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Analysis of Cardiac Physiology

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BM2102 Modelling and Analysis of Physiological Systems

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1 Introduction

The following report presents a detailed analysis of cardiac physiology under two specific conditions:

1. Normal cardiac rhythm
2. Cardiac rhythm in the presence of aortic valve stenosis

A single cardiac cycle in the human heart consists of three key phases: *atrial systole*, *ventricular systole*, and *complete cardiac diastole*. These phases are characterized by dynamic changes in physiological parameters such as chamber pressures, volumes, and valve function. Such changes can be effectively visualized and interpreted using tools like the *Wigger's Diagram*(wikipedia.org), which integrates electrical, mechanical, and acoustic events of the cardiac cycle.

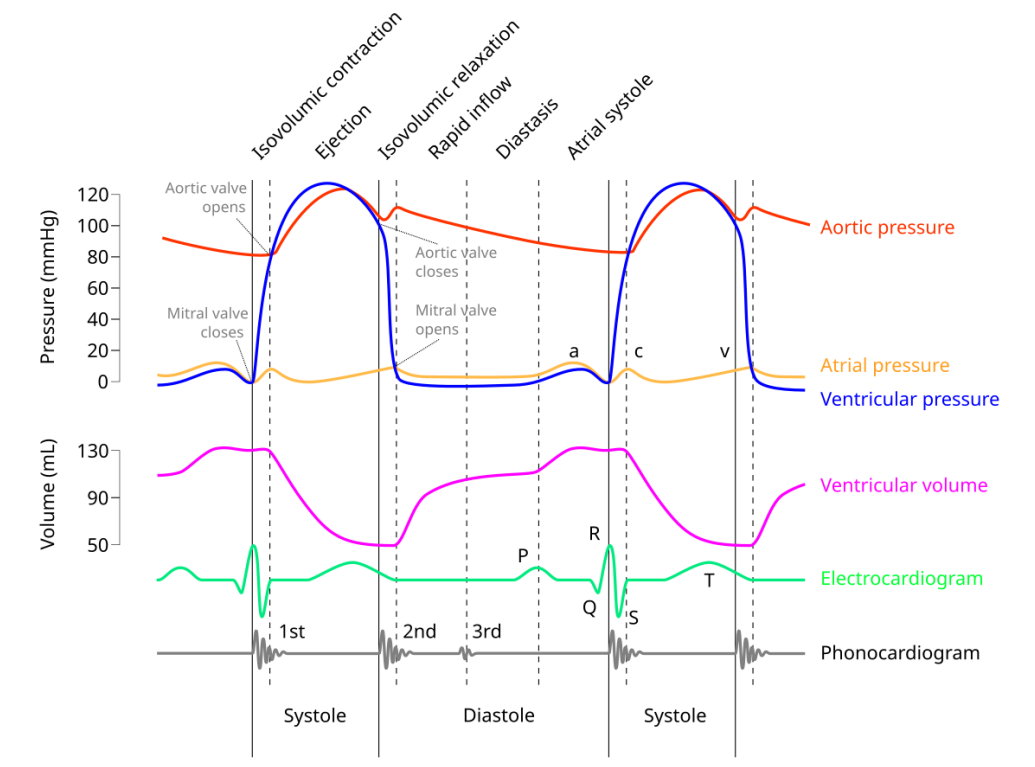


Figure 1: Wigger's Diagram

This analysis was conducted using the **CircAdapt Simulator**, a comprehensive cardiovascular simulation platform. All simulations and graphical observations were performed using **software version v1.1.0**, which offers interactive modules to examine pressure, volume, and flow relationships in a real-time model of the human heart.

2 Methodology

The **CircAdapt v1.1.0** simulator was downloaded and installed from the official website. The following graphs were observed during the simulation:

- Pressure vs Time for the left atrium, left ventricle, and aorta.
- Volume vs Time for the left ventricle.

- Flow velocities for the aortic and mitral valves.
- Pressure-Volume (P-V) loop for the left ventricle.

The **REFERENCE** mode was used to clearly identify the opening and closing events of the heart valves.

Aortic valve stenosis was simulated by increasing the valve stenosis percentage parameter in the simulator.

Various observations were documented throughout the simulation, including:

- Timing of valve opening and closing
- Labeling of events on the pressure-volume (P-V) loop
- Flow velocity profiles for both valves
- Pressure differences between chambers and vessels

3 Normal Sinus Rhythm

3.1 Aortic valve opening and closing

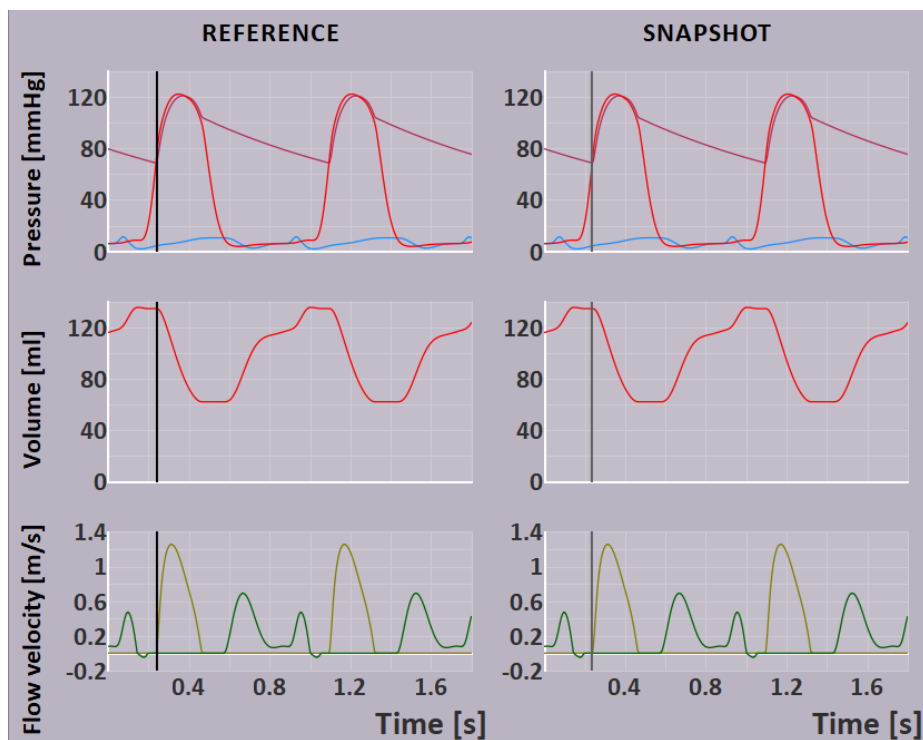


Figure 2: Opening of the aortic valve

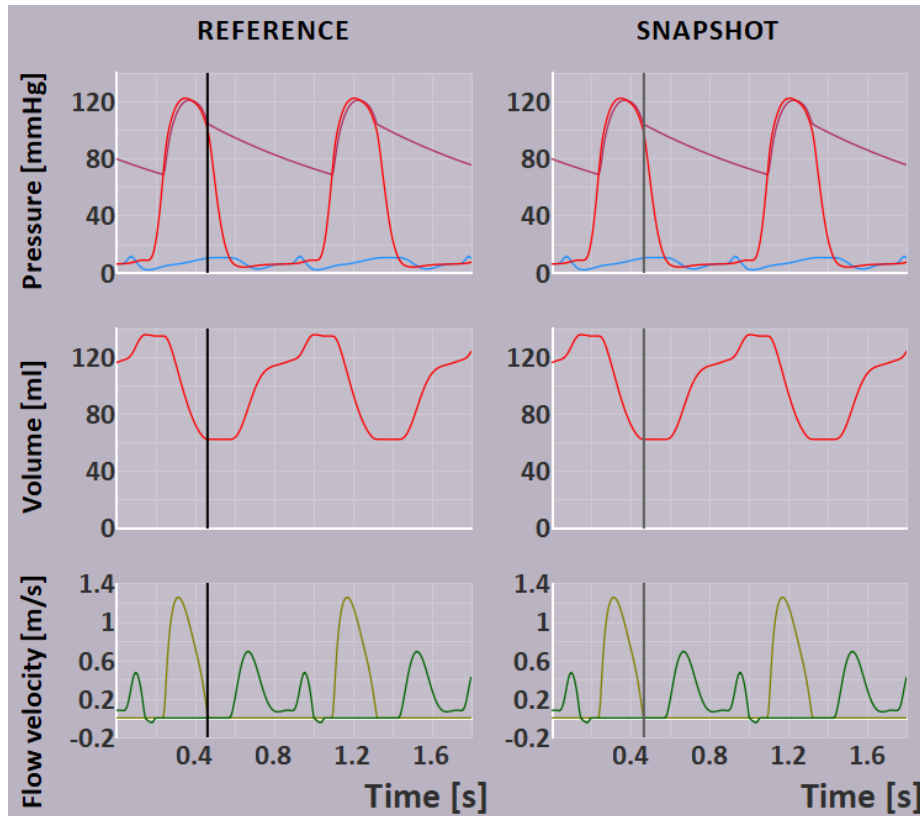


Figure 3: Closing of the aortic valve

Table 1: Aortic Valve Opening and Closing Parameters

| Parameter | Valve Opening | Valve Closing |
|---------------------------|---------------|---------------|
| Timestamp | 0.24 s | 0.46 s |
| Aortic Pressure | 70 mmHg | 104 mmHg |
| Left Ventricular Pressure | 76.2 mmHg | 99.7 mmHg |

By analyzing the generated graphs, the opening and closing of the aortic valve can be distinctly identified. During this interval, the left ventricular pressure rises sharply, reaching its peak—indicating the moment of maximum contractile force required to propel blood into the systemic circulation. Simultaneously, the left ventricular volume decreases significantly, confirming the ejection of blood into the aorta. This observation is further supported by the flow velocity graph, where the aortic valve flow (highlighted in lime green) shows a pronounced spike. This sudden increase indicates a rapid ejection phase, consistent with the systolic outflow into the aorta.

3.2 Mitral valve opening and closing

Table 2: Mitral Valve Opening and Closing Parameters

| Parameter | Valve Opening | Valve Closing |
|---------------------------|---------------|---------------|
| Timestamp | 0.58 s | 1.01 s |
| Left Atrial Pressure | 10.4 mmHg | 2.15 mmHg |
| Left Ventricular Pressure | 9.94 mmHg | 8.62 mmHg |

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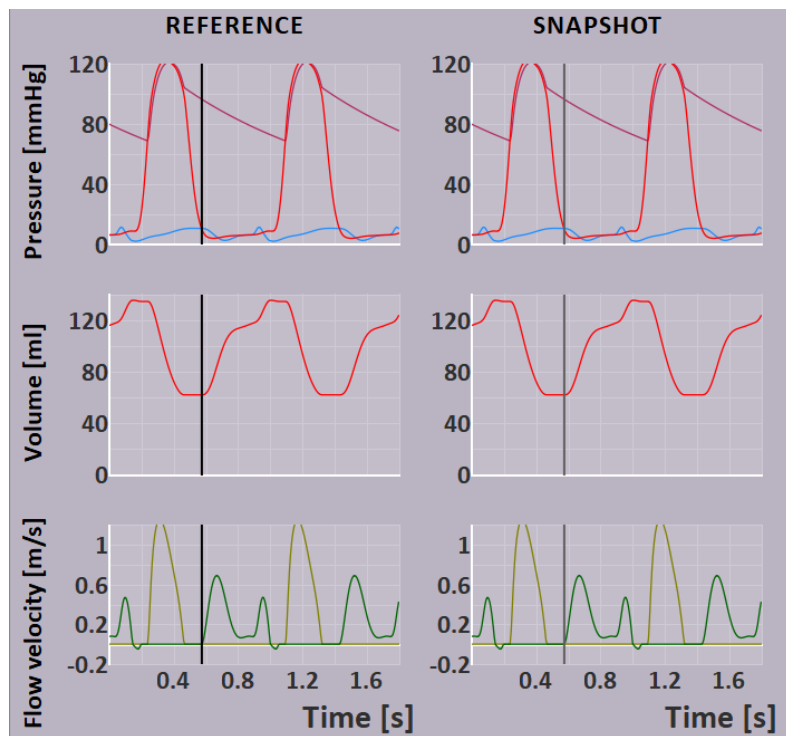


Figure 4: Opening of the mitral valve

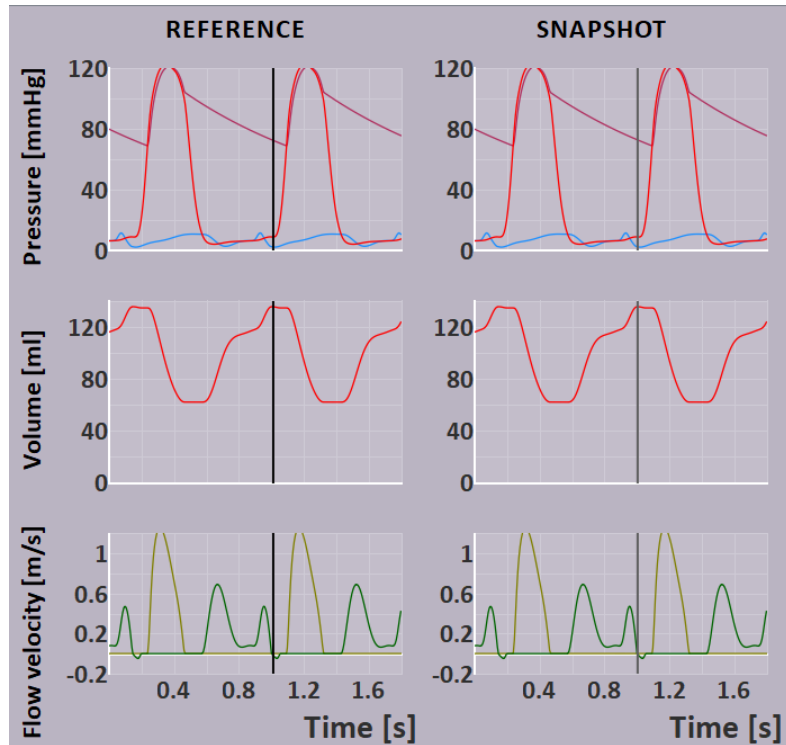


Figure 5: Closing of the mitral valve

3.3 Identification of points of the pressure-volume relation

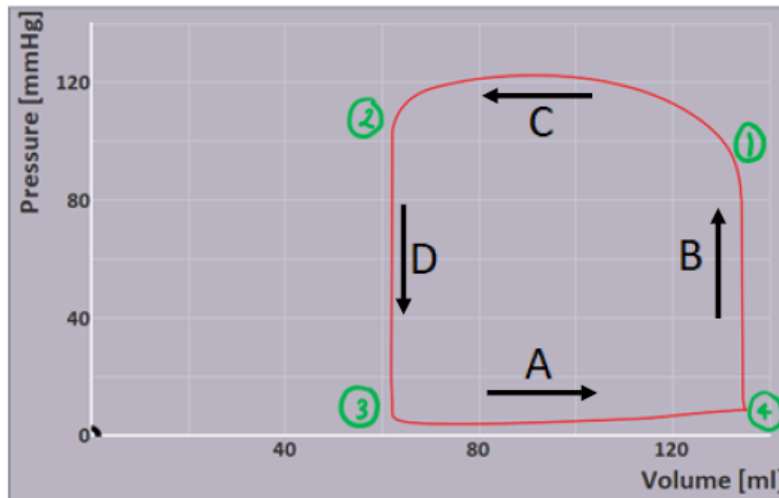


Figure 6: Pressure-volume graph

- 1 – Aortic valve opening
- 2 – Aortic valve closing
- 3 – Mitral valve opening
- 4 – Mitral valve closing

The positions corresponding to the opening and closing of the aortic and mitral valves on the pressure-volume (P-V) loop can be accurately determined by analyzing the changes in left ventricular volume throughout the cardiac cycle.

- The **aortic valve** opens when the left ventricular pressure exceeds the aortic pressure, initiating the *ejection phase*. During this period (from 1 → 2), the **ventricular volume decreases sharply**, indicating that blood is being expelled into the aorta. Hence, point 1 marks the start of ejection (aortic valve opening), and point 2 marks the end of ejection (aortic valve closing).
- The **mitral valve** opens when the left atrial pressure becomes higher than the ventricular pressure following isovolumic relaxation. This initiates the *filling phase* (from 3 → 4), during which the **ventricular volume increases**. Therefore, point 3 marks the beginning of ventricular filling (mitral valve opening), and point 4 marks the end of filling (mitral valve closing), just before the next systole begins.

This valve timing is critical for labeling the characteristic phases of the cardiac cycle on the P-V loop.

3.4 Phases of pressure-volume relation

- A – Filling
- B – Isovolumic Contraction
- C – Ejection
- D – Isovolumic Relaxation

3.5 Comparison of flow velocity patterns in aortic and mitral valves

The difference in the flow velocity patterns of the aortic and mitral valves is rooted in their distinct functional roles and the timing of pressure changes during the cardiac cycle.

The **aortic valve** exhibits a *single hump* in its flow velocity graph. This is because the aortic valve opens only once per cardiac cycle during the ventricular systole when the left ventricular pressure exceeds the aortic pressure. Once opened, there is a rapid and forceful ejection of blood from the left ventricle into the aorta, resulting in a sharp, singular peak in the velocity curve. As the ventricular pressure drops below the aortic pressure near the end of systole, the valve closes, and no further forward flow occurs until the next cycle.

In contrast, the **mitral valve** shows a *double-hump (biphasic)* flow velocity pattern. This occurs due to two distinct phases of blood inflow from the left atrium into the left ventricle:

- **Early passive filling:** Immediately after the isovolumic relaxation phase, the left ventricular pressure falls below the left atrial pressure, allowing the mitral valve to open. Blood passively flows into the ventricle, producing the first hump in the velocity curve.
- **Atrial contraction (atrial systole):** Just before the onset of ventricular systole, the atrium contracts to push the remaining blood into the ventricle. This produces the second hump, which is typically smaller but still distinct.

This biphasic pattern reflects the passive and active components of ventricular filling, in contrast to the unidirectional and singular nature of ventricular ejection seen in the aortic valve.

3.6 Explanation of atrial pressure change

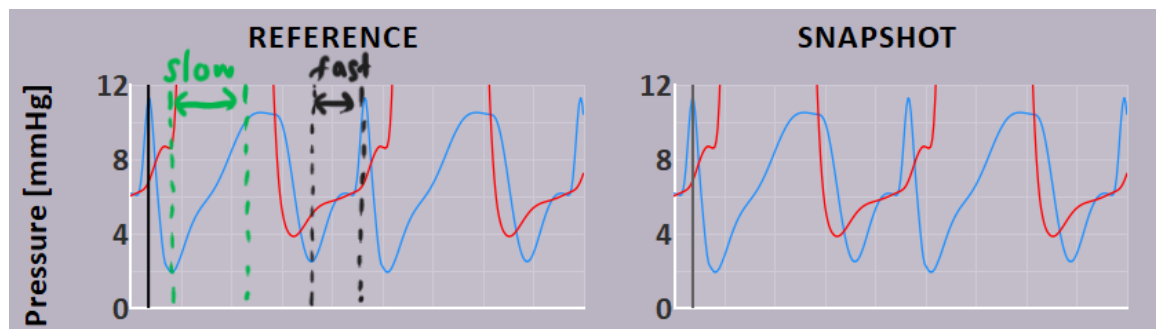


Figure 7: Different phases of atrial pressure increase in the left atrium

To analyze the atrial pressure waveform in detail, the y-axis scale of the graph was adjusted, allowing for clear identification of two distinct phases of pressure increase within a single cardiac cycle: one slow and one fast (steep) rise. These two phases correspond to different physiological mechanisms in the heart.

The slow increase in left atrial pressure occurs during the ventricular systole and early diastole, when both the atria and ventricles are relaxed. During this time, blood from the pulmonary veins enters the left atrium passively. Since the mitral valve remains closed until the ventricular pressure falls below atrial pressure, this passive filling leads to a gradual accumulation of blood in the left atrium, causing a gentle rise in pressure. This period is marked as the green region in the figure.

In contrast, the fast (steep) increase in atrial pressure happens due to atrial systole; the active contraction of the left atrium. Just before the ventricles begin contracting, the atria contract to push the remaining blood into the ventricles through the mitral valve. This rapid ejection of blood creates a sudden and sharp rise in atrial pressure, visible as the black region in the figure.

The ECG waveform that corresponds to this steep rise in atrial pressure is the QRS complex. Although atrial systole itself follows the P-wave, the hemodynamic effect of atrial contraction peaks

during ventricular depolarization, which is marked by the QRS complex. Therefore, the steep pressure rise in the atrium aligns closely with this ECG segment.

3.7 Identification of the E and A waves of the mitral blood flow velocity signal

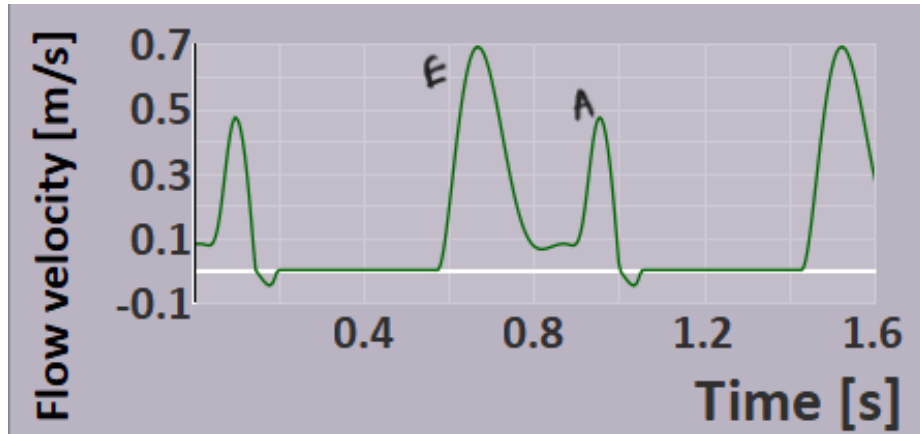


Figure 8: Early Rapid Filling (E) and Atrial Contraction (A) representation in the mitral valve blood flow graph

3.8 E/A ratio of the current simulation

| Peak | Timestamp (s) | Mitral valve flow velocity (m/s) |
|--------|---------------|----------------------------------|
| E peak | 0.67 | 0.691 |
| A peak | 0.95 | 0.471 |

Table 3: Readings related to the calculation

$$\text{E/A ratio} = \frac{0.691}{0.471} = 1.467$$

Since the calculated E/A ratio is above 1, it indicates that the diastolic function is within a healthy range.

3.9 Active and passive filling of left ventricle

| Instance | Timestamp (s) | Left Ventricular Volume (ml) |
|------------------------------------|---------------|------------------------------|
| Mitral valve closed | 0.58 | 62 |
| After early rapid filling (E peak) | 0.90 | 118 |
| After atrial contraction (A peak) | 1.01 | 135 |

Table 4: Left ventricular volume readings

Filling due to passive filling:

$$118 - 62 = 56 \text{ ml}$$

Filling due to active filling:

$$135 - 118 = 17 \text{ ml}$$

It can be observed that the volume gained through passive filling is nearly 3 times the volume gained through active (atrial) filling.

3.10 Relationship between flow velocity and flow rate

To convert the blood flow velocity (V_{valve}) through a cardiac valve into the corresponding flow rate (Q_{valve}), it is essential to know the **cross-sectional area** of the valve. The flow rate is defined as the product of velocity and cross-sectional area:

$$Q_{\text{valve}} = A_{\text{valve}} \times V_{\text{valve}}$$

Here, A_{valve} represents the anatomical or geometrical cross-sectional area of the valve orifice. The units of velocity are typically in meters per second (m/s), and the area is in square meters (m²), resulting in flow rate units of cubic meters per second (m³/s), which is consistent with volumetric flow.

4 Aortic Valve Stenosis

4.1 Preload and Afterload

Preload, or otherwise known as *Ventricular End Diastolic Volume (VEDV)*, is the volume of blood present in the ventricles at the end of diastole, just before contraction. It reflects the initial stretching of the cardiac muscle fibers due to the incoming blood. Preload is primarily influenced by **venous return**, **total blood volume**, and **atrial contraction**, all of which contribute to the filling of the ventricles.

Afterload is the *resistance* that the ventricles must overcome to eject blood during systole. It represents the load against which the heart must pump. Afterload is determined by factors such as **arterial blood pressure**, and the **compliance (distensibility and elasticity)** of arteries and arterioles. Increased vascular resistance or stiffness leads to a higher afterload, making it more difficult for the heart to eject blood.

4.2 Aortic Valve Stenosis (AS) Simulation

The simulation of aortic stenosis (AS) was performed by incrementally increasing the stenosis percentage in 5% steps, up to a maximum of 80%, using the “valves” tab. For each step, the corresponding hemodynamic parameters specifically, the peak flow velocity across the aortic valve, the peak pressure within the left ventricle, and the peak left ventricular volume were recorded and analyzed.

| Stenosis (%) | Peak Velocity (m/s) | Peak Pressure (mmHg) | Peak Volume (ml) |
|--------------|---------------------|----------------------|------------------|
| 0 | 1.26 | 122 | 135 |
| 5 | 1.32 | 123 | 135 |
| 10 | 1.38 | 123 | 135 |
| 15 | 1.46 | 124 | 135 |
| 20 | 1.54 | 125 | 135 |
| 25 | 1.63 | 125 | 135 |
| 30 | 1.72 | 126 | 135 |
| 35 | 1.84 | 127 | 136 |
| 40 | 1.97 | 128 | 136 |
| 45 | 2.11 | 130 | 136 |
| 50 | 2.28 | 132 | 136 |
| 55 | 2.47 | 135 | 136 |
| 60 | 2.69 | 139 | 136 |
| 65 | 2.96 | 143 | 137 |
| 70 | 3.28 | 150 | 137 |
| 75 | 3.68 | 158 | 139 |
| 80 | 4.20 | 171 | 141 |

Table 5: Change of peak flow velocity through aortic valve, peak pressure, and peak volume in the left ventricle

As shown in Table 5, the peak flow velocity increases steadily with each step of stenosis. This aligns with the continuity equation:

$$Q_{\text{valve}} = A_{\text{valve}} \times V_{\text{valve}}$$

As the cross-sectional area of the valve (A_{valve}) decreases due to stenosis and assuming the flow rate (Q_{valve}) remains relatively constant, the velocity (V_{valve}) must increase to compensate. Additionally, the left ventricular peak pressure begins to increase significantly beyond 20% stenosis. Meanwhile, the left ventricular volume shows only a modest and gradual rise. The pressure-volume relation shows the increasing afterload imposed on the left ventricle.

Maximal left ventricular pressure observed is 171 mmHg at 80% stenosis.

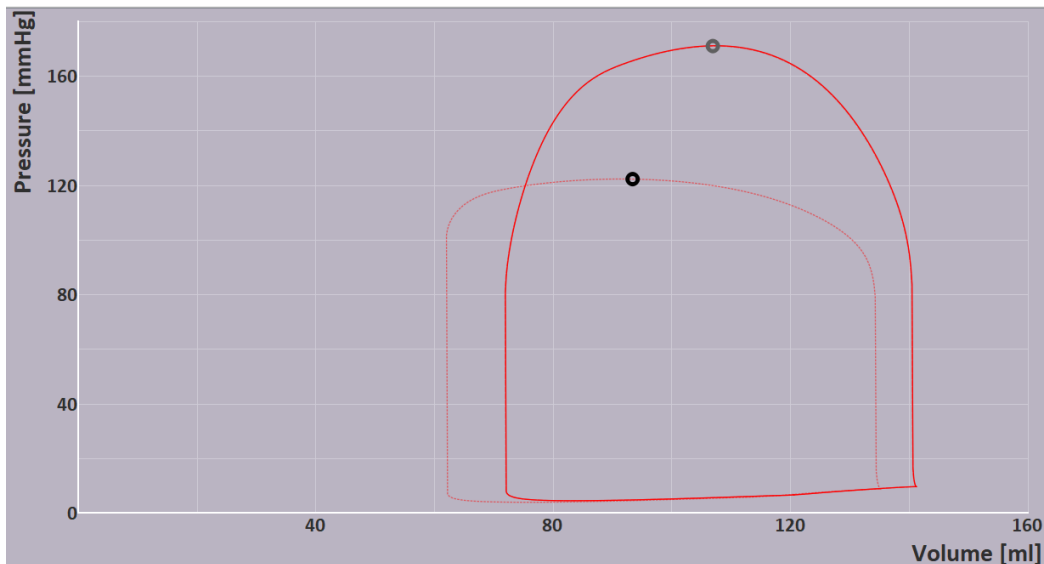


Figure 9: left ventricular pressure-volume relation of AS

The dashed line represents the normal condition (0% stenosis), where the heart pumps blood efficiently with a larger pressure-volume loop. In contrast, with 80% stenosis (red solid line), the loop becomes smaller, indicating reduced stroke volume and increased systolic pressure. The heart faces more resistance, leading to less blood being ejected with each beat.

4.3 Effect of Aortic Valve Stenosis on Preload, Afterload and Cardiac Output

| Variable | Effect of Stenosis |
|----------------------------|--|
| Preload | Aortic valve stenosis narrows the valve opening, making it difficult for the left ventricle to eject all the blood during systole. As a result, a portion of the blood remains in the ventricle. With each subsequent filling, this residual volume adds up, leading to an increase in preload . |
| Afterload | The stenotic (narrowed) aortic valve increases the resistance the left ventricle must overcome to eject blood. This increased resistance elevates the afterload , making it harder for the ventricle to pump blood into the aorta. |
| Cardiac Output (CO) | Cardiac Output = Stroke Volume \times Heart Rate. Although increased preload can enhance stroke volume via the Frank-Starling mechanism, the elevated afterload opposes ejection. Over time, the ventricle may weaken, and stroke volume can decrease, ultimately leading to reduced cardiac output , especially in severe cases. |

4.4 Pressure Calculation

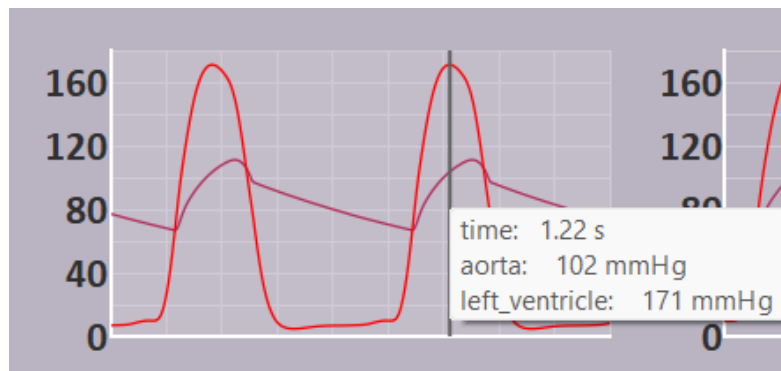


Figure 10: Readings for pressure calculation

Aortic pressure during maximum left ventricular pressure is 102 mmHg.

Pressure drop = $171 - 102 = 69$ mmHg

4.5 Pressure calculation using the given formula

Maximum flow velocity of the aortic valve is $v = 4.20 \text{ ms}^{-1}$.

$$\Delta P = 4v^2 = 4(4.2)^2 = 70.56 \text{ mmHg}$$

This calculated value is closer to the measured value.

4.6 Duration of ejection

The following shows the time taken for ventricular blood to be ejected through the constricted aortic valve.

$$\text{Duration of ejection} = 0.52 \text{ s} - 0.23 \text{ s} = 0.29 \text{ s}$$

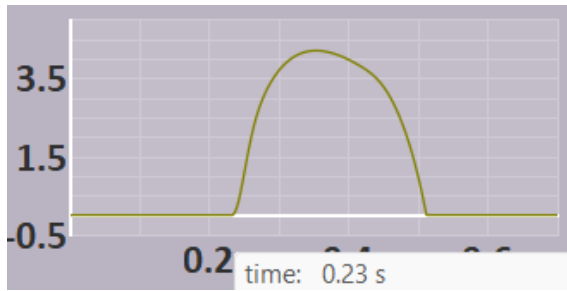


Figure 11:

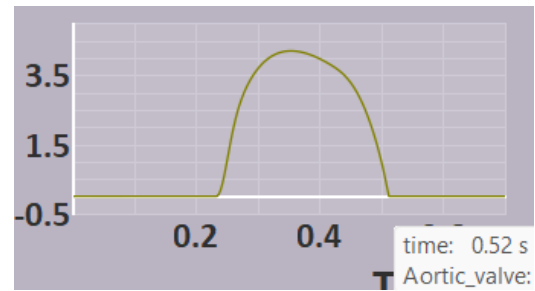


Figure 12:

4.7 external pump work generated by the left ventricle

Using Figure 7 in Page 10:

External pump work in a normal person = 20 squares

External pump work at 80% stenosis = 24 squares

Increase in pump work = $24 - 20 = 4$ squares = $4 \times 20 \times 20 = 1600 \text{ ml} \cdot \text{mmHg}$

4.8 Adaptation of left ventricle to generate the chronically increased pump work

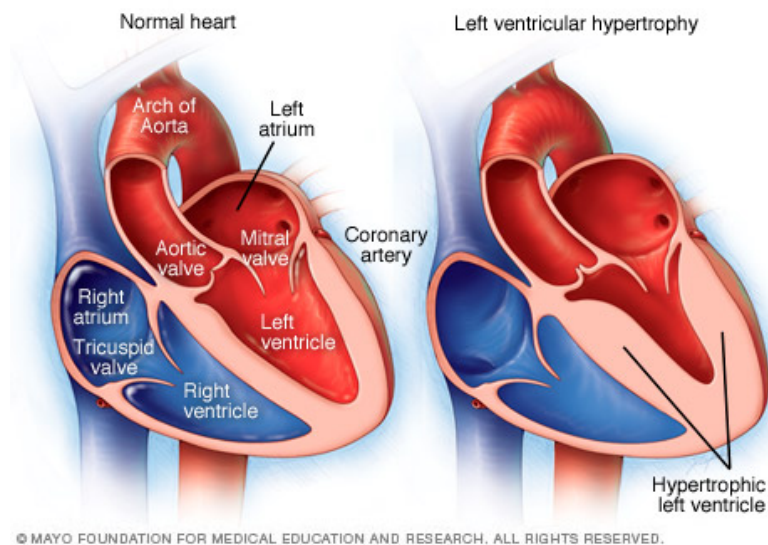


Figure 13: Normal Heart vs. LVH Heart

Initially, the myocardium of the left ventricle adapts to the increased pump work caused by aortic stenosis through hypertrophy. Specifically, the heart undergoes concentric hypertrophy of the left ventricle, commonly referred to as Left Ventricular Hypertrophy (LVH). This is characterized by an abnormal increase in myocardial wall thickness without chamber dilation, as the muscle mass grows to compensate for the chronic pressure overload. This structural change enables the left ventricle to generate higher pressures to overcome the elevated resistance.

This adaptation does not directly reduce the afterload, as afterload is defined by the resistance to blood flow through the narrowed aortic valve. However, the ventricle becomes more capable of coping with the elevated afterload, maintaining cardiac function in the early stages of the disease.

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