



Assignment 3 :Analysis of Cardiac Physiology

BM2102 - Modelling and Analysis of Physiological Systems
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May 15, 2025

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1 Introduction

This report discusses the analysis of cardiac physiology in the following 2 sections as follows.

1. Normal Sinus Rhythm
2. Aortic Valve Stenosis

A cardiac cycle in the human heart comprises three main phases: atrial systole, ventricular systole, and complete cardiac diastole. Variations in physiological parameters, such as pressure and volume, can be analyzed using a Wigger's Diagram [ref1]. For analysis **CircAdapt Simulator** software is used.

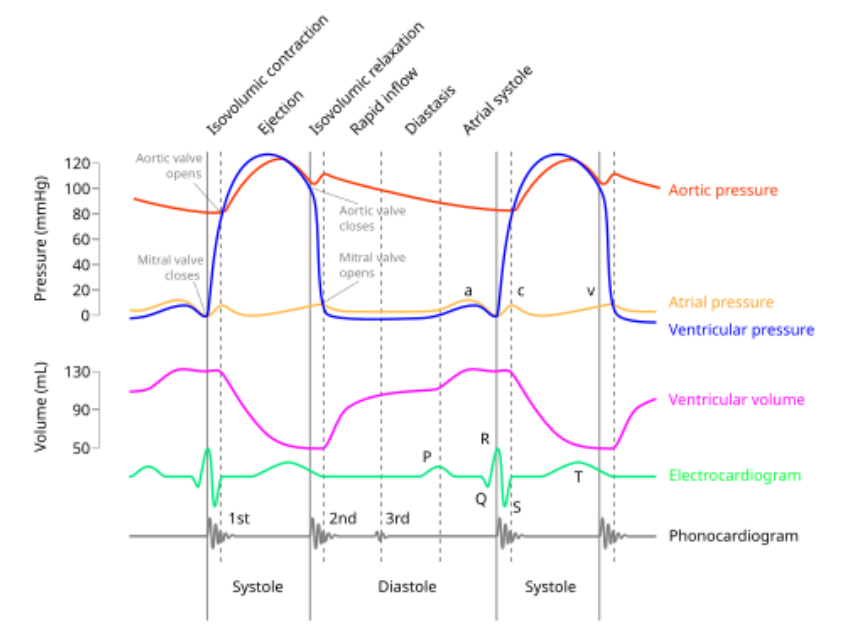


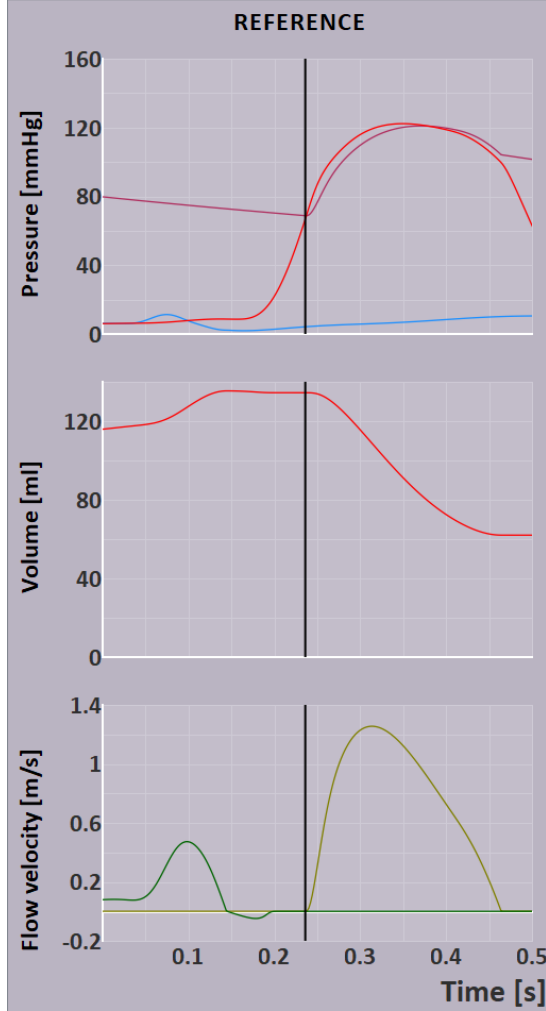
Figure 1: Wiggers diagram

In this simulation we are focused on the following physiological parameters.

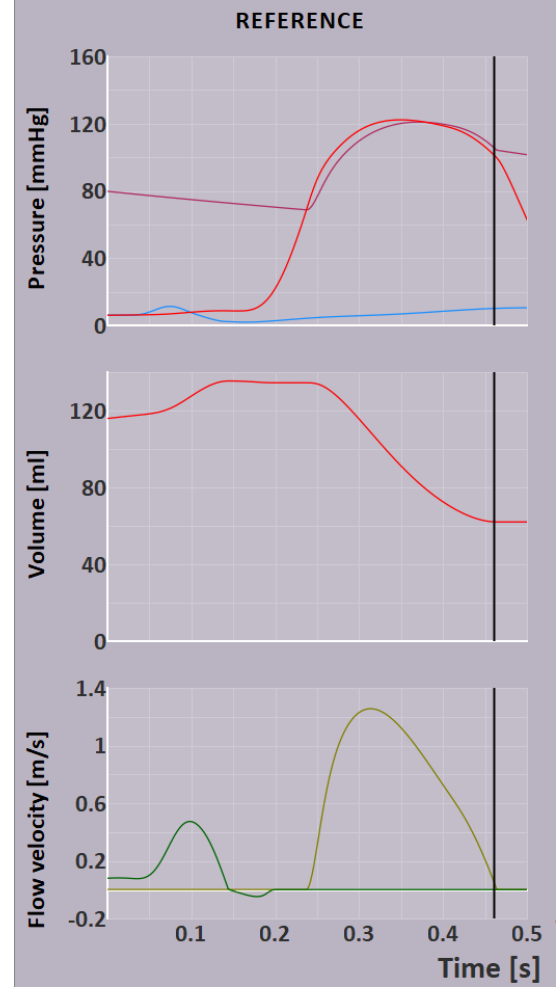
1. Left ventricular pressure
2. Left atrial pressure
3. Aortic pressure
4. Left ventricular volume
5. Flow rate of Aortic valve
6. Flow rate of Mitral valve

2 Normal Sinus Rhythm

2.1 (a) Opening of aortic valve



(a) Aortic valve open



(b) Aortic valve close

Figure 2: Timestamp of opening and closing of aortic valve

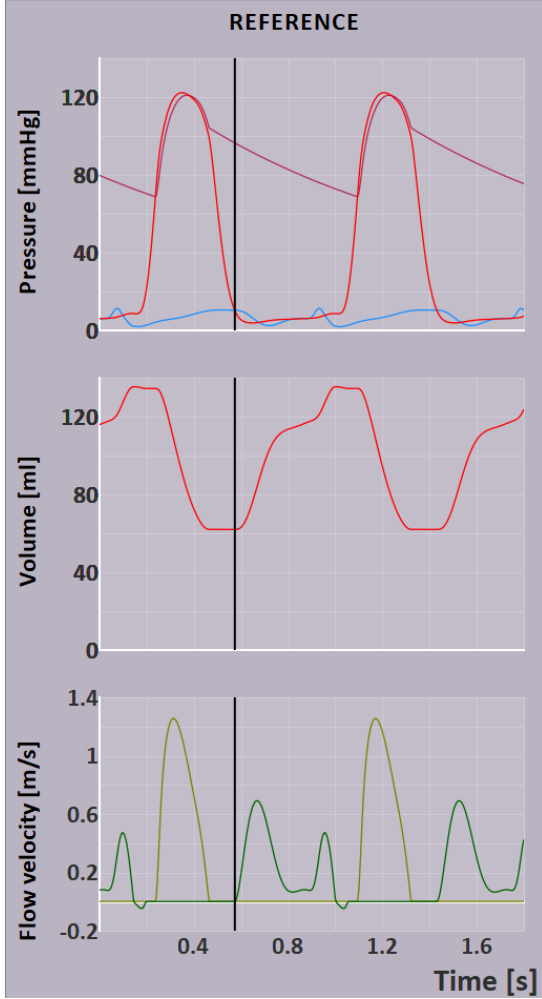
Parameter	Valve open	Valve close
Time(s)	0.24	0.47
Aortic pressure(mmHg)	68.7	104
Left ventricular pressure(mmHg)	68.4	99.1
Left ventricular volume(ml)	134	62.1

Table 1: Aortic valve state determining parameters

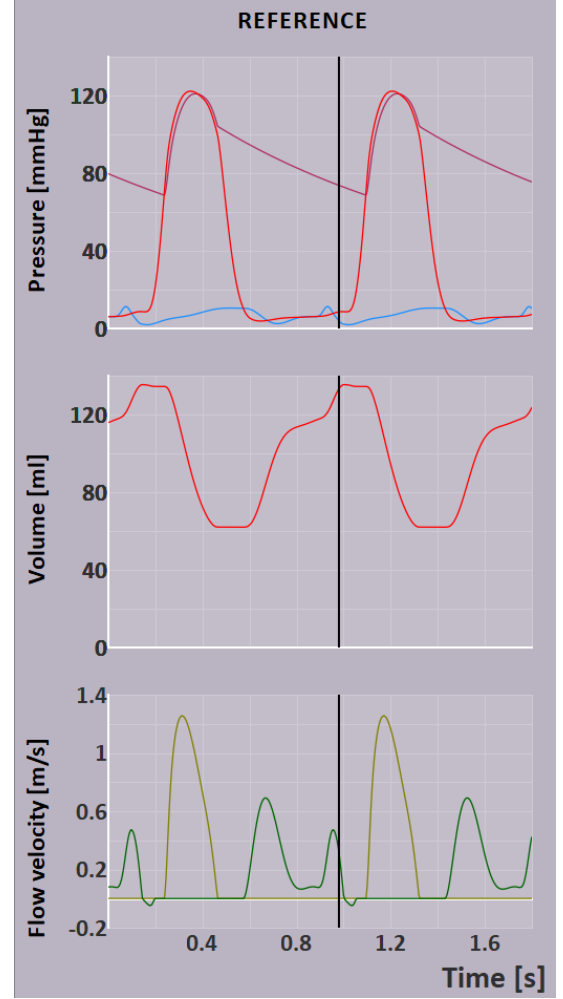
By examining the graphs, it's evident when the aortic valve opens and closes. During this period, the pressure in the left ventricle reaches its peak, indicating the maximum force required to propel blood into systemic circulation. Simultaneously, the volume of the left ventricle decreases, confirming that blood is being ejected into the aorta. This

observation is further supported by the third graph, where the aortic valve flow rate, shown in lime green, displays a sharp spike—signifying a rapid and forceful outflow of blood into the aorta.

2.2 (b) Opening of Mitral Valve



(a) Mitral valve open



(b) Mitral valve close

Figure 3: Timestamp of opening and closing of mitral valve

Parameter	Valve open	Valve close
Time(s)	0.58	1.01
Left atrial pressure(mmHg)	10.4	2.13
Left ventricular pressure(mmHg)	8.89	8.61
Left ventricular volume(ml)	62	135

Table 2: Mitral valve state determining parameters

The opening of the mitral valve is indicated by the left atrial pressure briefly exceeding the left ventricular pressure (see Table 2). This pressure difference allows the left ventricle

to begin refilling, which is visible in the volume graph (Graph 2 of Figure 3). The valve's state is further validated by the dark green curve in Graph 3, representing the mitral valve's flow rate. Notably, the flow curve shows two distinct peaks, indicating that ventricular refilling occurs in two phases—a pattern that is clearly reflected in the volume changes shown in the graph.

2.3 (c) Pressure-Volume graph of left ventricle

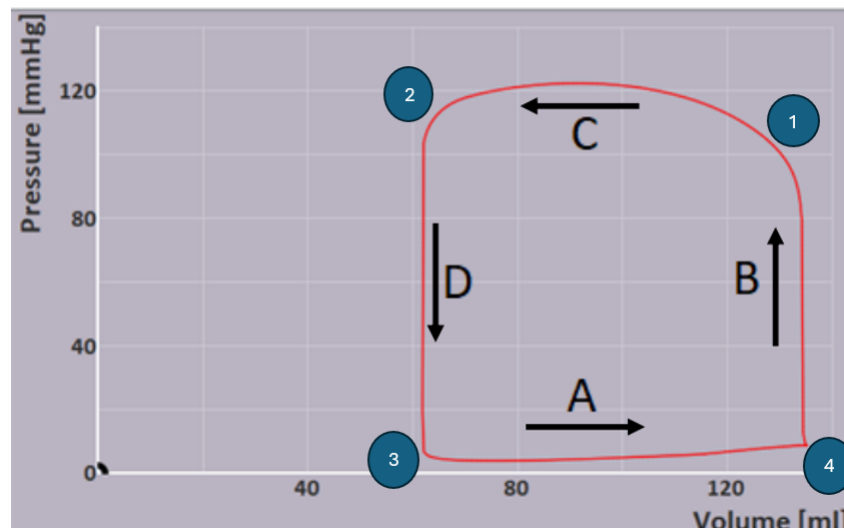


Figure 4: Pressure-Volume graph of left ventricle

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

The timing of aortic and mitral valve events can be inferred from the changes in left ventricular volume. The period between point 1 and point 2 corresponds to the phase when the aortic valve is open, during which the ventricular volume decreases as blood is ejected. Conversely, between point 3 and point 4, the mitral valve is open, and ventricular volume increases as the chamber refills.

2.4 (d) Rectangular approximation of cardiac cycle

Cardiac Cycle Phases:

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

2.5 (e) Comparison of flow velocity pattern humps

The aortic valve opens during ventricular systole, allowing blood to be forcefully ejected into the aorta. This results in a single prominent peak in the aortic flow graph. In contrast, the mitral valve opens during diastole, and its flow occurs in two distinct phases. Initially, blood flows rapidly from the left atrium to the left ventricle due to a pressure gradient—this is known as Early Rapid Filling (E) and is passive. Subsequently, during atrial systole, the atrium contracts to actively push the remaining blood into the ventricle, referred to as the Atrial Contraction phase (A). These two stages appear as two separate peaks in the mitral valve flow curve.

2.6 (f) Atrial pressure check

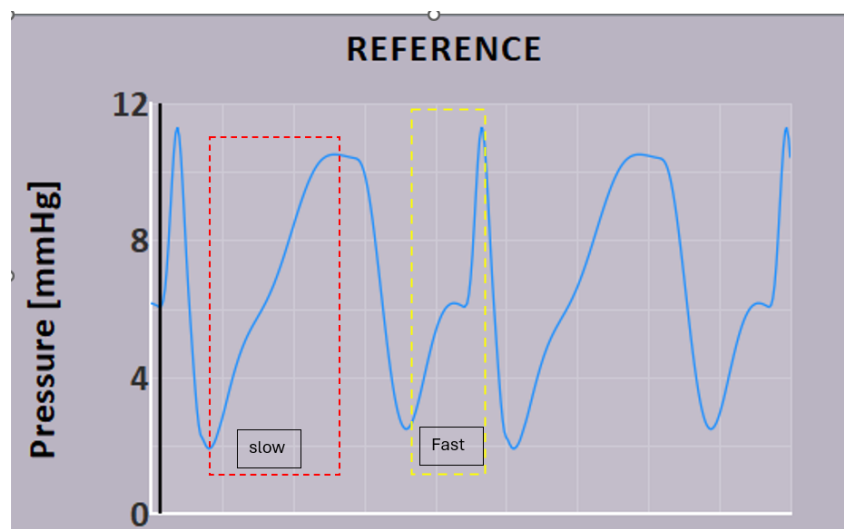


Figure 5: The Early Rapid Filling (E) and Atrial Contraction (A)

The sharp rise in atrial pressure corresponds to atrial systole, during which the atria actively contract to push the remaining blood into the ventricles. In contrast, the gradual increase in atrial pressure occurs during diastole, a phase when both the atria and ventricles are relaxed. During this time, the left atrium fills passively with blood returning from the pulmonary veins, leading to a slow buildup of pressure. The ECG segment that aligns with the steep increase in atrial pressure is the QRS complex, which also marks the onset of ventricular contraction.

2.7 (g) E, A waves of Mitral valve flow

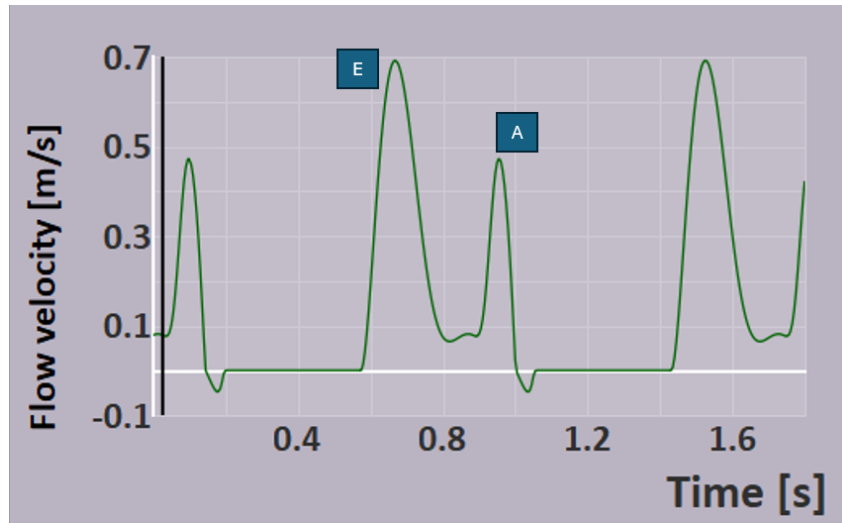


Figure 6: The slow and fast phases of atrial pressure increase in the left atrium

2.8 (h) E/A ratio calculation

Peak	Time(s)	Flow velocity(m/s)
E	0.68	0.683
A	0.96	0.46

Table 3: Mitral valve flow

The E/A ratio is defined as:

$$\text{E/A Ratio} = \frac{E}{A}$$

where E is the peak velocity of early (passive) filling, and A is the peak velocity of atrial (active) filling.

$$\text{E/A Ratio} = \frac{E}{A} = 1.485$$

The calculated E/A ratio is above 1 therefore diastolic function is healthy.

2.9 (i) Passive and Active filling of left ventricle

Instance	Time(s)	Left Ventricular Volume(ml)
Mitral valve closed	0.58	65
E peak	0.81	114
A peak	1.06	134

Table 4: Volume of Left Ventricle

Filling due to passive filling = $114 - 65 = 49ml$

Filling due to active filling = $134 - 114 = 20ml$

Passive filling volume is about twice the active filling volume.

2.10 (j) Relation between blood flow velocity and flow rate

$$Q = v \cdot A$$

where Q is the flow rate (e.g., in L/min), v is the blood flow velocity (e.g., in cm/s), and A is the cross-sectional area of the blood vessel (e.g., in cm^2) [ref2].

3 Aortic Valve Stenosis

3.1 (a) Preload and afterload

Preload refers to the initial stretching of the cardiac myocytes prior to contraction. It is closely related to the end-diastolic volume (EDV), which determines the amount of ventricular filling. **Afterload** is the pressure the heart must work against to eject blood during systole. It is influenced by factors such as systemic vascular resistance and aortic pressure.

3.2 (b) Simulating Aortic Valve Stenosis (AS)

Stenosis (%)	Peak Pressure (mmHg)	Peak velocity(m/s)	Peak Volume (ml)
0	121	1.25	135
5	122	1.30	135
10	122	1.38	135
15	123	1.46	135
20	124	1.53	135
25	125	1.63	135
30	126	1.73	135
35	127	1.84	136
40	128	1.97	136
45	130	2.11	136
50	132	2.28	136
55	135	2.47	136
60	139	2.69	136
65	143	2.95	137
70	149	3.28	138
75	158	3.68	139
80	171	4.2	141

Table 5: change of peak pressure , peak velocity, peak volume with stenosis

According to Table 5, the peak flow velocity through the aortic valve increases progressively with the percentage of stenosis. This observation is expected based on the equation

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

where the cross-sectional area of the valve (A_{valve}) decreases as stenosis worsens, while the flow rate (Q_{valve}) remains relatively constant. Consequently, the flow velocity (V_{valve}) must increase. Additionally, the peak left ventricular pressure shows a marked rise beyond 20% stenosis, whereas the increase in peak left ventricular volume is more gradual. This pressure-volume relationship is further illustrated in Figure 7. At 80% stenosis, the maximal left ventricular pressure reaches 171 mmHg.

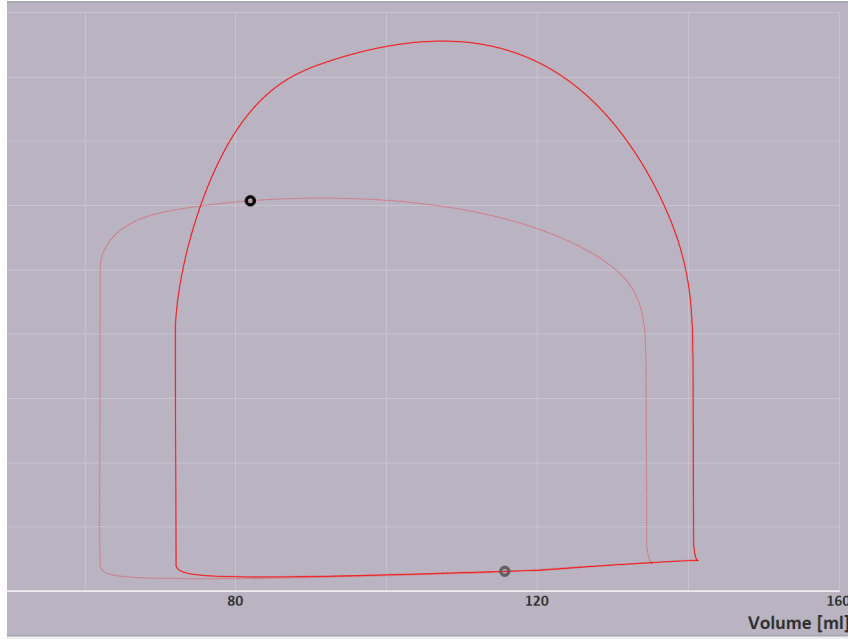


Figure 7: P-V diagram at 80% stenosis (dashed line normal condition)

3.3 (c) Effect of Aortic Stenosis on Preload, Afterload and Cardiac Output

Aortic stenosis (AS) significantly impacts cardiac function by altering preload, afterload, and cardiac output.

- **Preload:** The narrowing of the aortic valve limits the amount of blood the ventricles can eject. Consequently, some blood remains in the ventricles, leading to accumulation and increased preload.
- **Afterload:** Afterload increases directly due to the higher resistance against blood flow caused by the reduced cross-sectional area of the stenotic valve.
- **Cardiac Output (CO):** Since cardiac output is the product of stroke volume and heart rate, the increased preload can elevate stroke volume, potentially increasing cardiac output despite the stenosis.

3.4 (d) Pressure drop across the stenotic aortic valve

Aortic pressure during maximum left ventricular pressure is **103 mmHg**.

$$\text{Pressure drop} = 171 - 103 = 68 \text{ mmHg}$$

3.5 (e) Maximal pressure drop using formula

Maximum flow velocity of the aortic valve is $v = 4.2 \text{ m/s}$.

Pressure drop (ΔP) is calculated using the formula:

$$\Delta P = 4v^2 = 4 \times (4.2)^2 = 4 \times 17.64 = 70.56 \text{ mmHg}$$

This calculated value is close to the measured value.

3.6 (f) Calculation of duration of ejection

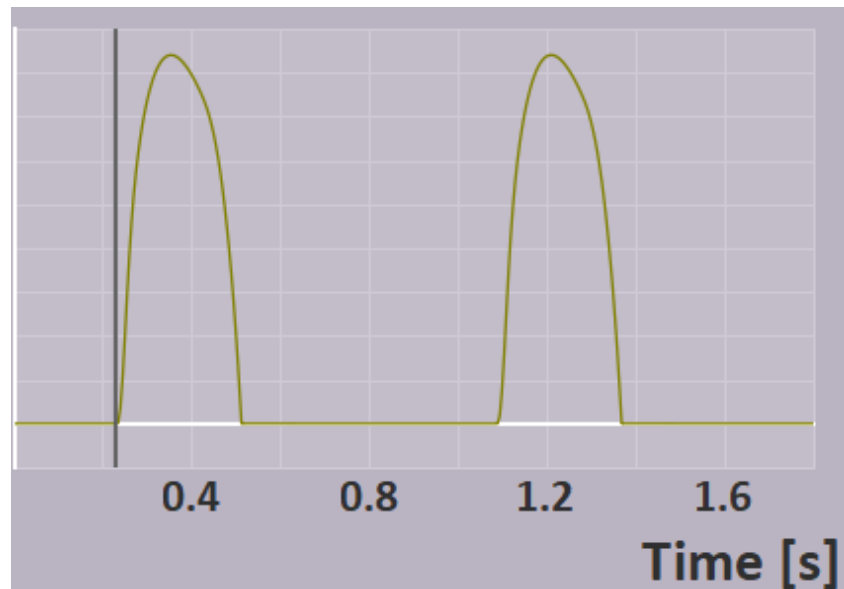


Figure 8: flow velocity diagram with 80 % stenosis

Time taken to eject ventricular blood through the narrowed aortic valve is calculated as follows:

$$\text{Duration of ejection} = 0.53 \text{ s} - 0.23 \text{ s} = 0.30 \text{ s}$$

3.7 (g) External pump work by left ventricle

Using Figure 7:

- External pump work in a normal person ≈ 20 squares
- External pump work at 80% stenosis ≈ 23 squares
- Increase in pump work ≈ 3 squares

3.8 (h) Change in the myocardial tissue of left ventricle [ref3]

To cope with the chronically increased pump work caused by aortic stenosis, the myocardial tissue of the left ventricle undergoes **hypertrophy**, resulting in thickening and enlargement of the ventricular muscle. This adaptation increases the contractile strength, allowing the ventricle to generate the higher pressures required to overcome the elevated afterload.

However, ventricular hypertrophy leads to a stiffer ventricular wall, which may impair relaxation and filling. Additionally, the increased muscle mass raises myocardial oxygen demand and can further increase the **afterload**, thereby imposing an even greater workload on the heart.

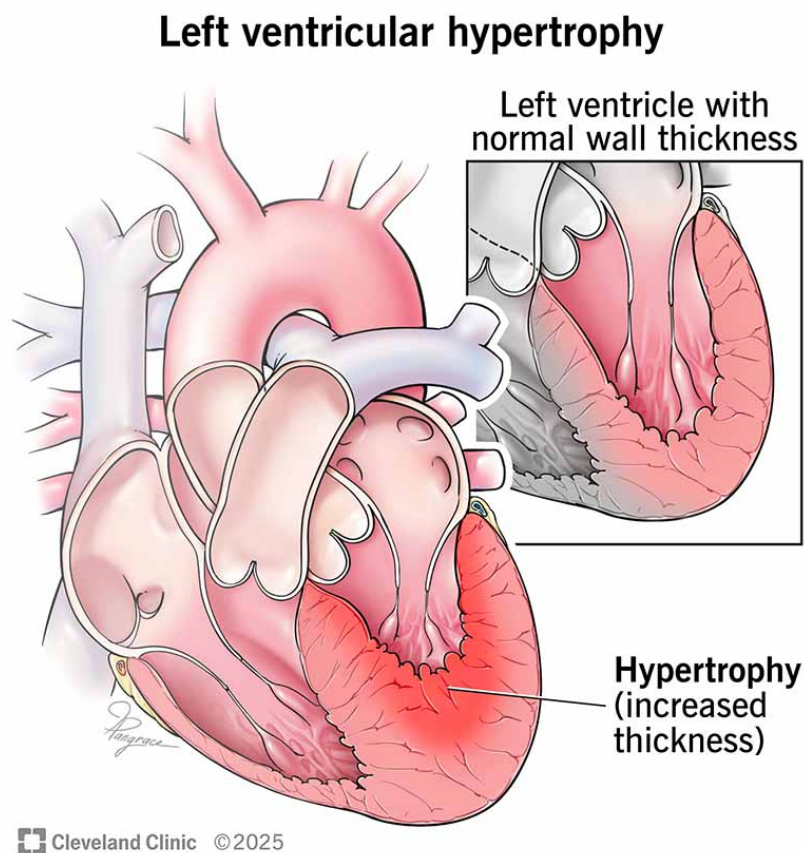


Figure 9: Left Ventricular Hypertrophy [4]

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