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ANALYSIS OF THE STUDY PERFORMED WITH CFD SOFTWARE IN VEINS SUBMITTED TO VALVULOPATHIES

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Abstract. *Nearly 50% of American adults have at least one form of cardiovascular disease (CVD). It has been observed an increase in the incidence of cases, especially among pregnant women, people over the age of fifty-five, and conditions such as obesity, diabetes, and high cholesterol, among others. The sooner the detection of CVD, the easier is to treat it. Therefore, diseases developed in the cardiovascular system have been significantly discussed and studied in the past years. Valvulopathies are diseases involving the valves in which blood fluid flows. This dysfunction is responsible for restricting and reducing the useful internal section of the tube, preventing the fluid from circulating freely and reaching the organs efficiently. Flow pressure and velocity are directly related, as described by Bernoulli's equation, with the tendency for the velocity to decrease when the pressure increases. The present work seeks to demonstrate, under the principles and guidelines of fluid mechanics, how the blood fluid reacts to the restrictions present in the valves and the possible consequences to the human body. Recently, several studies are being carried out to predict the behavior of the blood fluid inside the cardiovascular system. Using a commercial code of computational fluid dynamics (CFD), the objective of this paper is to analyze the blood fluid in a tube with boundary conditions like the ones in a blood vessel and to predict its behavior when there is a narrowing of the tube cross-section, which happens when the human body is subject to a state of stenosis and/or insufficiency.*

Keywords: *valvulopathy, CFD, cardiovascular system, pressure.*

1. INTRODUCTION

Cardiovascular diseases are a group of disorders of the heart and blood vessels. They represent the leading cause of global death, accounting for 32 percent of total deaths, an estimated 17.9 million people each year, with dramatic socioeconomic impact (Altamura et al., 2023). Valvulopathies are diseases that affect the cardiac valves, which are important anatomical structures in the heart responsible to regulate the flow of the blood. There are two types of dysfunction of the cardiac valves: the stenosis, or the narrowing of the valve, when the valve does not open sufficiently; and the insufficiency, or leakage, which occurs when the valve does not close properly, and the heart must work more to compensate. Valvulopathies can lead to serious disturbances in blood circulation, including the backflow of blood into the veins and the formation of blood clots, which can compromise heart function and lead to serious complications, such as heart failure. (MSD Manual, 2023).

To better understand the nature of valvulopathy and its effects on blood circulation, the study of fluid dynamics can be applied to analyze the pressure and velocity of the blood flowing through the veins. With the aid of computational fluid dynamics (CFD) software, it is possible to determine the velocity of blood, the direction of blood flow, and the controlled pressure on the walls of blood vessels. This analysis can help identify areas of blood flow stagnation, indicating the presence of blood clots, as well as backflow of blood caused by failing cardiac valves. In addition, analysis of blood pressure and velocity can guide the development of effective treatment strategies for valvular heart disease, such as cardiac valve repair or replacement surgery. (MSD Manual, 2023).

Mechanical engineering plays an important role in medicine and biology. Many common types of medical devices are purely mechanical. Furthermore, the circulation of blood inside the human body can be predicted using fluid mechanics equations. The fluid dynamics of the blood flow in arterial geometries most relevant in the context of atherosclerosis were experimentally and numerically evaluated by (Trigui et al., 2021). 3D axisymmetric 50% and 75% diameter reduction stenotic blood flows were investigated on steady and pulsatile flows. Measurement of ultrasound and shadow particle image velocimetry for a left ventricular outflow tract were compared to simulation model results obtained using CFD techniques by (Leinan et al., 2022). The CFD results coincide very well with the PIV flow field maximum velocities and curl intensity, as well as with the detailed vortex structure, however, this correspondence is subject to exact boundary conditions. A comparison of a 3D morphologic modeling and computational flow analysis of bicuspid aortophaty was performed by (Pasta et al., 2017). The authors developed a parametric program for 3D representations of aneurysmal aorta and bicuspid aortic valve phenotypes based on measurements derived by computed tomography angiography. The software provided a fast tool for 3D anatomic representations of bicuspid aortopathies. The change in the wall shear stress and intramural wall stress of patient-specific ascending thoracic aortic aneurysm models with different degrees of aortic stenosis was studied by (Cosentino et al., 2020) using computational analysis.

Although several studies concerning the use of CFD in medicine are found in the literature, the number of studies predicting the effect of stenosis in the cardiac valve is not large. Most works use very robust software to perform the analysis, which requires computational time and effort (Vellguth et al., 2018). The objective of this paper is the analysis of blood in a cardiac valve subject to stenosis, using simple CFD techniques. The velocity and wall pressure on a geometry of a valve with and without the presence of an obstruction were predicted and compared to each other.

2. MATHEMATICAL MODEL

2.1 Fluid flow and properties

Newtonian fluids show a linear relation between applied shear stress and the resulting strain rate (Fox et al., 2018). Non-Newtonian fluids are particular kinds of fluids in which the relationship between shear stress and velocity gradient is nonlinear and do not follow Newton's law of viscosity. In addition, the viscosity of these fluids changes with the application of force. These fluids have many valuable applications in various sciences and industries, including chemistry, mechanics, medicine, and electronics, among others (Zhu et al., 2023). The study of non-Newtonian fluids is called rheology. The study of rheology emerged in the 20th century and has as its main study the analysis of the behavior of deformations and the flow of materials. (Macosco, 1994).

Human blood is a complex heterogeneous mixture, which consists of red blood cells, white blood cells, and platelets combined within the liquid plasma, as shown in Fig. 1. Blood consists of two phases: continuous and discrete aqueous. The continuous aqueous phase (plasma) contains proteins, sugars, and salts; the discrete phase encompasses red blood cells (erythrocytes), white blood cells (leukocytes), and plasma. Although blood behaves like a non-Newtonian fluid, plasma separately has characteristics of a Newtonian fluid. The properties of blood depend mainly on temperature, cell shape, shear rate, mechanical properties of red blood cells, and cell orientation. Specifically, blood can exhibit non-Newtonian behavior known as yield stress (Chandran, 2003).

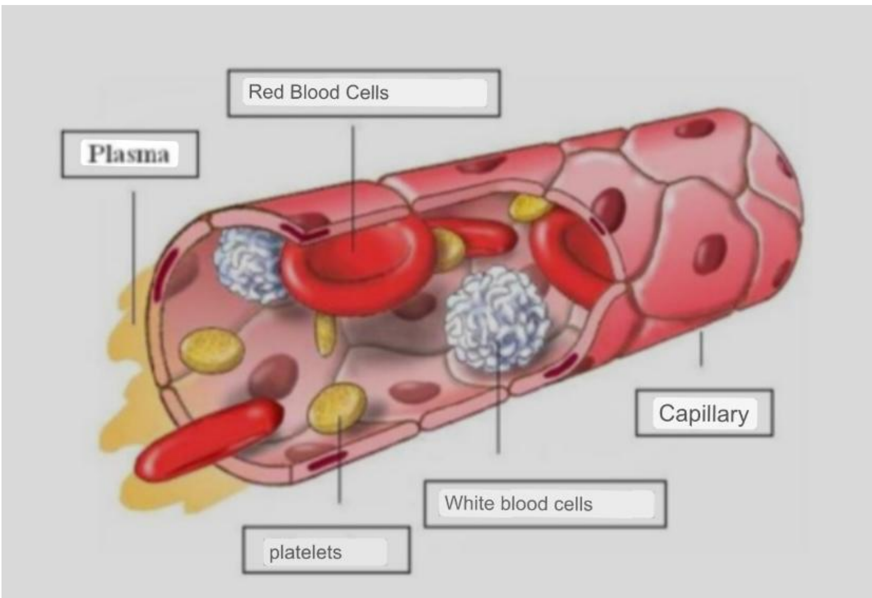

Figure 1: Structural representation of a blood vessel with the blood fluid inside.

Blood (originating from hematopoietic tissue) is a highly specialized tissue composed of various types of cells, forming the formed elements, dispersed in a liquid medium called plasma, which corresponds to the amorphous component. The cellular constituents include red blood cells (also known as erythrocytes) and white blood cells (also referred to as leukocytes) (Nader et al., 2019).

To account for the rheology of the blood, several non-Newtonian models have been proposed accordingly. However, despite several rheological models proposed in the literature, Carreau-Yasuda is among the most widely used ones (Seehanam et al., 2023).

2.2 Governing equations

The governing equations of fluid flow represent mathematical statements of the conservation laws of physics. The flow is assumed to be incompressible. The mass conservation and RANS (Reynolds-averaged Navier-Stokes) equations (Equations 1 and 2, respectively) can be written in Cartesian tensor notation as follows (Alfonsi, 2009):

$$\frac{\partial \rho}{\partial t} + \frac{\partial \underline{u}_i}{\partial x_i} = 0, \tag{1}$$

ρ is the density of the fluid, t is time, and $\underline{u}_i$ is the time-averaged velocity vector.

$$\frac{\partial \underline{u}_i}{\partial t} + \frac{\partial}{\partial x_i}\left(\underline{u_i u_j}\right) = -\frac{\partial p}{\partial x_i} + \nu \frac{\partial^2 \underline{u}_i}{\partial x_i \partial x_j}, \tag{2}$$

p is the pressure and ν is the kinematic viscosity of the fluid.

The turbulent properties are evaluated using the k-ε turbulence model. The turbulent kinetic energy k and the dissipation of the turbulent kinetic energy ε are determined through transport equations (Equations 3 and 4), as given in (Alfonsi, 2009).

$$\frac{\partial k}{\partial t} + \underline{u}_i \frac{\partial K}{\partial x_i} = -\tau_{ij} \frac{\partial \underline{u}_i}{\partial x_i} - \varepsilon + \frac{\partial}{\partial x_i}\left(\frac{\nu_t}{\sigma_k} \frac{\partial K}{\partial x_i}\right) + \upsilon \frac{\partial^2 K}{\partial x_i \partial x_j}, \tag{3}$$

$$\frac{\partial \varepsilon}{\partial t} + \underline{u}_i \frac{\partial \varepsilon}{\partial x_i} = -C_{\varepsilon 1} \frac{\varepsilon}{k} \tau_{ij} \frac{\partial \underline{u}_i}{\partial x_i} + \frac{\partial}{\partial x_i}\left(\frac{\nu_t}{\sigma_\varepsilon} \frac{\partial \varepsilon}{\partial x_i}\right) - C_{\varepsilon 2} \frac{\varepsilon^2}{k} + \upsilon \frac{\partial^2 \varepsilon}{\partial x_i \partial x_j}, \tag{4}$$

The Reynolds stress tensor is given by Equation (5)

$$\tau_{ij} = \frac{2}{3} k \delta_{ij} - \nu_t \left(\frac{\partial \underline{u}_i}{\partial x_j} + \frac{\partial \underline{u}_j}{\partial x_i}\right), \tag{5}$$

The eddy viscosity is given by Equation (6)

$$\nu_t = C_\mu \frac{k^2}{\varepsilon}, \tag{6}$$

In Equations 3-6, δ_{ij} is the Kronecker delta, and the constants assume the values:

$$C_{\varepsilon 1} = 1.44 \quad C_{\varepsilon 2} = 1.92 \quad C_\mu = 0.09 \quad \sigma_k = 1.0 \quad \sigma_\varepsilon = 1.3$$

A parameter to be evaluated is the shear stress. Blood is a living fluid, and if the forces applied to the fluid are sufficient, the resulting shearing stress can cause red blood cells to be destroyed. On the other hand, studies indicate a role for shear stress in modulating atherosclerotic plaques (Waite, 2007). In Figure 2, the shear stress on the top of the element results in a force that pulls the element downstream" The shear stress at the bottom of the element resists that movement (Waite, 2007).

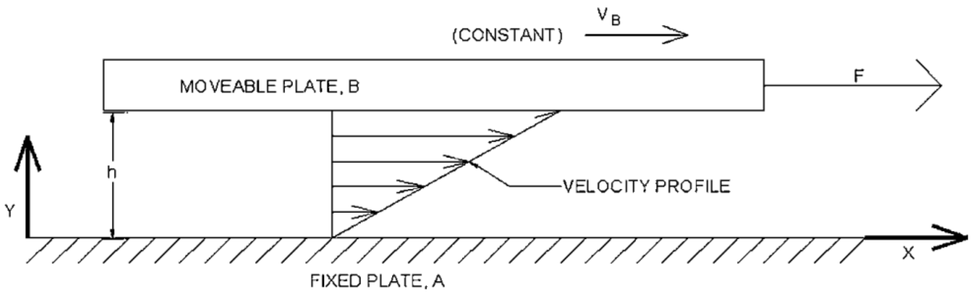

Figure 2: Velocity profile in a fluid between two parallel plates.

The shear stress on a fluid is related to the rate of shearing strain. If a very large force is applied to the moving plate B, a relatively higher velocity, a higher rate of shearing strain, and a higher stress will result. The relationship between shearing stress and the rate of shearing strain is determined by the fluid property known as viscosity (Waite, 2007).

3. METHODOLOGY

In his study, it was performed a simulation of the blood flow inside the cardiovascular system, using the commercial codes Fusion360 and Autodesk 2023 CFD. Fusion 360 is a commercial computer-aided engineering design software application, developed by Autodesk, used to generate the geometry of the flow. Autodesk 2023 CFD creates computational fluid dynamics and was used to generate the mesh and to simulate the flow. Two simulations were conducted, one with a constant diameter tube, and the other with a tube with a restriction, simulating the stenosis of the vessel.

The computational domain is shown in Figures 3 and 4. An external diameter of 250 mm and an internal diameter of 200 mm were adopted, with the diameter dropping to 100 mm in stenosis. The length is 1000 mm and the material chosen for the tube was titanium.

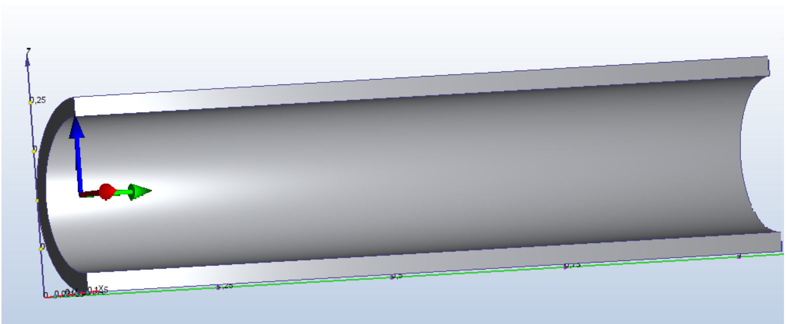

Figure 3: Normal blood vessel modeling

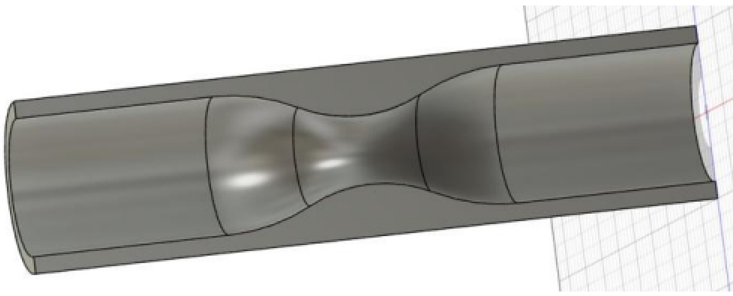

Figure 4: Blood vessel modeling with stenosis

The mesh used is shown in Figures 5 and 6, for the vein without and with stenosis, respectively. After a mesh test, it was defined a mesh of 5908 elements in both simulations.

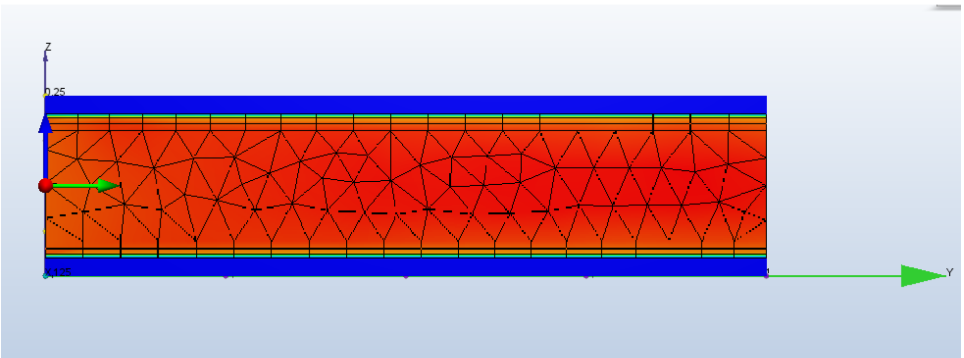

Figure 5: Mesh in normal vessel

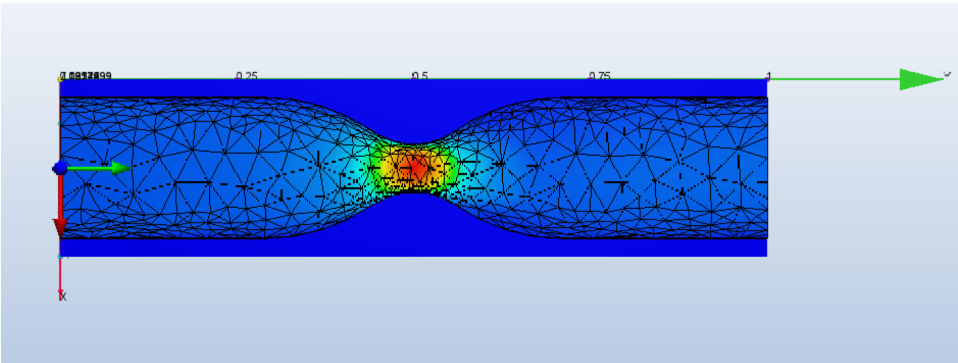

Figure 6: Mesh in stenosis vessel

As boundary conditions, an average velocity of 0.56 m/s was adopted at the inlet, and an outlet condition was assumed at the end of the tube, consistent with fully developed conditions. The pressure at the outlet was assumed as 19998. Pa, with a constant temperature of 37°C, as indicated by (Waite & Fine, 2007). The fluid was selected from the software library as “blood”, which presents non-Newtonian characteristics.

4. RESULTS AND DISCUSSION

This section presents the velocity and pressure fields obtained for a geometry with and without stenosis.

4.1. Blood flow through an unobstructed valve

For the valve without stenosis, the blood flows without any obstructions. Figure 7 shows the velocity field inside the vessel. The velocity is null at the wall, according to the boundary conditions, and is maximum at the centerline, reaching a value of 0.61 m/s. It can be seen that the flow presents characteristics of a fully developed flow.

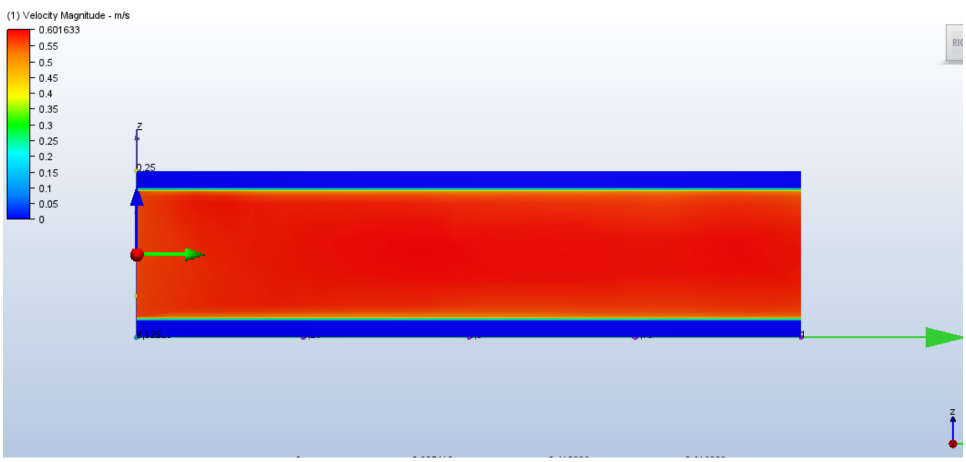

Figure 7: Velocity in the vessel without stenosis

The pressure field is shown in Figure 8. Since it was not imposed any obstruction, the pressure reduces only due to the head losses, and the pressure drop was low, of about 100 Pa in a length of 1 m.

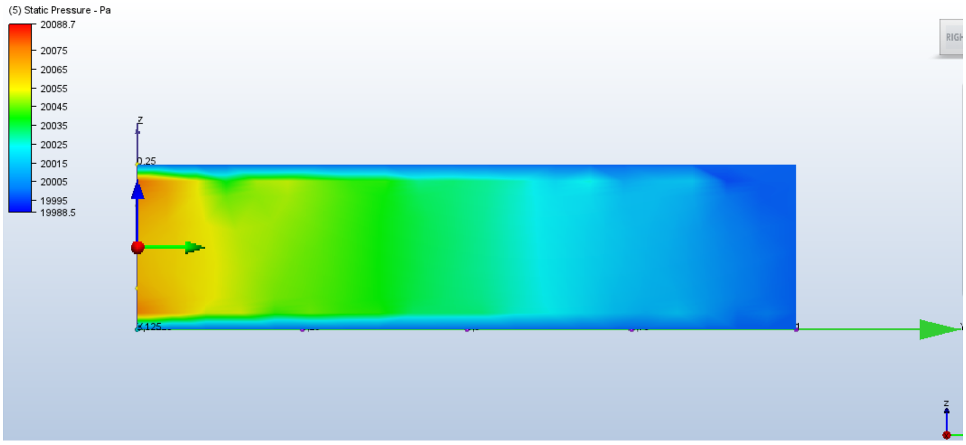

Figure 8: Pressure in the vessel without stenosis

The pressure gradient is not linear due to the variation in the viscosity of the fluid, presented in Figure 9. Since the fluid was selected as blood, the viscosity is not constant, and it is near the centerline. The maximum value found for the dynamic viscosity was 0.072 Pa.s.

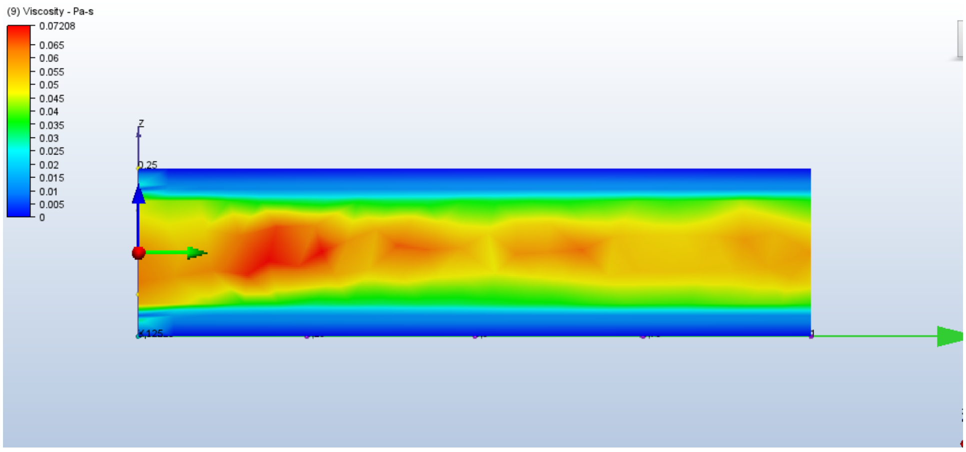

Figure 9: Dynamic viscosity of blood in the vessel without stenosis

As the shear rate increases, viscosity decreases (Leo; Simmonds; Sabapathy, 2019). For vein without any restriction, the shear stress was low (nearly 8 Pa), as seen in Figure 9, due to the low variation in the flow pressure.

Figure 10: Shear stress of blood in the vessel without stenosis

4.2 Blood flow through a valve with stenosis

For the valve with stenosis, there is a restriction imposed on the fluid. It is observed a narrowing of the tube diameter, which affects the flow. Figure 11 shows the velocity field inside the vessel. In most of the computational domain, the velocity is low, increasing significantly only in the restriction. The maximum velocity found was 5.62 m/s, in the throat. The simulations indicated that mitral stenosis led to an increase in blood flow velocity through the restricted mitral valve. This increase in velocity is a compensatory response of the cardiovascular system to ensure adequate blood flow to the systemic circulation. It is exposed by (Otto, 2006) that the velocity above 4 m/s represents a severe stenosis.

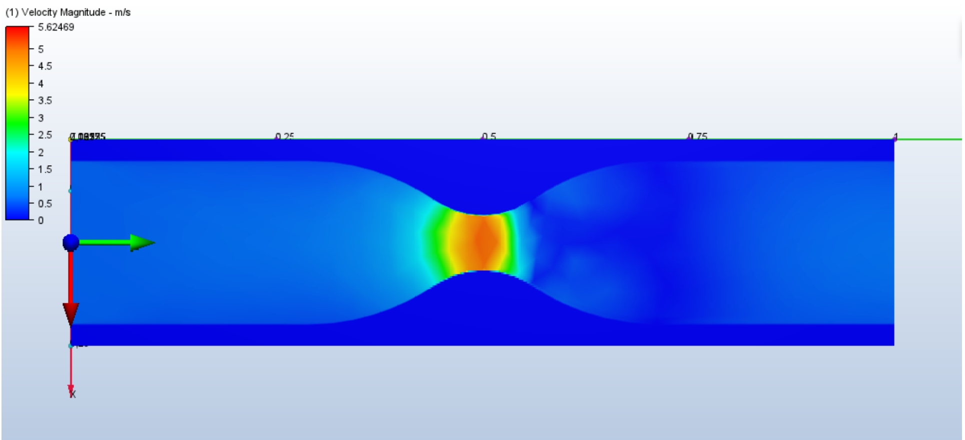

Figure 11: Velocity in the vessel with stenosis

The presence of mitral stenosis resulted in a narrowing of the mitral valve, hindering blood flow from the left atrium to the left ventricle. This restriction caused an increase in pressure in the left atrium, known as increased preload, as seen in Figure 12. The pressure difference between the inlet and outlet reached 6.36 kPa. As a direct result of the increase in aortic pressure due to mitral stenosis, there was also an increase in afterload, that is, the resistance that the ventricle must overcome to eject blood into the systemic circulation. These changes in preload and afterload conditions affected left ventricular performance. In the simulations, it was observed that the ventricle did not empty properly due to the increase in afterload. However, as the force of contraction of the ventricle increased, it was found that more blood was ejected during systole, resulting in more complete emptying of the ventricle. This allowed for more efficient filling of the ventricle in the next cardiac cycle.

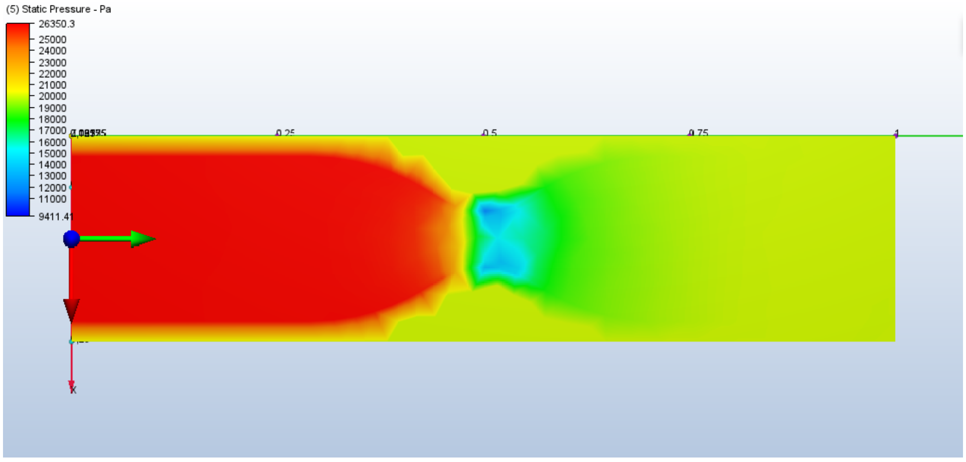

Figure 12: Pressure in the vessel with stenosis

The dynamic viscosity of the blood is shown in Figure 13. The maximum value found was 0.084 Pa.s, approximately 16% higher than the value found for the flow without stenosis. The higher velocities and pressures caused an increase in the dynamic viscosity. Due to the pattern of the flow, the viscosity distribution differs from the distribution found for the flow without stenosis.

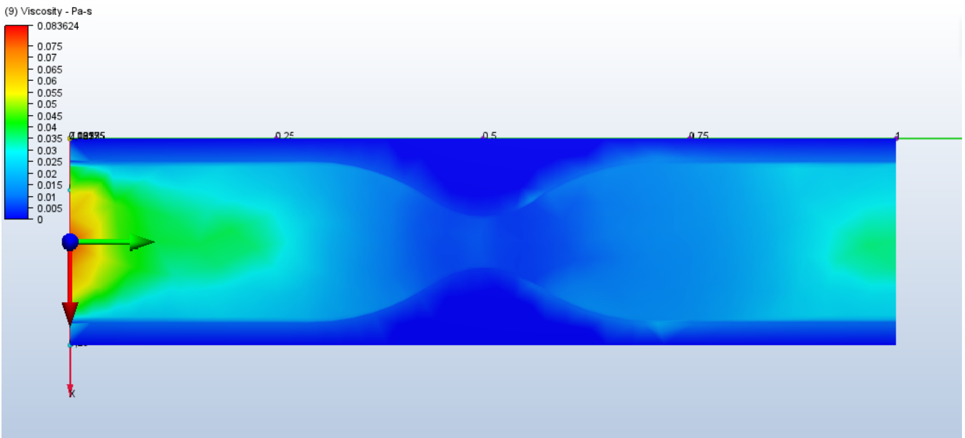

Figure 13: Dynamic viscosity of blood in the vessel with stenosis

It is more evident to note the decrease in viscosity and increase in shear stress (Figures 13 and 14) mainly due to the change in geometry (Shanmugavelayudam et al., 2010). The increase in the shear rate promotes greater effort in the region, contributing to the severity of the valvulopathy. The shear stress was significantly increased when compared to the values found without stenosis, from 8 Pa to 257 Pa.

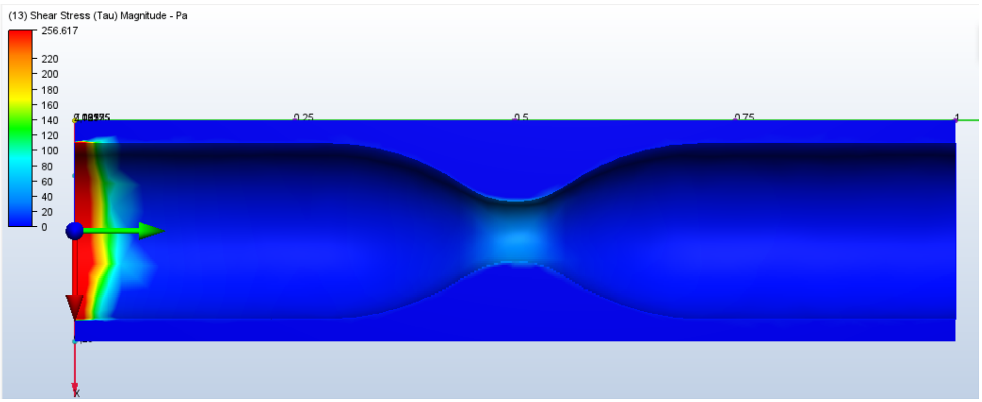

Figure 14: Shear stress in the vessel with stenosis

5. CONCLUSIONS

The study on valvular heart disease, specifically mitral stenosis, revealed many significant discoveries regarding hemodynamic behavior and its consequences on the cardiovascular system. This paper presented a numerical simulation of the stenosis in the blood flow, which allowed us to evaluate the effects of stenosis on pressure and velocity. It has been identified that the presence of mitral stenosis results in an increase in pressure in the left atrium due to the narrowing of the mitral valve opening, causing an increase in preload. In addition, increased aortic pressure as a result of stenosis creates greater afterload for the left ventricle, making it difficult for the ventricle to empty adequately.

For a reduction of 75% of the flow area, it was found that the maximum velocity increased from 0.56 to 5.62 m/s. The pressure difference between the inlet and the outlet increased from 100 Pa to 6.36 kPa. The shear stress increased from 8 Pa to 257 Pa.

These results reinforce the importance of early diagnosis and adequate treatment of mitral stenosis. Understanding the hemodynamic changes associated with valve stenosis allows for a better understanding of the pathophysiological mechanisms involved and can guide therapeutic approaches. However, it is important to emphasize that computer simulations provide a simplified view of the cardiovascular system and must be complemented with real clinical data for a more complete and accurate analysis.

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