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CHEG 341 011 - Fluid Mechanics
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Mini Project #3

EXECUTIVE SUMMARY

Two geometry models of coronary artery (stenosed artery without and with a bypass intervention) are used to model the blood flow in coronary artery disease (CAD) patients in SimScale. The first part of the simulation is performed by using a lower volumetric flow rate of $2.08 \times 10^{-6}\ \frac{m^3}{s}$ flowing into the left main coronary artery (LM), which is then bifurcated into the left circumflex artery (LCX) and left anterior descending (LAD). For the stenosed artery without and with the intervention of a bypass respectively, the blood is flowing with a volumetric flow rate (velocity) of 1.98×10^{-6} (0.591) and $1.47 \times 10^{-6}\ \frac{m^3}{s}$ ($0.348\ \frac{m}{s}$) at LCX and 6.39×10^{-8} (0.024) and $5.48 \times 10^{-7}\ \frac{m^3}{s}$ ($0.168\ \frac{m}{s}$) at LAD. The blood pressure at LM is determined to be 13,880 and 13,550 Pa respectively. Blood pressure for both types of simulations (without and with the bypass) at LCX and LAD are the same (13,330 Pa), with differing resistance, R. In the same order, R_{LCX} is 1.01×10^{9} and $1.36 \times 10^{9}\ \frac{kg}{m^4 \cdot s}$, while R_{LAD} is 3.13×10^{10} and $3.65 \times 10^{9}\ \frac{kg}{m^4 \cdot s}$ respectively. The second part of the simulation is performed by conducting the same analysis but increasing the volumetric flow rate into LM to $2.70 \times 10^{-6}\ \frac{m^3}{s}$. The volumetric flow rate (velocity) at LCX is found to be 2.56×10^{-6} (0.764) and $1.89 \times 10^{-6}\ \frac{m^3}{s}$ ($0.447\ \frac{m}{s}$), while LAD has it 9.07×10^{-8} (0.035) and $7.26 \times 10^{-6}\ \frac{m^3}{s}$ ($0.223\ \frac{m}{s}$), for the stenosed artery without and with the bypass respectively. In accordance, the flow resistance at LCX is found to be 7.82×10^{8} and $1.06 \times 10^{9}\ \frac{kg}{m^4 \cdot s}$, while the resistance at LAD is 2.21×10^{10} and $2.76 \times 10^{9}\ \frac{kg}{m^4 \cdot s}$ respectively, with the same blood pressure of 13,330 Pa. However, the blood pressure at LM for those without and with the bypass is higher than those at LCX and LAD, which is found to be 14,160 and 13,650 Pa respectively.

INTRODUCTION

Coronary artery disease (CAD) is developed through a process known as atherosclerosis, where the major blood vessels supplying blood to the heart become damaged [1]. Cholesterol deposited in the coronary arteries, forming a plaque, or inflammation are the typical factor of CAD. Coronary arteries supply blood, oxygen, and nutrients to the heart. Plaque buildup narrows these arteries, leading to an effect known as stenosis, thus decreasing the blood flow to the heart. Individuals who have CAD will experience angina, shortness of breath, or other CAD signs and symptoms [1]. In severe cases like a complete blockage, it can cause a heart attack.

There are many ways to reduce the risk of getting this heart disease or stroke. Changing one's lifestyle is one way to prevent getting CAD, such as exercising regularly and eating a healthy diet [2]. If the condition doesn't improve with lifestyle changes and medication, one should undergo surgical procedures recommended by doctors, such as coronary artery bypass graft (CABG) surgery [3]. The main goal of the surgery is to improve the blood flow to the heart. This surgical intervention will be the method of choice if the coronary arteries are so narrowed or blocked that it causes a high risk of heart attack to individuals.

Two major coronary arteries that branch off the aorta are the left main coronary artery (LM) and the right coronary artery (RCA), which feed blood to the left and the right side of the heart respectively. LM further bifurcates into two other arteries, the left anterior descending (LAD) artery and left circumflex (LCX) artery. For the case where the LAD artery is stenosed, to bypass the blocked portion of the artery, a healthy blood vessel from the leg known as saphenous vein is removed and attached one end of the vein to LM and the other end below the plaque region at LAD [3].

METHODS

A contract with the Food and Drug Administration (FDA) was made to update the guidelines for heart bypass surgery to understand the changes in blood flow in individuals who suffer from CAD. A stenosed and bypass coronary arteries in the heart are modelled by computational fluid dynamics (CFD) model in SimScale by using the geometry model as shown in **Figure 1** below.

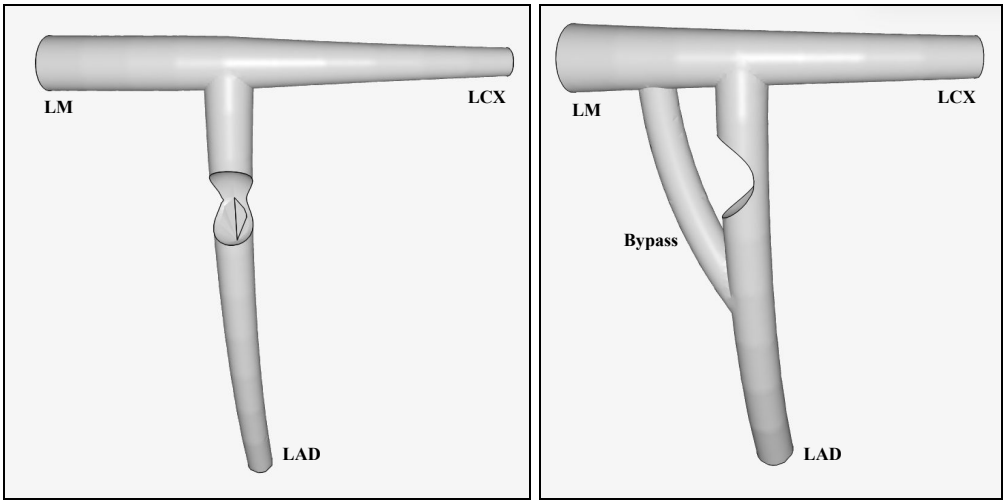

Figure 1: Geometry Model of Stenosed Artery With No Bypass (Left) and With A Bypass (Right)

The simulation was run twice for each part by using an incompressible Newtonian water as the fluid, with the density ($\rho = 1060\ \frac{kg}{m^3}$) and kinematic viscosity ($\eta = 3.3 \times 10^{-6}\ \frac{m^2}{s}$) changed to the values of blood for both geometry models. The model was scaled accordingly by a factor of 10^{-6} for the model with no bypass and 10^{-3} for the model with the bypass. Zero gauge pressure was set at the LAD and LCX outlet for all simulations. ***Part I*** of the analysis was performed by using a low volumetric flow rate of $2.08 \times 10^{-6}\ \frac{m^3}{s}$ entering the LM inlet. The analysis is then repeated by using a higher volumetric flow rate of $2.70 \times 10^{-6}\ \frac{m^3}{s}$ for ***Part II***. For each part, relevant data values from SimScales are obtained such as the volumetric flow rate, velocity, and relative pressure at respective locations by using appropriate visualization like cutting planes, isosurfaces, and iso volumes. By using the data obtained, the resistance of blood flow, R could be calculated by using **Eq. 1** and **2** (P: Relative Pressure; Q: Volumetric Flow Rate), and Reynolds number, Re, by **Eq. 3** (D: Diameter).

$$R_{LAD} = \frac{|P_C - P_{LAD}|}{Q_{LAD}} \quad \textbf{(Eq. 1)}$$

$$R_{LCX} = \frac{|P_C - P_{LCX}|}{Q_{LCX}} \quad \textbf{(Eq. 2)}$$

$$Re = \frac{D\rho v}{\mu} \quad \textbf{(Eq. 3)}$$

CFD is a common tool for generating solutions for fluid flows, with or without solid interaction [4]. The analysis is conducted in accordance with the fluid physical properties such as velocity, pressure, temperature, density, and viscosity. These properties should be considered simultaneously to generate an accurate solution, by using mathematical models and numerical methods in the software tools to analyze the flow such as the Navier Stokes equations. CFD allows the simulation of fluid motion using numerical approaches which cover a wide range of problems related to laminar and turbulent flows, incompressible and compressible fluids, multiphase flows and many more.

RESULTS AND DISCUSSION

Part I - Simulations with Low Volumetric Flow Rate Into LM Inlet

From **Figure 2** below which shows the convergence and residual plots of the simulation, the simulation is said to have a good performance and the values obtained are reliable and stable because all of the parameters converge towards certain values as time progresses and all the residuals are lower than 10^{-3}.

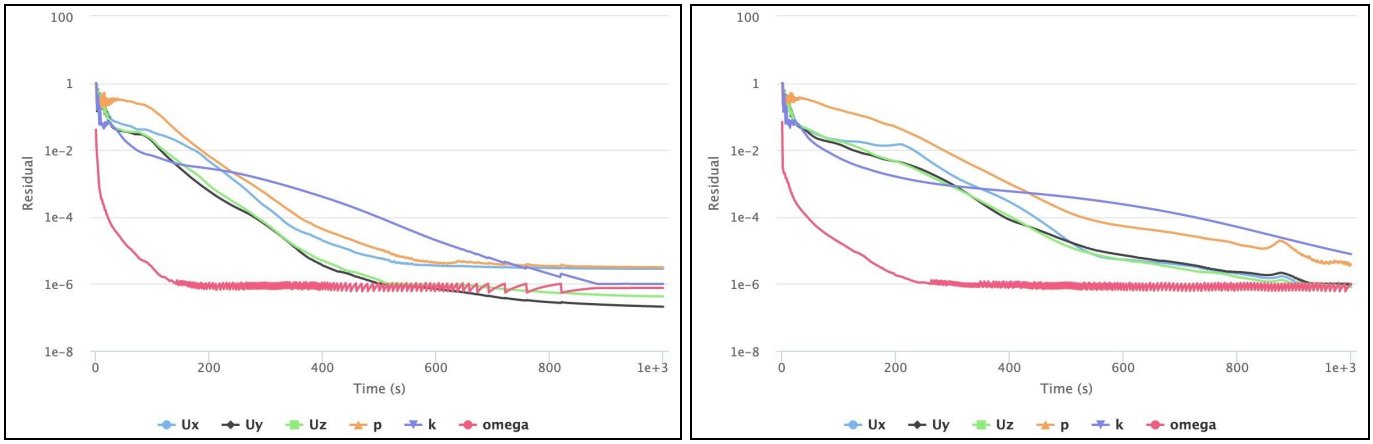

Figure 2: Residual Plots of Heart Stenosis Without A Bypass (Left) and With A Bypass (Right) for Part I

Illustrated in **Figure 3 - 9** and **Figure 10 - 16** are the relevant cutting planes, isosurfaces, and iso volumes of the anatomically accurate dimensions of the left coronary arteries, which have a stenosed LM artery bifurcation of 90 % occluded without and with the implementation of the bypass respectively.

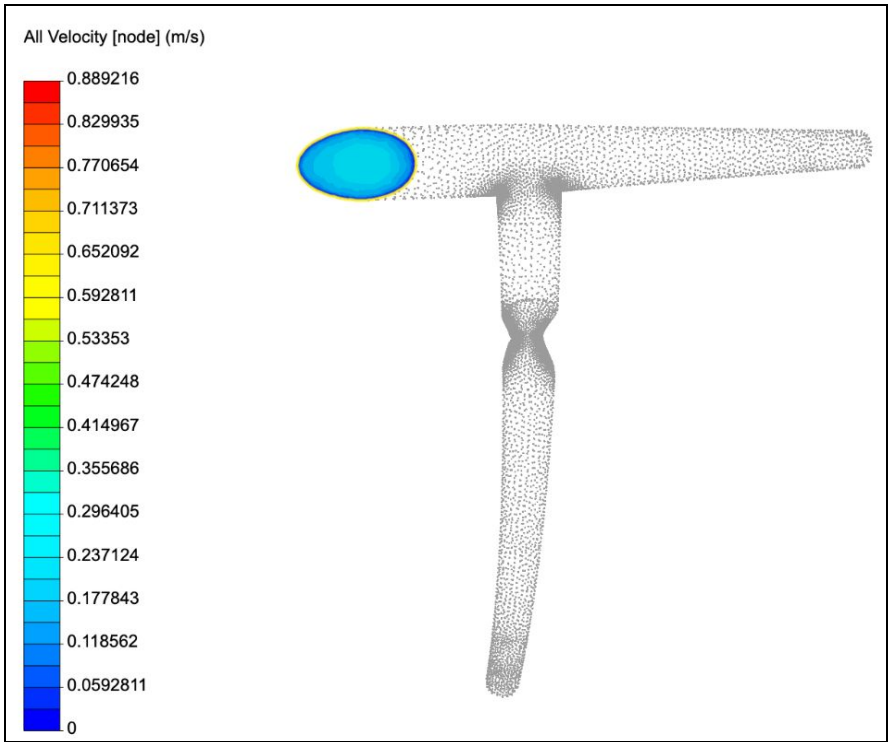

Figure 3: Cutting Planes of All Velocity [node] at LM Inlet of Heart Stenosis for Part I

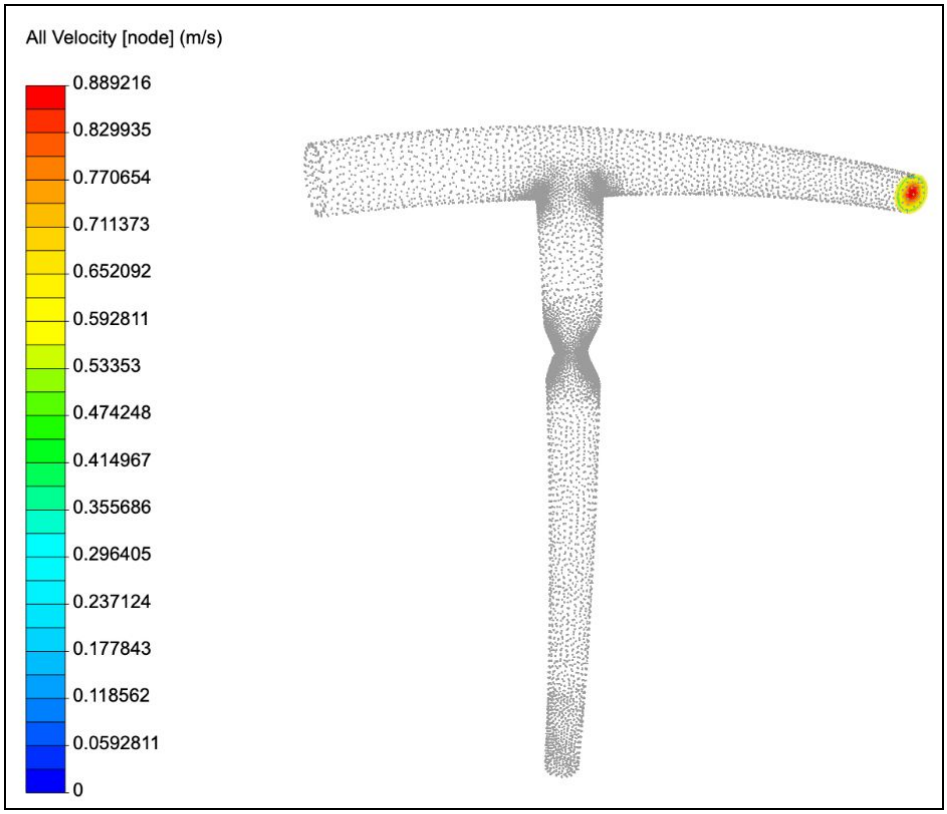

Figure 4: Cutting Planes of All Velocity [node] at LCX Outlet of Heart Stenosis for Part I

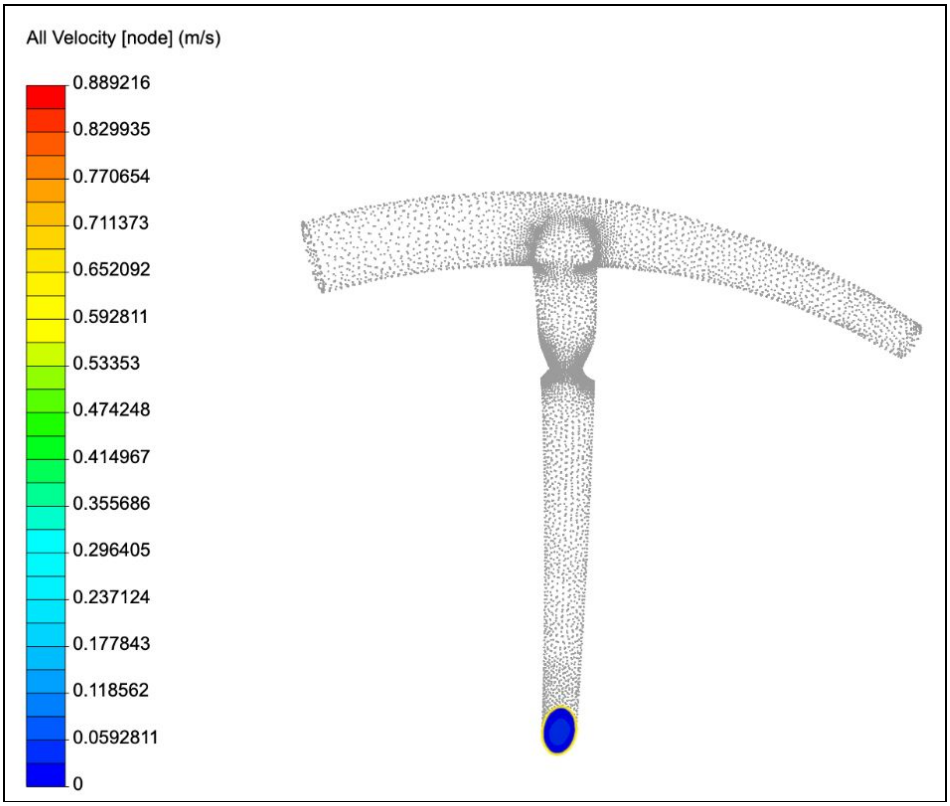

Figure 5: Cutting Planes of All Velocity [node] at LAD Outlet of Heart Stenosis for Part I

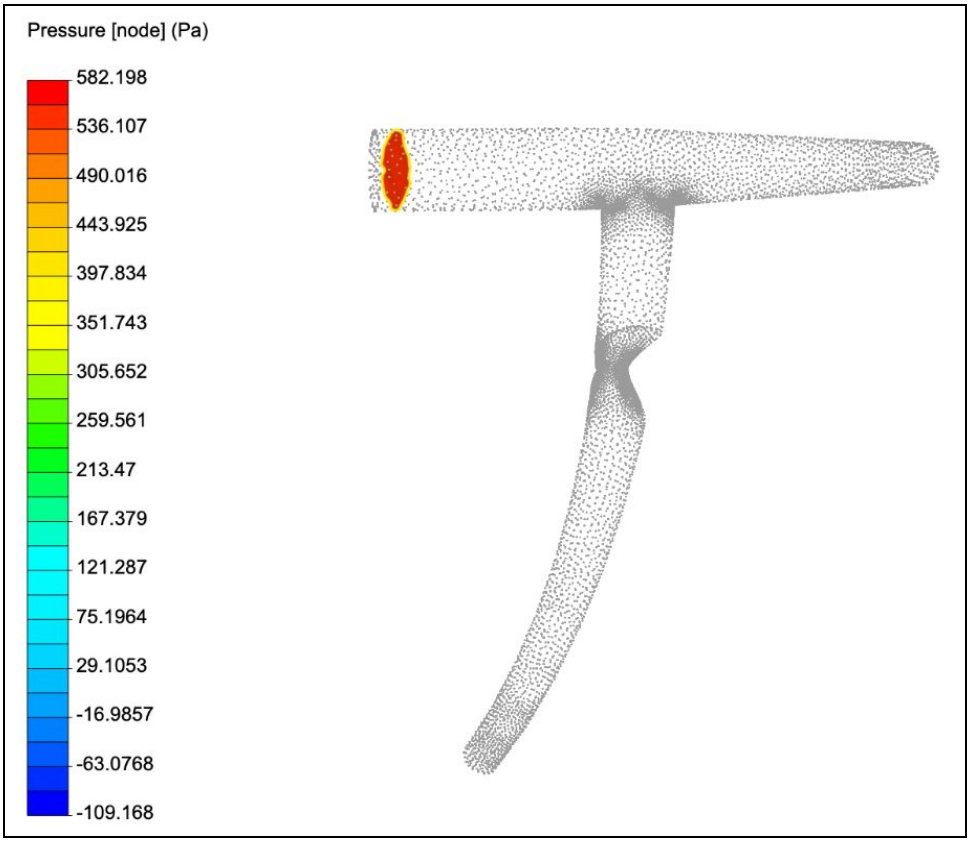

Figure 6: Isosurface of Pressure [node] at LM Inlet of Heart Stenosis for Part I

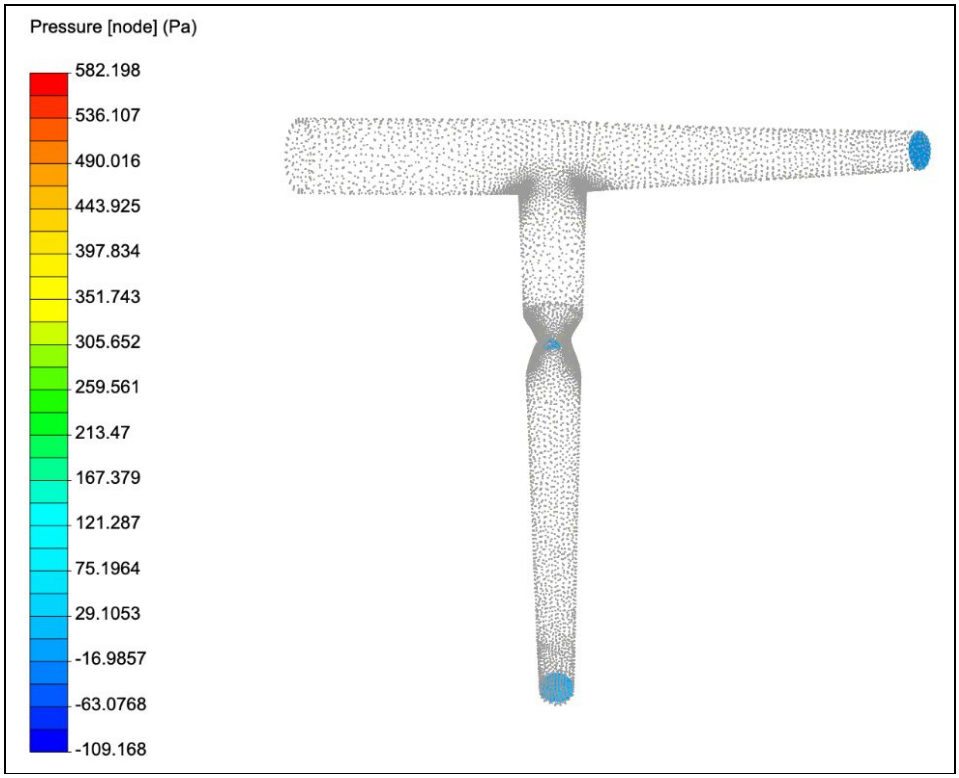

Figure 7: Isosurface of Pressure [node] at LCX and LAD Outlet of Heart Stenosis for Part I

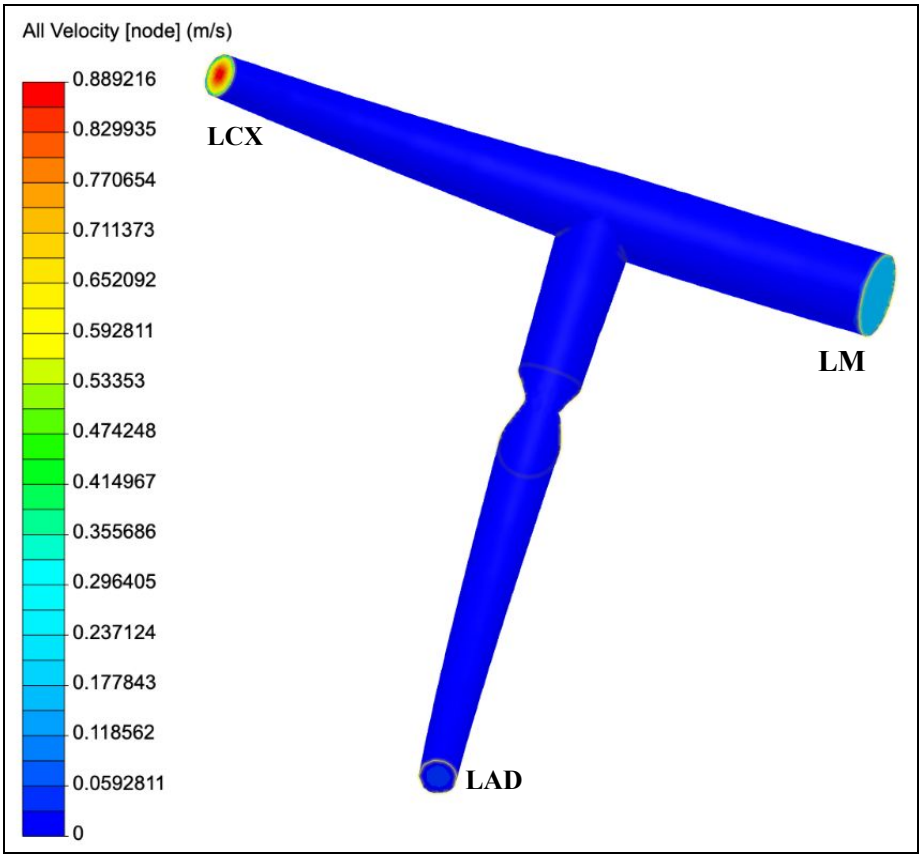

Figure 8: Isovolume of All Velocity [node] of Heart Stenosis for Part I

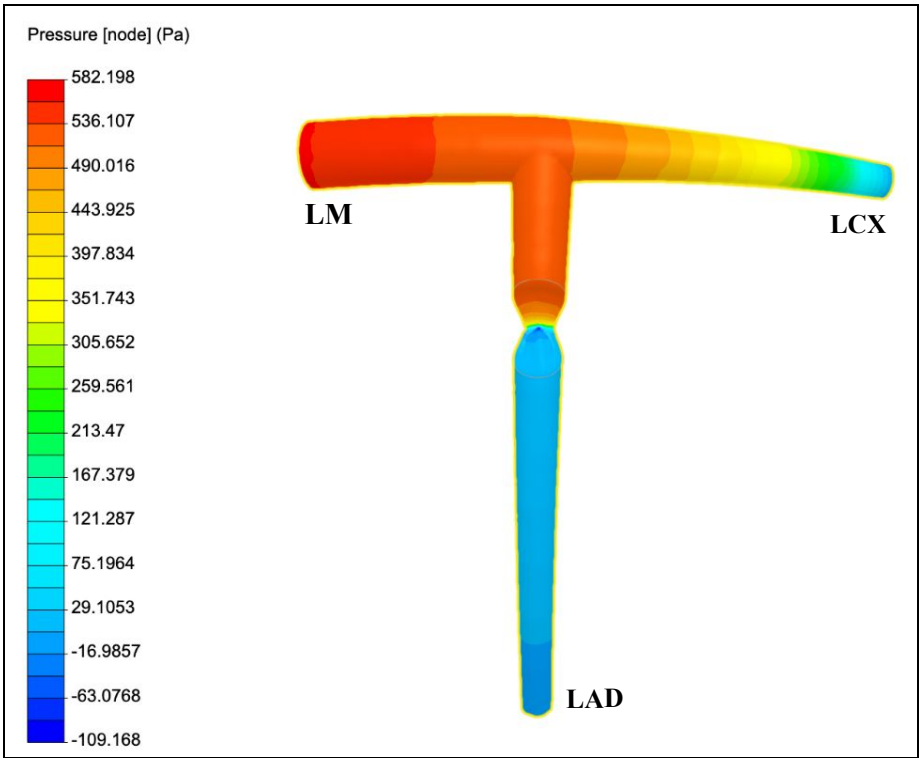

Figure 9: Isovolume of Pressure [node] of Heart Stenosis for Part I

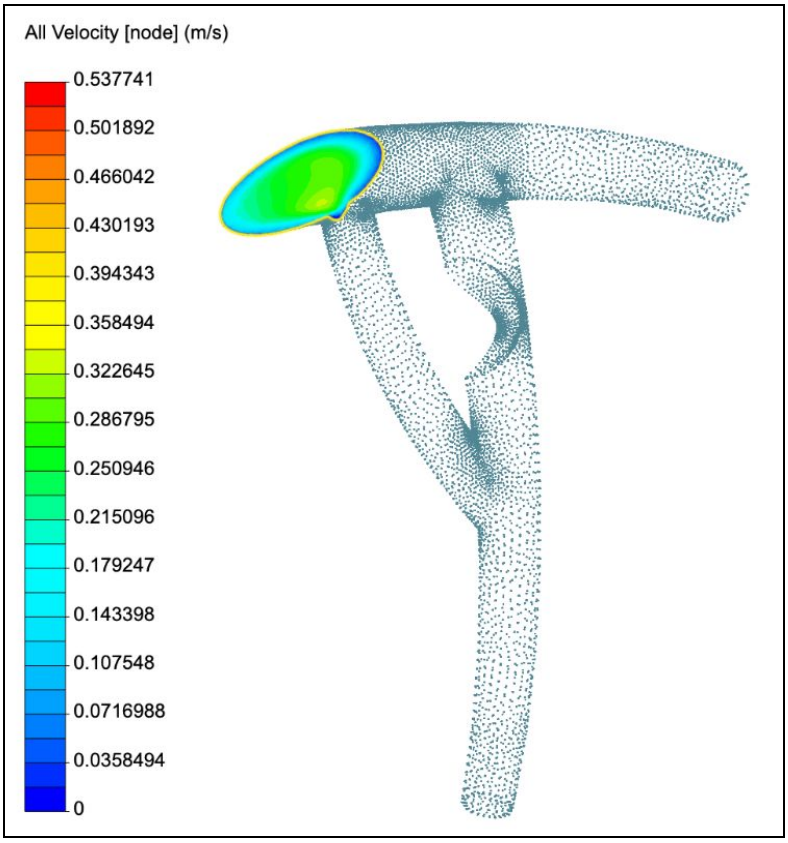

Figure 10: Cutting Planes of All Velocity [node] at LM Inlet of Heart Bypass for Part I

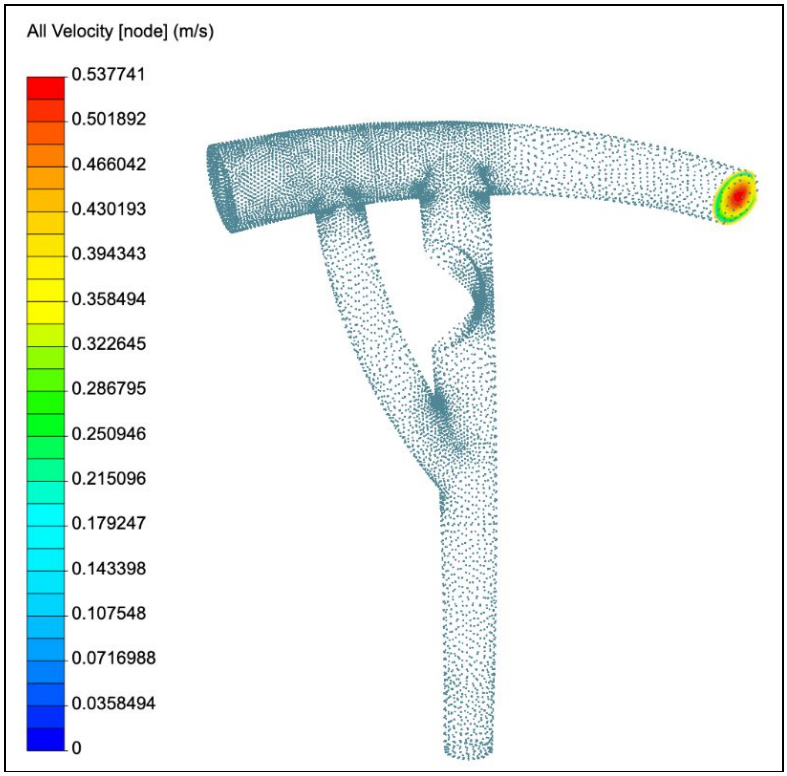

Figure 11: Cutting Planes of All Velocity [node] at LCX Outlet of Heart Bypass for Part I

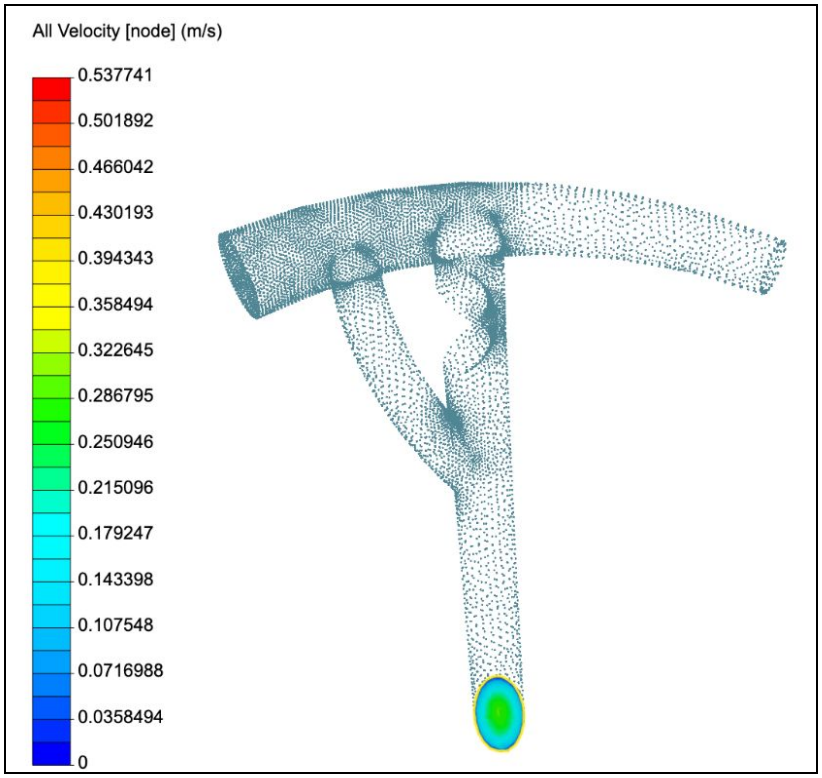

Figure 12: Cutting Planes of All Velocity [node] at LAD Outlet of Heart Bypass for Part I

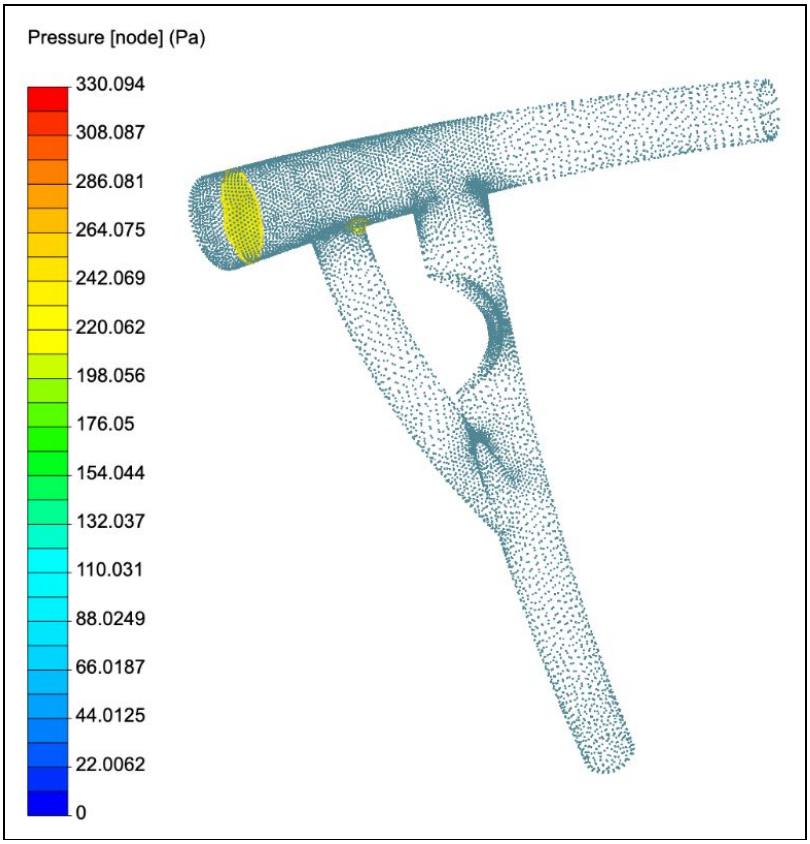

Figure 13: Isosurface of Pressure [node] at LM Inlet of Heart Bypass for Part I

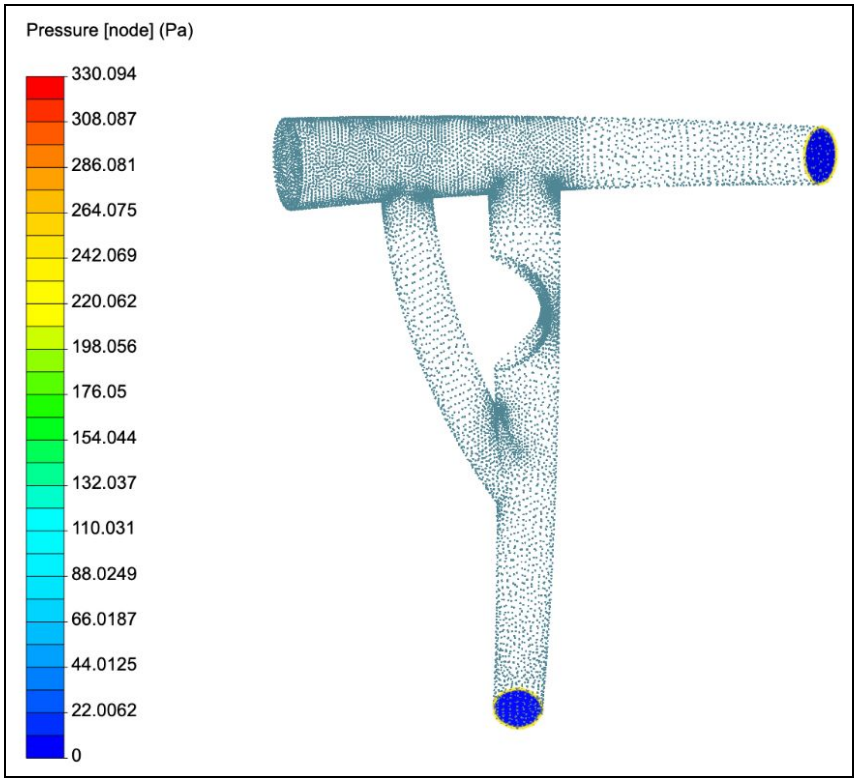

Figure 14: Isosurface of Pressure [node] at LCX and LAD Outlet of Heart Bypass for Part I

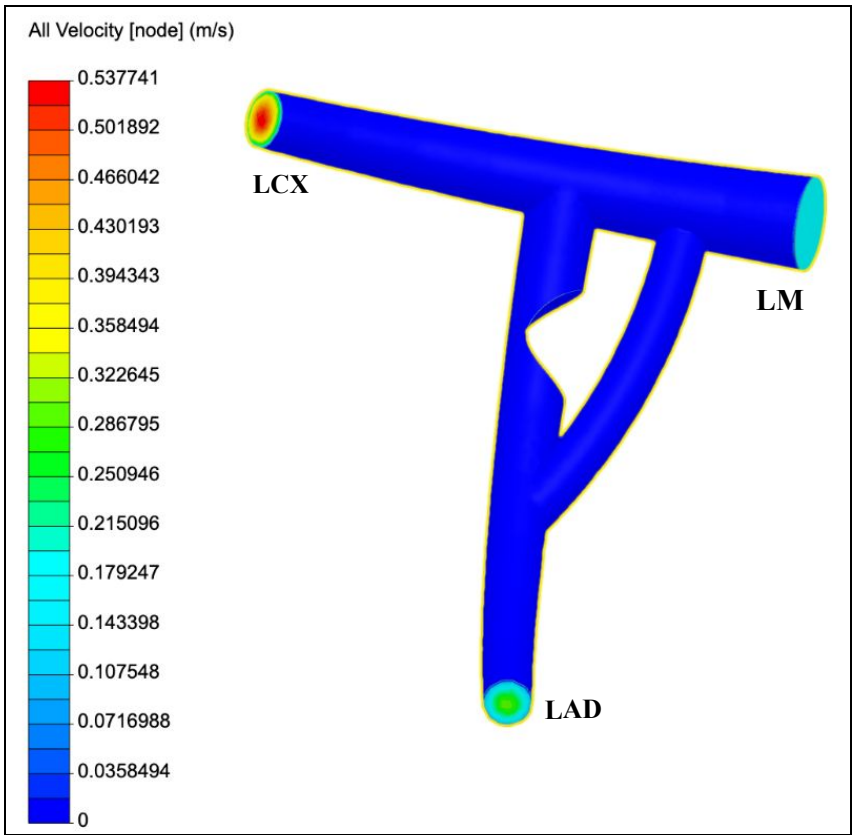

Figure 15: Isovolume of All Velocity [node] of Heart Bypass for Part I

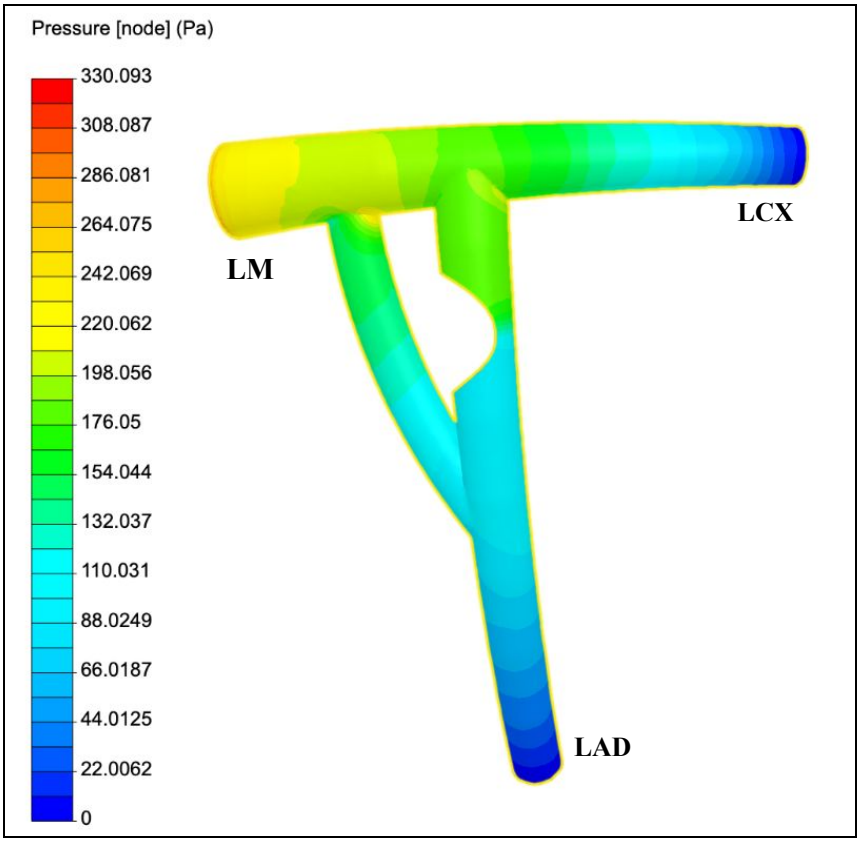

Figure 16: Isovolume of Pressure [node] of Heart Bypass for Part I

Table 1 below summarizes the relevant data values obtained from the analysis of the figures previously mentioned in SimScale with the relevant parameters used to obtain the resistance at LAD and LCX from **Eq. 1** and **2** respectively, and the Reynolds number, Re from **Eq. 3**.

*** Table 1: Data and Simulation Results of Both Simulations for Part I**

Type of Simulation	Without Bypass			With Bypass		
Location	LM	LCX	LAD	LM	LCX	LAD
Relative Pressure, P (Pa)	550	0	0	220	0	0
Volumetric Flow Rate, Q ($\frac{m^3}{s}$)	2.08×10^{-6}	1.98×10^{-6}	6.39×10^{-8}	2.08×10^{-6}	1.47×10^{-6}	5.48×10^{-7}
Velocity, v ($\frac{m}{s}$)	0.166	0.591	0.024	0.206	0.348	0.168
Diameter, D (m)	0.0040	0.0018	0.0020	0.0040	0.0018	0.0020
Reynolds Number, Re	201	314	15	250	185	102
Blood Pressure, P_B (Pa)	13880	13330	13330	13550	13330	13330
Resistance, R ($\frac{kg}{m^4 \cdot s}$)		1.01×10^{9}	3.13×10^{10}		1.36×10^{9}	3.65×10^{9}

* Blood Density, $\rho = 1060 \frac{kg}{m^3}$

* Blood Dynamic Viscosity, $\mu = 3.5 \times 10^{-3}$ Pa · s

* Capillaries Pressure at LAD and LCX, $P_C = 2000$ Pa

By comparing the velocity at the inlet and outlets from **Figure 3 - 5** and **Figure 10 - 12,** it is found that it is the highest relatively at LCX (0.591 and 0.348 $\frac{m}{s}$), and the lowest at LAD (0.024 and 0.168 $\frac{m}{s}$) for the simulation of heart stenosis without and with the bypass respectively. For the case of no bypass intervention, the velocity at LAD is expected to be lower than other places as the occlusion blocks the flow of blood through it (only allowing a small portion of the blood to flow through; blood backflow), making most of the blood flowing from LM to be distributed out of LCX, with higher velocity by mass balance analysis. This is also reflected in the volumetric flow rate out, Q of LCX and LAD. Q_{LAD} is much smaller than Q_{LCX} due to the same reason. However, the velocity at LAD is relatively higher for bypass in comparison to the stenosed arteries with no bypass. This should be true as the bypass creates an alternative path for the blood to flow through, without having to pass through the occlusion which prevents the overall blood circulation to the heart in the first place. By mass balance computation, it is expected to have a lower velocity at LCX than the one without bypass to compensate for the high velocity out of LAD. This is again reflected in the volumetric flow rate at LCX and LAD. Q_{LAD} is still lower than Q_{LCX}, however it was found that Q_{LAD} of the arteries with bypass is higher than those without bypass, due to the same reason explained previously. The relative pressure at LM as shown in **Figure 6** and **13** is found to be 550 Pa with blood pressure of 13,880 Pa for the case of no bypass, and 220 Pa with the blood pressure of 13,550 Pa for the one with the bypass attached. The simulation was set to have a gauge pressure of 0 Pa at both LCX and LAD, which is then confirmed with the isosurface analysis as shown in **Figure 7** and **14**, with a blood pressure of 13,330 Pa. **Figure 8** and **15** show the overview of the velocity trend, while **Figure 9** and **16** show the trend for pressures in the capillaries for the simulation without and with the bypass intervention respectively. The pressure is found to be decreasing from LM towards LCX and LAD. In addition, it could be seen that the pressure has decreased throughout the arteries in comparison to the one with no bypass.

Figure 17 and **18** as provided below shows the shear stress at the arteries' walls without and with the bypass respectively.

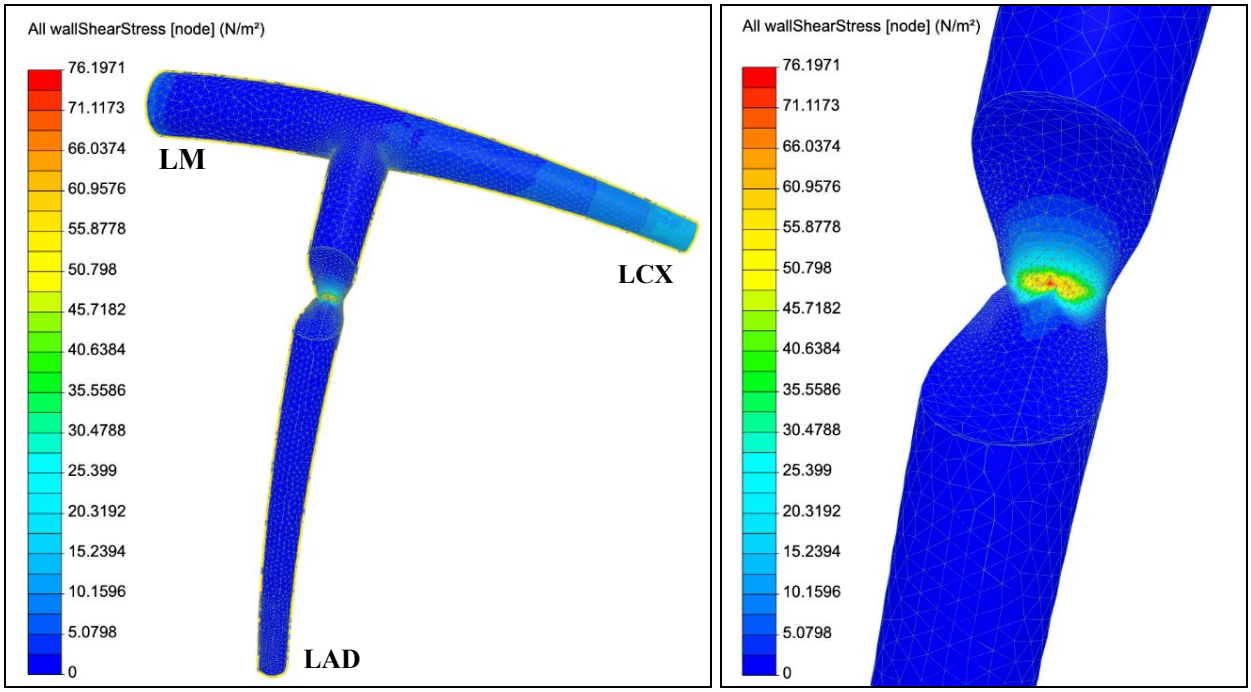

Figure 17: Isovolume of All Wall Shear Stress [node] of Heart Stenosis for Part I (closed up view on the right)

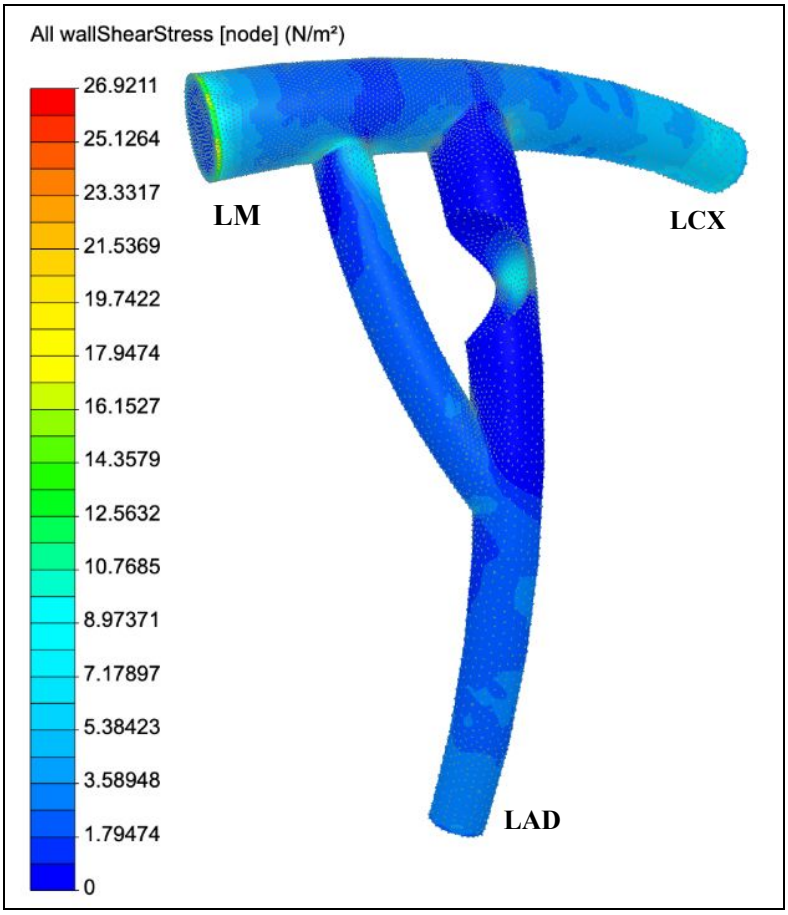

Figure 18: Isovolume of All Wall Shear Stress [node] of Heart Bypass for Part I

From **Figure 17** for the arteries with no bypass, it could be seen that the shear stress is the highest at the occlusion region, and lower everywhere else. This is because shear stress is directly proportional to the velocity of the blood and inversely proportional to the diameter of the cross-sectional area of the occlusion region. The region is expected to have fluid flowing through (in low rate, due to blood backflow as discussed previously) with high velocity due to the small opening before slowing down again to the LAD. From **Figure 18**, with the presence of bypass, the shear stress at the occlusion is reduced and distributed evenly throughout the arteries. There would be more shear stress at the arteries with bypass than the other scenario. Although there is no clear relationship with the intensity of the shear stress on the arteries on someone's health, from a study conducted in 2017, low shear stress can lead to the alteration of the endothelial cells (phenotype, cell signaling, gene expression, etc), which then promotes early plaque development [5]. High shear stress environment is then associated with certain biological processes which further promote plaque formation, vulnerability and destabilization. There is more shear stress based on **Figure 18** but with a lower magnitude that serves as "low shear stress" in comparison to **Figure 17** that has "high shear stress" at the occlusion. A normal carotid artery in a low shear stress environment will have a quiescent plaque, which will then form a stenotic plaque with narrowed coronary lumen, thick fibrous cap, and small lipid core [5]. For the patient's perspective, having stenotic plaque can provide a stable angina. High shear stress from stenotic plaque will lead to vulnerable plaque, which will cause acute coronary syndromes to patients if no further medication/care is done to reduce the stress. Essentially, having a bypass will ensure that the artery stays in a low-shear stress environment, preventing the development of more serious plaque. The blood flow in the arteries is similar to a flow of fluid in a cylindrical pipe having Poiseuille flow. With no-slip boundary conditions, the shear stress is the highest at the wall, which makes sense to have a plaque formation at the arteries' walls.

From these two simulations, FDA is recommended to implement the bypass in the arteries. The most compelling reasons to go for the suggested bypass design is that the overall blood pressure in the arteries can be reduced as clearly shown in **Figure 16**, in comparison to **Figure 9**. High-blood-pressure patients have higher risks to develop coronary artery disease [1]. High flow rate of blood exerts higher shear stress on the wall of arteries, inducing the development of plaque, as discussed earlier. The diameter of the bypass could not really be determined exactly, however the type of bypass used should be a decision factor made by the surgeons. While it is common to use the saphenous vein from the legs for the CABG surgery, radial artery in the wrists proven to be better although they differ in survival rates, which totally depends on the individuals' initial health conditions to begin with (not the type of bypass), due to the more flexible walls of arteries under high pressure compared to the veins [6]. The location wise of the bypass should be maintained as it should be connected to the aorta to the LAD to function properly.

Part II - Simulations with High Volumetric Flow Rate Into LM Inlet

Based on **Figure 19**, the simulations show a good performance and the values obtained are reliable and stable because the convergence and residual plots have all of the parameters converge towards certain values as time progresses and all the residuals are lower than 10^{-3}.

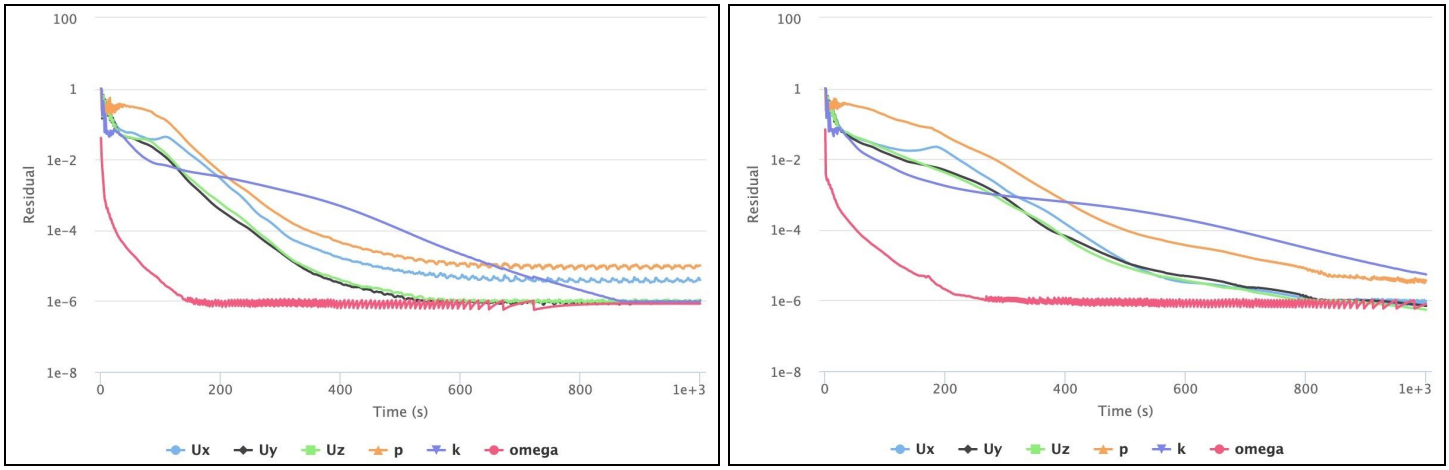

Figure 19: Residual Plots of Heart Stenosis Without A Bypass (Left) and With A Bypass (Right) for Part II

Figure 20 - 22 and **Figure 27 - 29** provided below are the relevant cutting planes, isosurfaces, and iso volumes of the anatomically accurate dimensions of the left coronary arteries, which have a stenosed LM artery bifurcation of 90 % occluded and of a bypass respectively, with a higher volumetric flow rate into LM priorly set up before running the simulation.

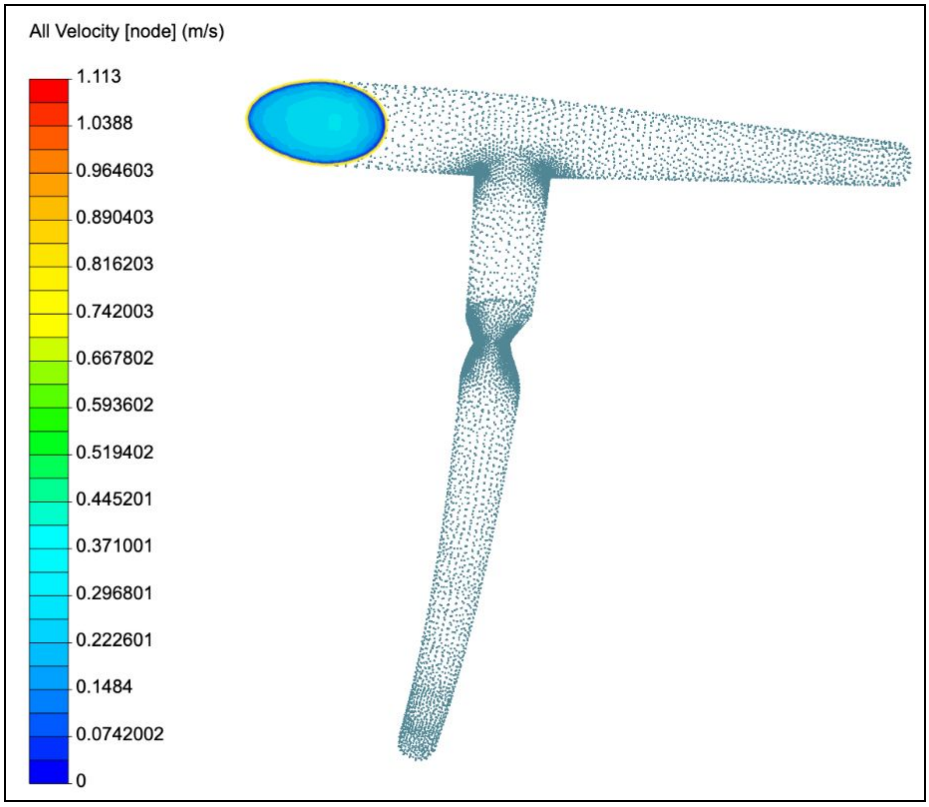

Figure 20: Cutting Planes of All Velocity [node] at LM Inlet of Heart Stenosis for Part II

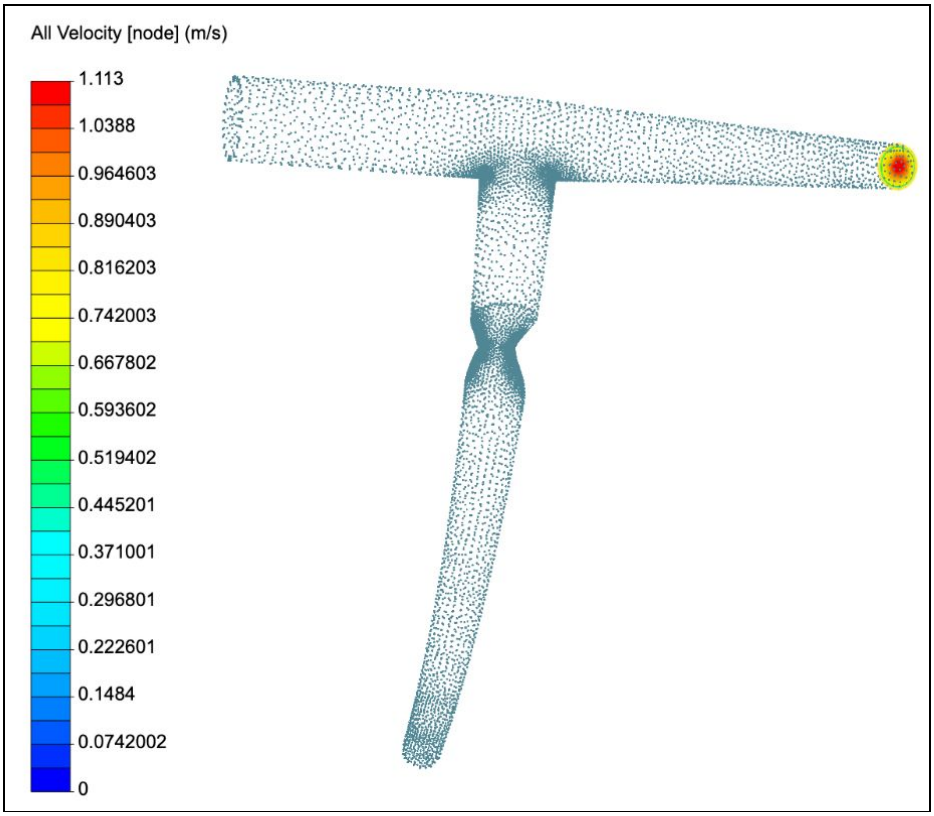

Figure 21: Cutting Planes of All Velocity [node] at LCX Outlet of Heart Stenosis for Part II

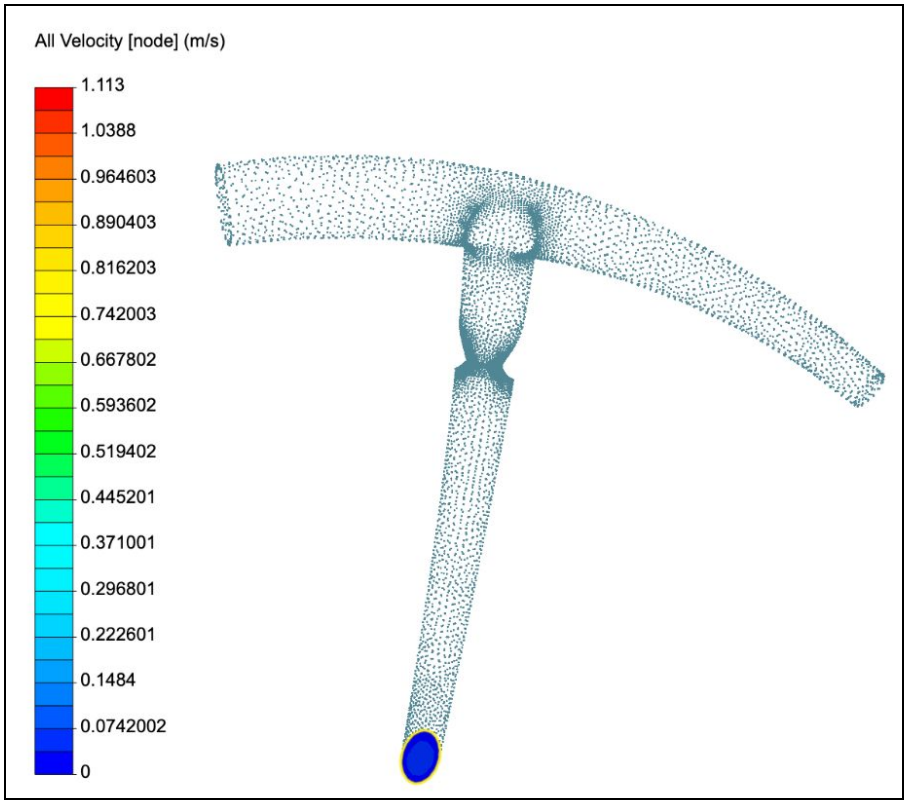

Figure 22: Cutting Planes of All Velocity [node] at LAD Outlet of Heart Stenosis for Part II

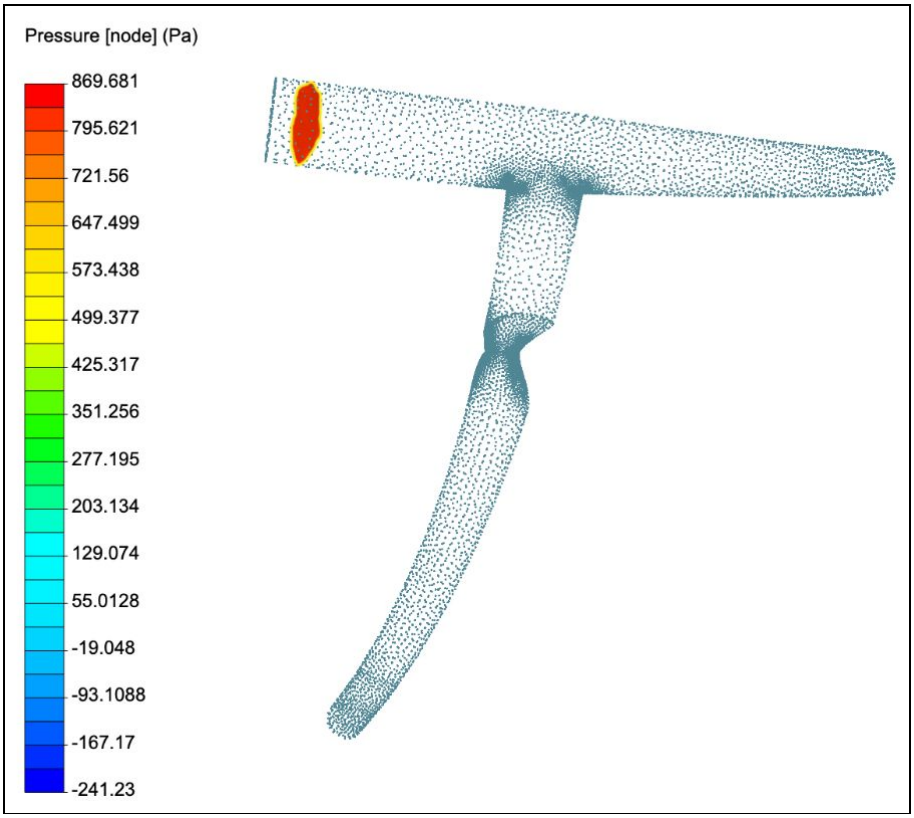

Figure 23: Isosurface of Pressure [node] at LM Inlet of Heart Stenosis for Part II

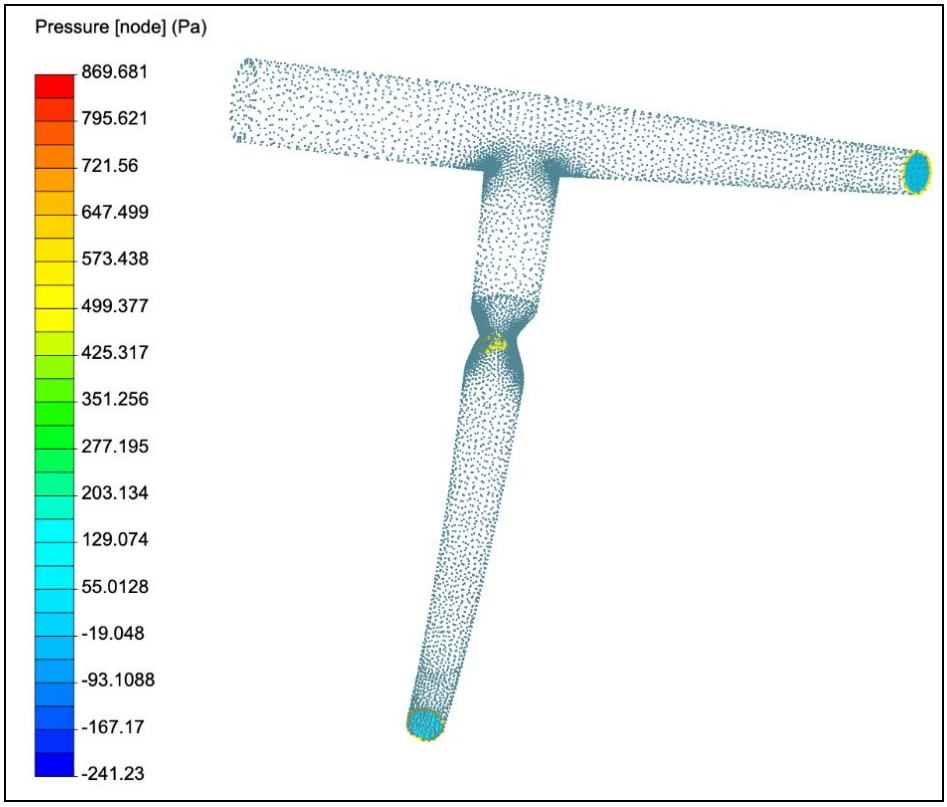

Figure 24: Isosurface of Pressure [node] at LCX and LAD Outlet of Heart Stenosis for Part II

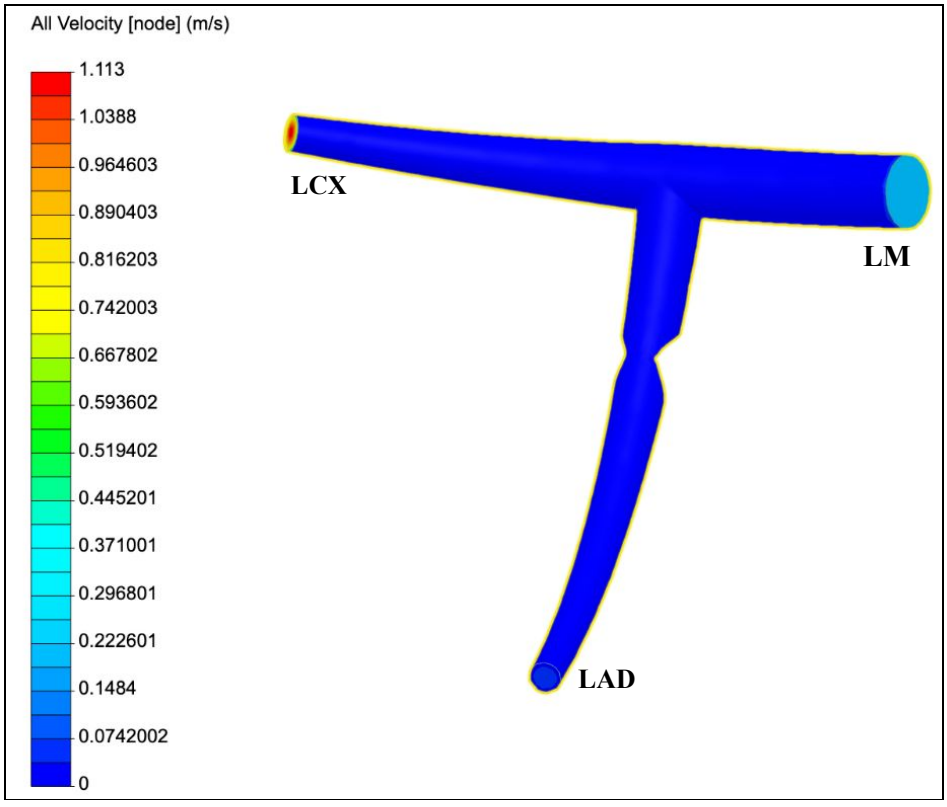

Figure 25: Isovolume of All Velocity [node] of Heart Stenosis for Part II

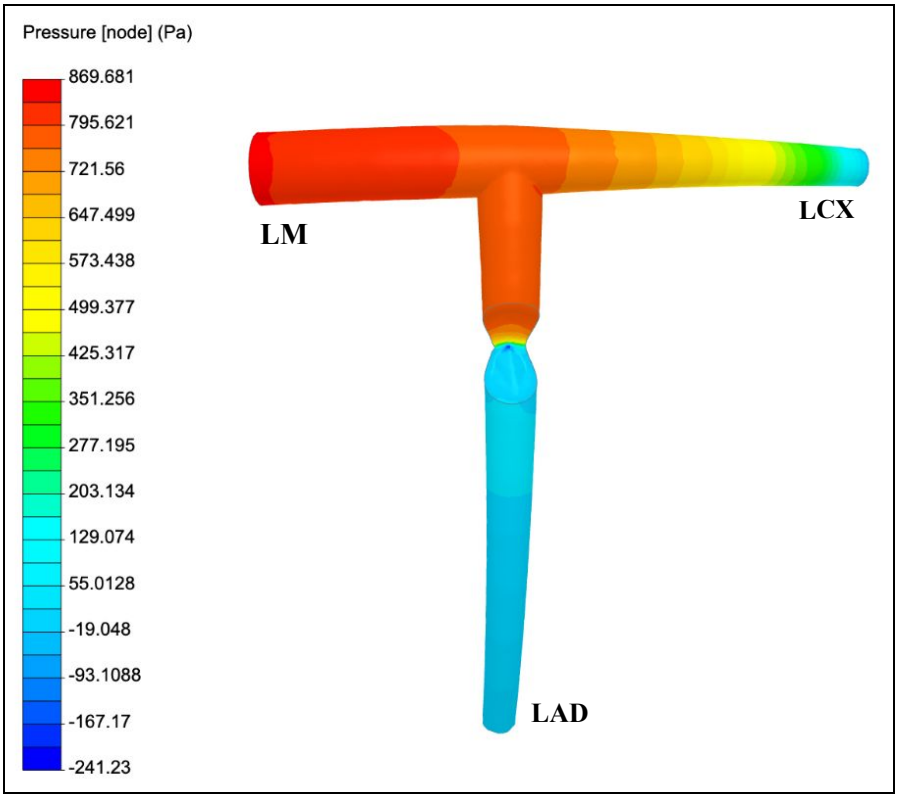

Figure 26: Isovolume of Pressure [node] of Heart Stenosis for Part II

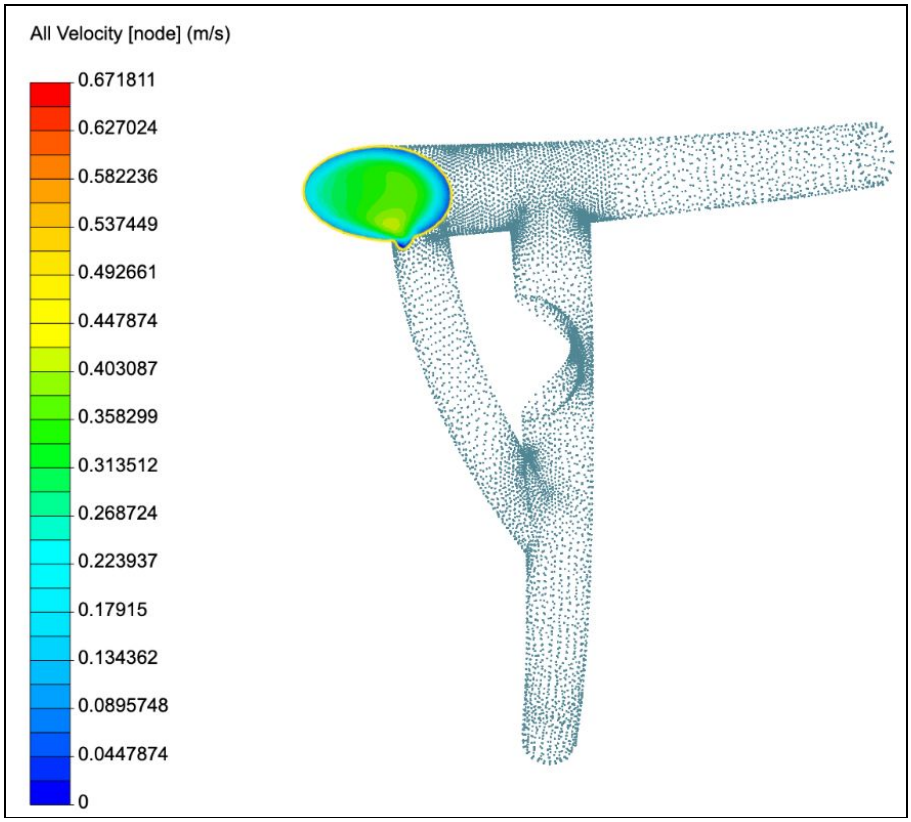

Figure 27: Cutting Planes of All Velocity [node] at LM Inlet of Heart Bypass for Part II

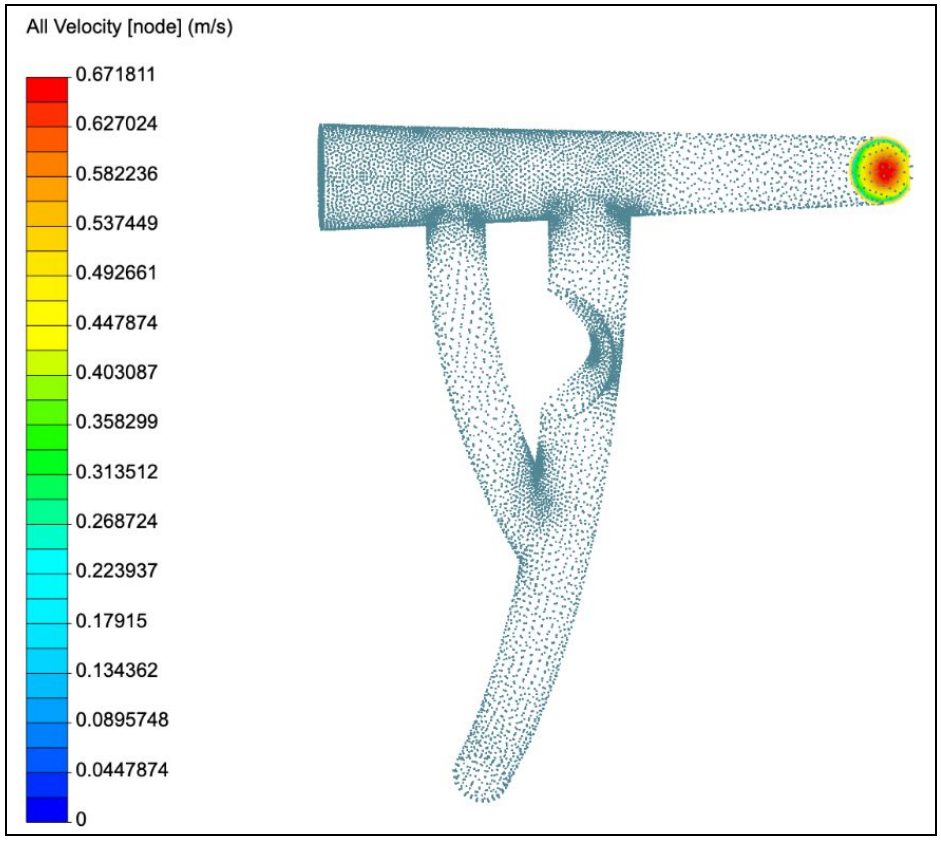

Figure 28: Cutting Planes of All Velocity [node] at LCX Outlet of Heart Bypass for Part II

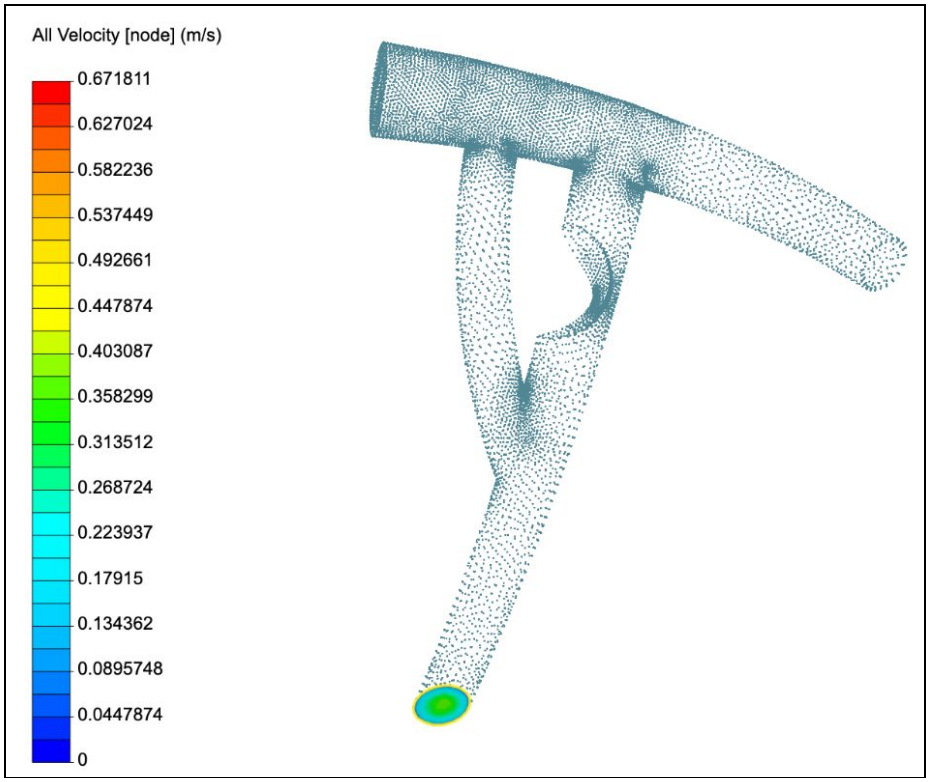

Figure 29: Cutting Planes of All Velocity [node] at LAD Outlet of Heart Bypass for Part II

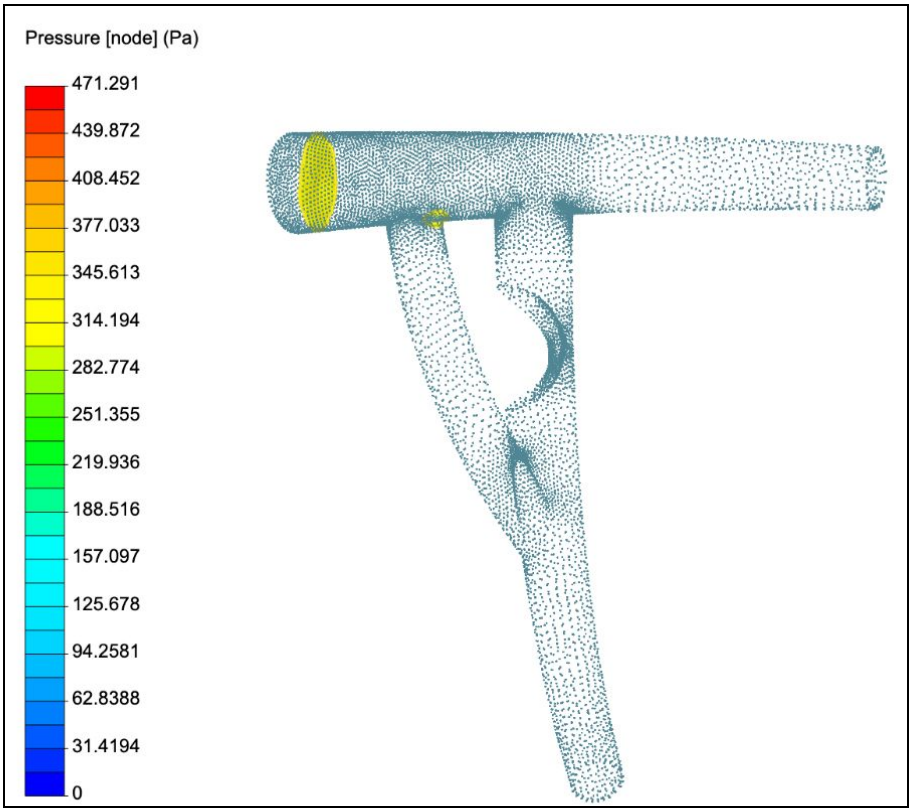

Figure 30: Isosurface of Pressure [node] at LM Inlet of Heart Bypass for Part II

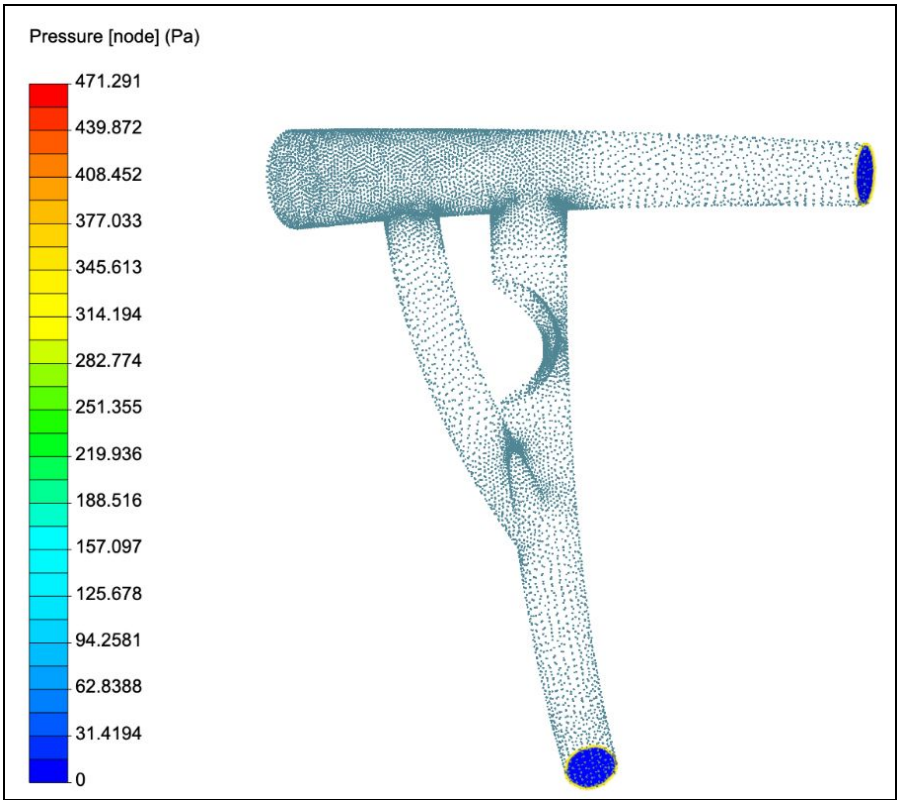

Figure 31: Isosurface of Pressure [node] at LCX and LAD Outlet of Heart Bypass for Part II

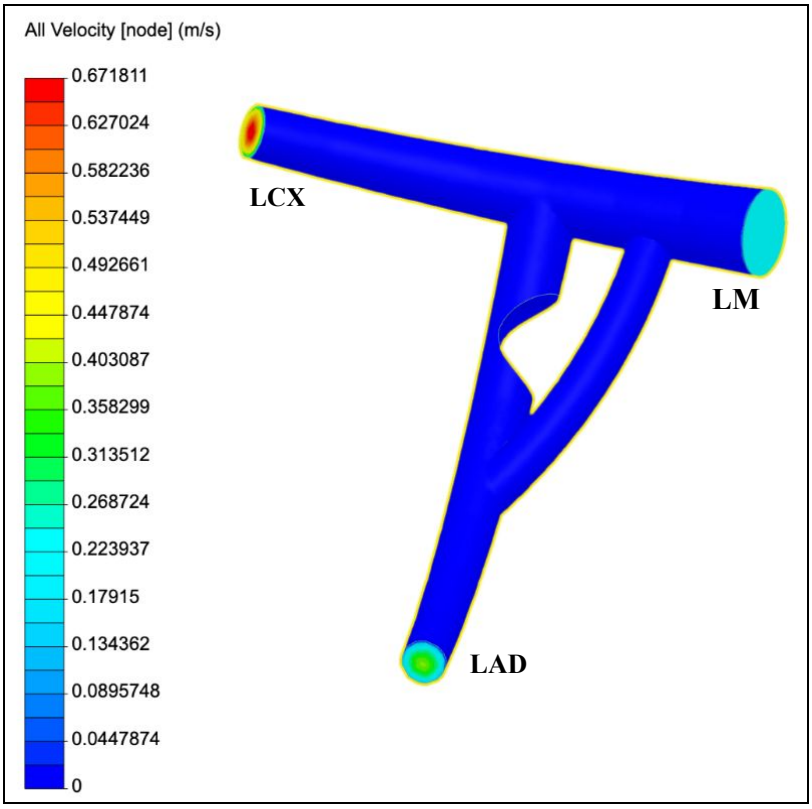

Figure 32: Isovolume of All Velocity [node] of Heart Bypass for Part II

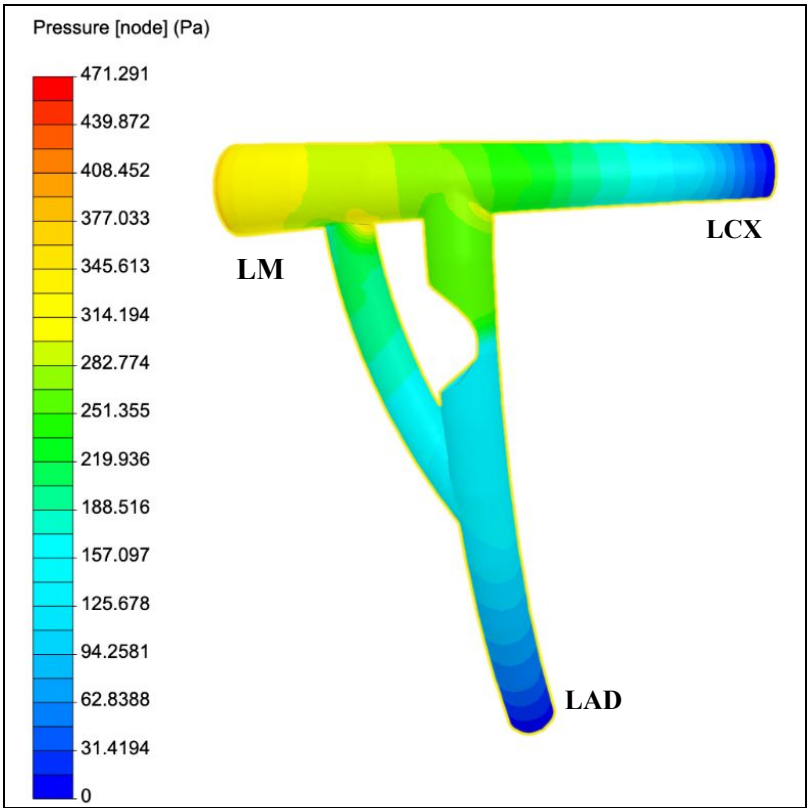

Figure 33: Isovolume of Pressure [node] of Heart Bypass for Part II

The data values obtained from the simulations are summarized as tabulated in **Table 2** below, with the relevant parameters needed to calculate the resistance and Re by using **Eq. 1 - 3** as introduced previously.

*** Table 2: Data and Simulation Results of Both Simulations for Part II**

Type of Simulation	Without Bypass			With Bypass		
Location	LM	LCX	LAD	LM	LCX	LAD
Relative Pressure, P (Pa)	830	0	0	320	0	0
Volumetric Flow Rate, Q ($\frac{m^3}{s}$)	2.70×10^{-6}	2.56×10^{-6}	9.07×10^{-8}	2.70×10^{-6}	1.89×10^{-6}	7.26×10^{-7}
Velocity, v ($\frac{m}{s}$)	0.215	0.764	0.035	0.267	0.447	0.223
Diameter, D (m)	0.0040	0.0018	0.0020	0.0040	0.0018	0.0020
Reynolds Number, Re	261	405	21	323	237	135
Blood Pressure, P_B (Pa)	14160	13330	13330	13650	13330	13330
Resistance, R ($\frac{kg}{m^4 \cdot s}$)		7.82×10^{8}	2.21×10^{10}		1.06×10^{9}	2.76×10^{9}

* Blood Density, $\rho = 1060 \frac{kg}{m^3}$

* Blood Dynamic Viscosity, $\mu = 3.5 \times 10^{-3}$ Pa · s

* Capillaries Pressure at LAD and LCX, $P_C = 2000$ Pa

From **Figure 20 - 22** and **Figure 27 - 29** for stenosed arteries without and with the bypass respectively, it could be seen that the velocity at LAD is relatively the smallest (0.035 and 0.223 $\frac{m}{s}$), and is relatively the highest at LCX (0.764 and 0.447 $\frac{m}{s}$) in comparison to all LM, LCX, and LAD. The same finding as ***Part I*** is observed for ***Part II***, where the velocity at LAD with a bypass is greater than that of no bypass, while the velocity at LCX is lower for a bypass than that of no bypass. The occlusion prevents the circulation of blood to pass through it to reach LAD, in which a lower velocity at LAD is expected for the case of no bypass. This backflow of blood will eventually flow back to LCX, making most of the flow rate to be distributed out of LCX. This same observation could be made from the volumetric flow rate, where Q_{LAD} without a bypass is smaller than that of a bypass due to the presence of occlusion preventing the smooth flow through it. To compensate for a greater flow rate at LAD of the bypass, a smaller flow rate than that of no bypass is needed at LCX by law of mass conservation. The velocity and volumetric flow rate at LM is relatively the same for both scenarios as there are no major changes at the inlet in both simulations. By referring to **Figure 23** and **24**, the relative pressure at LM of the case of no bypass is found to be 830 Pa with blood pressure of 14,160 Pa, and the pressure at both LCX and LAD are confirmed to be 0 Pa gauge pressure, as priorly set up before running the simulations. The same analysis is performed for the arteries with the bypass, in which based on **Figure 30** and **31**, the relative and blood pressure at LM is found to be 320 and 13,650 Pa respectively, with a confirmation of 0 Pa gauge pressure at LCX and LAD. From this observation, the relative pressure at LM of that with a bypass is smaller than that with no bypass. This is expected as the velocity at LM is slightly higher for the case of bypass than the one with no bypass (although the velocity is not much different). This is proven by Bernoulli's principle, where the fluid velocity is inversely proportional to the pressure. **Figure 25** and **32** represent the overall trend of velocities in the arteries, while **Figure 26** and **33** represents the overall trend of the pressures, for the case of having no and with the bypass respectively. The pressures are evenly distributed throughout the arteries for the case of heart bypass, thus explained the slight increase in velocity has a significant impact in the reduction of the relative pressure at LM.

Volumetric flow rate of blood varies across individuals. One of the factors affecting the blood flow is the blood viscosity [7]. Although the viscosity does not change gradually over a short period of time, there are two primary determinants of the viscosity, which includes the number of elements formed and plasma proteins in the blood. These said elements are referring to the erythrocytes, in which a condition affecting erythropoiesis such as anemia, will alter the blood viscosity. This condition is mostly affecting the women, young children, and people with long-term diseases [8]. In addition, any condition that is affecting the liver to produce less plasma protein, will change the viscosity slightly and increase the blood flow. Other than that, the length of vessels in the heart is also one of the factors. The length is directly proportional to the resistance flow. Individuals growing up increase the length of blood vessels, and remain unchanged in adults under normal physiological circumstances. An individual weighing 150 lbs has approximately 60,000 miles of vessels in the body [7]. Every 10 lbs increased in weight will add 2,000 to 4,000 extra miles of vessels in the body, increasing the resistance of blood flow; increasing volumetric flow rate. From this, one of the benefits of weight reduction is the reduced stress to the heart, as it is no longer required to overcome the additional resistance of many miles of vessels. Moreover, most common individuals who have high blood pressure, which is associated with high volumetric flow rate, are smokers [9]. The nicotine in cigarettes and other tobacco products constricts the blood vessels, causing the heart to beat faster. From a study comparing smokers to non-smokers in young adults (20 - 29 years old), smokers had a significantly higher heart rate than non-smokers at rest for both male and female [10]. For male and female respectively, the heart rate at rest is found to be 120.4 and 114.6 beats per minute (bpm), which are higher than the normal resting heart rate in adults, which ranges from 60 - 100 bpm [11]. Furthermore, not only the plaque can be formed naturally from the difference in shear stress intensity on the artery wall as previously discussed, the deposition of cholesterol on the artery wall is also a major reason that leads to atherosclerosis diseases. The “bad” cholesterol, known as low-density lipoproteins (LDL), can damage the arteries, in which further damages will cause LDL to keep on penetrating and building up in the artery walls [12]. When the body has started the defence attack on this buildup with white blood and other kinds of cells, over time, these cells will leave some debris and will become part of the buildup, in which will form a plaque as it grows larger over the years. This plaque formation will eventually start to block the flow of blood.

By comparing ***Part I*** and ***II*** of the artery simulations with low and high volumetric flow rate respectively, it is expected to have every parameter (flow rate, velocity, relative and blood pressure, at LM, LCX, and LAD) to be greater for ***Part I*** than ***Part II***. Due to that, the second approach is more accurate in comparison to the first approach because it is the closest one to resemble the actual blood pressure and flow rate in atherosclerosis patients. A normal blood pressure is less than 120/80 mmHg (16,000/10,670 Pa), where the first and second number represent the systolic and diastolic pressure respectively [13]. Blood pressure of higher than 130/80 mmHg (17,330/10,670 Pa) is categorized as high blood pressure, which is a major cause of atherosclerosis [14]. Regardless of these distinctions in representing the blood pressure, the blood pressure obtained in ***Part II*** is relatively closer to the pressure range of those who have atherosclerosis, in comparison to ***Part I*** data. Besides that, a study was conducted to measure the blood flow rate and velocity in the coronary artery stenosis using intracoronary frequency domain optical coherence tomography method on atherosclerosis patients [15]. It was found that most of the research participants who have this disease has blood flow rate of $4.81 \times 10^{-6} \frac{m^3}{s}$, and the flow rate in ***Part II*** is much closer to this value than that in ***Part I***.

CONCLUSION AND RECOMMENDATION

In ***Part I***, a simulation is run with both stenosed arteries with and without the bypass surgery, with a lower volumetric flow rate at the LM. It was found that the relative pressure at LM for the bypass artery is much lower than that of no bypass, as well as a higher velocity of fluid. The resistance of flow in the LAD and LCX is lower and higher in bypass respectively. The same simulation is conducted for ***Part II***, with a higher volumetric flow rate at LM. The same finding as in ***Part I*** is observed in ***Part II*** when comparing the bypass with no bypass artery. In contrast between the first and second part of the analysis, it is found that ***Part II*** is a more accurate approach than ***Part I***. This is because individuals suffering from CAD are expected to have a higher flow rate of blood and blood pressure in the body, where ***Part II*** has the closer simulation results to what was expected.

The bypass model used in this simulation is recommended to the FDA to be used as the guidelines for heart bypass surgery. The bypass should be located at the suggested location, where one end of the bypass is anastomosed to one of the aorta (which will be the LM), and the other one should be anastomosed to the end of the occlusion, at the LAD. The type of bypass used should be determined by the surgeons themselves through many years of experience in performing the surgeries [6]. Some find it easier to use a saphenous vein from the leg, but some surgeons think a radial artery in the wrist can do a much better job.

There are some limitations to this modelling approach. One of them includes the assumption of blood being a Newtonian fluid. Blood is a non-Newtonian fluid and only follows Newtonian nature when the shear rate is above 100 s^{-1} [16]. It is reasonable to assume blood as Newtonian in large blood vessels like aorta because the shear rate at that vessel is high. There is still some debate on whether to consider blood as Newtonian or non-Newtonian fluid in any cases, but it is safe to assume non-Newtonian behavior all the time [17]. From this, another simulation with high volumetric flow rate with an assumption of blood behaving as non-Newtonian fluid should be run and the results should be compared to those of Newtonian behavior. Moreover, the blood viscosity is different across individuals. This modelling assumes a constant temperature with constant density and viscosity. CAD patients come from different places around the world with different climates. Viscosity increases about 2 % for each degree of centigrade decrease in temperature [18]. One may argue that blood temperature does not change much in the body, however if one's hand is exposed to a cold environment, the blood temperature in the fingers will decrease and increase the blood viscosity (no longer constant). If the simulation could only be conducted isothermally (constant viscosity), the results obtained could not be generalized for all CAD patients as it will not be applicable to those who have much higher or lower blood viscosity than that of the simulations.

Mechanical or chemical engineering fields use CFD to analyze fluid flow, heat transfer, and associated phenomena by using computer-based simulation. In the biomedical field, utilization of CFD models is still emerging [19]. The human body fluid behavior is tremendously complex and careful understanding of fluid flow should be taken care of unlike other typical fluid. Only recently, due to high performance of hardware and software, CFD biomedical research is accessible and easily available. Many simulations and clinical results have been used to study congenital heart disease, heart failure, ventricle function, aortic disease, and many other cardiovascular diseases. CFD is an important methodology to understand the pathophysiology of the development and progression of cardiovascular disease. Computational simulations provide invaluable information that is difficult to obtain experimentally, such as blood flow through an abnormal artery. Medical simulations of blood circulation have many benefits, which include lowering the chance of postoperative complications, delivering good understanding of biological processes, as well as more efficient and less destructive medical equipment like blood pumps.

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