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Analysis of Physiological Systems

Assignment on Analysis of Cardiac Physiology

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

2024/03/11

Contents

1 The Cardiac Cycle

The cardiac cycle is a rhythmic sequence of events that describes the pumping action of the heart.

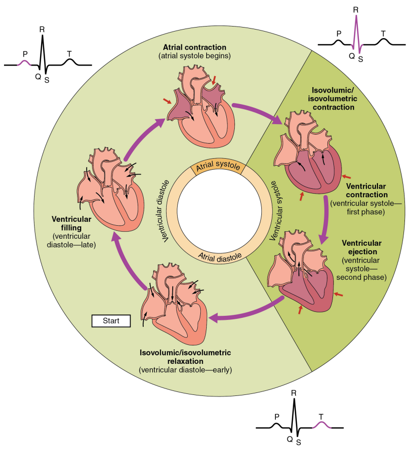

Figure 1: Cardiac Cycle
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1.1 Parts of Cardiac Cycle

1.1.1 Diastole

It begins with diastole, the relaxation phase. During diastole, the ventricles are filled with blood as the mitral and tricuspid valves open to allow blood to flow in from the atria.

1.1.2 Atrial Contraction

Following diastole, the atria contract in a coordinated manner, propelling additional blood into the ventricles. This phase, known as atrial systole, contributes to the completion of ventricular filling before the onset of ventricular contraction.

1.1.3 Ventricular Contraction (Systole)

The next phase of the cardiac cycle is ventricular systole, where the ventricles contract forcefully. This contraction leads to the closure of the mitral and tricuspid valves to prevent the backflow of blood into the atria. Simultaneously, the pressure within the ventricles rises, causing the aortic and pulmonary valves to open, allowing blood to be ejected into the aorta and pulmonary artery, respectively.

1.1.4 Ventricular Relaxation

Following the ejection of blood, the ventricles enter a phase of relaxation known as ventricular diastole. During this period, the aortic and pulmonary valves close to prevent blood from flowing back into the ventricles. The cycle then repeats, maintaining a continuous and coordinated flow of blood throughout the cardiovascular system.

1.2 Physiological Parameters

- Left Ventricular Pressure
- Left Atrial Pressure
- Aortic Pressure
- Left Ventricular Volume
- Blood Flow Rate of Aortic Valve
- Blood Flow Rate of Mitral Valve

2 Normal Sinus Diagram

2.1 Wiggers Diagram

The Wiggers diagram, named after Dr. Carl Wiggers, is a visual representation used in cardiovascular physiology to illustrate key events during a cardiac cycle. It includes waveforms of parameters like ECG, ventricular pressure, and volume changes, providing a comprehensive view of the heart's electrical and mechanical activities. This diagram aids in understanding the complex coordination of events during each heartbeat.

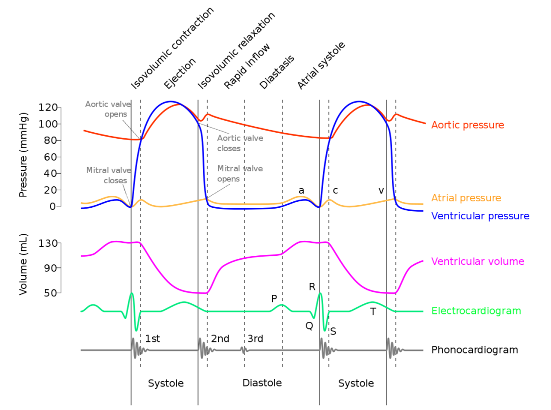


Figure 2: Wiggers Diagram of Cardiac Cycle

2.2 Identify the phases during which the Aortic valve is open and closed.

2.2.1 Aortic Valve Opening

The initiation of the aortic valve opening aligns with the contraction of the left ventricle, facilitating the pumping of blood into the aorta. During this phase, the left ventricular pressure, measured at 69.8 mmHg, surpasses the aortic pressure of 68.8 mmHg. This pressure differential dictates the one-way opening of the aortic valve, directing blood flow exclusively towards the aorta. As depicted in Figure 3, the specific moment of aortic valve opening corresponds to a left ventricular volume of 134 ml. This synchronized event ensures an efficient unidirectional flow of blood from the left ventricle into the aorta, maintaining a physiological balance.

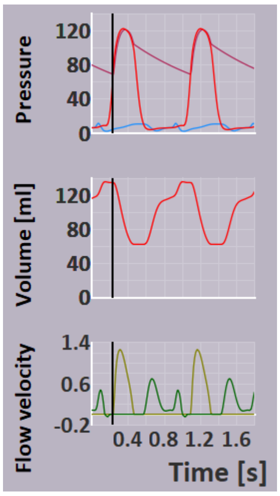


Figure 3: Aortic Valve Opening

2.2.2 Aortic Valve Closing

The closure of the aortic valve marks the conclusion of the left ventricular contraction, resulting in a reduction of left ventricular pressure. As illustrated in Figure 4, this closure leads to a state where the volume of the left ventricle remains constant. At the time of aortic valve closure, the left ventricular pressure decreases, creating a scenario where the aortic pressure exceeds that in the left ventricle. This prompts the aortic valve to swiftly close, preventing any back-flow of blood. Analysis of the cardiac physiology, considering the provided data, reveals that when the aortic valve closes, the left ventricular volume is measured at 62 ml. The difference in volumes during opening and closing indicates that 72 ml of blood has successfully passed through the aortic valve during this cardiac cycle, further highlighting the efficiency and precision of the cardiovascular system.

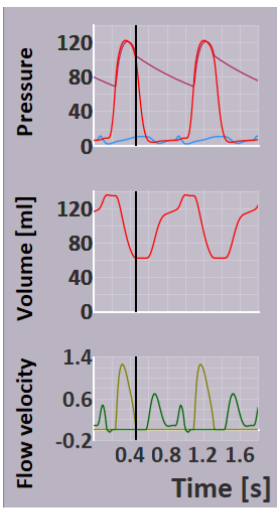


Figure 4: Aortic Valve Closing

2.3 Identify the phases during which the Mitral valve is in the open and closed states.

2.3.1 Mitral Valve Opening

The Mitral valve undergoes opening as the left ventricle enters its relaxation phase, allowing it to be filled with blood from the left atrium. During this stage, the left ventricular pressure measures 7.77 mmHg, which is notably lower than the corresponding left atrial pressure of 10.3 mmHg. This pressure gradient ensures that the Mitral valve exclusively opens towards the left ventricle, facilitating the unidirectional flow of blood into the ventricle. Simultaneously, the left ventricular volume is precisely measured at 62 ml, ensuring an efficient and controlled filling of the ventricle. This orchestrated sequence of events highlights the meticulous coordination between pressure differentials and valve dynamics during Mitral valve opening.

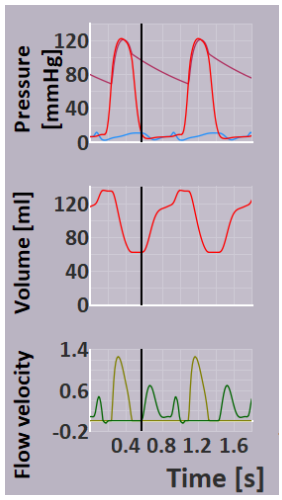


Figure 5: Mitral Valve Opening

2.3.2 Mitral Valve Closing

Conversely, the closure of the Mitral valve occurs when the left ventricle is fully filled with blood. At this point, the left ventricular pressure rises to 8.69 mmHg, while the left atrium maintains a pressure of 2.24 mmHg. The closure of the Mitral valve ensures the prevention of any back-flow of blood, maintaining the integrity of the cardiac cycle. The left ventricular volume during Mitral valve closure is precisely measured at 135 ml, denoting the completion of the filling phase. This detailed analysis elucidates the intricate dynamics involved in Mitral valve closure and underscores its crucial role in the regulation of blood flow within the cardiovascular system.

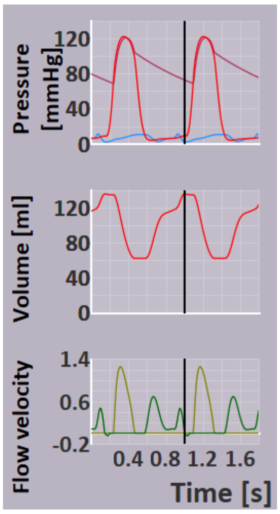


Figure 6: Mitral Valve Opening

2.4 Identify which points of the pressure-volume relation correspond to the closing and opening of the aortic and mitral valves

The left ventricular volume decreases during the opening of the aortic valve, while, conversely, the opening of the mitral valve leads to an increase in the left ventricular volume.

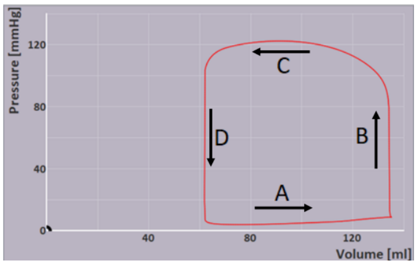
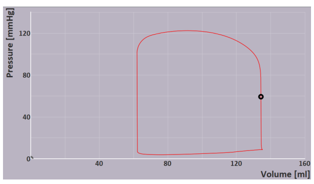


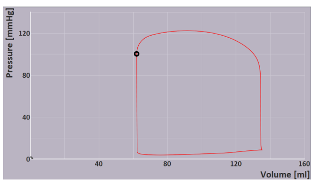
Figure 7: Cardiac Cycle

Path	Event
A	3 (mitral opening)
B	4 (mitral closing)
C	1 (aortic opening)
D	2 (aortic closing)

Table 1: Correspondence of Points to Cardiac Cycle Events

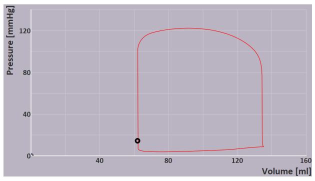


(a) Aortic Valve Opening

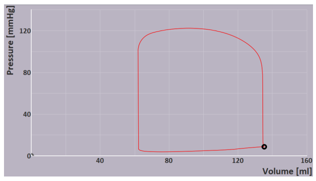


(b) Aortic Valve Closing

Figure 8: Opening and Closing of Aortic Valve



(a) Mitral Valve Opening



(b) Mitral Valve Closing

Figure 9: Opening and Closing of Mitral Valve

2.5 Identify in Relation to a Specific Stage of the Cardiac Cycle.

A:	Filling
B:	Isovolumic Contraction
C:	Ejection
D:	Isovolumic Relaxation

Table 2: Phases of the Cardiac Cycle

2.6 Reason Behind the Singular Peak in the Flow Velocity Pattern of the Aortic Valve and the Dual Peaks in the Pattern of the Mitral Valve.

The singular hump observed in the flow velocity pattern of the aortic valve is attributed to systolic activity, whereas the dual-hump pattern in the mitral valve arises from diastolic processes. The mitral valve opens during ventricular diastole, allowing blood to flow from the left atrium into the left ventricle. This distinctive two-hump pattern signifies the rapid initial filling of the ventricle during atrial contraction and the subsequent slower, passive filling phase driven by the pressure difference between the atrium and the ventricle. In contrast, the aortic valve's flow rate pattern reflects systolic activity, emphasizing the role of different cardiac phases in shaping the distinct characteristics of flow velocity patterns in these valves.

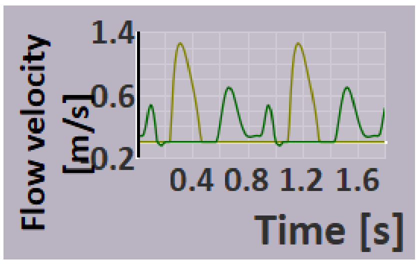


Figure 10: Flow Velocities of Aortic & Mitral Valves

2.7 Analysis of Atrial Pressure Changes in the Cardiac Cycle

In the cardiac cycle, the rapid surge in left atrial pressure during atrial systole results from atrial depolarization, indicating the forceful contraction of the atrial myocardium and coinciding with the P-wave on the electrocardiogram (ECG). Conversely, the gradual increase in left atrial pressure during ventricular systole, particularly during the isovolumetric contraction phase with the mitral valve closed, reflects passive atrial filling. This slower rise aligns with ECG changes and signifies blood inflow from the pulmonary veins as the ventricle contracts. These pressure variations offer a nuanced view of atrial dynamics, emphasizing the intricate interplay between active and passive phases in the cardiac cycle.

2.8 Identify the E- and A-waves of the Mitral blood flow velocity.

The 'E' of E-wave stands for 'early' / 'elastic' and that the 'A' of A-wave refers to 'atrial'/'active'.

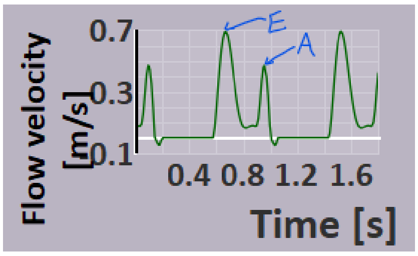
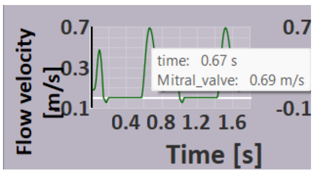
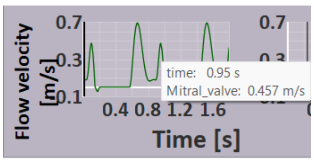


Figure 11: Flow Velocity of Mitral Valve

2.9 Calculate the E/A-ratio



(a) Velocity at E



(b) Velocity at A

Figure 12: Velocities at E & A

$$\text{E/A ratio} = \frac{0.692}{0.473} = 1.463$$

2.10 Amounts of Left Ventricular Filling

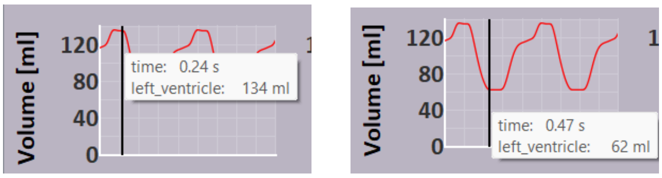


Figure 13: Respective Volume Measurements

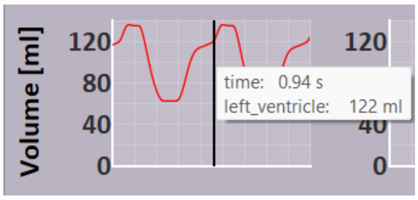


Figure 14: Respective Volume Measurements

$$\begin{aligned} \text{Due to active filling} &= 122\,\text{ml} - 62\,\text{ml} &&= 60\,\text{ml} \\ \text{Due to passive filling} &= 134\,\text{ml} - 122\,\text{ml} &&= 12\,\text{ml} \end{aligned}$$

To convert blood flow velocity (V_{valve}) into flow rate (Q_{valve}) through a valve, one needs to know the cross-sectional area (A) of the valve. The relationship between flow rate, velocity, and cross-sectional area is described by the equation $Q_{\text{valve}} = A \cdot V_{\text{valve}}$. Understanding the geometrical/anatomical property of the valve, specifically its cross-sectional area, is crucial for accurately estimating the flow rate based on the velocity of blood through the valve. The units of flow rate are typically expressed in volume per unit time (e.g., mL/s or L/min), while velocity is measured in distance per unit time.

3 Aortic Valve Stenosis

Preload, is the volume of blood in the heart before contraction, impacting the force of systolic contraction. Afterload is the pressure the heart faces to eject blood during systole, influenced by resistance in the circulatory system. Elevated afterload can strain the heart. Conditions like high blood pressure contribute to increased afterload.

Afterload is the pressure the heart must overcome to eject blood during systole, determined by the resistance encountered as blood leaves the heart. Elevated afterload, often due to conditions like high blood pressure, can strain the heart's pumping efficiency.

The below table examines how hemodynamic factors like peak ventricular pressure and maximum blood flow velocity through the aortic valve are impacted by gradual stenosis, increasing in 5% increments.

Percentage of Stenosis	Peak Flow Velocity (ms^{-1})	Peak Pressure (mmHg)
Normal	1.25	122
5%	1.31	123
10%	1.38	123
15%	1.46	124
20%	1.53	124
25%	1.62	125
30%	1.73	126
35%	1.84	127
40%	1.97	129
45%	2.11	130
50%	2.28	132
55%	2.47	135
60%	2.69	139
65%	2.96	143
70%	3.28	149
75%	3.67	158
80%	4.20	171

Table 3: Percentage of Stenosis, Peak Flow Velocity, and Peak Pressure

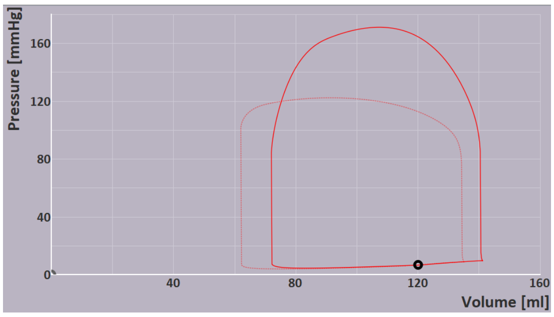


Figure 15: 80% Stenosis - Pressure Volume Diagram

- **Therefore, the maximum left ventricular pressure occurs at 80% stenosis equals to 171 mmHg.**

3.1 How does stenosis impact preload, afterload, and cardiac output?

3.1.1 Preload Impact

In AS, the narrowing of the aortic valve compels the left ventricle to exert more effort to pump blood through the restricted opening. This results in an increased volume of blood left in the ventricle after each heartbeat, elevating preload.

3.1.2 Afterload Impact

AS raises afterload, which represents the resistance the heart must overcome to pump blood during systole. The constricted aortic valve demands higher pressure generation, leading to an escalation in afterload as blood is forcefully pushed through the narrowed valve.

3.1.3 Cardiac Output Impact

Cardiac output, determined by Stroke Volume * Heart Rate, is affected in AS. Elevated afterload may decrease stroke volume as the left ventricle struggles to empty effectively. Simultaneously, an increased heart rate attempts to compensate, yet untreated AS may lead to a decline in overall cardiac output.

The maximum left ventricular pressure is 171 mmHg, corresponding to an aortic valve pressure of 103 mmHg. The pressure drop is determined by subtracting the aortic valve pressure from the left ventricular pressure:

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

This signifies a pressure difference of 68 mmHg across the aortic valve. Applying Bernoulli's equation allows us to estimate the maximum pressure drop:

$$\text{Maximum Flow Velocity} = 4.2\,\text{ms}^{-1}$$

$$\text{Pressure Drop} = 4 \times (4.2)^2 = 70.56\,\text{mmHg}$$

Comparing this with the previous calculation, the estimated pressure drop across the aortic valve is approximately 70.56 mmHg.

The calculation for the duration of ejection across the aortic valve is determined by subtracting the initial time of 0.24 seconds from the final time of 0.52 seconds:

$$\text{Duration} = 0.52\,\text{s} - 0.24\,\text{s} = 0.28\,\text{s}$$

Thus, the duration of ejection across the aortic valve is 0.28 seconds.

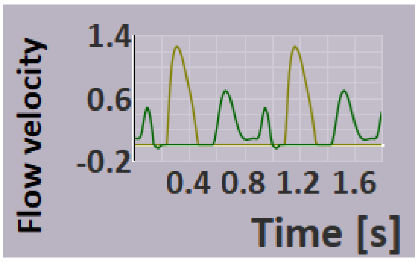


Figure 16: 80% Stenosis - Blood Flow Velocity Diagram

3.2 Determine the external work done by the left ventricle's pump

To determine the external pump work produced by the left ventricle, one can calculate the surface area enclosed by the pressure-volume relation. Estimating the increase in external pump work due to 80% aortic valve stenosis involves counting the effective number of squares enclosed by the pressure-volume curve.

1. External work done by the heart in a healthy individual: 20 squares.

2. External work done by the heart at 80% stenosis: 24 squares.

The increment in external work is computed as:

$$\text{Increase} = 24 - 20 = 4 \text{ squares.}$$

3.3 Myocardial Adaptations and Afterload Changes in Response to Increased Pump Work

The myocardial tissue of the left ventricle adapts to the chronically increased pump work by undergoing hypertrophy, where individual cardiac muscle cells enlarge, and there is an overall increase in the thickness of the ventricular wall. This hypertrophic adaptation is an attempt to enhance the contractile force of the left ventricle, allowing it to pump blood more effectively against the increased resistance.

The effect of this adaptation on afterload is twofold. On one hand, the hypertrophy allows the left ventricle to generate higher pressures during systole, aiding in overcoming the elevated afterload caused by conditions like aortic stenosis. However, on the other hand, the increased ventricular wall thickness can lead to stiffness, potentially impairing diastolic function and increasing overall afterload over time. This adaptation, while initially compensatory, may contribute to long-term cardiac challenges and potential complications.

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