Department of Electronic & Telecommunication Engineering,
University of Moratuwa, Sri Lanka.

A2: Analysis of Cardiac Physiology

I.A Withanawasam 210732H

Submitted in partial fulfillment of the requirements for the module
BM2102 Modelling and Analysis of Physiological Systems

11th March 2024

Contents

Introduction

In this report, we investigated the effects of aortic valve stenosis and normal heart rhythm using the CircAdapt simulation software. With the Wiggers diagram serving as a basis, we broke down a number of cardiac metrics, such as left ventricular pressure, atrial pressure, aortic pressure, and ventricular volume. Our comprehension of cardiac dynamics was further enhanced by the investigation of blood flow velocities and the pressure-volume relationship. We attempted to apply theoretical knowledge to real-world situations by using practical exercises like determining the opening and closing points of valves, figuring out the E/A ratio, and simulating an 80 percent aortic valve stenosis.

1 Normal Sinus Rhythm

1.1 Wiggers Diagram

The different pressures in the atrium, ventricle, and artery during a single cardiac cycle are shown in this Wigger's diagram.

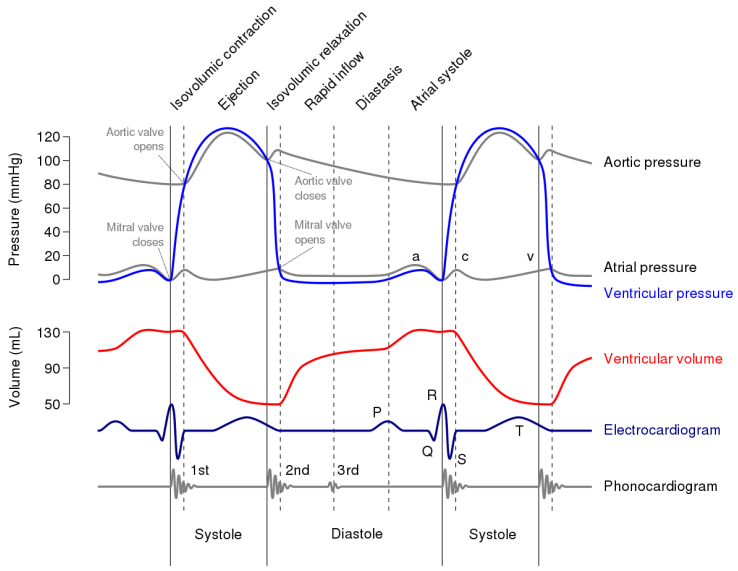


Figure 1: Wiggers Diagram

There are differences in intracardiac pressure between the left and right sides of the heart. Because the left side must pump blood throughout the body while the right side simply needs to pump blood through the lungs, the left side is under more pressure than the right. Nonetheless, each side's diagram would show the same shape.

1.2 Comparison

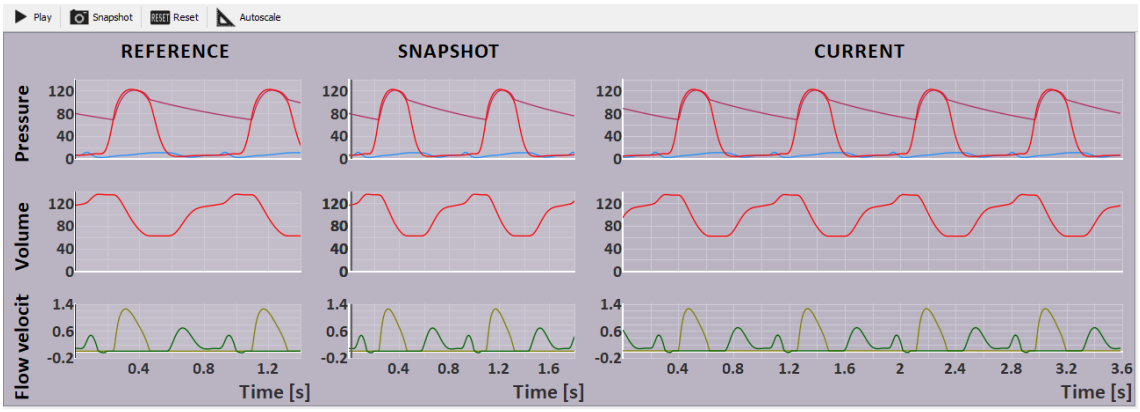


Figure 2: Simulation Output

In the simulation we have simulated,

- Pressure variations of Aortic, Left ventricle and Left atrium
- Volume variation of Left ventricle
- Flow velocity of Aortic valve and Mitral valve

By comparing with the Wiggers Diagram, we can see that the variations are equal in the two graphs, except for Electrocardiogram and Phonocardiogram which are not visible in the simualtion outputs.

Diastole (Relaxation Phase)

1. **Isovolumic relaxation**: Following ventricular contraction, this signals the start of diastole and the closing of the aortic valve. Because of the trapped blood, the ventricles are relaxing, but the pressure is still briefly high. During this stage, the mitral valve stays closed. The aortic valve closing causes the first heart sound, lub, to be produced.
2. **Rapid inflow**: When the pressure in the ventricles drops below the pressure in the atria, the mitral valve opens. This causes a sudden surge in ventricular pressure by generating a fast blood rush from the atria into the ventricles.
3. **Diastasis**: The pace of inflow decreases as the ventricles passively fill. The ventricular pressure gradually rises throughout this phase, although it is still lower than the aortic pressure.
4. **Atrial Systole**: The atria contract, completing the last push of ventricular filling. The ventricular pressure curve shows a slight increase in response to this (a wave). Atrial systole is represented by the P wave on the ECG.

Systole (Contraction Phase)

1. **Isovolumic contraction**: The blood is compressed inside the ventricles when they start to contract. But at this moment, the aortic and mitral valves are closed. With no change in volume, this contraction raises the ventricular pressure. The mitral valve closing is what produces the second heart sound, dub.
2. **Ejection**: Blood is expelled from the ventricles into the aorta when ventricular pressure surpasses aortic pressure, causing the aortic valve to open. The peak of the aortic pressure curve corresponds to this phase.

Explanations of graphs

1. Variations in Pressure
    - Aortic Pressure: Indicates the aorta's pressure. It increases in diastole and decreases in ventricular ejection during the cardiac cycle.
    - Left Ventricular Pressure: The left ventricle's internal pressure is reflected by the left ventricle pressure. increases during diastole, or rest, and drops during ventricular contraction, or systole.
    - Left Atrial Pressure: Shows the left atrium's pressure. When the atrium contracts to force blood into the ventricle during late diastole, it rises.
2. Variation in Left Ventricle Volume
    - Left Ventricular Volume: Shows how the left ventricle's volume varies during the cardiac cycle. It rises when the ventricle fills with blood during diastole and falls as blood is expelled into the aorta during systole.
3. Flow Velocity
    - Aortic Valve Flow Velocity: The speed at which blood passes through the aortic valve . During ventricular ejection, it peaks.
    - Mitral Valve Flow Velocity: The speed at which blood passes through the mitral valve. It is correlated with the left ventricle filling up during diastole.

1.3 Explanations on Normal Sinus Rhythm

â-01 - Aortic Valve Opening
The left ventricle contracts during systole, causing its pressure to rise quickly. The aortic valve opens when the pressure inside the left ventricle is higher than the pressure outside the vessel. At this critical point, the left ventricle's oxygenated blood can be released into the aorta, starting the ejection phase. In this period, the aortic valve's flow velocity peaks, guaranteeing effective blood ejection into the aorta.

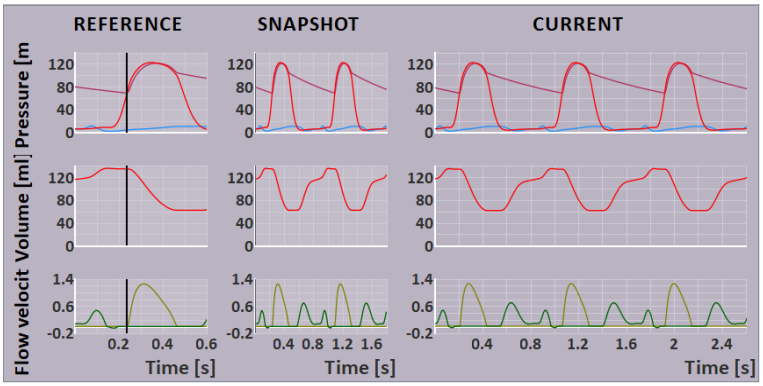


Figure 3: Aortic Valve Opening

a-02 - Aortic Valve Closing

The end of the cardiac cycle's ejection phase occurs during systole when the aortic valve closes. The pressure in the aorta eventually rises above the pressure in the left ventricle as a result of the left ventricle contracting and expelling blood into it. The aortic valve closes as a result of this pressure reversal, stopping blood from returning to the ventricle during diastole. As the ventricle relaxes and gets ready for the next cycle, the left ventricular pressure starts to drop. The aortic valve's flow velocity lowers in tandem with the decreasing pressure gradient between the aorta and left ventricle.

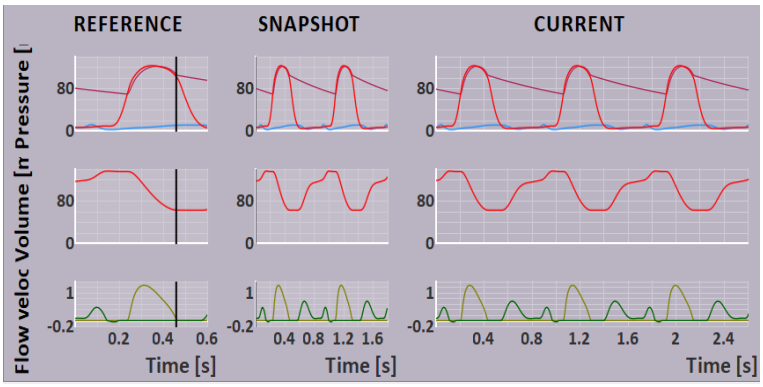


Figure 4: Aortic Valve Closing

b-01 - Mitral Valve Opening

The left ventricle's (LV) pressure decreases when it completes its contraction (systole) and starts to relax (diastole). The left atrium's pressure, which has been passively accumulating blood, is lower than the left ventricle's reduction in pressure. The mitral valve opens as a result of this pressure differential acting as a trigger. The left atrium's blood then swiftly flows into the left ventricle (LV) through the open mitral valve. The main process for filling the ventricle is this rapid input.

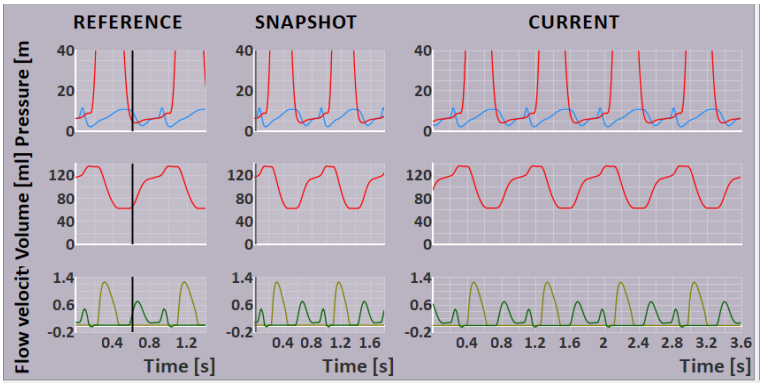


Figure 5: Mitral Valve Opening

b-02 - Mitral Valve Closing

The left ventricle (LV) experiences a spike in pressure during its forced contraction (systole) to expel blood. The left atrium's pressure, which has recently emptied, is less than the rise in left ventricular pressure. The mitral valve slams shut in response to this pressure reversal, stopping blood flow back into the atrium. Mitral valve closure, which effectively stops the flow from the atrial, is accompanied by a sudden drop in flow velocity through the valve.

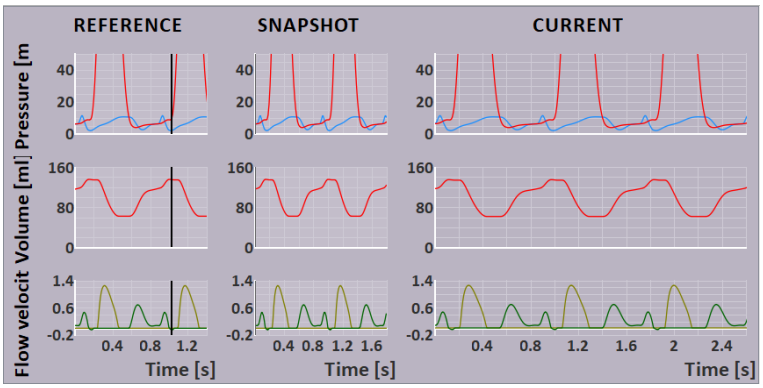


Figure 6: Aortic Valve Closing

c. Pressure Volume Relation

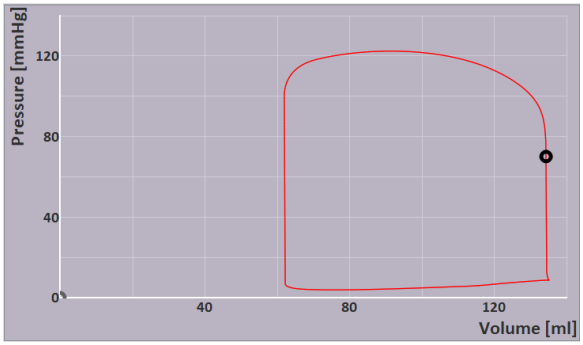


Figure 7: Aortic Valve Opening

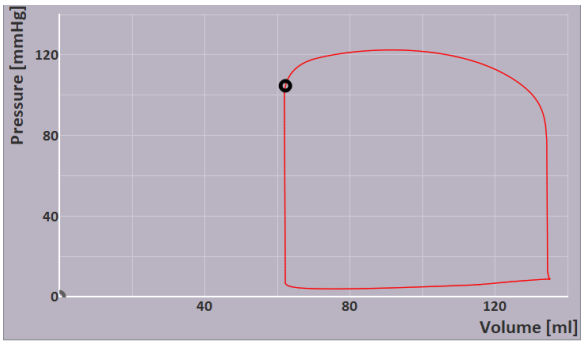


Figure 8: Aortic Valve Closing

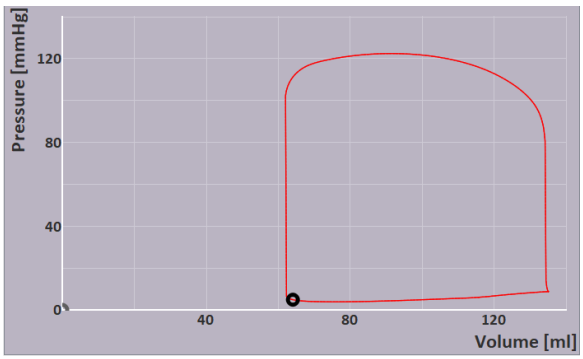


Figure 9: Mitral Valve Opening

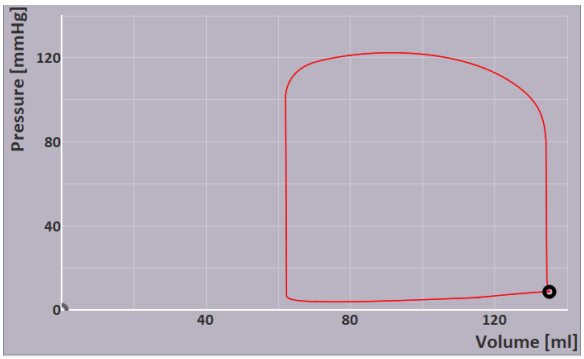
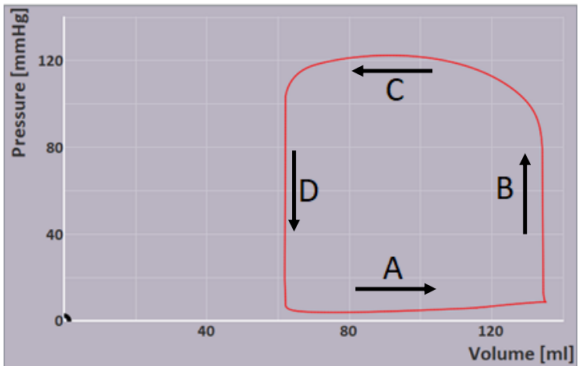


Figure 10: Mitral Valve Closing

d. Phases corresponding to the sides



- A - Rapid Inflow
- B - Isovolumic Contraction
- C - Ejection
- D - Isovolumic Relaxation

Figure 11: Pressure - Volume relationship

e. Aortic and Mitral Flow Velocity

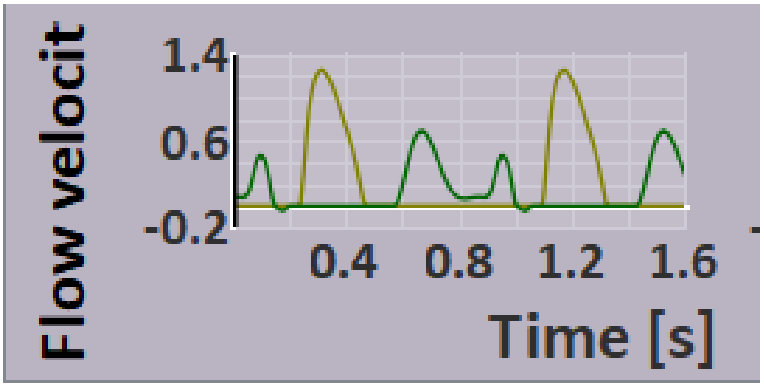


Figure 12: Flow Velocity graphs of Mitral and Aortic valves

The different processes controlling the opening and closure of the aortic and mitral valves can be responsible for their differing flow velocity patterns.

The aortic valve has a single-hump flow pattern and only opens during ventricular ejection when left ventricular pressure is higher than aortic pressure.

As opposed to this, the mitral valve opens in two stages during diastole: the first hump occurs during the passive phase, which occurs when left atrial pressure exceeds left ventricular pressure, and the second hump occurs during the active phase, which occurs during atrial contraction.

The interaction of passive and active filling mechanisms is reflected in this dual-hump flow pattern, which highlights the complex dynamics of blood flow control in the cardiac cycle.

f. Atrial Pressure

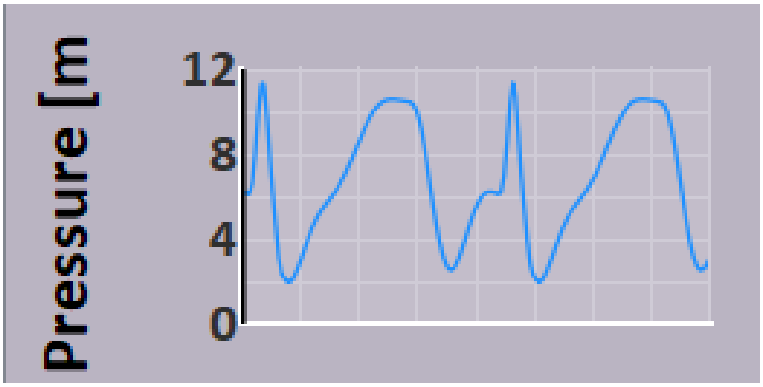


Figure 13: Atrial Pressure graph

Fast (Steep) Increase of Atrial Pressure

- Cause: During atrial systole, the atrial contraction is responsible for this sharp spike. Before ventricular systole starts, the atria constrict to force the remaining blood into the ventricles.
- ECG Correspondence: The P-wave is the ECG waveform that corresponds with this sharp rise. Atrial depolarization, or the electrical activity connected to atrial contraction, is represented by the P-wave.

Slow Increase of Atrial Pressure

- Cause: Diastasis, or the gradual and slower increase in atrial pressure, happens during the ventricles' filling phase. As blood flows from the atria, the ventricles passively fill during this period.
- Correspondence of ECG: There may not be a distinct ECG waveform that is directly connected with the steady increase in atrial pressure that corresponds with the diastasis phase. The ECG may show a reasonably constant or flat segment during diastasis.

g. E and A waves of Mitral Blood Flow

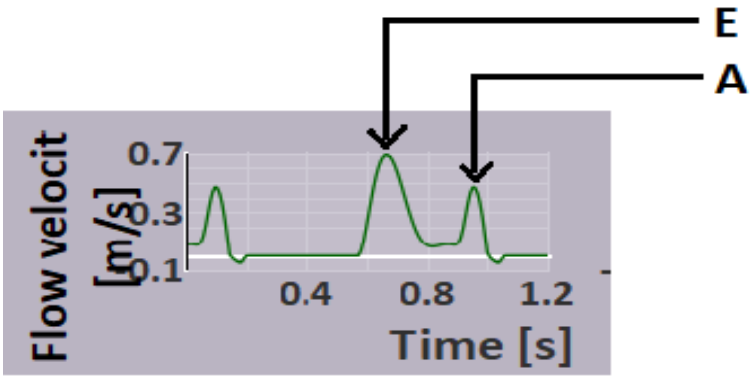


Figure 14: E and A waves in Mitral valve flow velocity

h. E/A Ratio

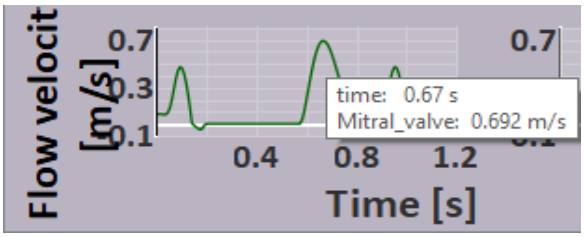


Figure 15: E wave parameters

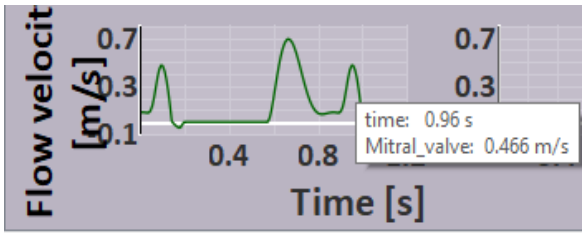


Figure 16: A wave parameters

$$\frac{E}{A} = \frac{0.692}{0.466} = 1.485$$

i. Volume

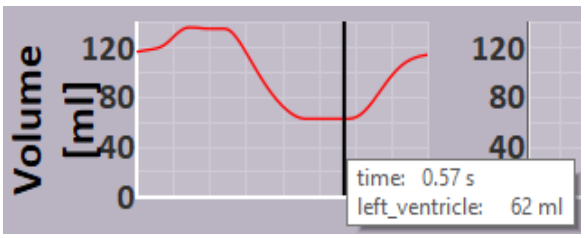


Figure 17: Starting of the E wave

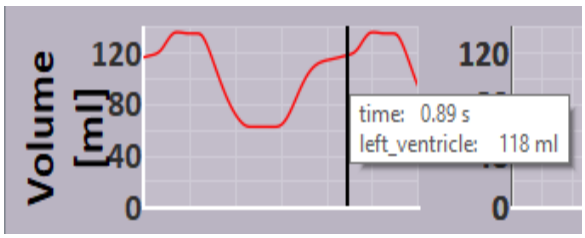


Figure 18: Starting of the A wave

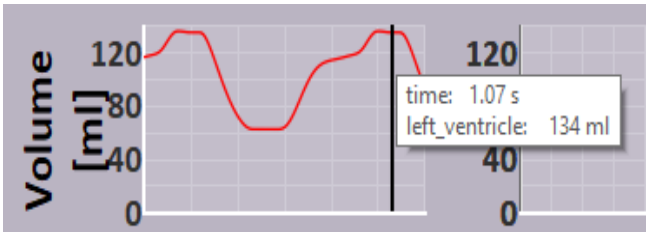


Figure 19: Ending of the E wave

1. Amount due to passive filling = 118 ml - 62 ml = 56 ml
2. Amount due to active filling = 134 ml - 118 ml = 16 ml

j. To convert the blood flow velocity into flow rate we need to know the cross sectional area of the valve.

2 Aortic Valve Stenosis

2.1 Explanations on Aortic Valve Stenosis

a. Preload and Afterload

- **Preload**: Preload, which is based on the volume of blood in the ventricles at the conclusion of diastole, is the stretch of the cardiac fibers immediately prior to the heart contracting. According to the Frank-Starling mechanism, more preload causes a stronger contraction during systole, which maximizes cardiac output. Venous return and blood volume are important variables that affect preload and provide information about how well the heart can pump incoming blood. In diseases like heart failure, where abnormalities in volume regulation affect cardiac function, preload monitoring is essential.

- **Afterload**: The resistance the heart encounters when pumping blood into the arteries is known as afterload, and it is indicated by the pressure required to open the aortic or pulmonary valve. Afterload is influenced by arterial resistance, compliance, and pressure. Conditions such as hypertension increase afterload, which makes it more difficult for the heart to pump blood efficiently. Increased cardiac effort from elevated afterload may result in an increased demand for oxygen by the heart. It's crucial to comprehend and control afterload, particularly in cardiovascular diseases like aortic valve stenosis where the heart's ability to pump blood efficiently is hampered by increased resistance.

b. Simulation of Aortic Stenosis(AS) of 80 percent

Results of pressure and velocity changes when we gradually increase the percentage of stenosis from 5% to 80%

Percentage of Stenosis	Left Ventricular maximum pressure (mmHg)	Aortic pressure (mmHg)	Aortic valve flow velocity (m/s)
0%	121	121	0.93
5%	123	120	1.17
10%	123	119	1.33
15%	124	119	1.4
20%	124	118	1.45
25%	125	117	1.57
30%	126	117	1.64
35%	127	116	1.74
40%	128	116	1.93
45%	130	116	2.05
50%	132	115	2.25
55%	135	114	2.45
60%	138	111	2.67
65%	143	112	2.85
70%	150	110	3.21
75%	158	105	3.68
80%	171	104	4.16

Figure 20: Pressure and flow veloctiy changes

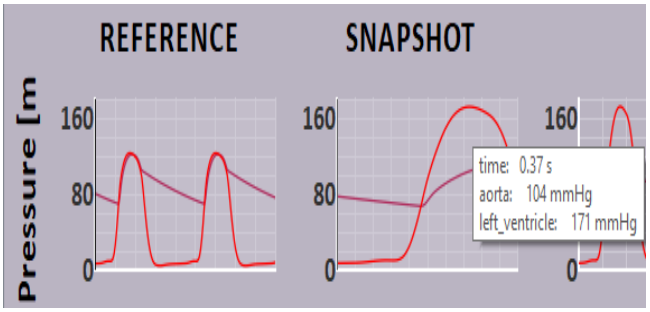


Figure 21: Pressure variation of normal condition and AS 80%

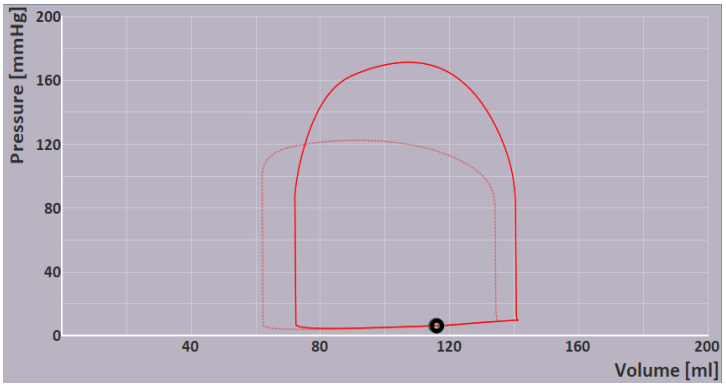


Figure 22: Pressure - Volume realtionship variation of normal condition and AS 80%

There are notable hemodynamic alterations in the case of severe aortic stenosis (AS) with an 80 percent reduction in the aortic valve area. Because of the higher resistance, the left ventricle, which pumps blood through the constricted aortic valve, experiences greater pressure. In an attempt to remove the obstruction, this increased left ventricular pressure may eventually cause the ventricular walls to enlarge.

Simultaneously, the decreased effectiveness of blood ejection into the aorta causes an increase in pressure in the left atrium. This elevated left atrial pressure has the potential to cause volume overflow and eventually atrial hypertrophy.

There is a pressure drop in the aorta downstream of the stenotic valve. The constricted valve prevents effective ejection, which lowers aortic pressure even when the heart tries to pump blood into the aorta.

An analysis of the left ventricle's pressure-volume relation shows a changed pattern. Higher pressures must be produced by the left ventricle in order to force blood through the constricted valve, which increases afterload. This higher pressure-volume relationship is the heart's way of making up for the extra effort that the severe aortic stenosis is putting on it. These hemodynamic alterations over time may be a factor in symptoms like exhaustion, chest pain, and impaired heart function.

c. Effects of Aortic Valve Stenosis

- **Preload in Aortic Valve Stenosis**: The left ventricle works harder when the aortic valve narrows in AS. The stenosis prevents blood from being ejected into the aorta efficiently, which results in partial left ventricular emptying during systole. Preload rises as a result of an increase in the left ventricular end-diastolic volume. The narrowed valve creates resistance for the heart to beat against, which causes the ventricular muscle fibers to stretch more during diastole.

- **Afterload in Aortic Valve Stenosis**: The left ventricle's afterload is markedly increased with aortic valve stenosis. The heart has to beat over extra resistance caused by the constricted valve in order to pump blood into the aorta. Because of the increased force required during systole due to this elevated afterload, the left ventricle finds it more difficult to effectively pump blood into the systemic circulation. Ventricular hypertrophy, the body's compensatory response to the stenotic valve's higher strain, is facilitated by the increased afterload.

- **Effect on Cardiac Output**: Heart output is negatively affected by the higher preload and afterload associated with aortic valve stenosis. The left ventricle finds it difficult to continue ejecting blood efficiently due to both increasing volume and resistance. When the heart loses its ability to pump enough blood to meet the body's needs, this might eventually cause a decrease in cardiac output. Severe aortic valve stenosis can cause symptoms including exhaustion and chest pain, which can eventually impair heart function as a whole.

d. Pressure difference across the stenotic aortic valve

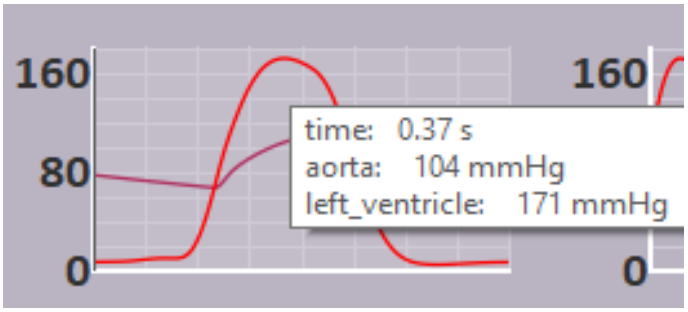


Figure 23: Pressure Values when having 80% Aortic Stenosis

Maximum left ventricle pressure = 171 mmHg

Aortic pressure when maximum pressure occurs in the left ventrcile = 104 mmHg

Pressure difference across the stenotic aortic valve = 171 mmHg - 104 mmHg = 67 mmHg

e. Alternative method to calculate Pressure difference across the stenotic aortic valve

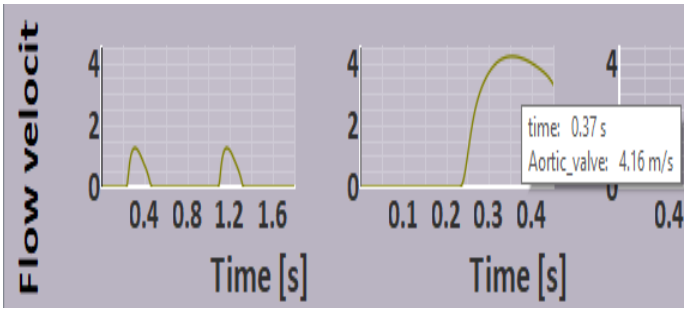


Figure 24: Aortic Valve Flow Velocity when AS 80%

$$\Delta P \approx 4V^2$$
$$\Delta P \approx 4X4.16^2$$
$$\Delta P \approx 69.224\ mmHg$$

The two methods for determining the maximum pressure drop across the stenotic aortic valve agree, as seen by the numbers, which are rather similar.

e. Time Duration of Ejection

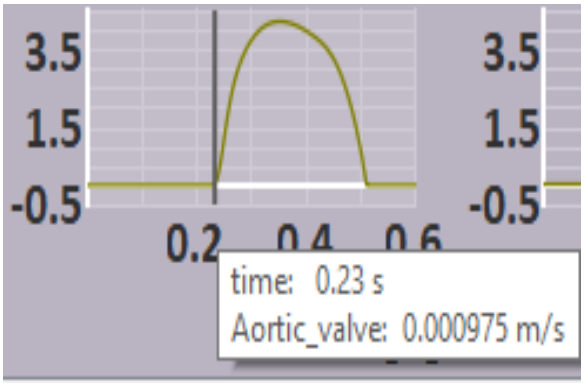


Figure 25: Beginning of the ejection - Aortic Valve Flow velocity

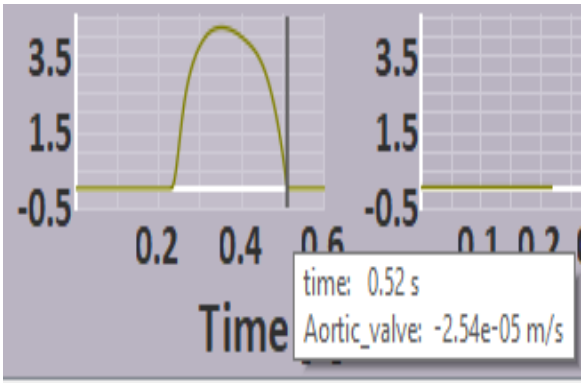


Figure 26: Ending of the ejection - Aortic Valve Flow velocity

The time duration = 0.52 s - 0.23 s = 0.29 s

e. Pressure Increase

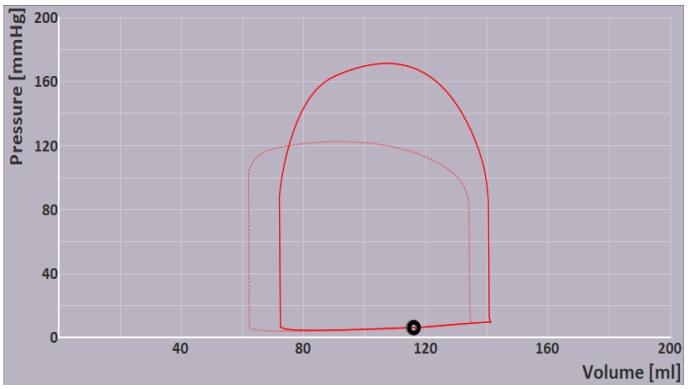


Figure 27: Pressure - Volume realtionship variation of normal condition and AS

Number of squares without Aortic stenosis(AS) $\approx 20 squares$

Number of squares with 80% AS $\approx 24 squares$

Increase of pressure $\approx 4 squares \approx \times 20 \times 20 \approx 1600$ mmHg(squares)

Chronically increased pump work in the left ventricle (LV), which is common in situations such as aortic stenosis (AS), causes the myocardial tissue to develop a process called hypertrophy. An adaptive reaction called cardiac hypertrophy aims to make the left ventricle thicker and more muscular. The goal of this adaptation is to improve the heart's capacity to produce the force required to overcome the elevated afterload brought on by pumping blood through the constricted aortic valve.

Myocardial hypertrophy mostly affects the left ventricle in the following ways:

- **Increased Muscle Mass**: Hypertrophy, or the enlargement of individual cardiac cells within the left ventricle, results in an overall increase in muscle mass. This facilitates the ventricle's ability to contract with more force.
- **Ventricular Wall Thinning**: In order to handle the increasing strain, the left ventricle's walls thicken. Maintaining cardiac output in the face of the stenotic aortic valve's increased resistance requires this structural alteration.
- **Improved Contractility**: A higher contractile function is linked to myocardial hypertrophy. Because of this adaptation, the left ventricle can contract with greater force, making up for the increased afterload and promoting efficient blood ejection into the systemic circulation.

References

1. The Cardiac Cycle `https://geekymedics.com/the-cardiac-cycle/`
2. Left Ventricular Pressure and Volume Changes (Wigger's Diagram) `https://ditki.com/course/physiology/glossary/term/left-ventricular-pressure-volume-changes`
3. Preload and Afterload: A Cardiac Output Overview `ttps://simplenursing.com/preload-and-afterload/`
4. Tricuspid Regurgitation `https://www.youtube.com/watch?v=cO6B9Q3At1Y`