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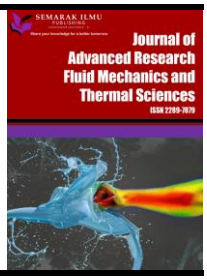

Computational Analysis of Hemodynamic Blood Flow through Stenotic Human Artery

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ABSTRACT

Cardiovascular system conditions, such as aneurysms, narrowing in coronary arteries, heart attacks, and strokes, have been identified as the major cause of death globally. The aorta of the heart, coronary, carotid, and femoral arteries are the arteries that are most often impacted. It is a crucial field of research for understanding blood circulation behavior and viscosity under shear. The mathematical evaluation and modeling of blood circulation via stenosis artery walls at various velocity levels have been investigated during this research. The hyperbolic trajectory is employed at the input, whereas fixed pressure is applied at the outlets. For motion, uniform Neumann parameters are utilized, and for vascular stress, no-slip parameters are utilized. Using an expression of normalized primitive variables, the laws of momentum as well as mass equations are presented. Using the Galerkin weighted residual approach of the finite element method; the laws of motion that are free of dimensions are solved. The mathematical models of blood flow's results are shown in terms of streamlines and directions along the vessel axis, pressure graphs, and velocity contour lines. The graphical analysis has demonstrated the impacts of blood speed, arterial pressure across the vessel axis, wall shear stress, and governing variables for the model, specifically Reynolds number *Re.* It is observed that there is great agreement between the findings of code validation and previously published research. The findings show that variables like the Reynolds value Re and the length of the stenosis have a significant impact on blood flow and pressure. This study will assist doctors in better identifying any CVD conditions associated with stenosis and will help them provide treatment.

1. Introduction

In the fields related to cardiovascular pathophysiology and biological mechanics, hemodynamic blood circulation is a crucial topic of research. An artery can become stenosis, or narrowed, as a result of the accumulation of cholesterol or other factors. This stenosis may cause decreased

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circulation, elevated pressure, and turbulent motion, which may raise the probability of clotting and other problems. In accordance with Yilmaz *et al.,* [1], it has been established that cardiovascular conditions include aneurysms, atherosclerosis (stenosis), coronary artery disease, and stroke as the most common cause of mortality worldwide by the World Health Organization (WHO). The thoracic aorta, carotid, coronary, and femoral arteries are the ones that are the most frequently damaged. Different mathematical frameworks that replicate blood flow via an artery may be utilized to investigate how stenosis affects circulation. Different mathematical approaches may be utilized to mimic the movement of blood via an artery in order to better comprehend the impact of narrowing on blood flow. These models frequently take the morphology of the blockage in addition to the blood's flow rate, pressure, and disturbance into account. Once stenosis intensity has changed and other parameters have been taken into account, the simulations may be used to assess how the blood flow has changed. There are no significant disruptions in the circulatory system's big velocity flow must be introduced whenever blood circulation is switched in any form (prosthesis, stent, and fistula) in order to cure disease [2]. Analyses based on computational fluid dynamics, also known as CFD, are a widely used technique for simulating physiological blood circulation via constricted arteries. Simulations such as these may be used to analyse big and complicated systems since they represent blood flow using mathematical formulas. In addition to quantifying adjustments to blood flow and instability, the findings of these computations may be utilized to visualize trends in flow including pressure gradients. It is also possible to combine mathematical models with a particular patient's radiology and empirical evidence. The optimum course of therapy may be determined by the doctor with the use of computerized models, which may in this situation have predictive capacity [3]. A different strategy is to employ computational models built on the Navier-Stokes formulas and a Reynolds transports theorem, which explain both the movement of fluid and the interactions between forces that control its behaviour. These simulations may be utilized to explore how stenosis affects blood flow in addition to assessing the effects of different therapies like angioplasty or stenting. In conclusion, research into the study of hemodynamic blood circulation via stenosis-prone arteries is extensive and continuous. A variety of mathematical models and methods are employed to simulate and comprehend the impact of stenosis on blood circulation. The interchange of information and expertise that may be utilized to model medical conditions has attracted more interest in recent years among bioengineers, computational investigators, and medical professionals. These research findings are essential for generating novel therapies to enhance the results for patients as well as bettering our comprehension of cardiovascular disorders.

A number of studies exist on the blood flow through the artery with symmetric stenosis but no study found on the blood flow through the artery with realistic composite stenosis. The purpose of this work is to quantitatively examine circulation dynamics while taking into account stenosis of various realistic composite geometries. Results will be discussed in terms of streamlines, variations in pressure, and shear stress on walls for various parameters, including the Reynolds numbers (Re), Weissenberg Number (Wi), etc. The principal objectives of the suggested work are nonetheless to choose an anatomical representation of an artery with composite geometry stenosis, create and mimic a computerized representation of blood flow within an artery with combined geometry congestion, resolve the equations that govern using the technique of finite element analysis (FEM), and demonstrate the findings with prior hypothetical and computational aspects work.

2. Literature Review

Aberrant tumours of the walls of arteries can develop in a variety of locations across the circulatory system. Atherosclerosis and aneurysms are the primary causes of arterial disease. According to theory, inadequate cellular development and cholesterol build-up in the artery wall are the two main causes of abnormal expansions in the arterial lumen. Lower-density lipoproteins (LDL) and other compounds are accumulating in the artery wall at an increasing rate, which causes the cavity to shrink and significantly alters blood flow, resulting in the underlying cause of this condition. Atherosclerosis, which results in aberrant hemodynamic circumstances in arteries, accounts for over 31% of all fatalities globally from cardiovascular disease [4-6]. In a word, atherosclerosis is a silent, multifactorial, and complex disease that emerges as plaque when cholesterol and immune system cells clump together in the arterial wall [7]. The circulation of blood decreases significantly whenever an artery is totally stopped, which may eventually cause chest discomfort or a cardiac event [8,9]. Such arterial diseases progress in part due to fluid dynamic variables. Given that a number of cardiovascular diseases are closely linked to conditions involving arterial flow, there has been a sharp increase in the need for hemodynamic research in recent years [2]. Moreover, a recent study revealed that coagulant venous and arterial occlusions were highly likely among COVID-19 carriers. Recent studies also showed that individuals with COVID-19 had a high risk of coagulant venous and arterial occlusions. If we take into account the number of fatalities the virus has yet to cause and its connection to the emergence of atherosclerosis, the picture gets considerably more problematic [10]. Although rather substantial damage results when this disease grows in the afflicted arteries, the circumstance is considerably worse when it does so in the arteries that supplies the heart. The accumulation of fats creates an obstacle that restricts blood circulation to the chambers of the heart muscle whenever they are injured, which results in a reduction in the amount of pus, oxygen consumption, along other nutrients essential for the heart muscle's functioning effectively. A cardiac event or chest discomfort may eventually result from an artery that is fully paused if the circulation is sufficiently limited [11,12]. In the event the fact that the artery is still patent, blood flow can be resumed; nevertheless, the danger of restlessness is still present [13,14]. Due to the disease's widespread effects, it is crucial to have a complete grasp of how blood flow is affected by hemodynamic. As a result, several studies have been conducted in the area using either empirical or numerical techniques. Since numerous cardiovascular disorders are directly connected to flow conditions, there has been an increase in interest in hemodynamic research recently [15]. Vessels that carry blood under the use of an analytical technique, in a study by Kumar [16] thoroughly investigated the continuous flow by using an axisymmetric confinement and demonstrated how the movement patterns are highly influenced by the symmetry of the confinement and its preceding Reynolds number that is present. This can be treated with angioplasty using balloons and intracoronary stenting. Numerous mathematical investigations and practical research have been conducted on the flow of fluid through the blockage since it is obvious that the pulsatile character of the stream cannot be disregarded [17-19]. The blood flow is modelled as Newtonian in the majority of these investigations. When it concerns high-velocity flow via bigger arteries, the Newtonian behaviour of blood hypothesis is appropriate. In smaller arteries, it is now widely understood that blood, which is a cell being suspended, behaves like a low-shear fluid that does not obey Newton's laws under specific flow circumstances [20,21]. According to Sankar and Nagar [22], and Razhali and Taib [23], the blood passing through tiny capillaries contains two layers: an anatomical layer of suspended haemoglobin and the plasma barrier that is free of erythrocytes and located next to the capillary wall (the Newtonian layer). Uddin *et al.,* [24] and Karim *et al.,* [25], who agreed with this idea, stated in their test findings that when applied to

human blood, Casson's fluid model would yield the most accurate portrayal of blood. The goal of this work was to develop a model that could replicate the patterns of blood flowing via a portion of a human being's artery that had symmetric stenosis [26].

3. Methodology

3.1 Artery Modeling

The lower segment of the aorta and the cardiac muscle that surrounds it are the focus of this model's study of the vascular system. The surrounding muscles and interior surfaces of the artery are under pressure from the blood flow, which damages the tissue. The investigation consists of two distinct but connected steps: first, a fluid mechanics assessment which involves determining the blood's velocity field as well as pressure distribution; and second, a mechanical assessment of the tissue and artery's displacement. The heart muscle must be taken into account during the mechanical investigation because of its rigidity, which prevents arterial distortion brought on by applied pressure. The following figure illustrates the shape of the regular (Figure 1) and stenosed (Figure 2) arteries under consideration.

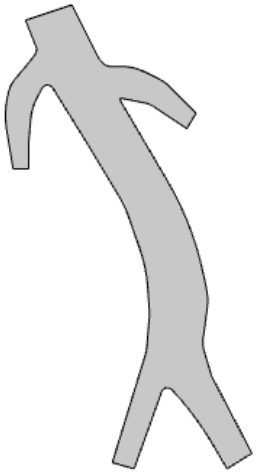

Fig. 1. Computational domain of the regular artery

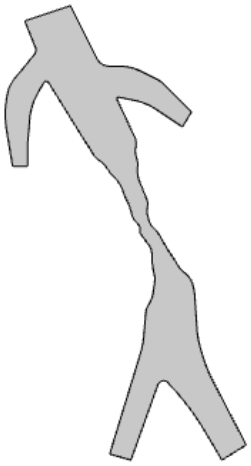

Fig. 2. Computational domain of the patient-based artery with stenosis

3.2 Flow Modeling

The basic formulas for modelling fluid flow are known as the Navier-Stokes equations. Conserving mass and momentum using the simplified Navier-Stokes formulae is

$$\frac{\partial u_i}{\partial x_i} = 0$$

When the external gravitational force is assumed to remain constant and the material is incompressible.

$$\frac{\partial u_i}{\partial x_i} = 0 \tag{1}$$

$$\rho u_j \frac{\partial u_j}{\partial x_j} = -\frac{\partial p}{\partial x_i} + \mu \frac{\partial^2 u_i}{\partial x_j^2} + F \tag{2}$$

where $u_i = (u, v, w)$ is the local velocity, $x_i = (x, y, z)$ is the length coordinate, p is the fluid pressure, μ is the dynamic viscosity. The attraction force acting on the vessel's wall is represented by the final statement in Eq. (2).

The blood-like fluid was modelled under isothermal, which is incompressible, and Newtonian (constant viscosity) conditions. The parametric equations,

$$\sigma_{ij} = -p\delta_{ij} + 2\mu e_{ij} \tag{3}$$

where σ_{ij} is the stress tensor, μ is the fluid viscosity, and δ_{ij} is the Kronecker delta.

3.3 Finite Element Mesh

Free-triangular moving mesh is applied to discretize the computational domain of the artery under consideration. The solver used the physics-controlled mesh which generates a grid of 7,769 elements for regular artery, as shown in Figure 3, and a grid of 11,153 elements for stenosed artery, as shown in Figure 4. Some other configurations are also used to simulate the flows which are not shown in the following figures.

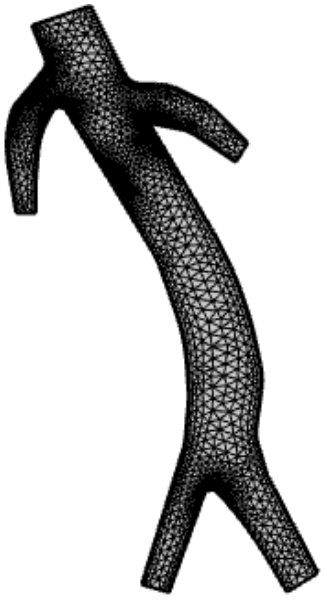

Fig. 3. Free-triangular moving mesh of the regular artery (7,769 elements)

Fig. 4. Free-triangular moving mesh of the stenosed artery (11,153 elements)

4. Results

The present framework made an effort to predict a few key aspects of blood flow following artery stenosis while accounting for body displacement and pulsatile pressure gradient. Applying a particular pressure gradient at the intake and exit at zero pressure results in pulsatile blood flow, which is believed to be homogeneous and Newtonian. No-slip circumstances are employed to

calculate the velocity within the vessel, with the artery walls and neighbouring muscle modelled as hyperplastic Neo-Hooke materials.

4.1 Stenosis Effect on Blood Flow

A gradient in pressure is created at the entrance and exit of this ascending tube where blood flows via a parabola velocity diagram from the left. The fluid velocity is quite varied in the stenosis zone and rises (high-velocity gradient) as it approaches the throat. Figure 5 and Figure 6 illustrate the distribution of blood flow velocity at the stenosed and normal arteries. The region whereby the flow divergence takes place immediately following the formation of stenosis is the region that's of interest. The recirculation zone contains a significant amount of particles.

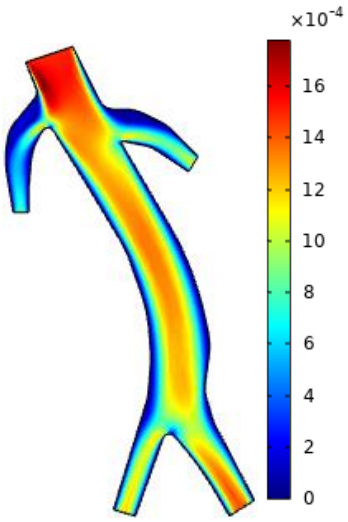

Fig. 5. Blood flow field for regular artery

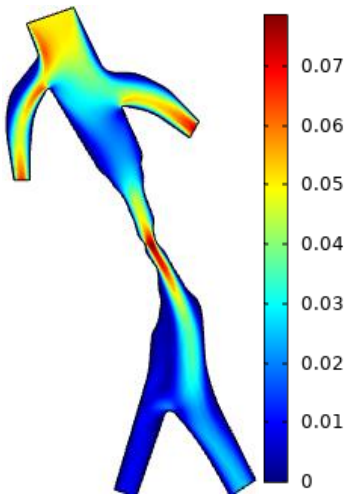

Fig. 6. Blood flow field for stenosed artery

4.2 Pressure Contour

The fluid domain's pressure contours are depicted in the accompanying Figure 7 and Figure 8. The findings demonstrate that as the blood vessel travels through the stenosis zone, it progressively rises relative to the regular arteries and takes on a parabolic shape. The following section of the artery has a significant reduction in pressure, but its pressure contour also changes, which is understandable as shown in Figure 9 and Figure 10.

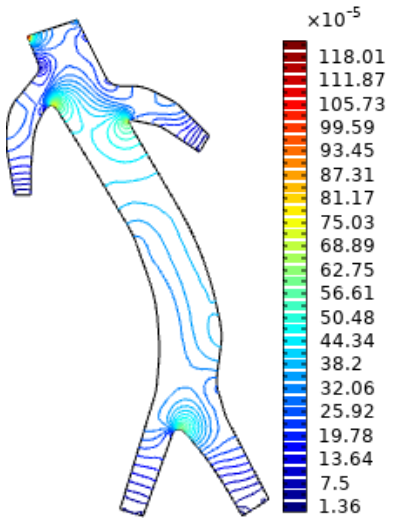

Fig. 7. Pressure contour for regular artery

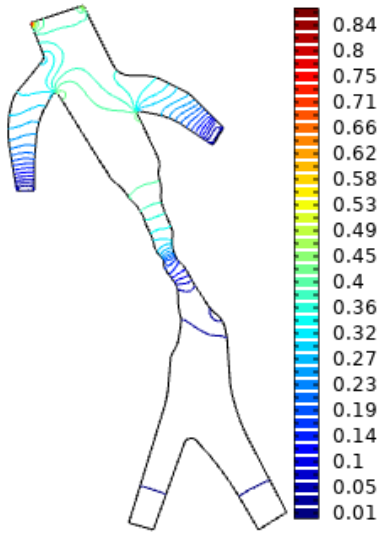

Fig. 8. Pressure contour for stenosed artery

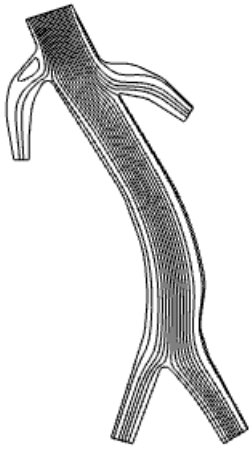

Fig. 9. Streamlines for regular artery

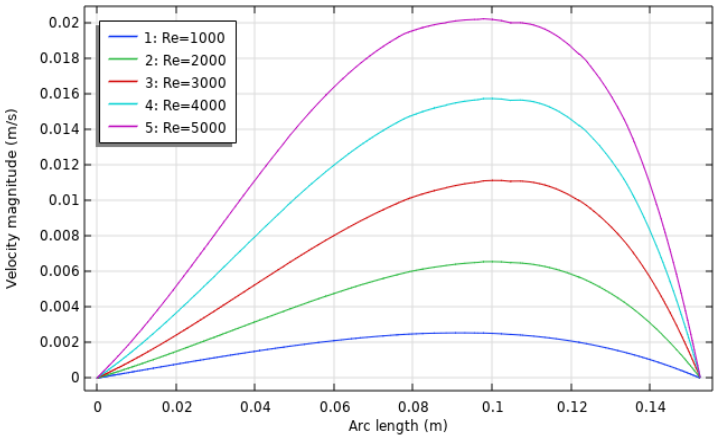

Fig. 10. Velocity plot group for regular artery

The streamlined profiles for normal and stenosed arteries and the velocity graph groups for the blood flow profile relative to the thickness of the artery are shown in Figure 11 and Figure 12. Five Reynolds values, $Re = 1000, 2000, 3000, 4000, and\ 5000$, are used to determine the plot groupings. The figures demonstrate that before the stenosis region, the velocity profile still takes the shape of a Hagen-Poiseuille flow. Blood no longer flows laminarly and disturbances develop whenever it passes through the stenosis geographical region.

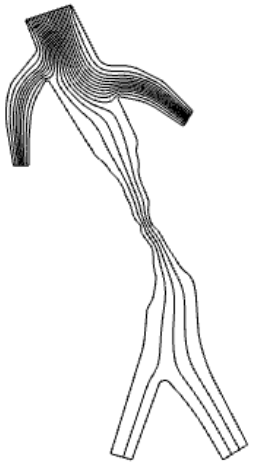

Fig. 11. Streamlines for stenosed artery

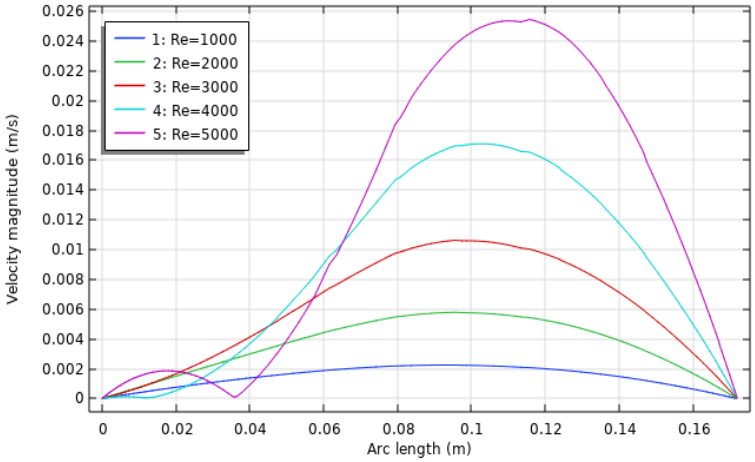

Fig. 12. Velocity plot group for stenosed artery

4.3 Wall Shear Stress

One of the most significant physiologic factors influencing how an atherosclerosis progresses is wall shear stress (WSS). The blood flow's transverse force on the capillary endothelium's interface is referred to as WSS. The figures shown in Figure 13 displays the preset stress component (τ_{ij}) as well as the shear component (σ_{ij}). WSS is subsequently enhanced at the stenosis' neck and noticeably decreased at its distal side. Stenosis and the walls of vessels are continually changing as a result of vascular impedance and fluidity. More deformities compared to an ordinary artery happen in the stenosis section of the arterial as a result of the stenosis's higher stress.

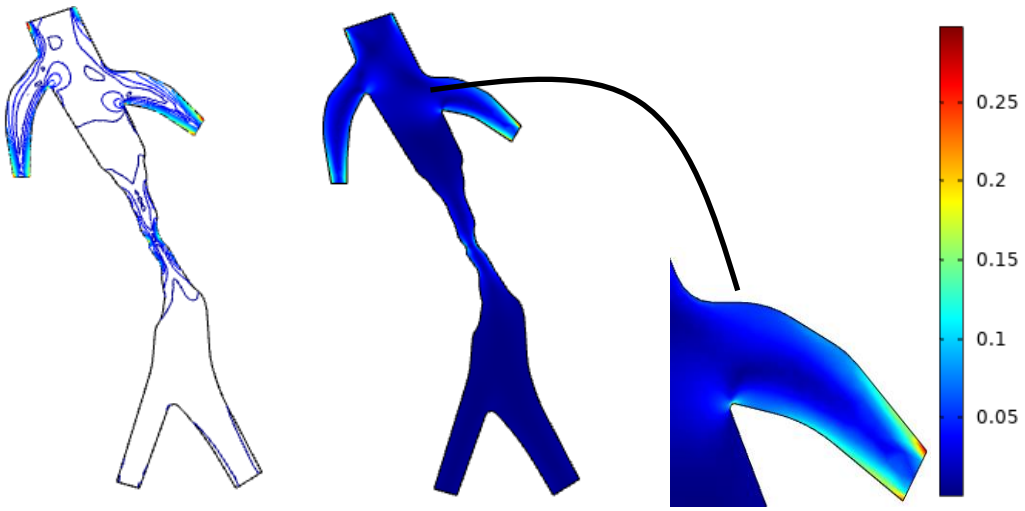

Fig. 13. Wall shear stress for stenosed artery

4.4 Effect of Reynolds Number (Re)

The Reynolds number has a limit beyond which the flow entering the artery produces turbulence. The "essential Reynolds value" is the term used to describe this limit. With various shapes and flow circumstances, the essential Reynolds number's value changes. The essential Reynolds number for interior flow through pipes is typically $Re = 2300$. For laminar in nature compression, and turbulence flows, precise Reynolds numbers are obviously preferred, but in

actuality, it's not constantly necessary. Substrate imperfections, pipeline movement, and circulation disruptions brought on by flow variations all play a role in the change between laminar into turbulence. The next Reynolds number range illustrates the border between the various flow states. $Re = 4000$ represents a turbulent flow. Figure 14 depicts a velocity curve representing the impact of the Reynolds numbers $Re\,(2000, 3000, 4000, 5000)$ on circulation. The graphic illustrates the impact of various Reynolds numbers (0–5000) on circulation. This is where Casson's theory includes an embolus. Model length, blood viscosity, and various entrance velocities are taken into account while calculating different *Re.* In any instance, recirculation zones develop close to the thrombus. As the quantity of Re rises with stenosis, the form of the circulation changes from oval to elliptical. The blood flow rates in the inflow and outflow remained the same when Re = 1000. The velocity divergence area resembles the laminar flow of blood $(Re = 1000)$ at $Re = 2000$, and the recirculation region widens. The circulation regions in the blood flow in a turbulent $(Re = 3000, 4000, 5000)$ took on the appearance of lengthy elliptical forms as the blood flow speed progressively decreased due to thrombosis development brought on by the stenosis. In a turbulent body, the circulation regions are larger, take up more room for internal blood flow, and expand quickly in a restricted throat.

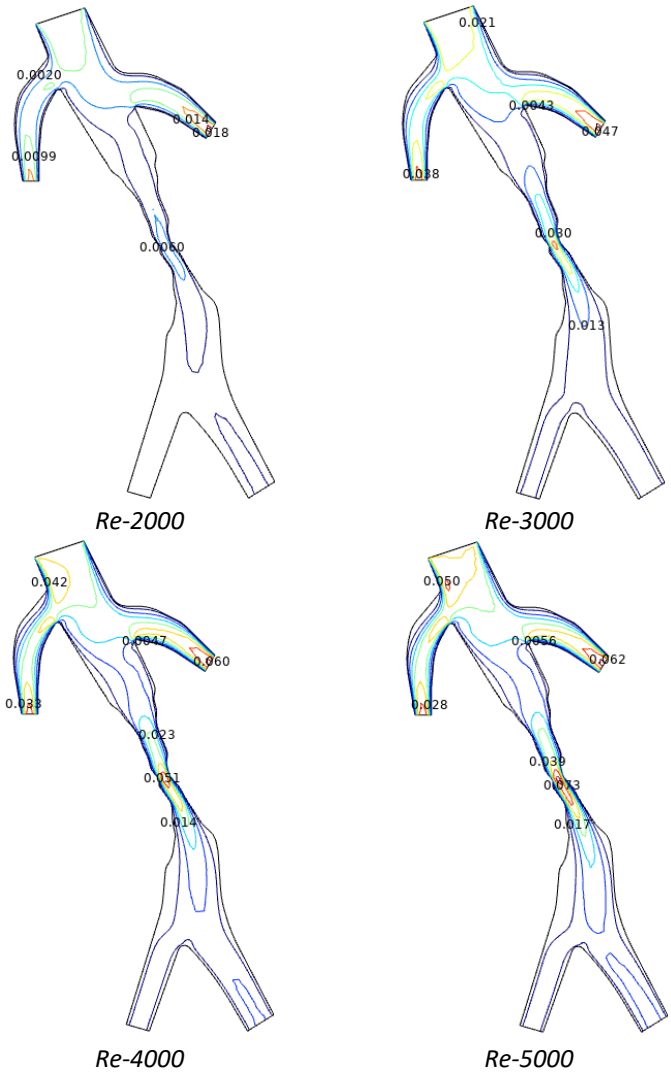

Fig. 14. Velocity contours for different values of Re

However, in laminar blood flow, as opposed to different Re numbers, the blood speed is greater in the restriction region, which causes the creation of mini-circulation regions and a gradual reduction in blood flow, which is seen on the velocity contours. In conjunction with arterial stenosis, a thrombus reduces the blood flow's turbulence, which in all situations results in the circulation's consistency. Stenotic areas are lower relative to the surrounding areas, causing the flow to be quicker than in unconventional areas and its patterns to stay irregular. Since the pressure contours for all Re values are practically identical at the centre of the barrier, this indicates that the blood pressure there is lower.

The loops formed by the pressure contours, shown in Figure 15 of dissolved blood as the Re rise denote the lowest blood pressure level. The blood clot's low-shear zone has the lowest pressure, whereas the stenotic artery's high-shear region has the greatest pressure. In terms of the Reynolds numbers, pressure is nearly uniform.

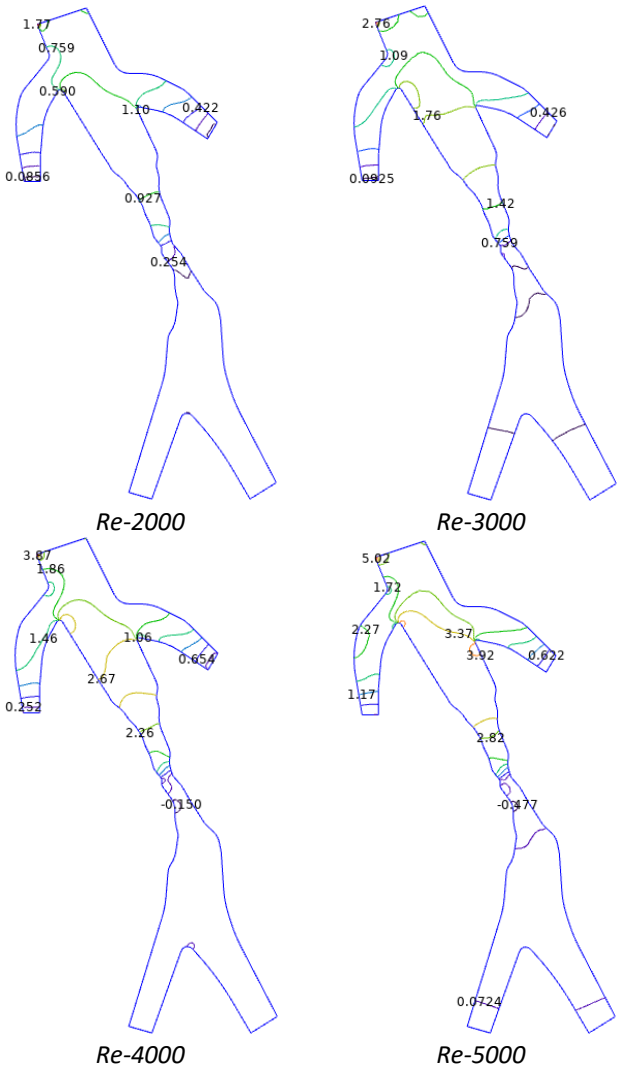

Fig. 15. Effects of Re on pressure distribution (pressure contour) of blood flow

5. Conclusions

Due to considerable developments made lately in the creation of CFD techniques, it has recently become feasible for researchers to swiftly and precisely learn more about atherosclerosis. The following hemodynamic investigations must address several concerns. The application of computer modelling under varied biological blood flow variables, distinct rheological scenarios, and boundary situations was covered in the present review. As computational approaches continue to advance, even more remarkable and difficult hemodynamic studies will be carried out in the next years. Significant data on blood flow may be obtained from these models, both statistically and qualitatively for forthcoming studies on the clinical evaluation and management of atherosclerosis. The research is designed to build and simulate a computer model of blood.

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