DEPARTMENT OF ELECTRONIC AND TELECOMMUNICATION ENGINEERING

UNIVERSITY OF MORATUWA

BM2101

Modeling and Analysis of Physiological Systems

Analysis of Cardiac Physiology

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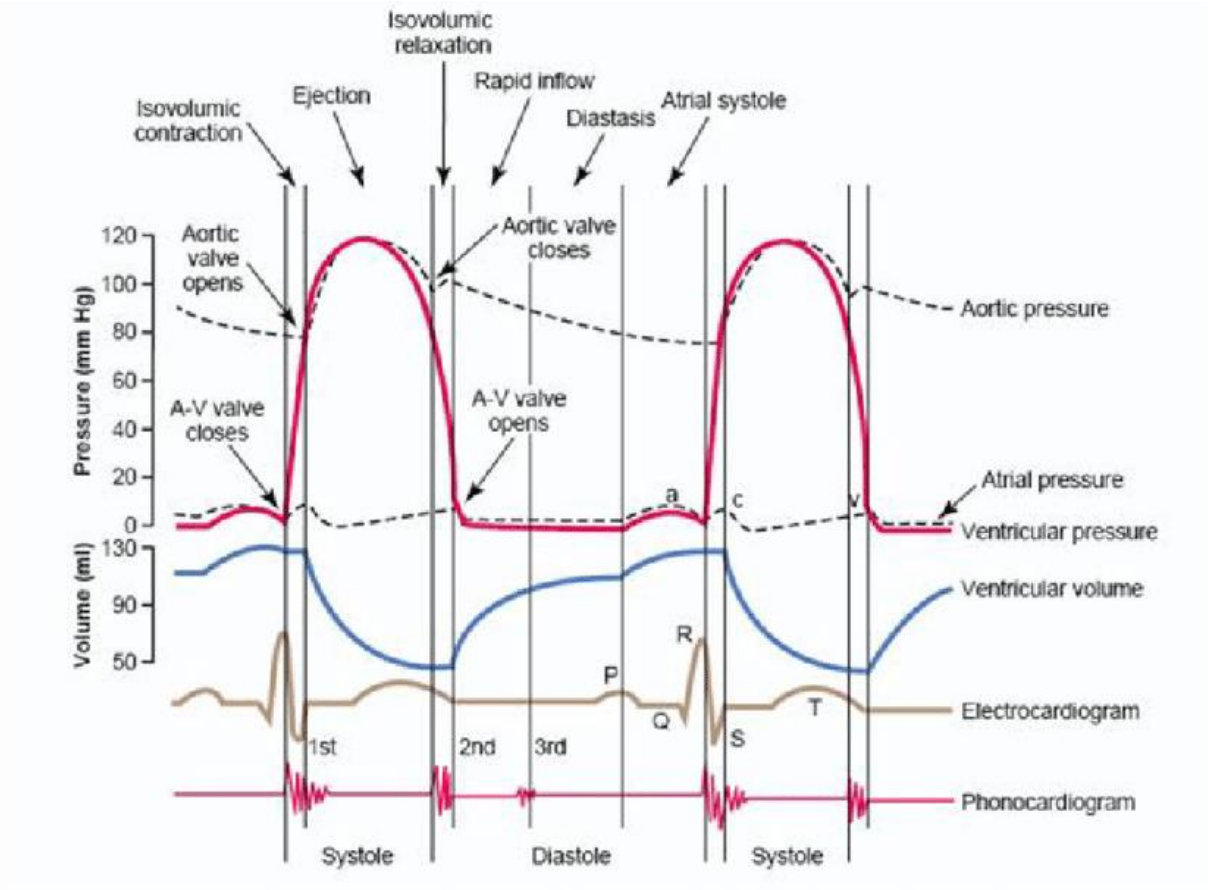
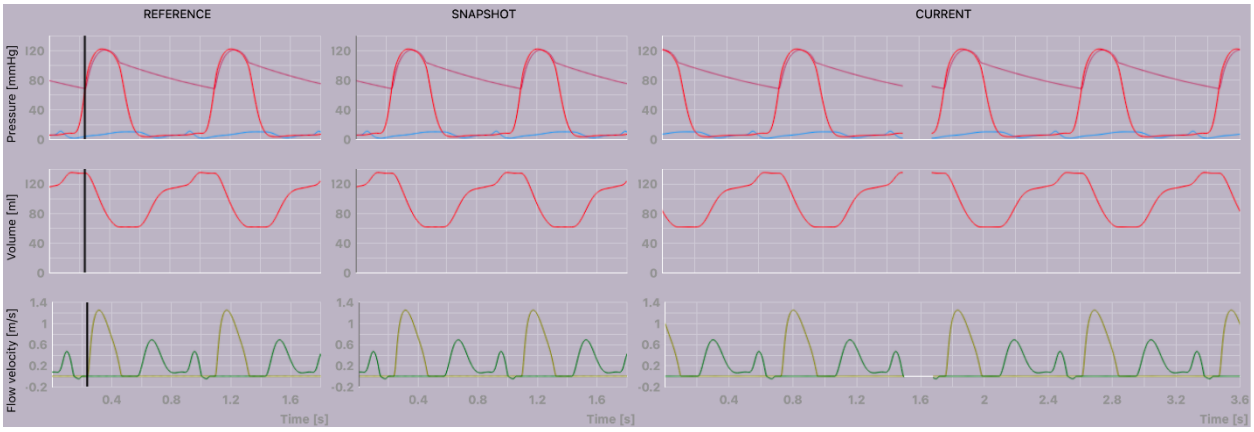
200664P

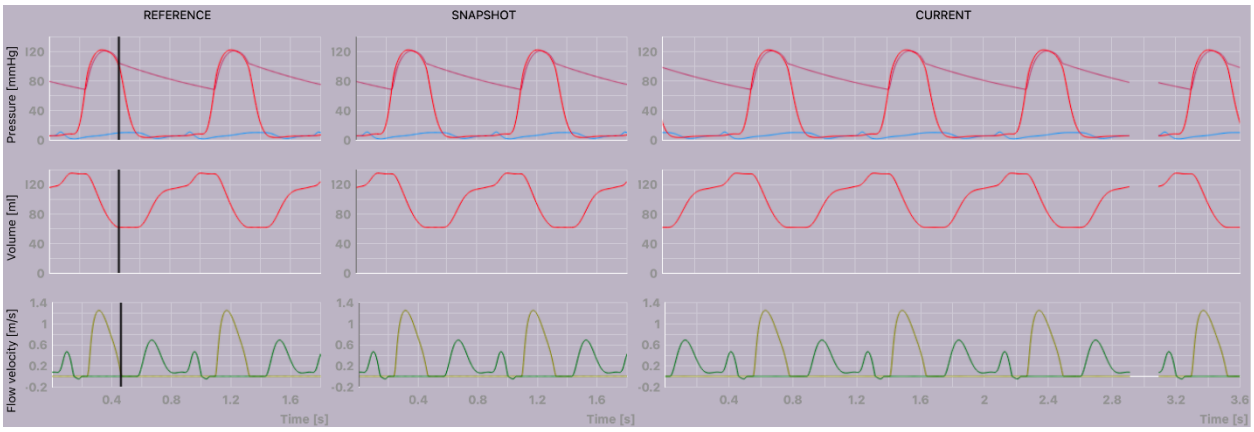
Wiggers Diagram:



a. During the isovolumic contraction ventricles start to contract and pressure within the ventricles increases rapidly. Since both aortic and mitral valves are closed during this phase the ventricular volume doesn't change. But when ventricular pressure goes beyond the aortic pressure with the increase of ventricular pressure the aortic valve opens and blood flows into the aorta. With that we can observe the ventricular volume decreases rapidly. Subsequently the pressure in the ventricles start to reduce and when it falls below the aortic pressure again the aortic valve closes and enter to the isovolumic relaxation phase in that phase again both valves close, and ventricular volume doesn't change. But ventricular pressure rapidly decreases.

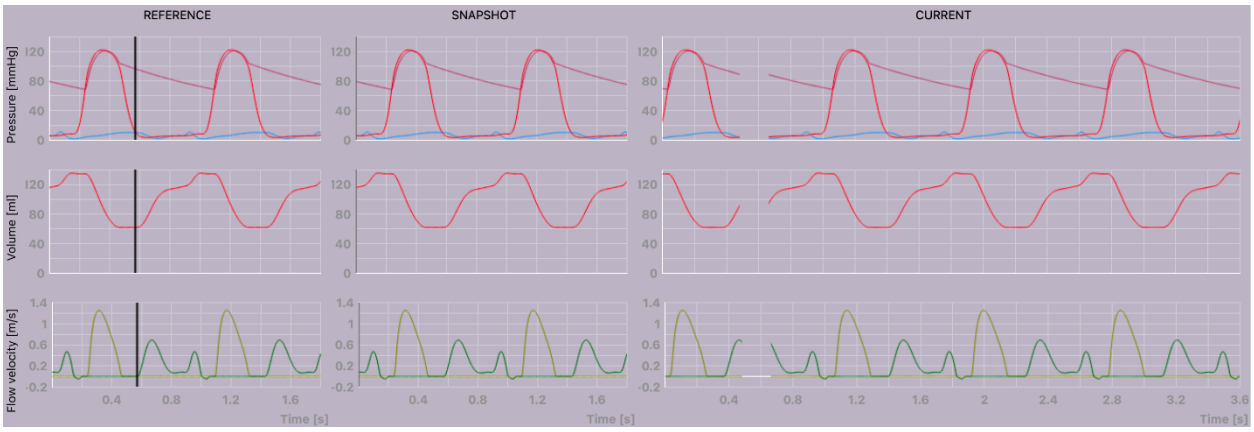


Opening

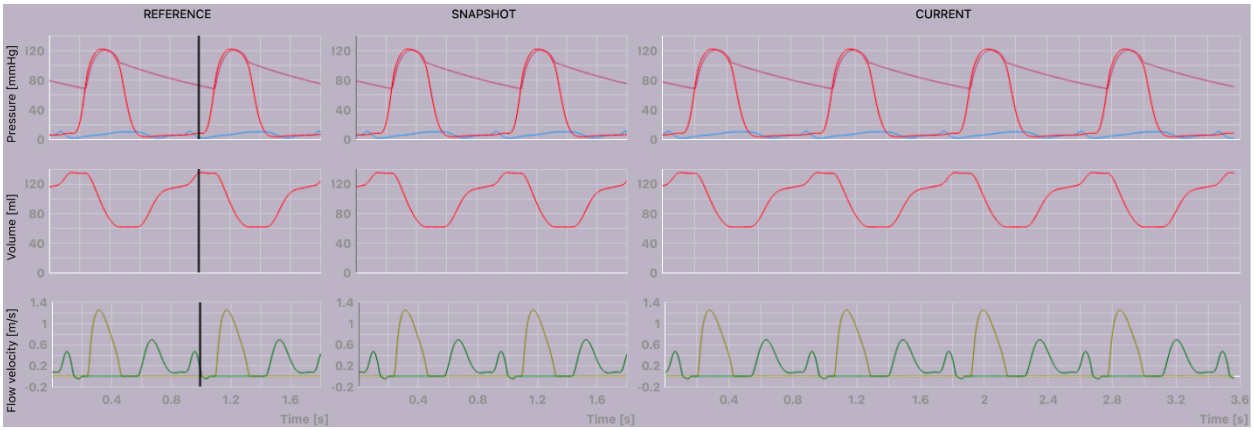


Closing

b. As explained in the above question ventricular pressure decreases rapidly and when it goes below the atrial pressure the mitral valve opens. Then blood filled in the atria flows into the ventricles. Due to that ventricular volume increases. Consequently, the pressure in the ventricles rises again. Also, with the atrial systole extra blood will be pumped into the ventricles and pressure rises further in the ventricles this causes mitral valves to close again.



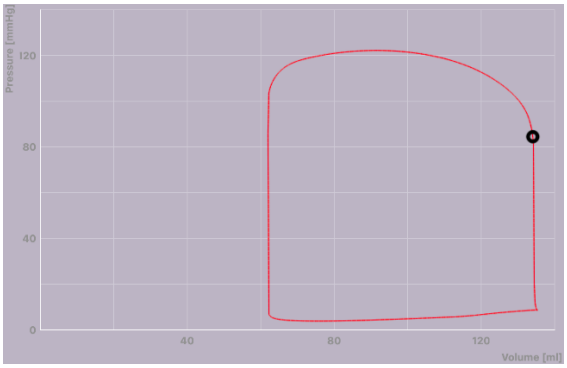
Opening



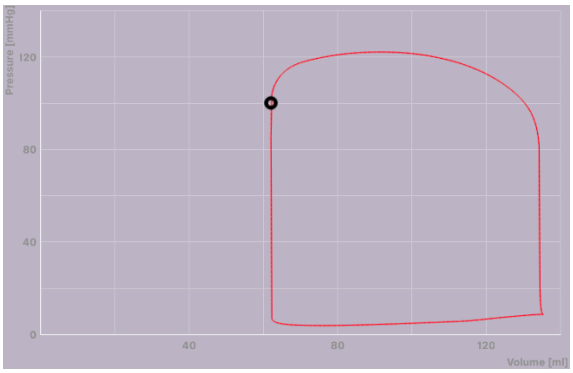
Closing

c.

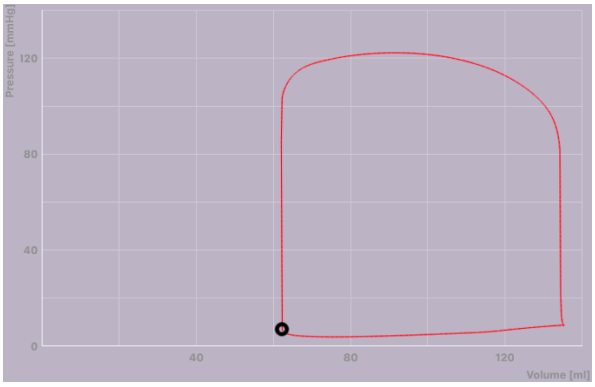
Aortic Opening:



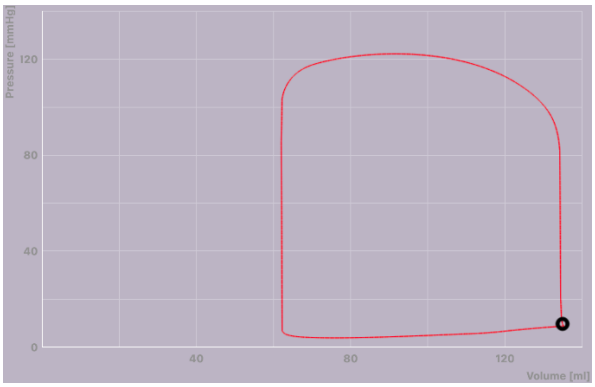
Aortic Closing:



Mitral Opening:



Mitral Closing:



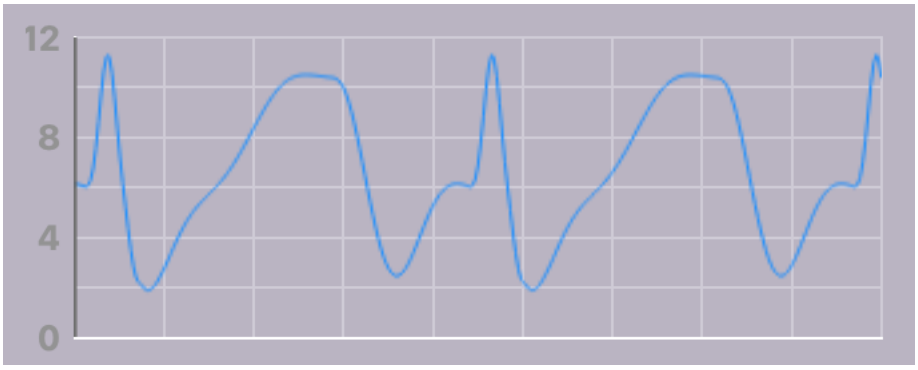
d.

A- Filling
B- Isovolumic contraction
C- Ejection
D- Isovolumic relaxation

e. During Ventricular contraction the aortic valve opens because of increased pressure of ventricles than the aorta. Blood flows rapidly into the aorta and then reduces since the decrease pressure of ventricles. Then when blood pressure reduces and closes the aortic valve causing the blood flow cease. The characteristic one hump is due to that opening and closing of aortic valve.

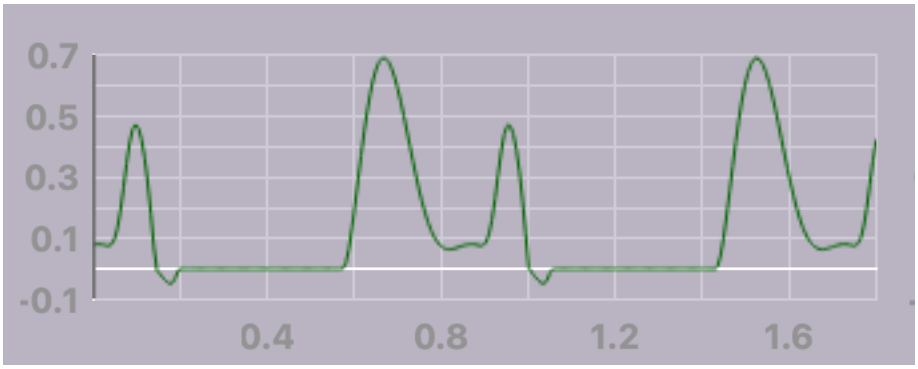
In Mitral valve the first hum is due to inflow of blood to the ventricles from atria as a result of opening the mitral valve. The second hump is due to the atrial contraction and flow of additional blood to the ventricles.

f.



The sharp edge in the pressure curve corresponds to the pressure increase during atrial contraction (depolarization), aligning with the P wave on the ECG waveform. After atrial contraction, there is a rapid decrease in pressure due to ventricular filling and atrial emptying. The gradual rise in atrial pressure is due to blood flowing into the relaxed atria. Simultaneously, the ventricles relax, and as their pressure drops below the atrial pressure, the mitral valve opens, causing a rapid decrease in pressure following the slow peak of atrial pressure.

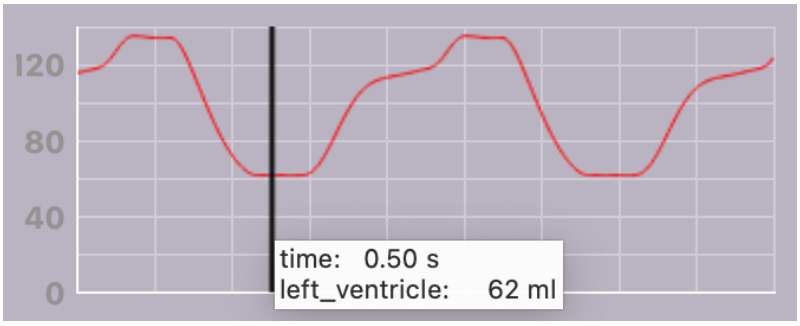
g.

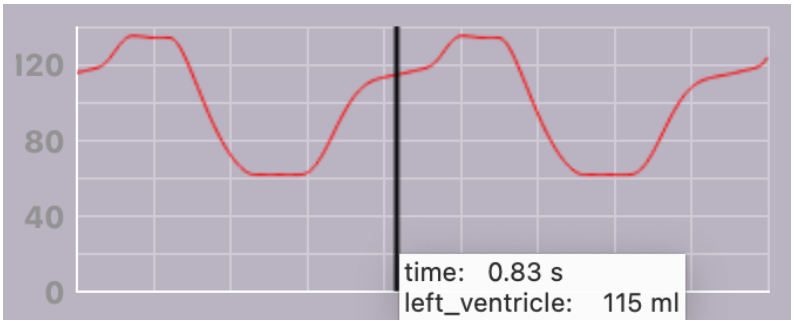


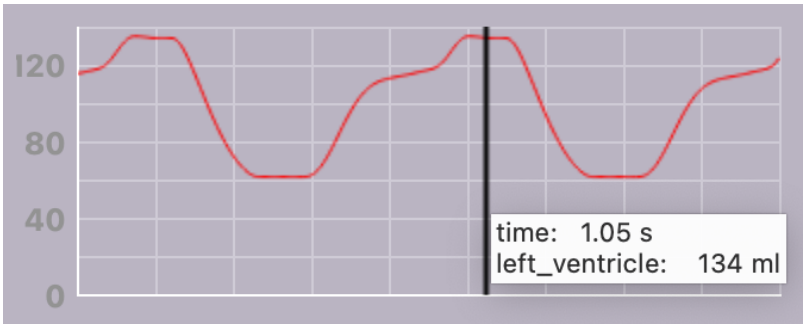
h.

$\frac{E}{A} = \frac{0.691}{0.473} = 1.46$

i.







left ventricular filling due to active filling = 135- 115 = 20ml

left ventricular filling that is due to passive filling. = 115-62 =53ml

j. Cross sectional area of the valve.

2.

a. Preload refers to the initial stretching of the heart muscle fibers just before contraction, which is determined by the volume of blood filling the ventricles during diastole. It represents the degree of cardiac muscle fiber stretch and influences stroke volume—the amount of blood ejected from the heart during systole.

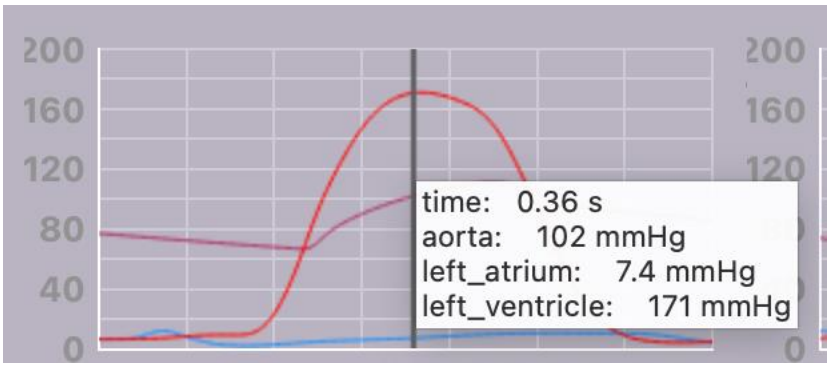
Afterload is the resistance that the heart must overcome to eject blood during systole. It is primarily determined by the pressure in the arteries and the peripheral vascular resistance. Increased afterload requires the heart to work harder to pump blood, affecting stroke volume and cardiac output.

b.

As aortic stenosis (AS) worsens, the left ventricle faces greater challenges in pumping blood through the narrowed aortic valve, leading to higher left ventricular pressure during systole. To compensate for the obstruction and maintain adequate cardiac output, blood flow through the narrowed valve accelerates, resulting in increased flow velocity. This causes the pressure-volume loop, representing left ventricular pressure and volume during a cardiac cycle, to shift rightward and upward. The increased area under the curve indicates greater left ventricular work and myocardial energy expenditure required to overcome the stenosis and effectively eject blood. In essence, the heart has to exert more effort to cope with the narrowing and maintain proper blood flow.

c.

Aortic Valve Stenosis (AS) causes a slight increase in the volume of blood in the left ventricle before contraction, leading to elevated preload. However, the narrowed valve obstructs blood ejection into the aorta, increasing the afterload. This elevated afterload challenges the heart's ability to pump blood effectively. As a compensatory mechanism, the stroke volume remains relatively constant, maintaining cardiac output. Nevertheless, over time, the ventricular walls may thicken and become less flexible due to the increased workload.



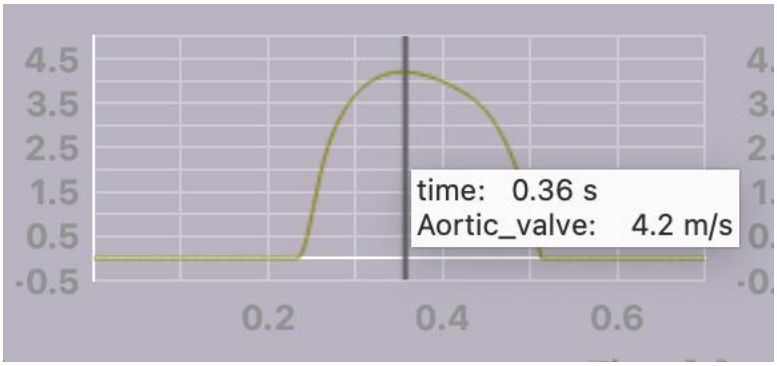
d.

Maximal left ventricular pressure = 171 mmHg

Aortic blood pressure at the moment of maximal left ventricular pressure = 104 mmHg

Pressure drop across stenotic valve = 171-104 = 67 mmHg

e.



$$\Delta p \approx 4v^2$$

$$\Delta p \approx 4 * 4.18^2$$

$$\Delta p = 69.8896 mmHg$$

f.

g.

Duration of Ejection = 0.53S – 0.24S = 0.29S

h. Chronic pressure overload in the left ventricle due to aortic stenosis prompts the heart muscle to adapt by thickening (hypertrophy). This hypertrophy is a compensatory mechanism to enhance the heart's contractile force and cope with the increased workload. However, the thickened myocardial tissues result in reduced ventricular compliance, impacting the heart's ability to relax and fill adequately during diastole. This alteration in diastolic function can contribute to a condition known as heart failure with preserved ejection fraction (HFpEF), where the heart struggles to efficiently fill with blood despite a relatively normal ejection of blood during systole.

increase in external pump work = 4*20*20 = 1600mlmmHg