A Numerical Analysis of Blood Flow in Clogged Artery

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Abstract The artery wall gets stressed due to the variation in pressure of the blood flow passing through it. When this pressure subsides, these arterial walls dilate in reaction to increasing blood pressure and contract. The additional simulation focused on determining when there is a blockage inside an artery, the velocity and wall shear stress. The current simulation focused on determining the velocity, pressure and wall shear stress when there is a different stages of blockage present inside an artery. This paper has extended the simulation with the blockage developed at the symmetric or asymmetric condition inside the arteries. The analysis of blockage expressed with flow contour, pressure, wall shear stress and velocity. This work explains completed simulations with 10% to 80% blockage at different Reynolds number as 50, 100, 500, 1000, and 4000. More particularly, focused on understanding the functioning and morbidity related to vital organs of the human anatomy with maximum shear stress. The result suggested that how these parameters vary according to the above stated cases and the stage of critical situation. The parameters set in the study at hand aim to resolve the complexity of the procedure by eons and can be utilized in placing a design of set apparatus which will greatly help in increasing the mortality among ailing humans.

Key Words: Arterial walls, blood, Newtonian, Non-Newtonian, blockage, wall shear stress

1. Introduction:
There exists a network of blood vessels in the form of veins and arteries which work in consonance with the heart to deliver and retrieve oxygen, the life-giving element, in addition to various micro-nutrients and macro nutrients synthesized by our body. While one set of blood vessels i.e., the arteries, are in charge of carrying blood away from the heart; the second set of blood vessels i.e., the veins are accountable to perform the opposite. As the path of the veins and arteries progress from the heart towards the extremities, they resemble like the glacial path of a flowing river which divulge into tributaries and streams, as such, the narrowest of such blood vessels are termed to form a “capillary network”. The genesis of the cycle of a blood circulation is said to take place at the fraction between two heartbeats. The cardiac muscle is engulfed by bifurcated aortas and branching blood vessels. As the blood flow creates pressure on the artery’s internal surfaces and its branches, the said muscles change their shape. In knowing the variables that cause cardiovascular diseases and deformation of the aorta, the results of the analysis at hand may be extremely helpful. Furthermore, clinical applications could also utilize the findings to better understand and predict such dictum. As design by the auto-immune, the artery attempts to fight back such ‘foreign’ deposits by galvanizing a seal of fibrous material penultimate to the fatty core (Fig.1).
The blood which flows in human arteries is a non-Newtonian fluid. The computational work is maximized to investigate the three-dimensional FSI problem in its entirety, which is beckoning to how the fluctuation in pressure interacts with the cardio-vascular system. The most challenging aspect of studying said phenomenon is to undertake the changing properties of vessels and blood. The mechanical simulation and relations between the cardiac cycle and time are taken into consideration. It has been concluded that different people have different elasticity in veins. Simulation as a flow of viscous in-compressible fluid through the network of elastic tubes. The non-Newtonian behavior of blood plays an important effect of non-Newtonian theology occurs in the flow of blood through small arteries and in the blood circulation through porous tissue.

In the research paper, Thomas concluded that the blood which flows in human arteries is a non-Newtonian fluid. The most challenging aspect of studying said phenomenon is to undertake the changing properties of vessels and blood [1]. Gamilov and Simakov have expressed the mechanical simulation exhibited and the relation between time and the cardiac cycle [2]. Chow conclusively theorizes the pressure gradient and the pressure downstream of the blood taken continuously with the aid of a 2-D model [3]. Moreover, from the study of an estimation of the wall shear tension for an average adult human was stipulated to be 0.076 to 0.76Pa [4] illustrates a scatter plot in this paper that narrates the relationship between the velocity of blood flow and wall shear stress at maturation. In the research paper, Sochi describes how the non-Newtonian behavior of blood plays an important effect on non-Newtonian theology [5]. From the research paper by Bernsdorf with the assistance of the Lattice Boltzmann process, he was inspired to conduct the simulation in the weakest artery section. He estimated WSS for non-Newtonian fluid and compared an overestimation of the WSS results for any Reynolds number when the effects of blood properties are ignored in the simulation [6]. Aleksandra analyzes how subsequent fracture is produced in patients due to continuous strain, creep deformation due to axial and radial, kink, is scoped [7]. The paper by Biomed studies Artery buckling, the phenomenon through which changes in the flow of lumen blood can result [8]. Lee measured the critical pressures of the veins from the artery buckling processes, with approximate axial stretch ratios [9]. In the paper by Vinoth et al. [10], the effect of yield stress on the velocity value at peak systole has been discussed. The wall shear stress moves significantly due to the non-Newtonian property of blood [10]. Benim and Nahavandi observed the time-dependent, loss coefficients of MRI data were established. By doing the estimates, it was found that the reliance of the loss coefficients on the Reynolds number was very poor and could be ignored without a substantial loss of precision [11]. Selmi and Belmabrou show that during a cardiac period, the velocity field, strain, and displacement distributions were studied. The modeling is useful for CVD investigation and can be an efficient medical stream instrument [12]. Pantya and Harvey, an observation was made of the shape of the arteries, such as straight, twisting, branches, and the main arterial network models, as to the significance of blood velocity transition [13]. In the paper by Velikov [14], a summary of pathological changes during venous disease is drawn. From the paper by Morris, a (VIRTU heart) which is a non-invasive computational method was developed which is said to have the capability of helping cardiologists make more objective and precise treatment decisions to determine pressure drop across an arterial blockage [15]. From the work reported by Zou, according to a 2018 study, almost 30% of all death are caused by cardiovascular diseases. It requires only angiogram images and CFD simulations [16].

Nanotechnology in CAD, Robotics in CAD, Stem Cells. Thrinayan in his paper transcribes that most of the dynamics are studied by the use of three-dimensional image-based modeling of the original model of arteries using magnetic resonance imaging and computational fluid dynamics studies of blood [17]. Hajar
summarizes - A family history, Risks to reduce disability & premature deaths from CVD [18]. In the paper by Malota, demonstrates that there is a drastic change in the coronary hemodynamics indices valves with regards to variable flow conditions and geometry [19]. From the paper by Xunjie “COW (Circle of Willis)” shows that on the contralateral side, the outward hypertrophic remodeling of the COA (communicating artery) can compensate for blood flow [20]. In the paper by Nadeem elucidates the stenosis region, whereby the viscosity values decrease due to increase in shear rate which leads to increased blood flow velocity [21]. From the paper by Ramesha concluded that the differential pressure produced by blood pressure on stent implants should be considered [22]. Keerthana in her paper details the outcome which indicates that PEEK450G was found to be more versatile with more displacements for sudden shifts within the artery [23]. In the paper by Kanzaki the remodeling, streamline of flow, WSS, and OSI were optimally observed. The side-to-side approach for IMA-LAD anastomosis is proposed to have no major drawback over the end-to-side methodology [24]. It was found in the paper by Roy that the rupture plague in stenosis can potentially lead to blood clotting or thrombosis [25]. In the illustrative paper by Ahmed, following factors were examined, such as: Increase the level of cholesterol, Hypertension, Diabetes, Obesity, Stress. From this research paper, it is shown how these above factors are responsible for leading to a cardiovascular disease inside the LAD artery and creating a blockage [26].

![Figure 1](image.png)

**Figure. 1:** Blockage development inside Artery. (a) Free flow of blood (b) On set of blockages (c) observing flow in 80% blockage.

The main focus of the present study is to analyze and optimize the detection and treatment of cardiovascular disease which statistically is the major cause death in developed and developing countries. Stenosed vessels are chargeable for the circulatory network and thus forms the crucial part of making said analysis. Additionally, the reasonable nexus which exists between blood pressure and narrow vessels have also been ancillary part of the research. When taking accountability of the pressure created by blood circulation, one cannot fail to examine the nature of fluid so responsible. Inquiries in studies owing to their behavior as “Newtonian” and “Non-Newtonian” Fluids has been assumed and analyzed. Even though, the topic at hand is a widely researched and examined subject, the aspects so analyzed are one of the fewer so touched area, whereby the researched seek to delve into the unchartered territories and provide a mathematical analysis of the same. By means of a simulation assuming that blood is directed as a non-Newtonian fluid, the researchers intend to analyze the unsteady state of blood flow through a stenotic artery of different severity. The use of three different stenosis straight tubes, 20 %, 40 %, 60 %, and 80 %, numerical simulations were performed for the flow field. In short, this is the approach used to understand the change in flow topology in conjunction with the blockage's magnitude.
2. Simulation Methodology:
Geometry in the study is taken as a rectangular domain which is two-dimensional cut plane of artery wall. Its height is kept at 4.9mm which is denoted by D. Its length is 42mm which is denoted by L. The dimensions were obtained through a literature survey. The flow is developed through the entire geometry. The left side is flow inlet and right side is outlet whereas top and bottom is wall. The blood is non-Newtonian and properties are taken from published paper [2]. The computational geometry was modeled in Ansys 18.1. The standard governing equation was applied for incompressible laminar flow. For non-Newtonian fluid, viscosity is varying with shear stress (Carreau model, Ansys-Fluent).

\[
\frac{\partial \vec{u}_i}{\partial x_i} = 0 \quad (1)
\]

\[
\frac{\partial \vec{u}_i}{\partial t} + \vec{u}_j \frac{\partial \vec{u}_i}{\partial x_j} = -\frac{1}{\rho} \frac{\partial \rho}{\partial x_i} + \nu \frac{\partial^2 \vec{u}_i}{\partial x_j \partial x_j} - \frac{1}{\rho} \frac{\partial \rho}{\partial x_j} \quad (2)
\]

The simulation was done in Ansys workbench 18.1 and shown in Table 1. The high-quality structured mesh was created. While adding blockage, there was skewness observed in mesh which is correctly using refinement. Mesh details are provided

- Sizing of mesh - 1e-4, Size function – curvature, Relevance center – coarse, Span angle center – fine
- Quality: Smoothing – medium, Mesh metric - none, Statistics: Nodes – 19649, Elements – 19138.

**Table. 1:** Grid independent

| Face Sizing Element Size [m] | Mesh Nodes | Mesh Elements | Out-velocity [m s\(^{-1}\)] | Out-pressure [Pa] | max-velocity [m s\(^{-1}\)] |
|-----------------------------|------------|---------------|-----------------------------|-------------------|-----------------------------|
| [m s\(^{-1}\)]             |            |               |                             |                   |                             |
| 0.0006 604                  | 530        | 0.024632052   | 0.37824445                 | 0.036806416       |
| 0.0005 856                  | 771        | 0.024720467   | 0.37938947                 | 0.03744293        |
| 0.0004 1236                 | 1132       | 0.024785159   | 0.38798511                 | 0.037258584       |
| 0.0003 2436                 | 2283       | 0.02484286    | 0.39319145                 | 0.036967982       |
| 0.0002 5304                 | 5075       | 0.024924233   | 0.3858567                  | 0.036679413       |
| 0.0001 19716                | 19275      | 0.024971637   | 0.39184528                 | 0.036228422       |

**Figure. 2:** Computational Domain (D= 4.9mm, L=42mm).
3. Validation of CFD results
This work was taken for comparing the current simulation scheme. It is found that the matching of velocity contour is pretty good (Fig.3). Hence, it can be concluded that the numerical scheme is robust and can capture the flow physics of non-Newtonian fluid flow.

![Figure 3](image)

Figure 3: Velocity contours are compared with the work by (a) Chaw [2] (b) present simulation.

4. Results

4.1 Effect of Reynolds number on flow patterns (no blockage)

![Figure 4](image)

Figure 4: (a) Velocity contour and (b) Pressure contour at Reynolds no 50.
Figure 5: (a) Velocity and (b) Pressure contour at Reynolds no 4000.

Figure 4 and 5 show the contours for pressure and velocity. Figure 4 consists the velocity contour on the left and pressure contour on the right. Both the contours are obtained for Reynolds no 50 with no blockage. Similarly figure 5 consists velocity contour on the left and pressure contour on the right. While these contours were obtained for Reynolds no 4000 with no blockage.

4.2 Analyzing the pressure and velocity distribution for vascular blockage in the arteries: Asymmetric knots

In the simulation, Reynolds number vary from 50 to 4000 and blockage 10% to 80%. Figure 6 and figure 7 shows the velocity contour and pressure contour at Reynolds number is kept constant as 50, the blockage is varied as 10% and 80%. Figure 8 and figure 9 shows velocity contour and pressure contour respectively. These figures indicate the contours while keeping the blockage constant at 80% and varying the Reynolds no 50 and 4000. By comparing side by side, the difference in contours is prominent.

Figure 6: Velocity contour for Reynolds no 50, blockage (a) 10% & (b) 80%.
Figure 7: Pressure contour for Reynolds no 50, blockage (a) 10% & (b) 80%.

Figure 8: Velocity contour for blockage 80%, Reynolds no (a) 50 & (b) 4000.

Figure 9: Pressure contour for blockage 80%, Reynolds no (a) 50 & (b) 4000.
4.3 Analyzing the pressure and velocity distribution for vascular blockage in the arteries: symmetric knots

Figure 10 and figure 11 shows the velocity contour and pressure contour respectively. While the Reynolds no is kept constant as 50, the blockage is varied as 20% and 80%. Figure 12 and figure 13 shows velocity contour and pressure contour respectively. These figures indicate the contours while keeping the blockage constant at 80% and varying the Reynolds no 50 and 4000. By comparing side by side, the difference in contours is prominent.

Figure. 10: Velocity contour for Reynolds no 50, blockage (a) 20% & (b) 80%.

Figure. 11: Pressure contour for Reynolds no 50, blockage (a) 20% & (b) 80%.
Figure 12: Velocity contour for blockage 80%, Reynolds no (a) 50 & (b) 4000.

Figure 13: Pressure contour for blockage 80%, Reynolds no (a) 50 & (b) 4000.
4. 4 Wall Shear Stress: Asymmetric knots

![Graph (a)](image1.png) ![Graph (b)](image2.png)

**Figure. 14:** Wall shear stress vs position of isoplane on last blocking knot for Reynolds no. (a)50 & (b) 4000 - 10% blockage.

Figure 14 represents graph between wall shear stress and position on the isoplane. The Isoplane was created along the last knot (blockage). In this figure the blockage is 10% while the Reynolds no is varied by 50 and 4000 for a better comparison. Similarly, in figure 15 the blockage is considered in this case is 80% and Reynolds number is similar as in the figure 13.

![Graph (a)](image3.png) ![Graph (b)](image4.png)

**Figure. 15:** Wall shear stress vs position of Iso-plane on last blocking knot for Reynolds no. (a) 50 & (b) 4000 - 80% blockage.

4. 5 Wall Shear Stress: symmetric knots

Figure 16 represents graph between wall shear stress and position on the isoplane. The Isoplane was created along the last knot (blockage). In this figure the blockage is 20% while the Reynolds no is varied
by 50 and 4000 for a better comparison. Similarly, in figure 17 the blockage is considered in this case is 80% and Reynolds no is similar as in the above figure.

(a)  
(b)  

**Figure. 16**: Wall shear stress vs position of Iso-plane on last blocking knot for Reynolds no. (a) 50 & (b) 4000 - 20% blockage.

(a)  
(b)  

**Figure. 17**: Wall shear stress vs position of Iso-plane on last blocking knot for Reynolds no. (a) 50 & (b) 4000 - 80% blockage.

4. 6 Discussion

Varied cases of artery blockages are simulated for a deeper understanding of the issue and its effect on the subject. The 3 cases undertaken include:

1. Healthy artery.

2. Artery with blockage (asymmetric) for different blockage percentages.

3. Artery with blockage (symmetric) for different blockage percentages.

These cases are not only indicative of the factors partaking in artery blockages but also the magnitude of their contribution. For reference to a simpler understanding, the case of no artery blockage has also been considered. For a broader coverage of the matter, shown below is compiled information on the different
parameters affecting the simulation namely the velocity of the blood flow in the artery in each case, wall shear stress, and the absolute pressure. These parameters show the disastrous impact of blocked arteries.

4. 7 Results

In the study at hand, a two-dimensional simulation of the blood flow inside the human artery was performed using Ansys workbench. The properties of blood were taken into consideration and the dimension of the human artery was taken from various research papers and properly validated. It is possible for the artery of a human being to have three scenarios in terms of blockage; be it either asymmetric blockage, symmetric blockage or no blockage at all. Therefore, the authors created a two-dimensional simulation showing types of blockage with different Reynolds numbers ranging from 50 to 4000. The hemodynamics of the artery has been explained in term of maximum pressure and shear stress. Consecutively, there was addition of different percentage of asymmetric blockages set at parameters of 20%, 40%, and 80% for each Reynolds number as above. In the second case, symmetric blockages were added with 20% blockage for each Reynolds number as 50, 100, 500, 1000, and 4000 respectively. Thereafter there was an addition of a different percentage of symmetric blockages as 40%, 60%, and 80% for each Reynolds number that was used in the first case. After concluding the results of both asymmetric and symmetric cases, a trend was to be observed that after increasing the blockage percentage the maximum velocity and pressure of blood were also increasing. It was also opined that even after increasing the Reynolds number the maximum velocity and pressure were again in turn increasing. This information will be helpful for the analysis of blockage in artery.

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