Between Atrial and Ventricular Systole and Diastole

The durations of atrial and ventricular activity are different.

Atrial diastole includes the entire period of ventricular systole and a substantial portion of ventricular diastole.

In contrast, atrial systole occurs during the **final portion of ventricular diastole**.

Therefore, atrial contraction contributes to the completion of ventricular filling immediately before ventricular contraction begins.

Cardiac event	Duration
Atrial systole	0.1 seconds
Atrial diastole	0.7 seconds
Ventricular systole	0.3 seconds
Ventricular diastole	0.5 seconds

"وتَبكي بُكاء المُضطر و تَنـامّ
والله لا ينامُ عن تدبير أمرك."

Ventricular Pressure-Volume Loop

The **ventricular pressure-volume loop** represents the changes in ventricular pressure and volume throughout the cardiac cycle.

It is constructed using two axes:

- **X-axis:** Ventricular volume (mL).
- **Y-axis:** Ventricular pressure (mmHg).

The doctor explained the pressure-volume loop using four points: **A, B, C, and D**.

In this example:

- Point A represents an end-systolic volume of approximately **80 mL**.
- Point B represents an end-diastolic volume of approximately **160 mL**.

The loop also relates to two curves:

- **Diastolic function curve:** Represents the ventricular filling relationship.
- **Systolic function curve:** Represents ventricular systolic performance.

1. A → B: Ventricular Filling

Point A represents the **end-systolic volume (ESV)**.

V_A=80mL

During ventricular diastole, blood enters the ventricle, increasing ventricular volume until it reaches point B.

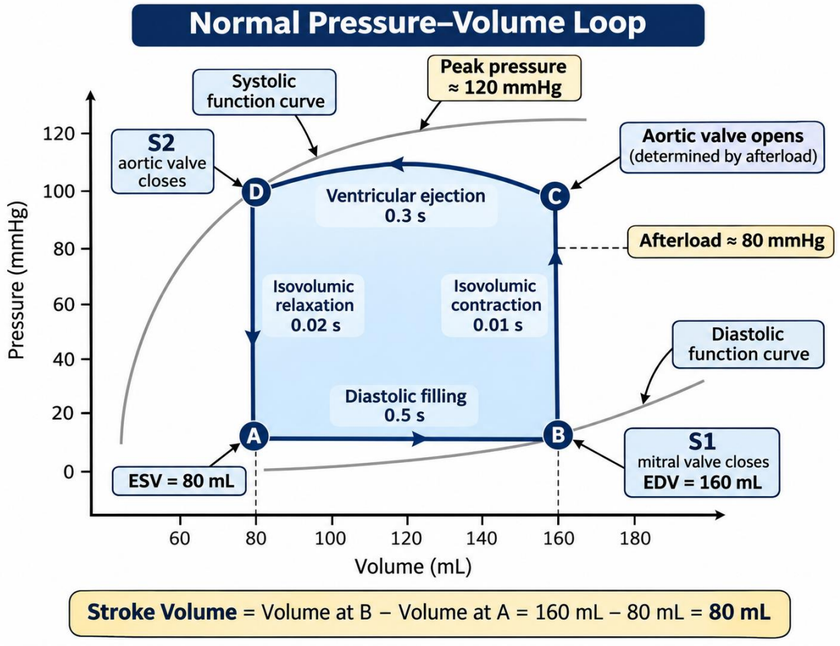
Point B represents the **end-diastolic volume (EDV)**.

V_B=160mL

The segment A → B follows the diastolic function curve and represents ventricular filling.

At point B, ventricular filling has ended, and the ventricle begins to contract.

As ventricular pressure increases above atrial pressure, the **mitral valve closes**, producing the first heart sound (S1).



2. B → C: Isovolumetric Contraction

After mitral valve closure, ventricular contraction continues.

Because both the mitral and aortic valves remain closed, ventricular pressure increases without a change in ventricular volume.

Therefore, the segment B → C represents **isovolumetric contraction**.

The doctor mentioned an approximate duration of **0.01 second** for this phase.

Point C is determined by **afterload**.

For example, if aortic pressure is 80 mmHg, ventricular pressure must rise sufficiently to overcome 80 mmHg before the aortic valve opens.

If the afterload is 90 or 100 mmHg, ventricular pressure must increase accordingly before the valve can open.

At point C, the **aortic semilunar valve opens**.

This opening is normally silent.

3. C → D: Ventricular Ejection

Once the aortic valve opens at point C, blood begins flowing from the left ventricle into the aorta.

During this phase, ventricular volume decreases as blood is ejected.

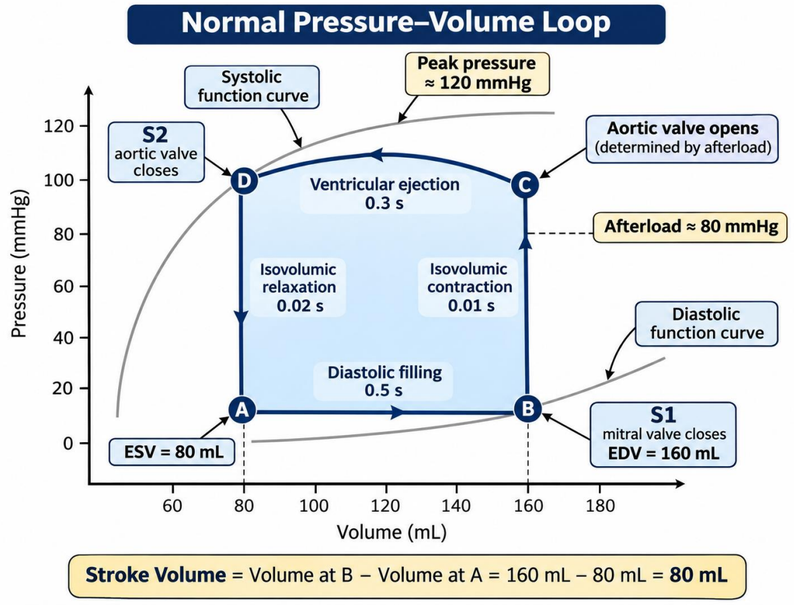
Ventricular pressure rises toward approximately **120 mmHg** during systole.

The doctor related point D to the **systolic function curve**, which represents the mechanical performance of the ventricle during contraction.

The segment C → D represents ventricular ejection.

At point D, the aortic valve closes, producing the **second heart sound (S2)**.

The doctor referred to approximately **0.3 seconds** when discussing the duration of ventricular systole.



4. D → A: Isovolumetric Relaxation

Following aortic valve closure at point D, the ventricle begins to relax.

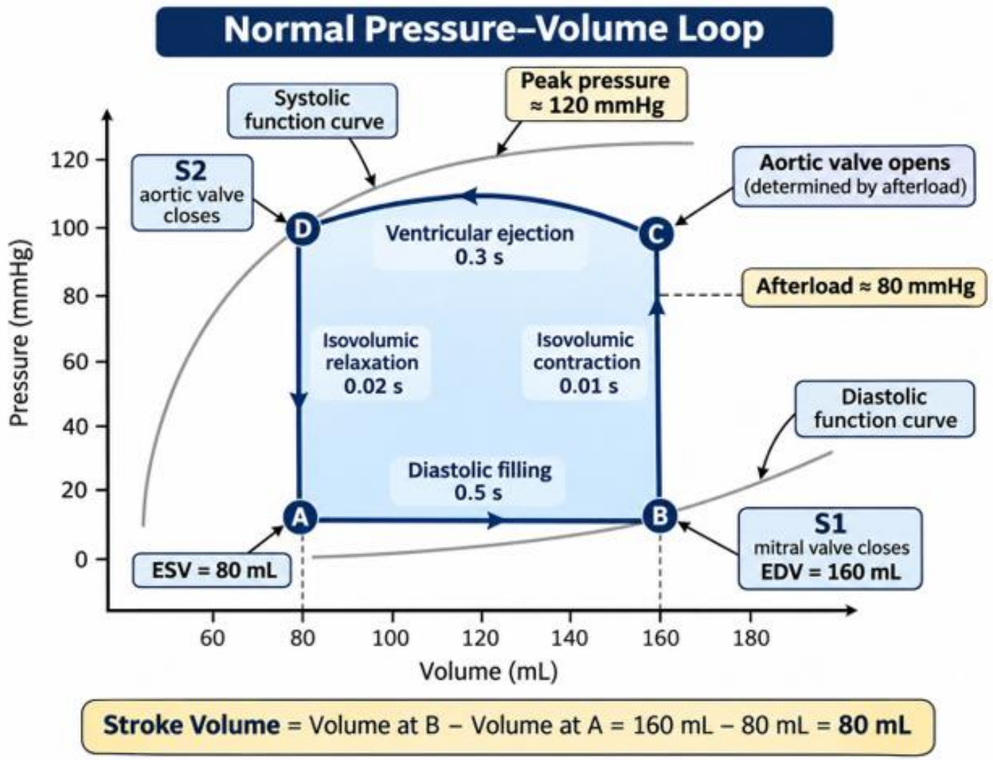
Ventricular pressure decreases, but ventricular volume remains constant because the valves are closed.

Therefore, the segment D → A represents **isovolumetric relaxation**.

The doctor mentioned that the interval from S2 to point A lasts approximately **0.02 seconds**.

When ventricular pressure falls below atrial pressure, the mitral valve opens at point A, allowing ventricular filling to begin again.

This completes the pressure-volume loop



Stroke Volume from the Pressure-Volume Loop

Stroke volume is calculated by subtracting the ventricular volume at point A from the ventricular volume at point B.

SV=VB-VA

Using the doctor's example:

SV=160-80

SV=80mL

Therefore, the horizontal difference between the end-diastolic and end-systolic volumes represents the stroke volume

Important Examination Questions on the Pressure-Volume Loop

Question	Answer
What does the X-axis represent?	Ventricular volume
What does the Y-axis represent?	Ventricular pressure
What does point A represent?	End-systolic volume; mitral valve opens
What does point B represent?	End-diastolic volume; mitral valve closes (S1)
What happens at point C?	Aortic semilunar valve opens
What happens at point D?	Aortic semilunar valve closes (S2)
What does A → B represent?	Ventricular filling
What does B → C represent?	Isovolumetric contraction
What does C → D represent?	Ventricular ejection
What does D → A represent?	Isovolumetric relaxation
What determines point C?	Afterload
What is the relationship of point D to cardiac function?	It is associated with the systolic function curve
How is stroke volume calculated?	Volume at B – Volume at A

The doctor also mentioned the approximate durations of **0.01 second for isovolumetric contraction**, **0.02 seconds for isovolumetric relaxation**, **0.3 seconds for ventricular systole**, and **0.5 seconds for ventricular diastole**.
He emphasized that although the pressure-volume loop does not directly show time on its axes, students must understand the relationship between each segment and the corresponding phase of the cardiac cycle.

Cardiac Cycle

- Cardiac cycle refers to all events associated with blood flow through the heart
  - –Systole – contraction of heart muscle
  - –Diastole – relaxation of heart muscle

Cardiac Cycle

- Atrial systole 0.1 second
- Atrial diastole 0.7 second **the longest**
- Ventricular systole 0.3 second
  - Isovolumic contraction 0.01 seconds
  - Rapid ejection period
  - Slow ejection period
- Ventricular diastole 0.5 seconds
  - Isovolumic relaxation 0.02 seconds
  - Rapid filling
  - Slow filling (Diastasis)
  - Atrial contraction

During **tachycardia**, the duration of ventricular diastole decreases, resulting in a shortening of the **diastasis phase**.
As diastasis becomes shorter, the time available for coronary perfusion is reduced, which may compromise myocardial blood supply.

Cardiac cycle …cont

- End diastolic volume (EDV) – End systolic volume (ESV) = Stroke volume (SV)
- SV X heart rate (HR) = cardiac output (CO)
- Ejection fraction = SV/EDV
- Inotropic vs. Chronotropic
- Autonomic control of cardiac cycle (pump)

Effect of Increased Afterload on Stroke Volume

The doctor used **hypertension** as an example of increased afterload.

When arterial pressure increases, the ventricle must generate greater pressure to open the aortic valve and eject blood.

For example, if aortic pressure increases from 80 mmHg to 100 mmHg, the ventricle must overcome the higher pressure before ejection can begin.

As afterload increases, ventricular ejection becomes more difficult.

Consequently, **stroke volume decreases**.

The doctor emphasized this relationship:

$$\uparrow \text{Afterload} \longrightarrow \downarrow \text{Stroke Volume}$$

Effect of Sympathetic Stimulation on Ventricular Contractility

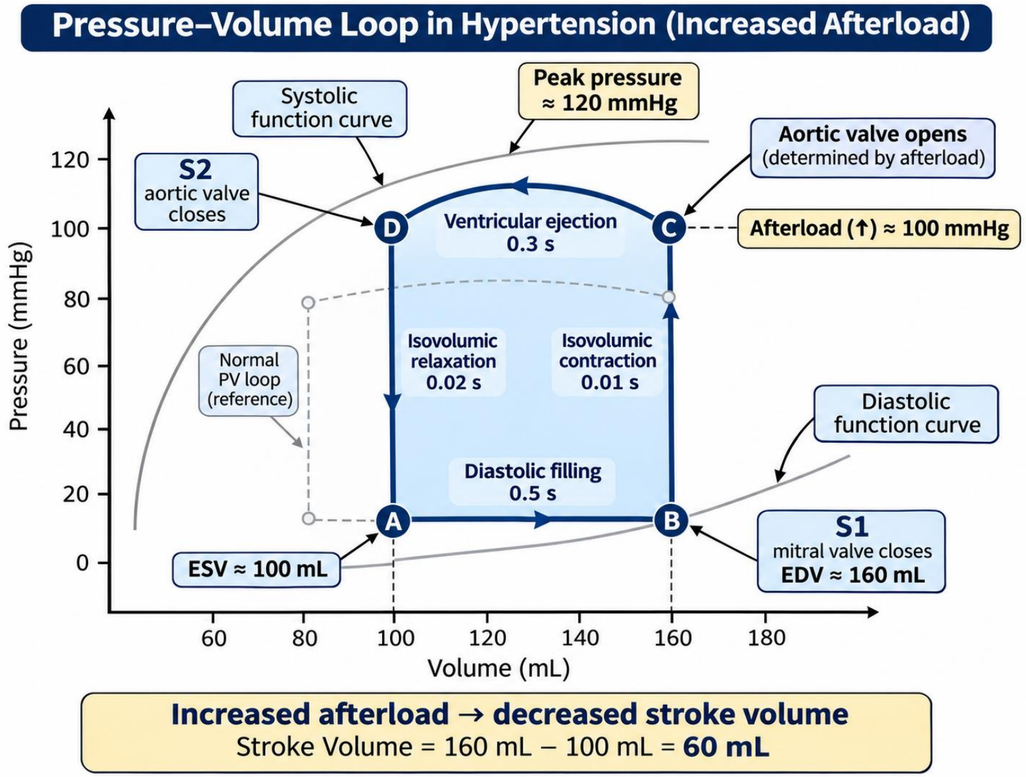
The doctor explained that sympathetic stimulation produces a **positive inotropic effect**, increasing the force of ventricular contraction.

When contractility increases, the systolic function curve shifts **upward and to the left**.

This allows the ventricle to generate greater contractile force and improve its ability to eject blood.

In the example of increased afterload, sympathetic stimulation can increase ventricular contractility to help maintain the stroke volume.

Therefore, the doctor emphasized that increased sympathetic stimulation can compensate for the greater load imposed on the ventricle.



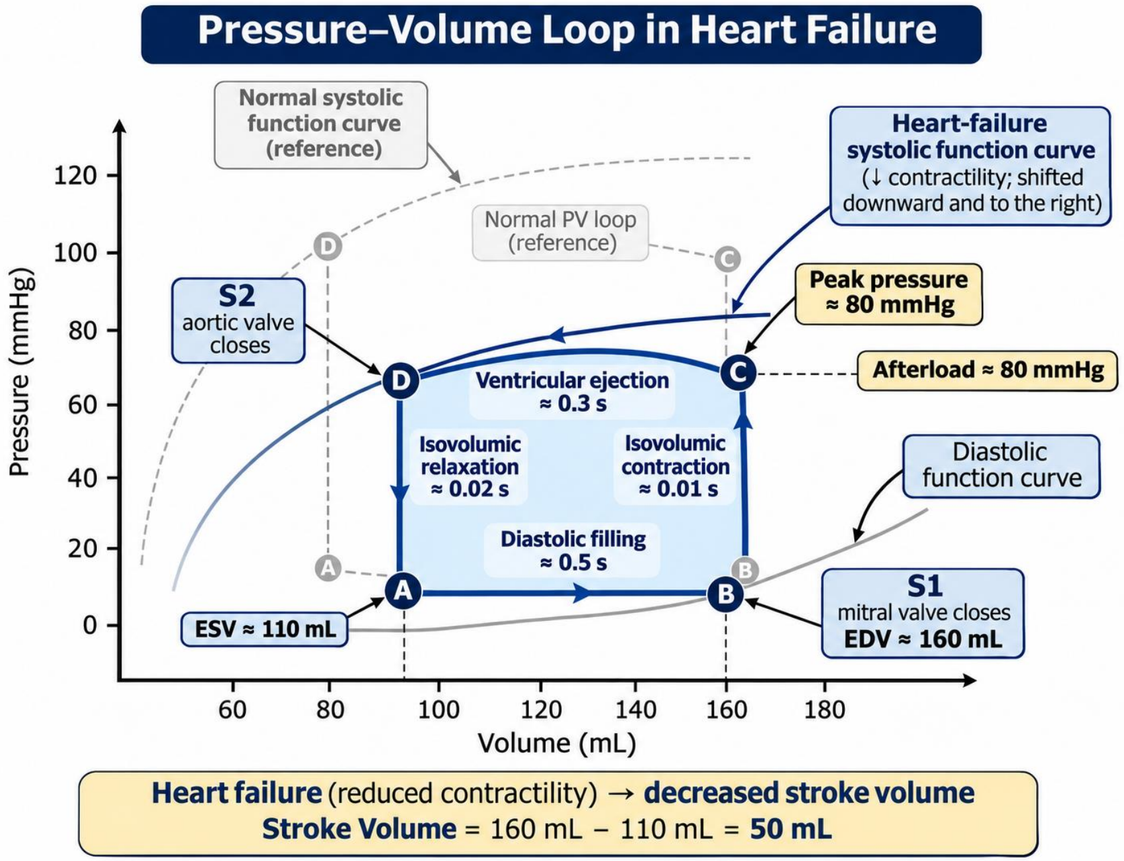
Effect of Heart Failure on the Pressure-Volume Relationship

In heart failure associated with reduced myocardial contractility, the heart generates less contractile force.

The systolic function curve shifts **downward and to the right**.

This represents reduced ventricular pumping performance.

The doctor contrasted this with increased sympathetic stimulation, in which the systolic function curve shifts upward and to the left because of increased contractility.



Definition of Heart Failure

Heart failure is a condition in which the heart is unable to supply the tissues with sufficient oxygen to meet their needs.

The doctor explained that heart failure can result from two major types of cardiac dysfunction:

1. **Systolic dysfunction (pump failure).**
2. **Diastolic dysfunction (impaired ventricular filling).**

Systolic Heart Failure – Pump Failure

Systolic heart failure occurs when the heart has difficulty contracting effectively and ejecting blood into the circulation.

The primary problem is therefore related to **ventricular systole**, during which the heart functions as a pump.

Because ventricular contraction is impaired, the heart cannot eject an adequate amount of blood.

This type is known as **heart failure with reduced ejection fraction (HFrEF)**.

The doctor mentioned ischemic heart disease and carditis as examples of conditions associated with impaired cardiac pumping function.

Diastolic Heart Failure – Impaired Ventricular Filling

In diastolic heart failure, the primary problem is not necessarily a reduction in the contractile ability of the cardiac muscle.

Instead, the ventricular wall may become **thicker, enlarged, or stiffer**, reducing its ability to stretch and accommodate blood during diastole.

This is described as **decreased ventricular compliance**.

As a result, the ventricle cannot fill normally because it does not relax or expand adequately.

Therefore, the main problem occurs during **ventricular diastole**, rather than during systolic contraction.

Effect on Ventricular Volume and Ejection Fraction

The doctor illustrated this concept by comparing a ventricle that normally fills with approximately 120 mL of blood to a less compliant ventricle that can accommodate only approximately 100 mL.

Despite the reduction in ventricular filling, the heart may still eject approximately 50–60 mL during contraction.

Consequently, the **ejection fraction may remain normal even though the patient has heart failure**.

This condition is known as **heart failure with preserved ejection fraction (HFpEF)**.

Clinical Assessment

The doctor explained that patients with this type of heart failure may present with the heart failure symptoms discussed previously.

On echocardiography, structural changes such as **cardiomegaly or ventricular hypertrophy** may be observed.

These findings were used to illustrate the impaired ability of the ventricle to receive blood during diastole.

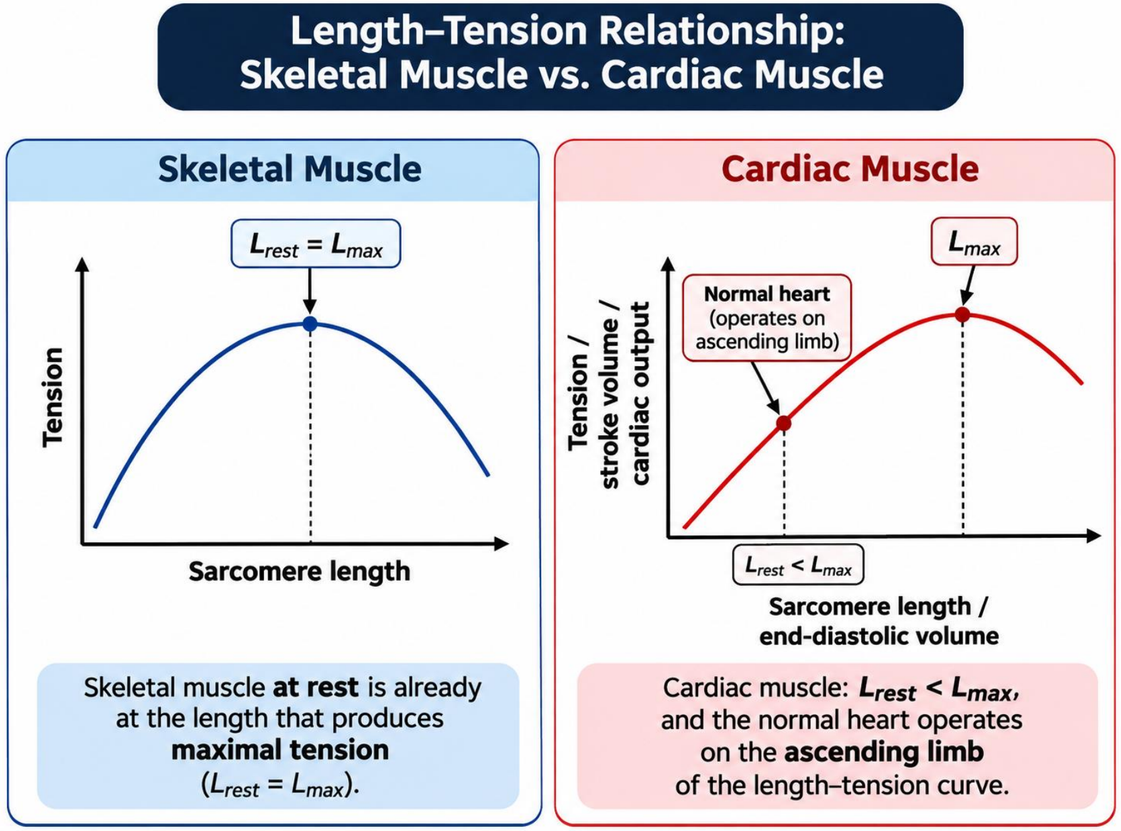
Type	Ejection fraction described by the doctor	Main dysfunction
Heart failure with reduced ejection fraction (HFrEF)	Less than 40%	Impaired ventricular contraction
Heart failure with preserved ejection fraction (HFpEF)	More than 40%	Impaired ventricular filling

The key distinction is that **systolic heart failure involves impaired pumping**, whereas **diastolic heart failure involves impaired filling despite a potentially normal ejection fraction**.

Most clinical studies investigating heart failure and its pharmacological management have focused on **heart failure with reduced ejection fraction**.
Although studies have also investigated heart failure with preserved ejection fraction, the available research has historically been less extensive.
The effectiveness of certain medications in preserved ejection fraction has therefore been a subject of investigation and discussion.
However, **some medications may be suitable for both types of heart failure**.

In **skeletal muscle**, the maximum tension is produced at **Lmax**.
In skeletal muscle, **Lmax is equal to Lrest**.
This means that when skeletal muscles are at rest, such as the muscles of the hands and legs, they are already at the length at which they can produce their maximum response.
Therefore, if a person suddenly needs to exert force, the skeletal muscles are already prepared to generate maximum tension.
In the **heart**, however, **Lrest is less than Lmax**.
This means that, at rest, cardiac muscle is not operating at the sarcomere length that gives maximum tension.

L corresponds to **end-diastolic volume**.
Therefore, increasing the length of the sarcomere means increasing ventricular filling.
As ventricular filling increases, the sarcomere length increases, and the heart can generate a greater response.



Heart failure with reduced ejection fraction.
If a person has a weak heart, for example because of ischemia, smoking, or another cause of cardiac weakness, the heart becomes less able to pump effectively.
In a normal person, the ventricle may contain **120 mL**, pump **70 mL**, and leave **50 mL**, giving an ejection fraction of about **55%**.
The doctor also mentioned that in adults the ejection fraction may be around **65%**.
In a person with a weaker heart, the heart tries to compensate by **increasing the sarcomere length**, which means increasing the **end-diastolic volume**.
So, instead of ejecting 70 mL from 120 mL, the heart may eject:

- **70 mL from 150 mL**, giving an ejection fraction of about **45%**.
- If the heart becomes weaker, it may eject **70 mL from 170 mL**, giving an ejection fraction of about **35%**.

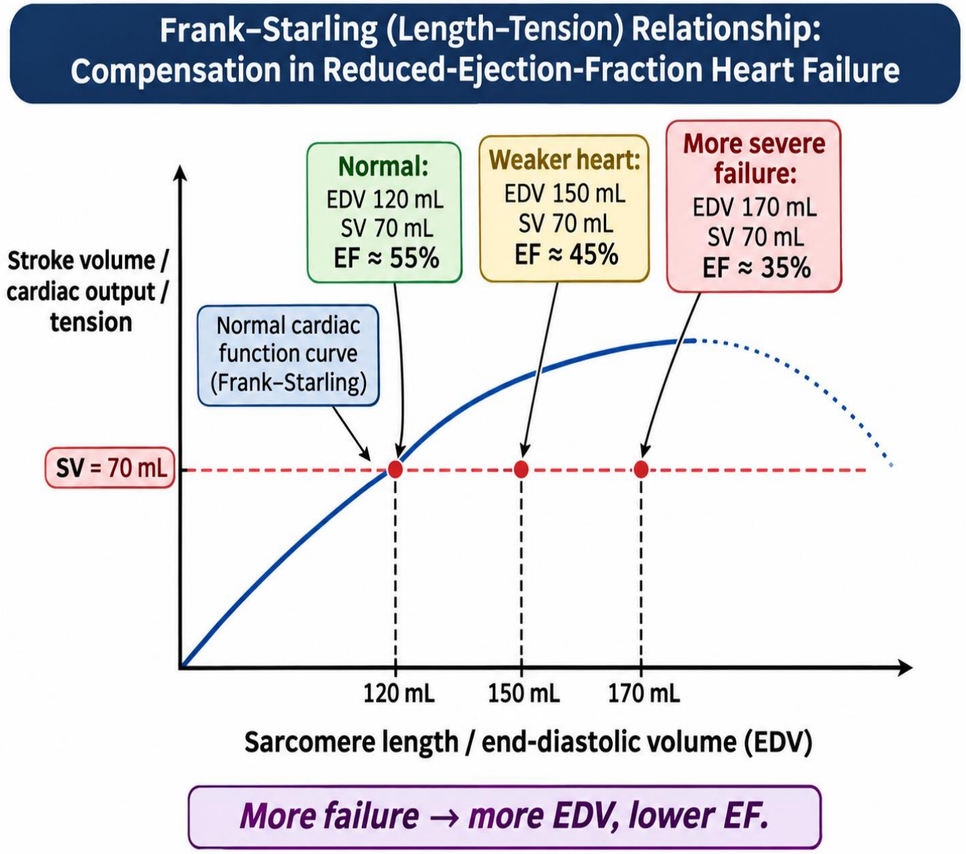
Thus, as the heart becomes weaker, it tries to maintain stroke volume by increasing end-diastolic volume.

5. Relationship Between Heart Failure and Ejection Fraction

The doctor's main idea is that, in **reduced ejection fraction heart failure**, the heart compensates by increasing sarcomere length and end-diastolic volume.
Therefore:

- The more severe the failure,
- the greater the end-diastolic volume,
- and the lower the ejection fraction.

So, in this type of heart failure, **ejection fraction reflects the condition of the heart and indicates what is happening in the patient.**



Third Heart Sound (S3)

The **third heart sound (S3)** occurs shortly after the second heart sound (S2), during the **rapid ventricular filling phase** of early diastole.

After the atrioventricular (AV) valves open, blood flows rapidly from the atria into the ventricles. This rapid filling produces vibrations as blood enters the ventricles and interacts with the ventricular walls, generating S3.

When S3 is audible, three heart sounds can be heard during the cardiac cycle: **S1, S2, and S3**.

Clinical Significance of S3

The third heart sound may be associated with conditions such as:

- Atrial enlargement.
- Mitral regurgitation.
- Heart failure.

However, **S3 may be normal in children** because their hearts are relatively hyperdynamic.

In adults, particularly older individuals, the presence of S3 may indicate an abnormal cardiac condition, including heart failure.

Gallop Rhythm

When S3 is heard in addition to S1 and S2, the resulting three-sound rhythm may be described as a **gallop rhythm**.

The term *gallop* refers to the resemblance between the rhythm of the heart sounds and the sound of a galloping horse.

The doctor emphasized that this rhythm may be physiological in children but can be pathological in adults.

2. Fourth Heart Sound (S4)

The **fourth heart sound (S4)** occurs immediately before the first heart sound (S1), near the end of ventricular diastole.

It is associated with **atrial contraction**.

Unlike S3, which occurs during rapid ventricular filling, S4 occurs during the final phase of ventricular filling when the atria contract.

The doctor described S4 as being associated with pathological conditions and emphasized that it is generally not audible during routine auscultation.

However, it may be detected and recorded using a **phonocardiogram**.

Dicrotic notch (incisura): A brief change in aortic pressure associated with aortic valve closure and elastic recoil of the aorta.

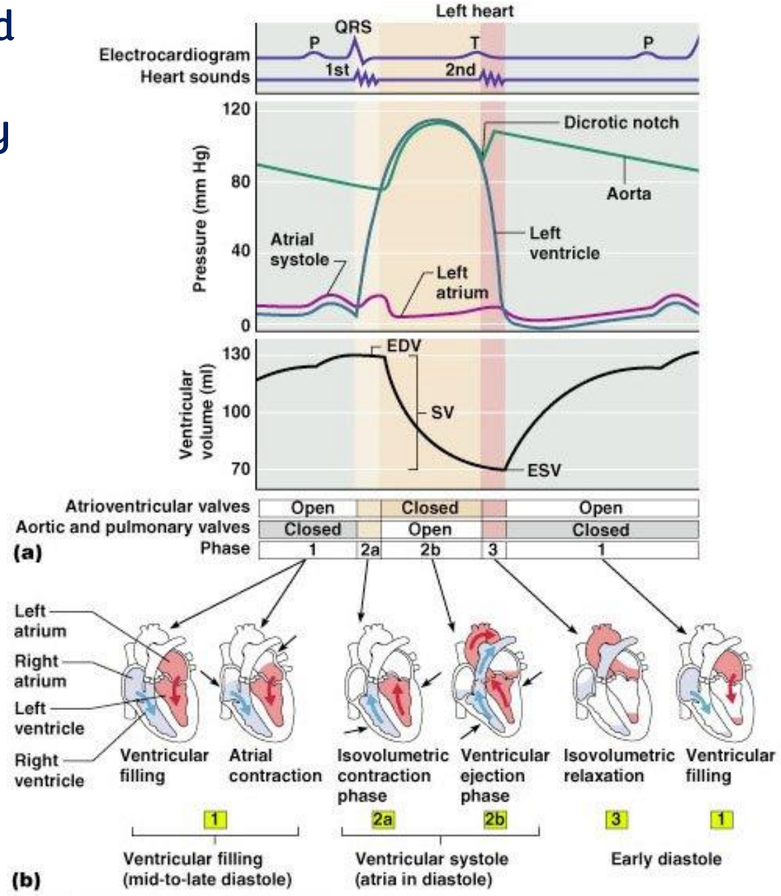
At the beginning of ventricular systole, aortic pressure is approximately **80 mmHg**.
During **isovolumetric contraction**, left ventricular pressure rises rapidly while the aortic valve remains closed.
When ventricular pressure exceeds aortic pressure, the aortic valve opens, allowing blood to enter the aorta.
As ventricular ejection continues, aortic pressure increases until it reaches approximately **120 mmHg**. It then begins to decrease toward the end of ventricular systole.
During **isovolumetric relaxation**, ventricular pressure decreases rapidly after the aortic valve closes.

2. Dicrotic Notch (Incisura)

The **dicrotic notch**, also called the *incisura*, is a small, brief change in the aortic pressure curve associated with closure of the aortic semilunar valve.

Mechanism of Formation

During ventricular ejection, the aorta dilates as it receives blood from the left ventricle.
When ventricular pressure decreases and the aortic valve closes, the elastic aortic wall tends to return to its original shape through **elastic recoil**.
This recoil compresses the blood within the aorta and promotes its movement.
Because the aortic valve is closed, blood cannot return freely into the left ventricle.
Consequently, blood is directed forward through the aorta.
The doctor explained that this process produces a brief increase in aortic pressure, forming the **dicrotic notch**.



Site of Recording

The doctor emphasized that the dicrotic notch is most clearly recorded near the **aortic valve**.
He explained that, as the recording site moves farther away toward the aortic arch or descending aorta, the notch may become less apparent.
Therefore, the location of the pressure recording influences the visibility of the dicrotic notch.

Phases of the Cardiac Cycle

- Ventricular filling – mid-to-late diastole
  - Heart blood pressure is low as blood enters atria and flows into ventricles
  - AV valves are open, then atrial systole occurs

Phases of the Cardiac Cycle

- Ventricular systole
  - Atria relax
  - Rising ventricular pressure results in closing of AV valves
  - Isovolumetric contraction phase
  - Ventricular ejection phase opens semilunar valves

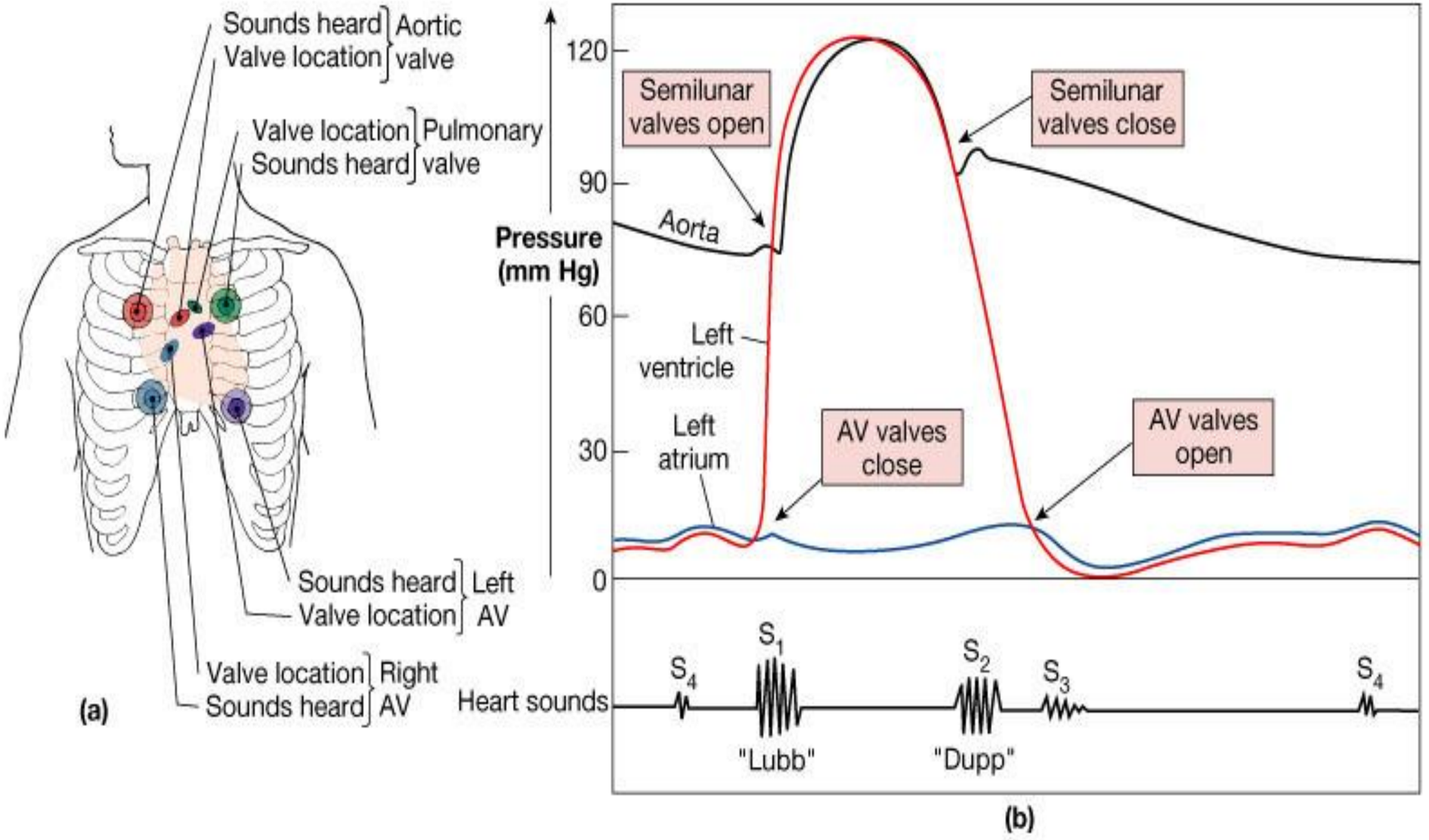
Phases of the Cardiac Cycle

- Isovolumetric relaxation – early diastole
  - Ventricles relax
  - Backflow of blood in aorta and pulmonary trunk closes semilunar valves
- Dicrotic notch – brief rise in aortic pressure caused by backflow of blood rebounding off semilunar valves

Changes during Cardiac cycle

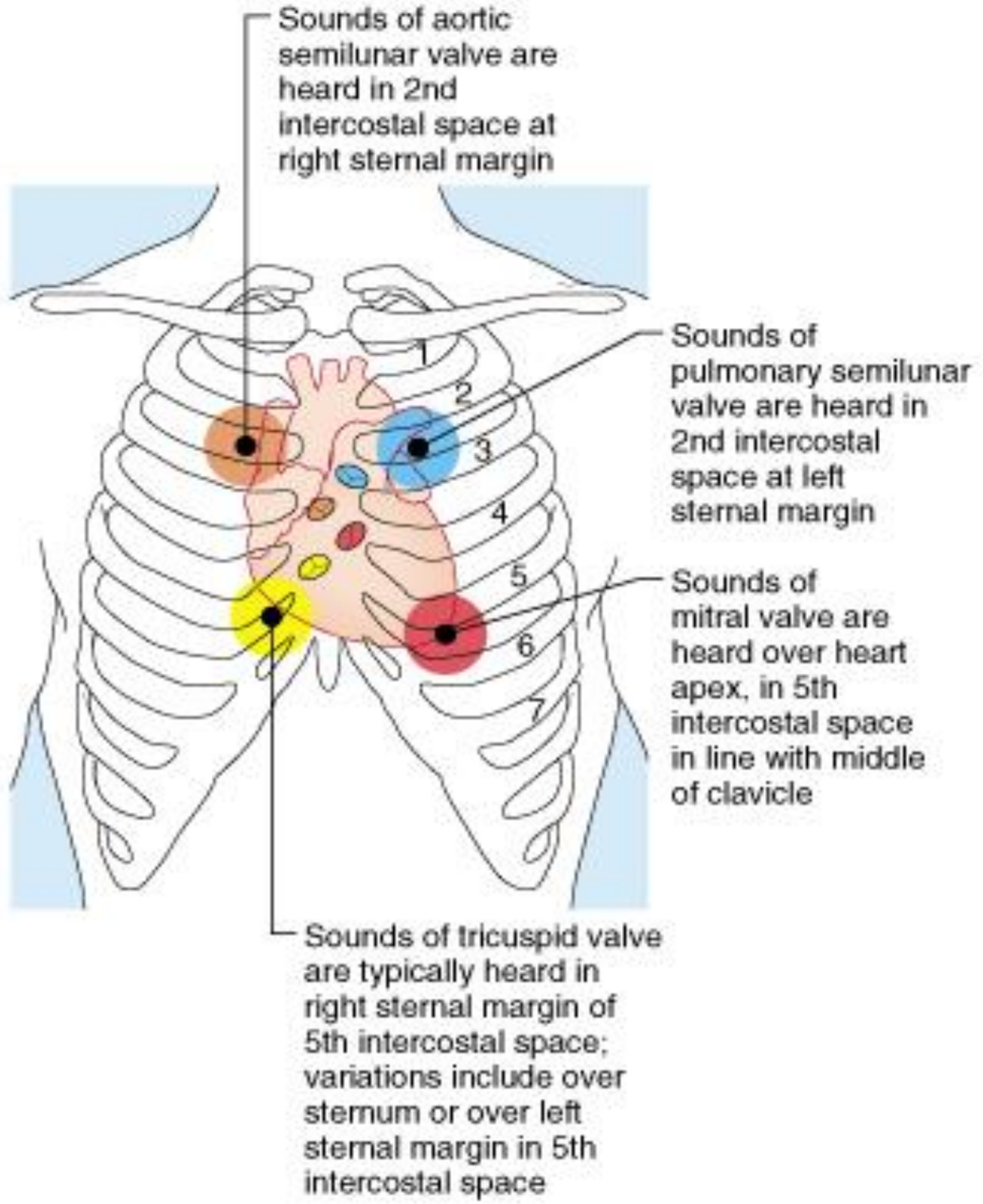
- Volume changes: End-diastolic volume, End-systolic volume, Stroke volume and Cardiac output.
- Aortic pressure: Diastolic pressure ~80 mmHg, Systolic pressure ~ 120 mmHg, most of systole ventricular pressure higher than aortic
- Ventricular pressure: Diastolic ~ 0, systolic Lt. ~120 Rt. ~ 25 mmHg.
- Atrial pressure: A wave =atrial systole, C wave= ventricular contraction (AV closure), V wave= ventricular diastole (Av opening)
- Heart sounds: S_1 = turbulence of blood around a closed AV valves, S_2 = turbulence of blood around a closed semilunar valves.

Heart Sounds



Heart Sounds

- Heart sounds (lub-dup) are associated with closing of heart valves



Heart sounds

- Auscultation – listening to heart sound via stethoscope
- Four heart sounds
  - S_1 – “lubb” caused by the closing of the AV valves
  - S_2 – “dupp” caused by the closing of the semilunar valves
  - S_3 – a faint sound associated with blood flowing into the ventricles
  - S_4 – another faint sound associated with atrial contraction

Cardiac Output (CO) and Reserve

- CO is the amount of blood pumped by each ventricle in one minute
- CO is the product of heart rate (HR) and stroke volume (SV)
- HR is the number of heart beats per minute
- SV is the amount of blood pumped out by a ventricle with each beat
- Cardiac reserve is the difference between resting and maximal CO

Cardiac Output: Example

- CO (ml/min) = HR (75 beats/min) x SV (70 ml/beat)
- CO = 5250 ml/min (5.25 L/min)

Regulation of Stroke Volume

- SV = end diastolic volume (EDV) minus end systolic volume (ESV)
- EDV = amount of blood collected in a ventricle during diastole
- ESV = amount of blood remaining in a ventricle after contraction

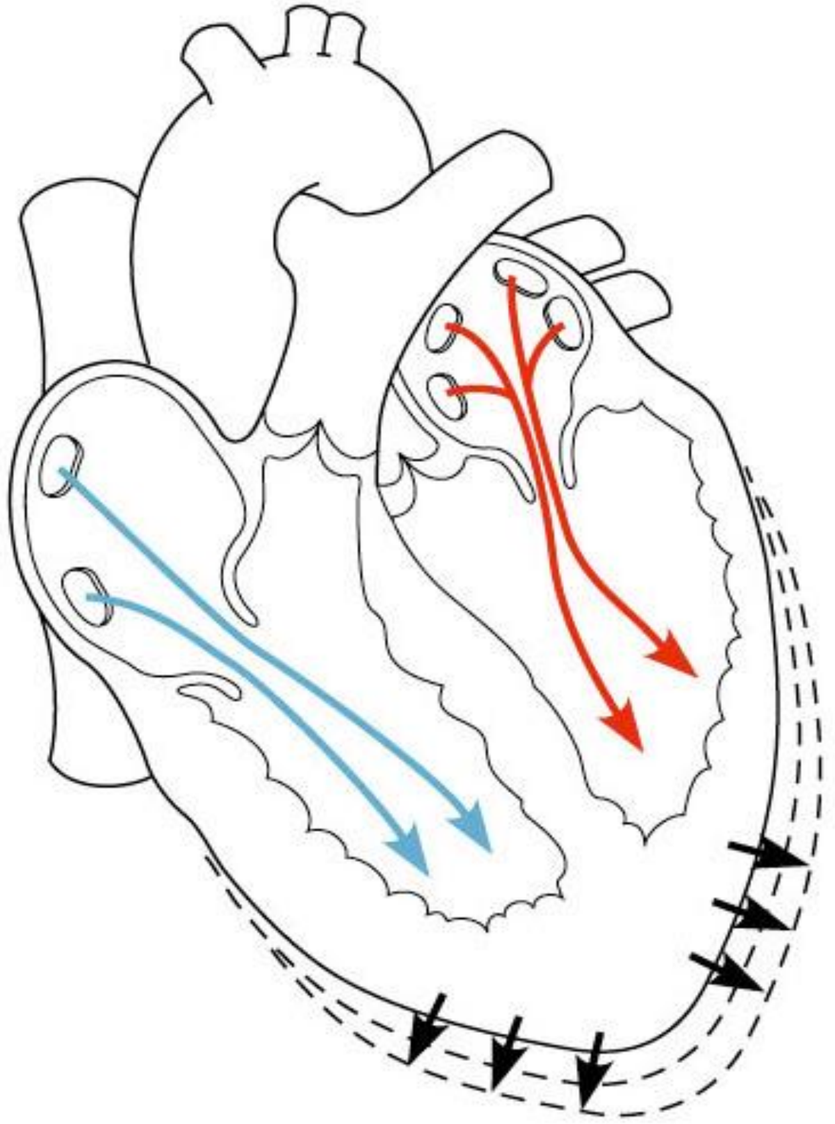
Factors Affecting Stroke Volume

- Preload – amount ventricles are stretched by contained blood
- Contractility – cardiac cell contractile force due to factors other than EDV
- Afterload – back pressure exerted by blood in the large arteries leaving the heart

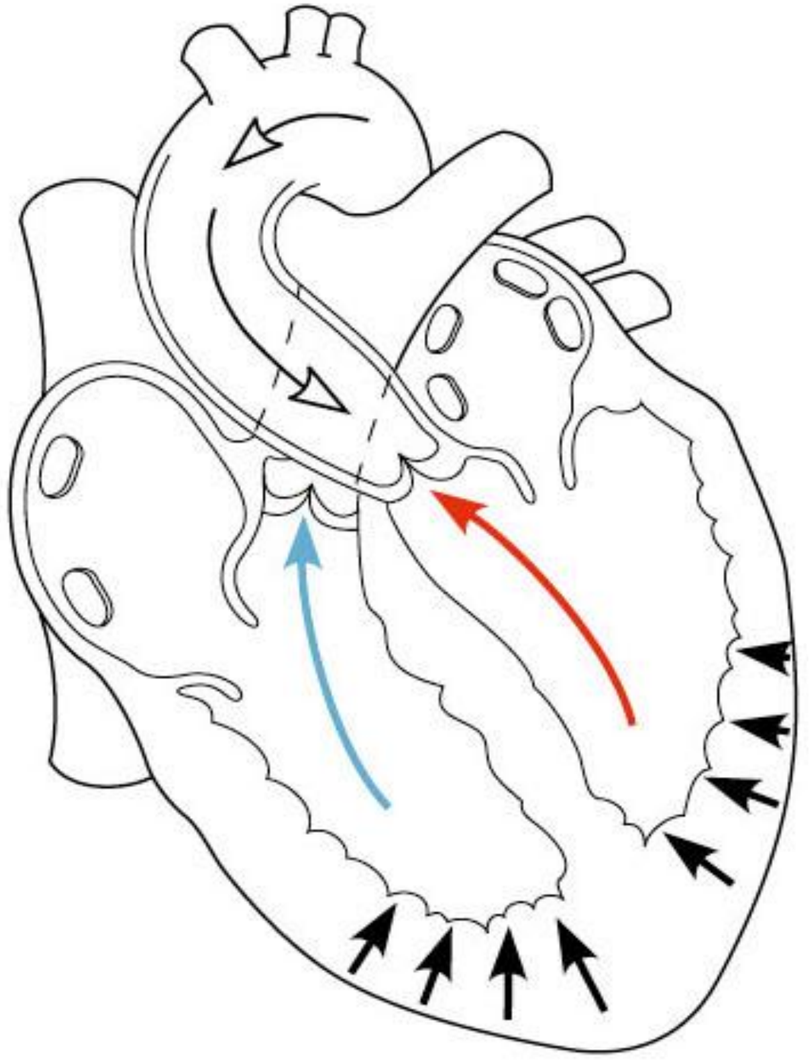
Frank-Starling Law of the Heart

- Preload, or degree of stretch, of cardiac muscle cells before they contract is the critical factor controlling stroke volume
- Slow heartbeat and exercise increase venous return to the heart, increasing SV
- Blood loss and extremely rapid heartbeat decrease SV

Preload

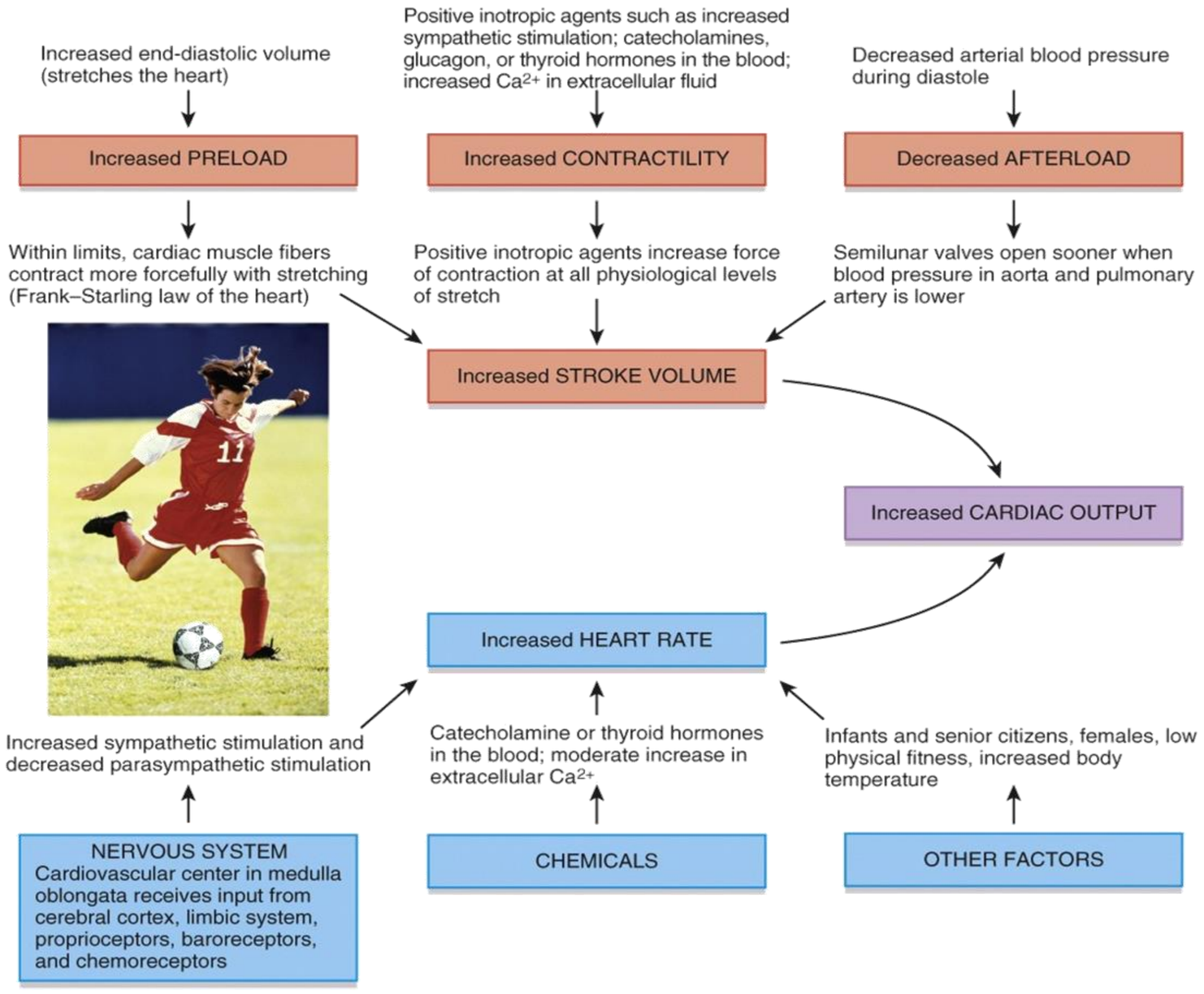


(a) Preload



(b) Afterload

Cardiac Output



Extrinsic Factors Influencing Stroke Volume

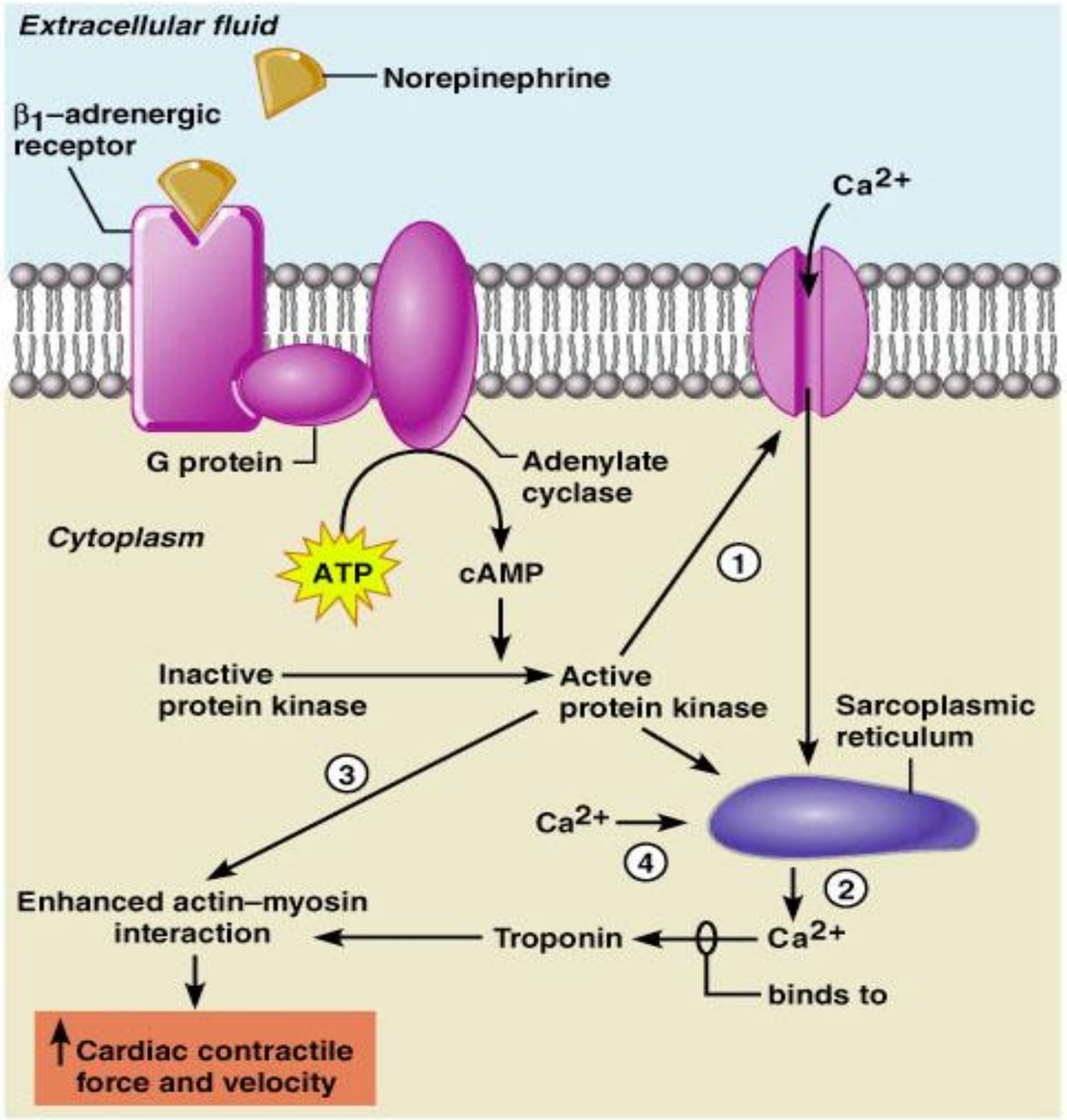
- Contractility is the increase in contractile strength, independent of stretch and EDV
- Increase in contractility comes from:
  - Increased sympathetic stimuli
  - Certain hormones
  - Ca^{2+} and some drugs

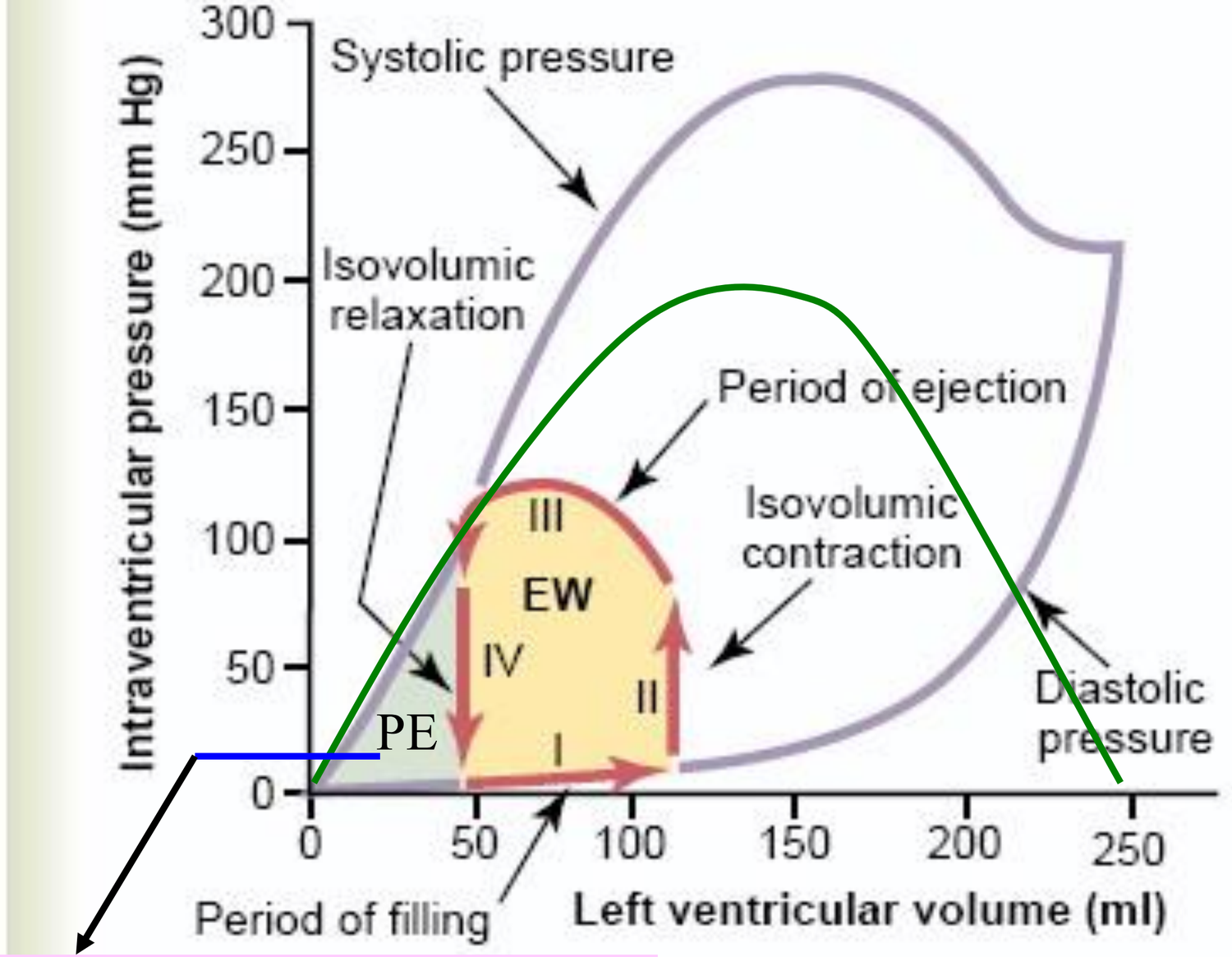
Extrinsic Factors Influencing Stroke Volume

- Agents/factors that decrease contractility include:
  - Acidosis
  - Increased extracellular K^+
  - Calcium channel blockers

Contractility and Norepinephrine

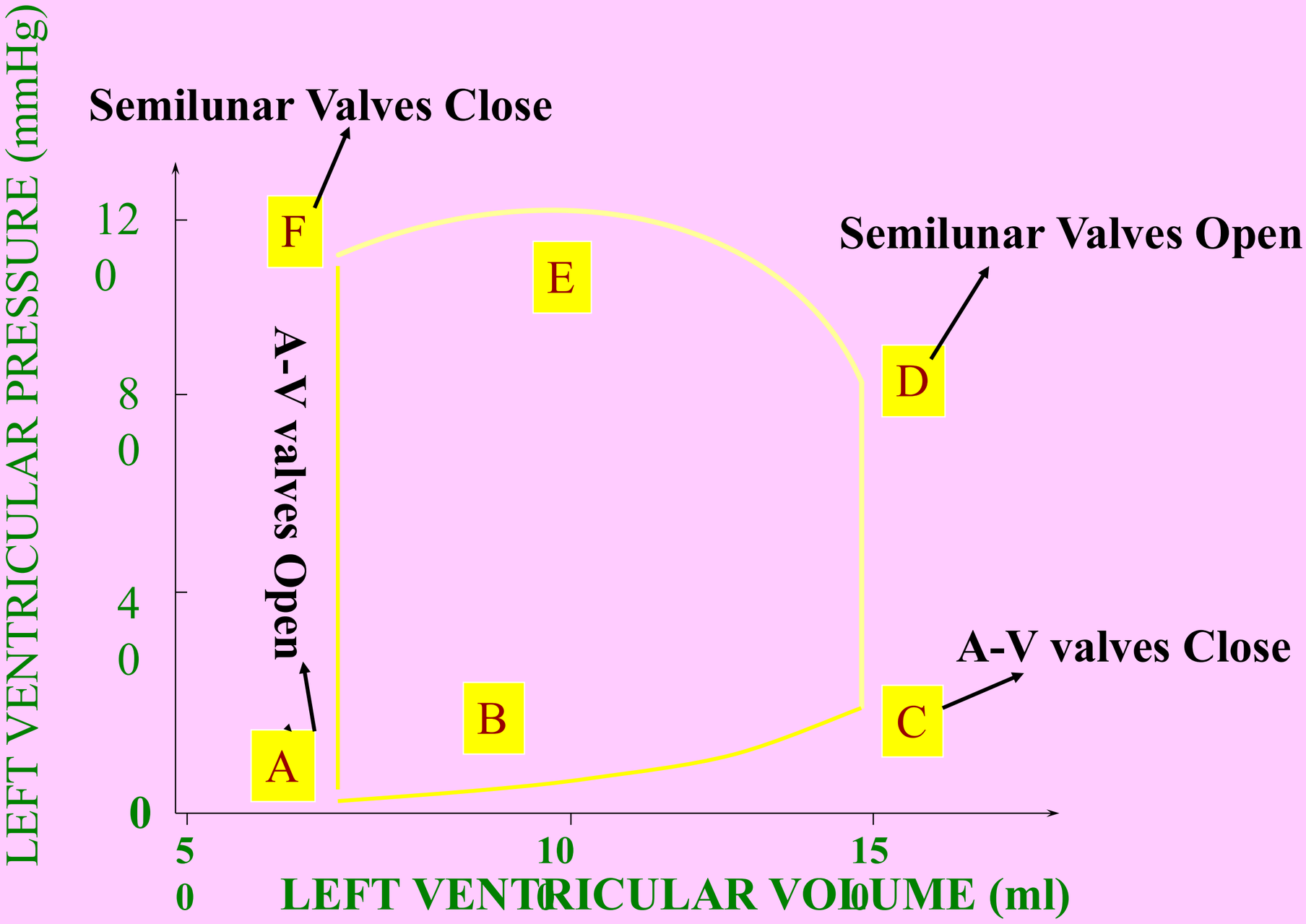
- Sympathetic stimulation releases norepinephrine and initiates a cyclic AMP second-messenger system





Potential Energy (PE)

LEFT VENTRICULAR PRESSURE/VOLUME P/V LOOP



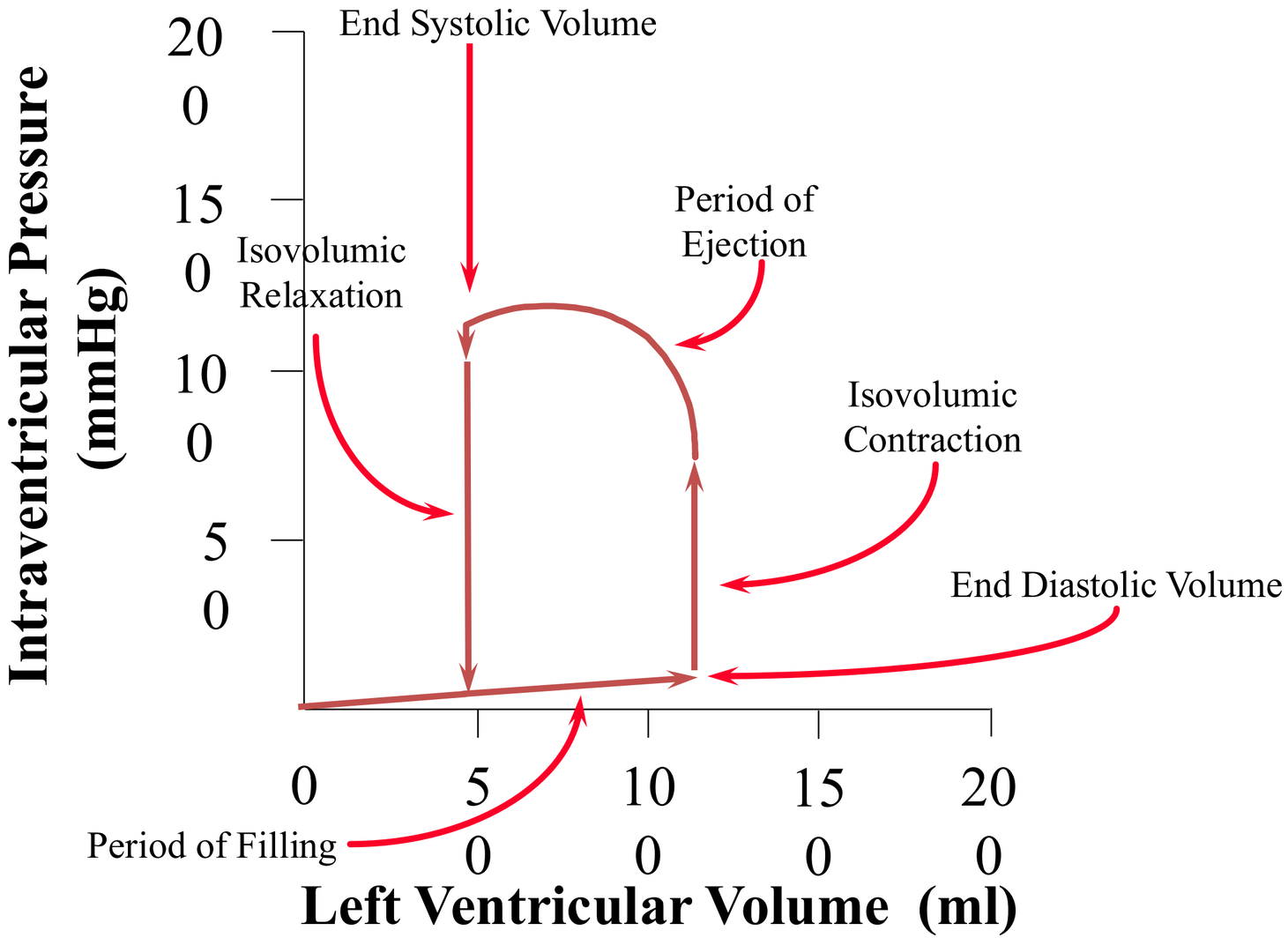
Valvular Function

- To prevent back-flow.
- Chordae tendineae are attached to A-V valves.
- Papillary muscle, attached to chordae tendineae, contract during systole and help prevent back-flow.
- Because of smaller opening, velocity through aortic and pulmonary valves exceed that through the A-V valves.

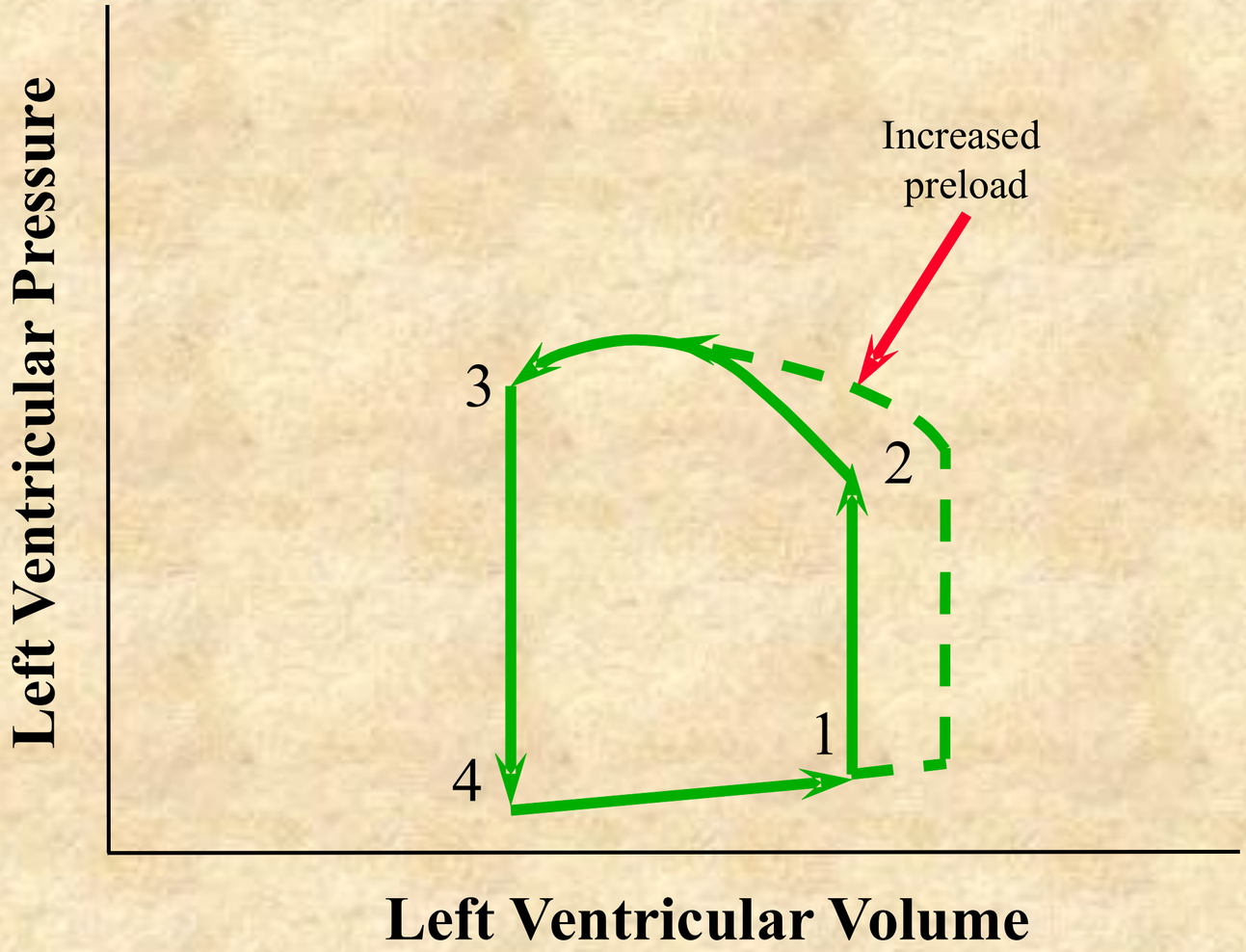
Valvular Function (cont'd)

- Most work is external work or pressure-volume work.
- A small amount of work is required to impart kinetic energy to the heart ($1/2\ mV^2$).
- What is stroke-volume in previous figure?
- External work is area of Pressure-Volume curve.
- Work output is affected by "preload" (end-diastolic pressure) and "afterload" (aortic pressure).

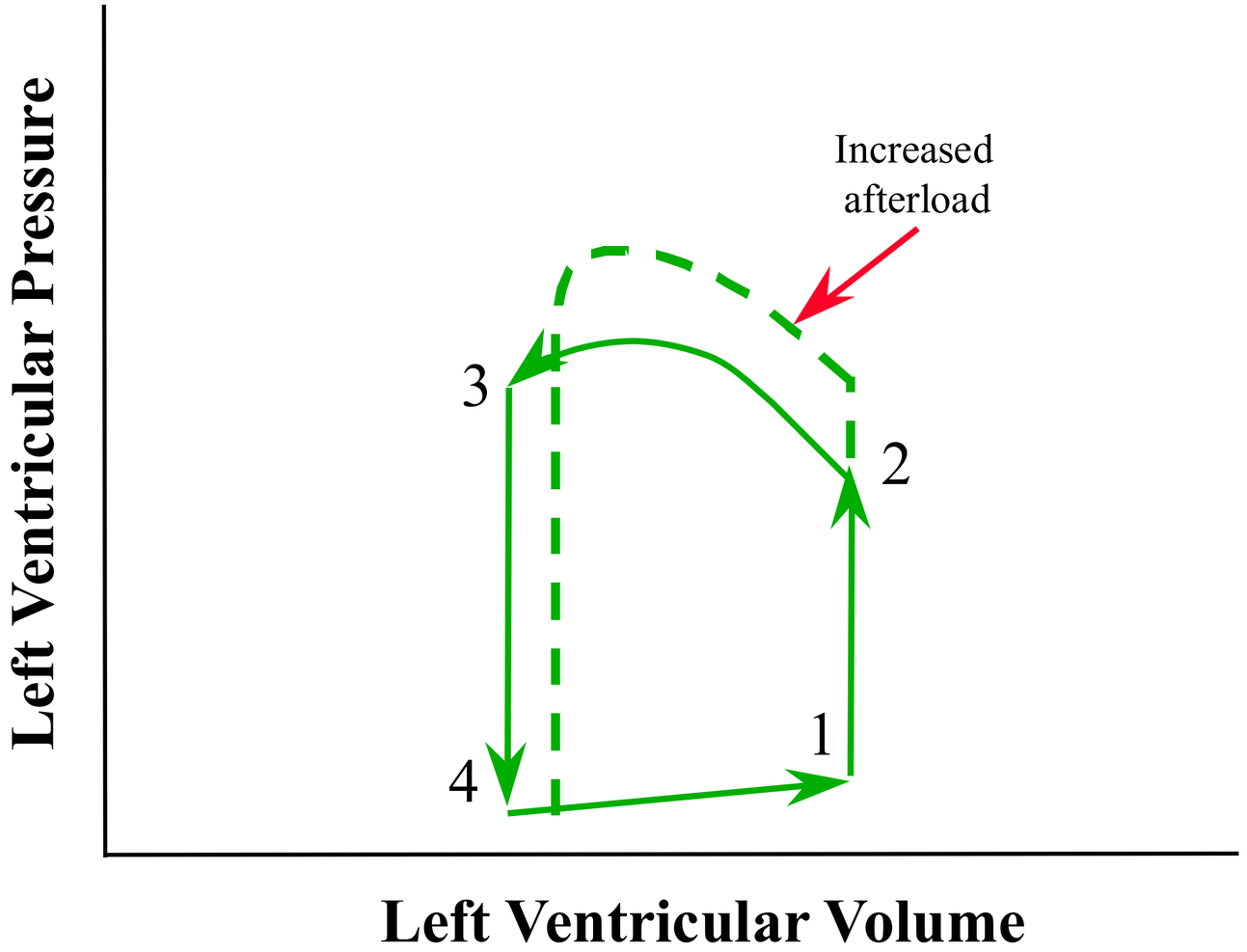
Work Output of the Heart



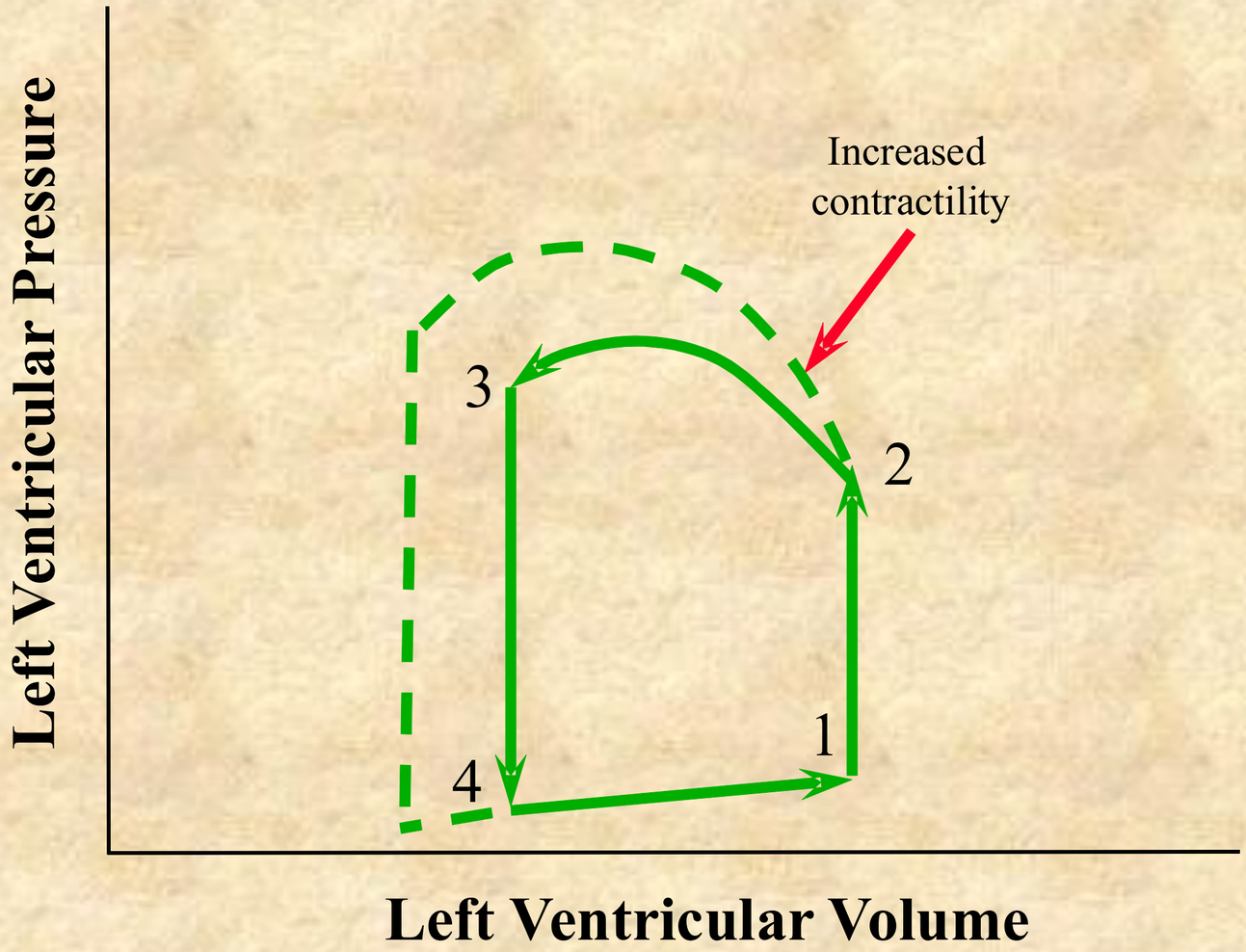
A


Left Ventricular Pressure
Increased
preload
3
2
1
4
Left Ventricular Volume

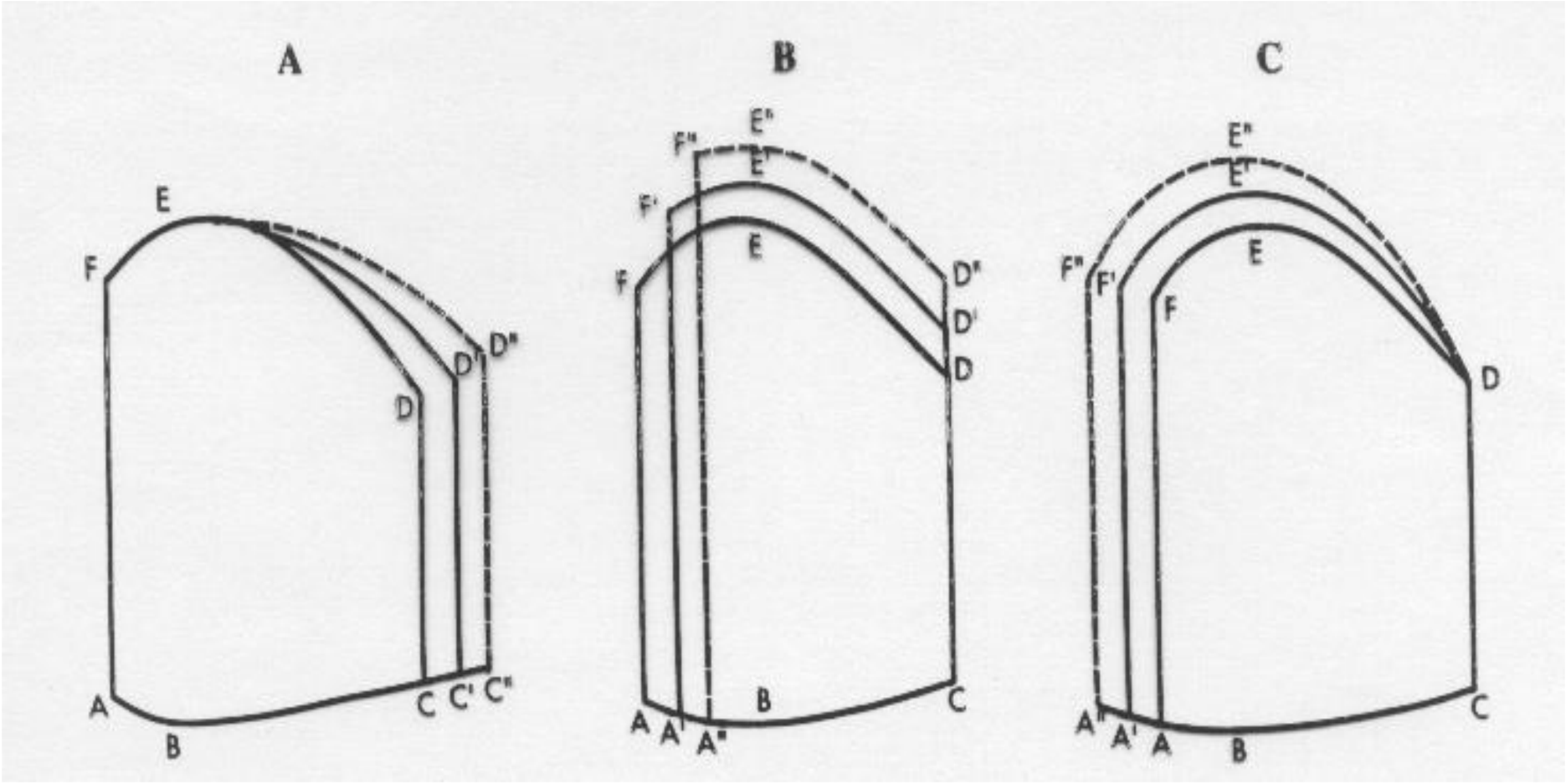
B


Left Ventricular Pressure
Increased
afterload
3
2
1
4
Left Ventricular Volume

c


Left Ventricular Pressure
Increased
contractility
3
2
1
4
Left Ventricular Volume

PRESSURE/VOLUME RELATIONSHIPS UNDER DIFFERENT CONDITIONS

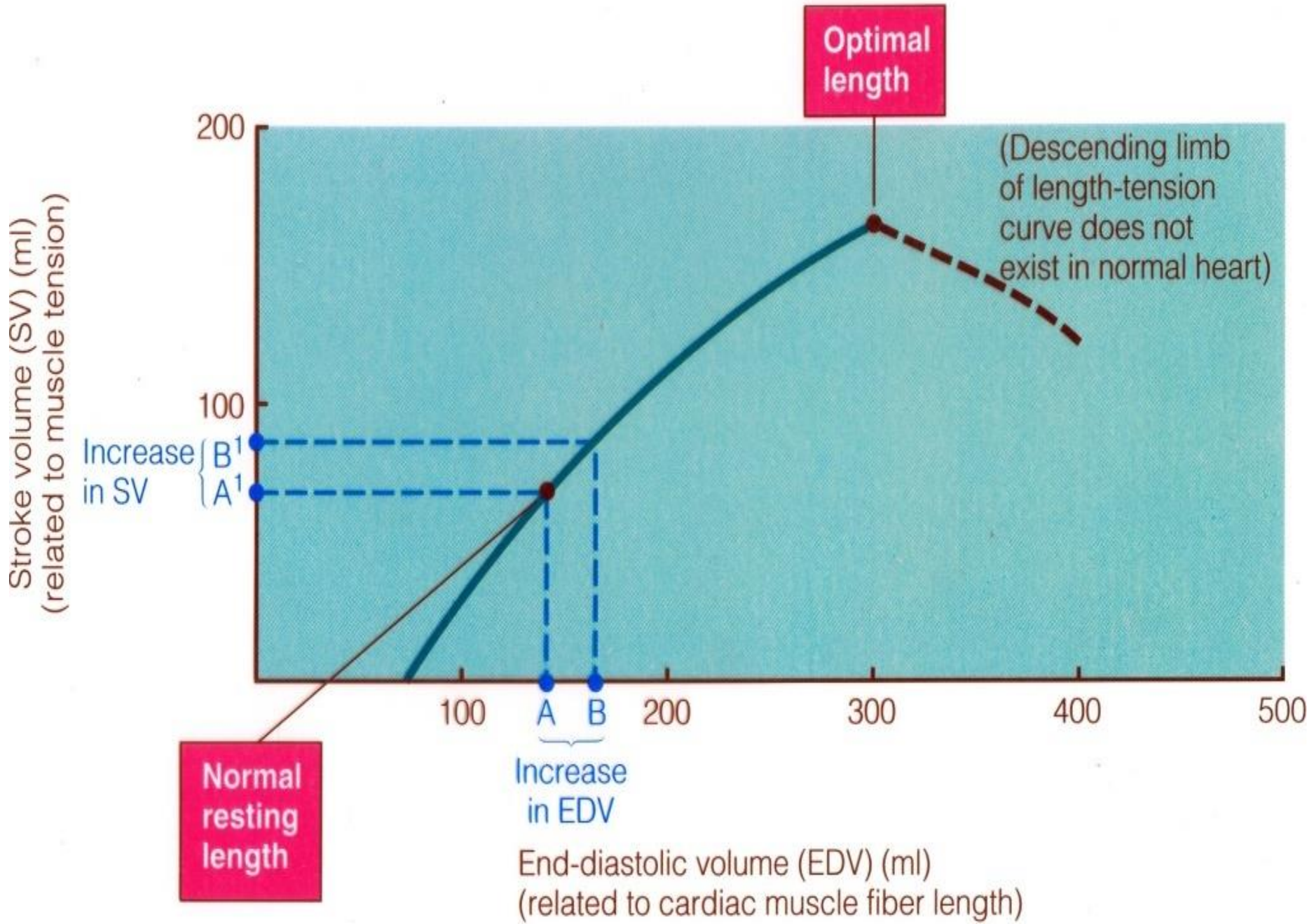


PRELOAD

AFTERLOAD

CONTRACTILITY

Intrinsic Control of Stroke Volume (Frank-Starling Curve)



Regulation of Heart Rate

- Positive chronotropic factors increase heart rate
- Negative chronotropic factors decrease heart rate

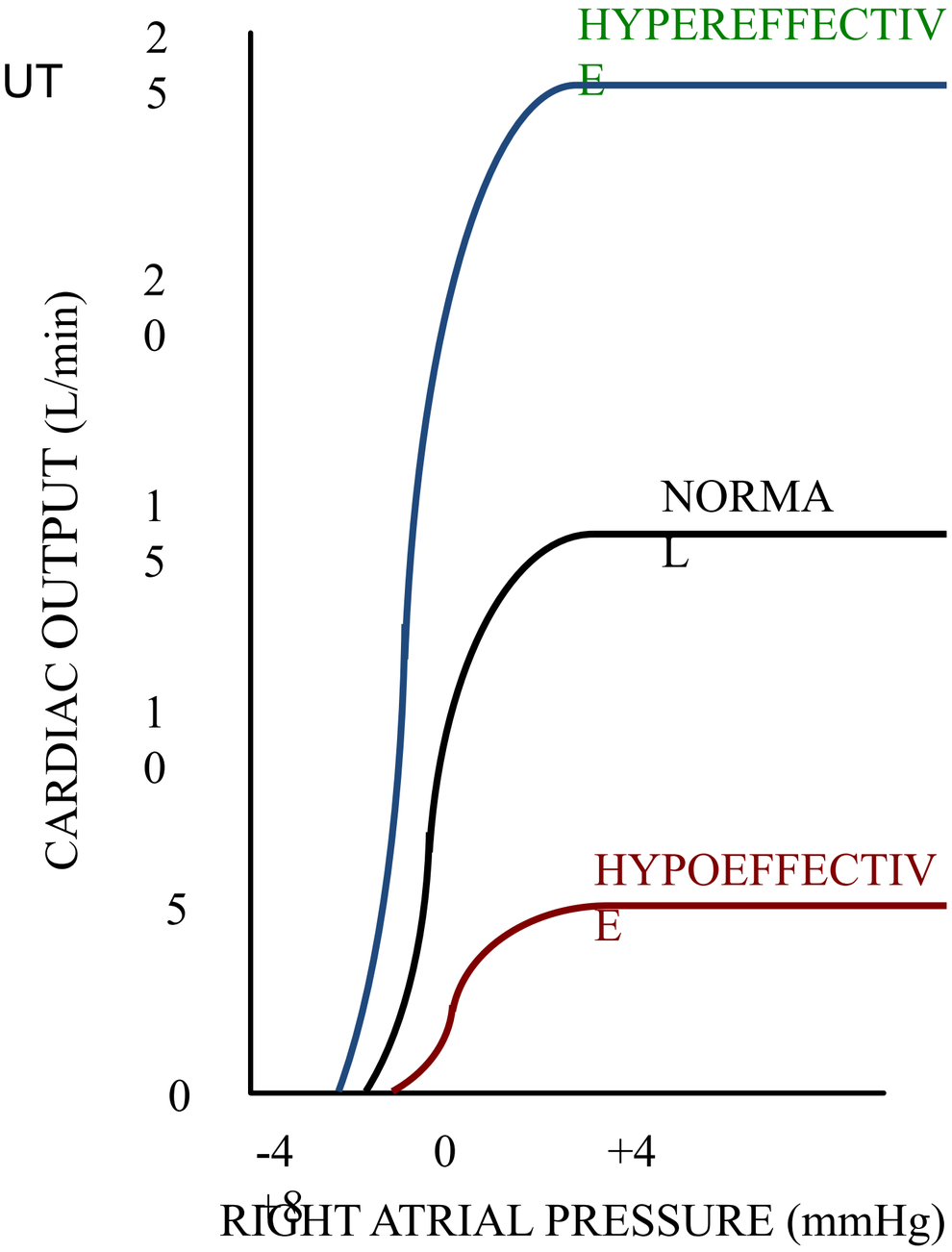
Regulation of Heart Rate: Autonomic Nervous System

- Sympathetic nervous system (SNS) stimulation is activated by stress, anxiety, excitement, or exercise
- Parasympathetic nervous system (PNS) stimulation is mediated by acetylcholine and opposes the SNS
- PNS dominates the autonomic stimulation, slowing heart rate and causing vagal tone

Chemical Regulation of the Heart

- The hormones epinephrine and thyroxine increase heart rate
- Intra- and extracellular ion concentrations must be maintained for normal heart function

CARDIAC OUTPUT CURVES



Ventricular Pressure and Volume Curves (cont'd)

- During the latter part of the ejection phase how can blood still leave the ventricle if pressure is higher in the aorta? Momentum of blood flow
- Total energy of blood = $P + mV^2/2$ =
  pressure + kinetic energy
- Total energy of blood leaving ventricle is greater than in aorta.

Ejection Fraction

- End diastolic volume = 125 ml
- End systolic volume = 55 ml
- Ejection volume (stroke volume) = 70 ml
- Ejection fraction = 70ml/125ml = 56% (normally 60%)
- If heart rate (HR) is 70 beats/minute, what is cardiac output?
- Cardiac output = HR * stroke volume
  = 70/min. * 70 ml =
  4900ml/min.

Ejection Fraction (cont'd)

- If HR =100, end diastolic volume = 180 ml, end systolic vol. = 20 ml, what is cardiac output?
- C.O. = 100/min. * 160 ml = 16,000 ml/min.
- Ejection fraction= 160/180%=~ 90%

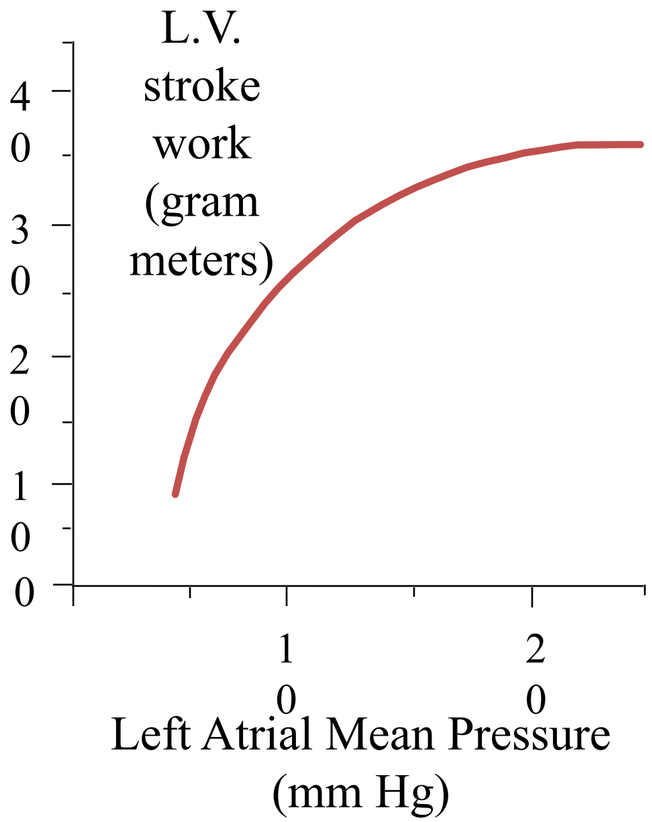
Aortic Pressure Curve

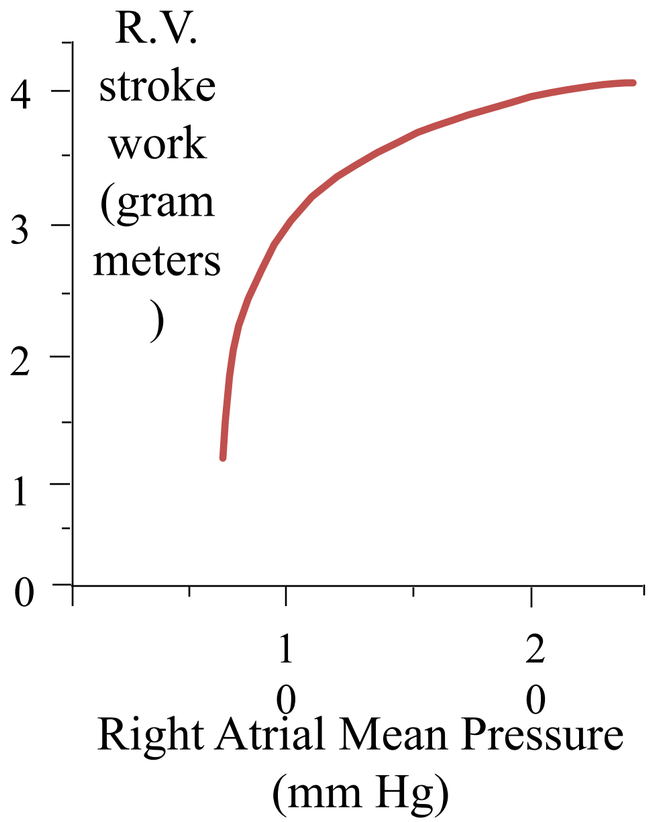
- Aortic pressure starts increasing during systole after the aortic valve opens.
- Aortic pressure decreases toward the end of the ejection phase.
- After the aortic valve closes, an *incisura* occurs because of sudden cessation of back-flow toward left ventricle.
- Aortic pressure decreases slowly during diastole because of the elasticity of the aorta.

Frank-Starling Mechanism

- Within physiological limits the heart pumps all the blood that comes to it without excessive damming in the veins.
- Extra stretch on cardiac myocytes makes actin and myosin filaments interdigitate to a more optimal degree for force generation.

Ventricular Stroke Work Output





Autonomic Effects on Heart

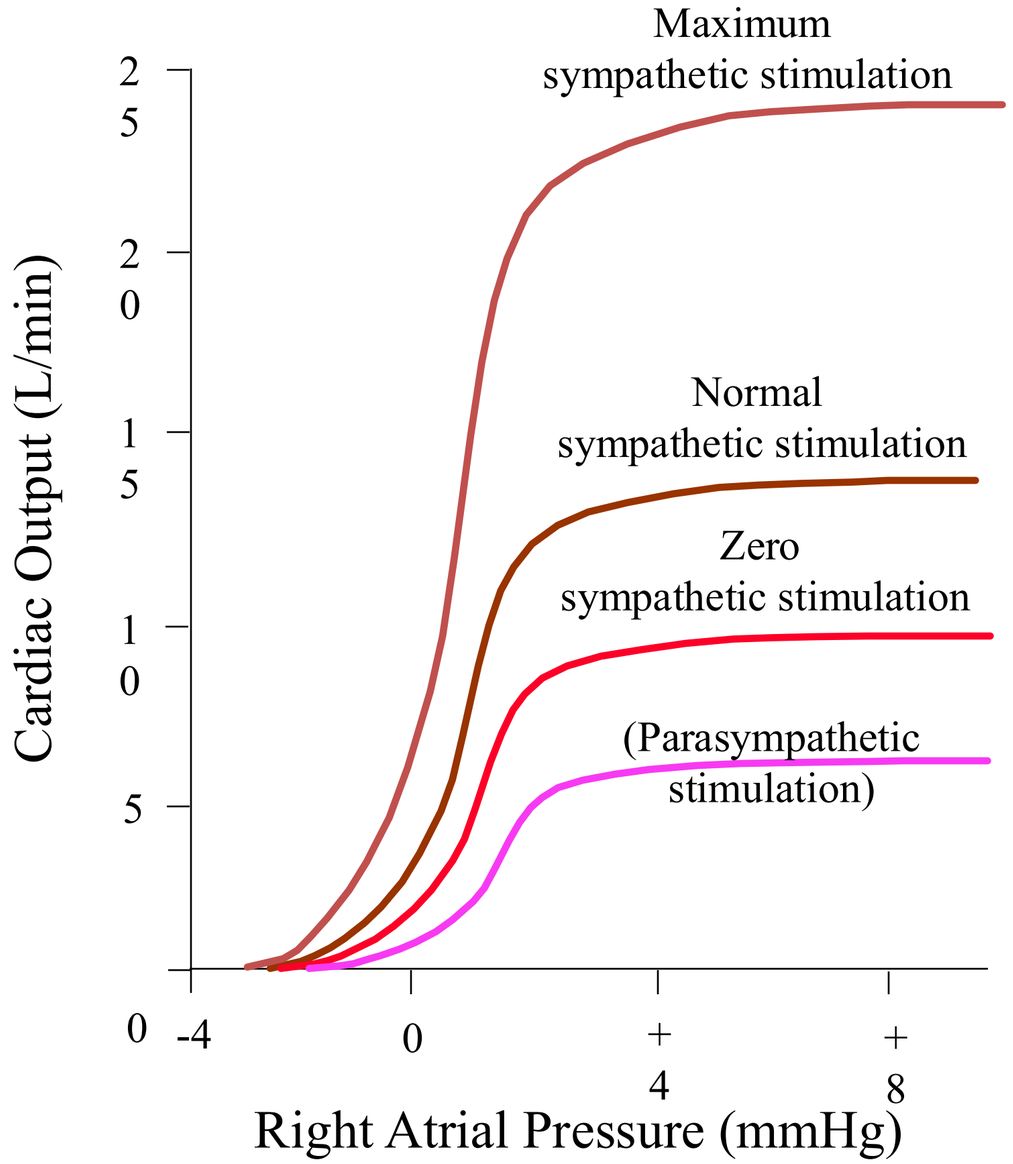
- Sympathetic stimulation causes increased HR and increased contractility with HR = 180-200 and C.O. = 15-20 L/min.
- Parasympathetic stimulation decreases HR markedly and decreases cardiac contractility slightly. Vagal fibers go mainly to atria.
- Fast heart rate (tachycardia) can decrease C.O. because there is not enough time for heart to fill during diastole.

At HR 72 bpm, systole occupies 40% of the cardiac cycle. At HR 200 bpm, it occupies 65% of the time.

Each person has his own optimal maxHR.

HR	Systole	Diastole
60	0.3	0.7
75	0.25	0.55
200	0.19	0.11

Effect of Sympathetic and Parasympathetic Stimulation on Cardiac Output



Thank You



رسالة من الفريق العلمي:

رَبِّ اشْـــــرَحْ
لِي صَـــــدْرِي
وَيَسِّرْ لِي أَمْرِي

وامضِ لحلمكَ واثقًا
كُن فى مقدّمة الصفوف
وامنح فؤادكَ أن يسافرَ
فى الأمانىَ دون خوف
فاليومُ تبذلُ للمنىٰ
وغدًا سترضيك القطوف
أنت الصّباحُ فلا تضِق
مهما تكدّرت الظروف

For any feedback, scan the code or click on it.

Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1			
V1 → V2			