A CFD Approach to the Fluid Dynamic Properties of the Human Carotid Bifurcation

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Abstract* — *Understanding haemodynamic mechanisms driving atherosclerosis and other vascular diseases requires precise modelling of the flow dynamics of blood in carotid artery bifurcation. By an emphasis on the carotid artery bifurcation model, this study compares non-Newtonian and Newtonian simulations of blood flow using ANSYS Workbench. For the Newtonian situation, blood was modelled like a constant viscosity incompressible fluid. However, for non-Newtonian case, the behaviour of shear-thinning of actual blood was reflected using Carreau-Yasuda viscosity model. Physiological inlet velocity is pulsatile flow and outlet pressure boundary condition was applied during the fully transient computational fluid dynamics (CFD) simulations. Important haemodynamic variables were examined and contrasted; including velocity profiles, and wall shear stress (WSS). The findings reveal notable variations in two models'; namely WSS distribution and flow patterns, particularly in the areas close to the bifurcation and along the arterial walls. The current findings demonstrate how important it is to use non-Newtonian features in vascular simulations to improve the accuracy of haemodynamic assessments and disease prognostication. This work clarifies the importance of fluid model selection in biomedical CFD studies and supports non-Newtonian models' usage for carotid artery simulations for more physiologically realistic findings.

Keywords: Haemodynamic; non-Newtonian flow; transient; blood flow; ANSYS Fluent.

I. INTRODUCTION

Haemodynamics study fluid dynamics in blood flow and forces that govern circulation. Cardiovascular system is regulated by homeostatic mechanisms that continuously respond to physiological demands. Blood vessels are geometrically complex structures, and blood flow behaviour within them depends on vessel geometry, boundary conditions, and the rheological properties of blood [1]. Blood was considered a Newtonian fluid in the early research, which is true for fluids with shear rates higher than 100 s−1. This only happens when blood flows via big arteries. Small isotropic (symmetric in structure and characteristics) molecules that are not orientated by flow make up Newtonian fluids in most cases [2]. Nevertheless, huge anisotropic molecules can also exhibit Newtonian behaviour. Blood was simulated in a few different experiments as a constant viscosity Newtonian fluid of 0.0345 Pa.s [3-5], blood with constant viscosity = 0.0035 Pa.s [6-9], and blood with different viscosity values [10, 11]. However, because Newtonian blood predictions varied greatly from reality, all of these investigations led to the use of alternative models to mimic blood. There is a lot of study interest in how this affects flow behaviour since blood flow in small arteries frequently displays non-Newtonian properties [12]. Non-Newtonian fluids are more suited for simulating biological fluids like blood because, in contrast to Newtonian fluids, they have a viscosity that varies with the shear rate. Specifically, blood has shear-thinning behavior. When shear rate rises, viscosity falls.
The Casson, Carreau-Yasuda, and Power-law models are some of the most popular; they all seek to get the intricate rheological blood properties in pulsatile and fluctuating flow scenarios. Several non-Newtonian models were examined to identify a good model aimed at replicating viscosity variations for bloodstream. [13–15]. Therefore, comparing non-Newtonian and Newtonian models of fluid is essential for accurately predicting parameters of hemodynamic; velocity profiles and wall shear stress (WSS), particularly in bifurcations, stenoses, or aneurysms. This paper presents a comparative analysis of non-Newtonian and Newtonian models of blood flow, highlighting their impact on flow behaviour in carotid bifurcation geometries.

II. PHYSICAL AND CFD MODEL

An idealised (non-patient-specific) three-dimensional model of human carotid artery bifurcation is made for all simulations. Blood flows from main carotid artery inlet (Figure 1 on left) through the bifurcation to two outlets representing the inner and outer carotid arteries (Figure 1 on right). The main carotid inlet diameter is 6.3 mm. Inner carotid artery (Outlet 1) diameter equals approximately 4.5 mm, and the outer carotid artery (Outlet 2) diameter equals approximately 3.0 mm. The bifurcation angles are

approximately 40° (inner carotid) and 60° (outer carotid) regarding to main carotid axis. No carotid sinus bulge is included in this idealised geometry. These dimensions are consistent with published anatomical data for adult human carotid bifurcations [5]. Blood has a density of 1060 kg/m^3 [1]. As a non-Newtonian fluid, blood's coefficient of viscosity is a function of velocity gradients rather than a constant. Here, blood's viscosity is modelled using Carreau-Yasuda model. The velocity profile at the inlet is a function of time because blood flow is cyclic and pulsatile. Outlet pressure is constant and equals 100 mm Hg. The geometry is an idealised (non-patient-specific) carotid bifurcation with a main carotid inlet diameter of 6.3 mm, outlet diameter of internal carotid of 4.5 mm, and outlet diameter of external carotid of 3.0 mm. The bifurcation angle between the two branches is approximately 40° (internal carotid) and 60° (external carotid) relative to the main carotid axis; no carotid sinus bulge is included in this idealised model. The arterial wall is considered as rigid wall with no-slip condition; fluid-structure interaction (FSI) and wall compliance are not modelled. This is a common simplification for a first-order CFD study, although it is acknowledged that wall compliance and FSI can affect WSS magnitude and flow separation, particularly during cardiac cycle deceleration phase [3].

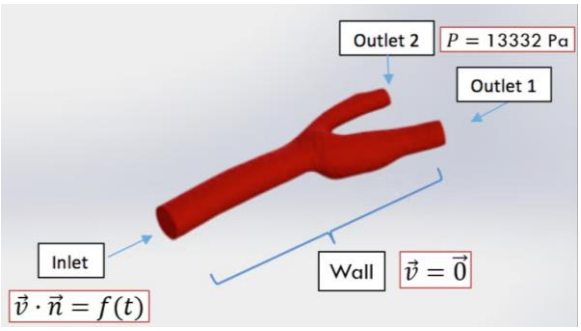

Fig 1. 3D model of a carotid artery bifurcation

III. BOUNDARY CONDITIONS

Figure 1 shows the boundary conditions. Three boundaries are considered; namely wall, inlet, and outlet.

A. Wall:

The arterial wall is the most straightforward boundary condition to define. All we have to do is set this model to "wall" and define its wall areas. The "wall" requirement, as seen from a physical perspective, with a velocity equals to zero. Arterial wall is treated as rigid (no wall compliance), with no fluid-structure interaction (FSI) is modelled. While this is an accepted simplification for a preliminary study, it is important to note that arterial wall compliance can reduce peak WSS and alter flow separation patterns, especially during diastole. Future work should consider FSI modelling to quantify these effects.

B. Inlet:

Human blood flow is pulsatile and cyclical, as is well known which is shown in figure 2. The simulations were performed as fully transient (time-dependent), with a time step of Δt = 0.005 s and three complete cardiac cycles simulated to ensure periodic convergence. Outputs are taken from 3rd cycle. In this instance, a time-varying periodic profile inlet velocity is rather than a constant. Within each period, two phases are combined in pulsatile profile. During the systolic period, nlet velocity fluctuates with sinusoidal pattern (a min. of 0.1 m/s and a max. of 0.5 m/s). A cycle takes 0.5 s, corresponding to a heart frequency of 120 beats/min, Equation (1).

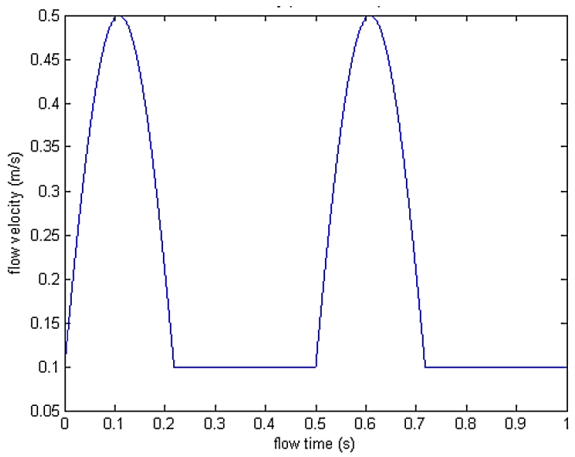

Fig 2. Profile of inlet velocity in two profiles

Profile is described by:

$$v_{inlet}(t) = \begin{cases} 0.5\sin[4\pi(t + 0.0160236)] & : 0.5n < x \le 0.5n + 0.218 \\ 0.1 & 0.5n + 0.218 < n \le 0.5(n+1) \end{cases}$$

n = 0, 1, 2, … (1)

C. Outlets:

A healthy person has about 120 mm Hg as a systolic pressure of and about 80 mm Hg as a diastolic pressure. As a result, outlets pressure are set to 100 mm Hg, or roughly 13,332 Pascal (two phases average pressure).

IV. THERITICAL ANALYSIS

Continuity Equation besides Navier-Stokes Equations are the governing equations.

Continuity Formula:

$$\frac{\partial \rho}{\partial t} + \nabla . (\rho\, v) = 0 \quad (2)$$

However, since blood is an incompressible fluid (density change is zero), Continuity Equation is reduced to:

$$\nabla .\ v = 0 \quad (3)$$

Navier-Stokes Equation:

$$\rho \left(\frac{dv}{dt} + v . \nabla v \right) = -\nabla p + \mu \nabla^2 v + f \quad (4)$$

For Newtonian flow simulation case, it's considered that viscosity is constant = 0.0035 kg/m.s & density = 1060 kg/m^3.

Viscosity coefficient (μ) is a function of shear rate rather than a constant. As the shear rate rises, blood becomes less viscous (shear thinning). Here, Carreau-Yasuda model is considered to simulate blood viscosity. Carreau-Yasuda equation is expressed mathematically as follows:

$$\mu_{eff}(\dot{\gamma}) = \mu_{inf} + (\mu_0 - \mu_{inf})(1 + \lambda\dot{\gamma})^2)^{\frac{n-1}{2}} \quad (5)$$

Where μ_{eff} is the effective viscosity. μ_{inf}, μ_0, λ, and n are material coefficients. The parameter values used here (μ_0 = 0.056 Pa·s, μ_{inf} = 0.0035 Pa.s, λ = 3.313s, n = 0.3568) are taken from Johnston et al. [3] and are commonly appropriate in carotid haemodynamics literature.

V. VALIDATION AND MESH INDEPENDENCE

Simulations were run as fully transient (time-dependent) using a segregated pressure-based solver in ANSYS Fluent. A time step of $\Delta t = 0.005$ s was used (100 time steps per cardiac cycle of T = 0.5 s), with a max. of 20 inner iterations/time step to achieve sub-iteration convergence (residuals below 10^{-4}). Three complete cardiac cycles were simulated; results presented are taken from the third cycle after periodic convergence was confirmed by monitoring peak velocity and WSS at the bifurcation apex between successive cycles (cycle-to-cycle variation < 0.5%). Second-order spatial discretisation was applied for both pressure and momentum equations. Pressure-velocity coupling was handled using the SIMPLE (Semi-Implicit Method for Pressure-Linked Equations) algorithm.

5.1 Validation with exact solution

comparison of exact solution and results from software is made to validate the present software. Both the non-Newtonian and Newtonian models are validated compared to analytical besides published numerical benchmarks to assess the accuracy of the CFD implementation in ANSYS Workbench/Fluent. For the Newtonian case, the fully developed velocity profile for a straight rigid pipe (Hagen-Poiseuille flow) is considered as exact analytical reference. Simulations were conducted at Re equals 100, 300, and 500, consistent with physiological carotid flow conditions (Womersley number Wo ≈ 3.5 for the 0.5 s period and 6.3 mm inlet diameter). The maximum deviation between the simulated centreline velocity and the analytical Hagen-Poiseuille profile was less than 2% across all tested Re. Considering non-Newtonian case, the simulated velocity profile was compared against published analytical solution for Carreau-Yasuda flow in a straight pipe, following the approach of Johnston et al. [3]. Deviations remained below 3% near the wall and below 1% at the centreline. It is acknowledged that these straight-pipe benchmarks do not directly validate bifurcation hemodynamics (recirculation, secondary flows, branch flow splitting).

Carreau-Yasuda model is used in non-Newtonian simulation to represent shear-thinning behaviour of blood. As described above, velocity profiles were compared against the published analytical/numerical solution for Carreau-Yasuda pipe flow [Johnston et al., 3], achieving deviations below 3%. These validation results establish that the CFD solver correctly implements both the Newtonian and Carreau-Yasuda constitutive models for internal flow. Specifically, the quoted figure of "variances < 8%" relative to Doppler/MRI data refers to the pipe-flow benchmark; no direct comparison against patient or phantom carotid bifurcation experimental data has been performed. Caution is therefore warranted when extrapolating these results to bifurcation-specific phenomena such as recirculation zones, flow separation, and secondary flows in the post-bifurcation region.

Used geometry in validation cases for Non-Newtonian and Newtonian models of blood flow is a rigid pipe with length of 0.3m and a diameter of 0.01m.

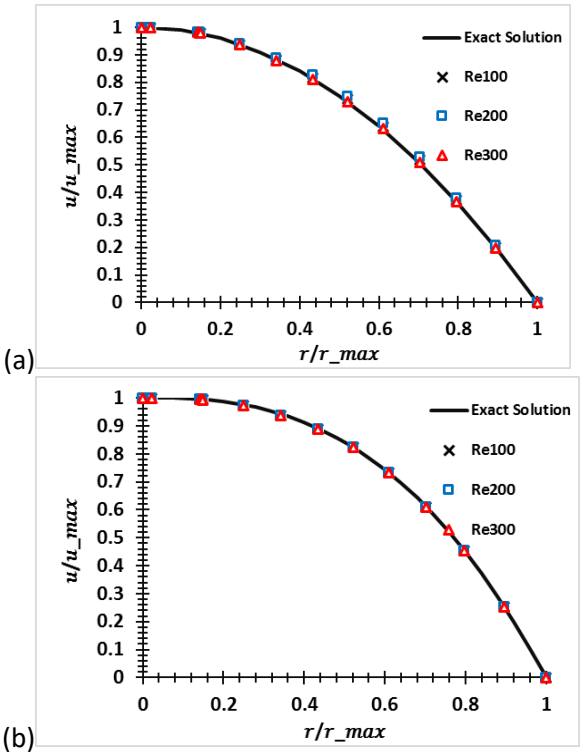

Fig 3. Velocity profiles comparison between CFD results and exact solution for (a) Newtonian and (b) Non-Newtonian flow models for various Reynolds numbers

A mesh independence examination was conducted to ensure results are insensitive to spatial discretisation. Three mesh densities were tested: Coarse (~200k elements), Fine (~500k elements), and Finer (~800k elements). Results for peak WSS, centreline velocity, and pressure drop at the bifurcation apex converged after approximately 500k elements, with changes between the Fine and Finer meshes of less than 1% for velocity and pressure, and less than 2% for WSS locally, as shown in Figure 5. Inflation layers were applied at the arterial wall to resolve the near-wall velocity gradient; five inflation layers were used with a first-layer thickness targeting a dimensionless wall distance of $y^+ < 1$, consistent with the requirements for accurate WSS resolution without wall functions. The near-wall mesh was refined further in the post-bifurcation region, where WSS gradients are highest. WSS grid convergence was verified locally at the bifurcation apex, in addition to global metrics.

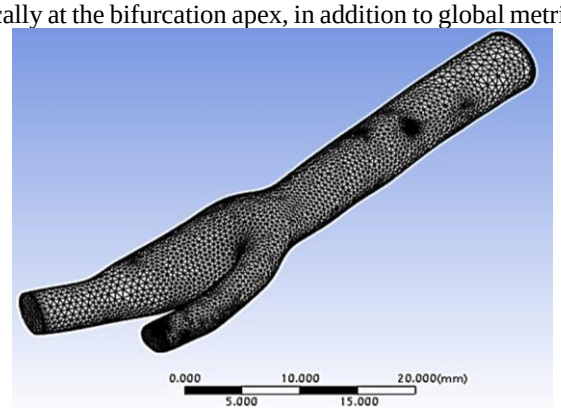

Fig 5. Mesh of the carotid bifurcation artery

For the reference values are set to be Area = 0.0013245 m^2, Density = 1060 kg/m^3 and velocity = 0.1 m/s.

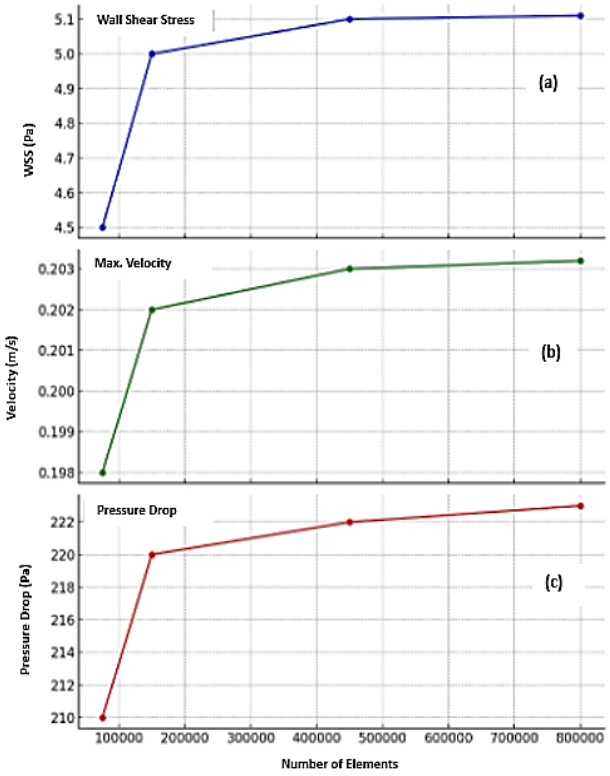

Fig 4. Mesh independency curves; number of elements vs (a) WSS, (b) Velocity and (c) Pressure at 0.2 m/s velocity.

VI. RESULTS AND DISCUSSION

The max. flow velocity within the carotid artery bifurcation of Non-Newtonian and Newtonian blood models differs noticeably, with respect to velocity comparison Figs. 7 and 8. Non-Newtonian model displayed a min. velocity of 0.2161m/s than the Newtonian simulation, which recorded a peak velocity of 0.2947m/s. This discrepancy is in line with the expected haemodynamic behaviour of blood. It demonstrates shear-thinning features that Newtonian assumption is unable to account for. Regardless of the local shear rate, blood is regarded like a constant viscosity fluid in Newtonian model. This causes velocity magnitudes to be overestimated, particularly in areas with strong shear rates, like the area close to the bifurcation or artery walls. Non-Newtonian model, instead, modifies viscosity in accordance to local shear rate. That is achieved using shear-thinning viscosity model, such as Carreau-Yasuda. Lower peak velocities are the result of a more spread and physiologically realistic flow profile caused by a drop in apparent viscosity at high shear rates.

A smoother velocity gradient is also reflected in the Non-Newtonian simulation's decreased peak velocity, which has significant clinical implications. It implies that in order to adequately depict flow separation, recirculation zones, and low shear regions linked to the onset of vascular diseases such as atherosclerosis, a Non-Newtonian model may be necessary.

All things considered, the comparison shows that although the Newtonian model might be easier to compute, it might result in large overestimations of important haemodynamic parameters. Even though it is more complicated, Non-Newtonian model offers an extra precise description of behaviour of blood flow, particularly in areas similar to carotid bifurcation that are geometrically intricate and prone to disease.

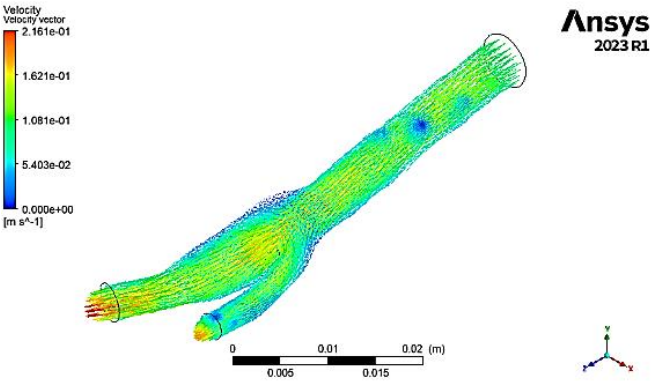

Fig 6. Velocity vectors of Newtonian Simulation at 0.2 m/s velocity.

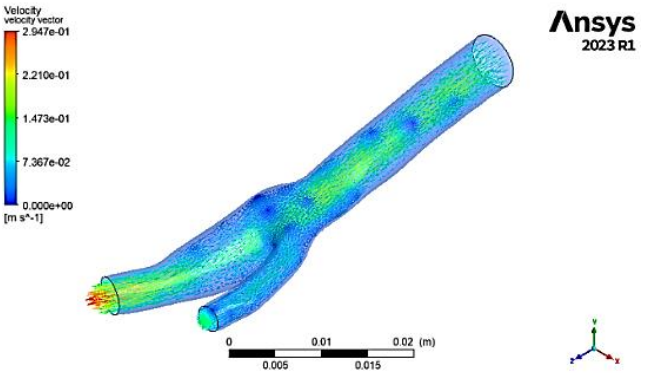

Fig 7. Velocity vectors of Non-Newtonian Simulation at 0.2 m/s velocity.

According to simulation results, Non-Newtonian model's maximum wall shear stress (WSS) (3.8009 Pa) is greater than the Newtonian model's (3.207 Pa) as shown in figures 8 and 9. This finding highlights a significant discrepancy between the two fluid models' representations of shear-dependent viscosity effects in arterial blood flow. Blood is modelled with constant viscosity in the Newtonian simulation, which tends to underestimate WSS, especially in areas with intricate flow dynamics like curvature or bifurcations. This is due to the fact that localised variations in shear rate, which are essential for more precise WSS prediction, cannot be taken into account by constant-viscosity models.

Non-Newtonian model, on the other hand, takes into account the fluctuating viscosity of blood that decreases by increasing shear rate and is generally guided by the Carreau-Yasuda or comparable shear-thinning equations. Higher velocity gradients close to the artery walls result from this behaviour, raising the computed WSS values. The Non-Newtonian case's rise in WSS implies that shear-thinning behaviour exacerbates endothelial surface stress, especially in high-shear regions such as post-bifurcation zone.

Given the important function WSS plays in vascular biology, this differentiation is essential. Plaque formation, endothelial dysfunction, and the progression of disease have all been linked to abnormally high or low WSS.

Therefore, in clinical or biomedical research, underestimating WSS using a Newtonian assumption may result in a misidentification of disease-risk zones.

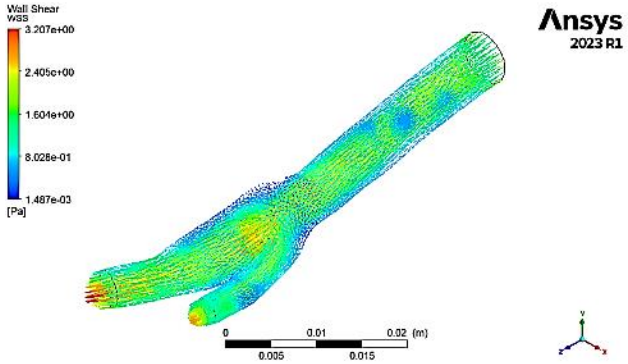

Fig 8. Wall Shear Stress WSS of Newtonian Simulation at 0.2 m/s velocity.

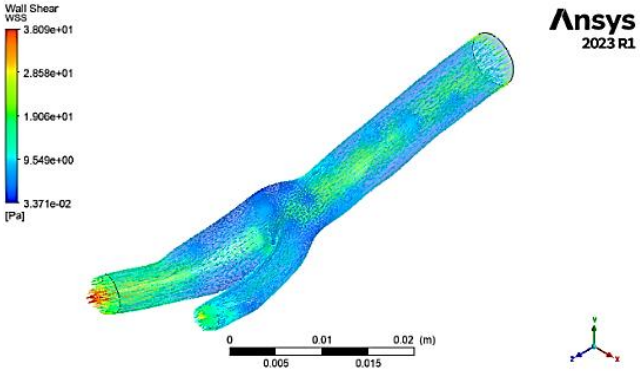

Fig 9. Wall Shear Stress WSS of Non- Newtonian Simulation at 0.2 m/s velocity.

Figure 10 illustrates the max. velocity comparison, confirming that Newtonian model overestimates flow velocity relative to Non-Newtonian model, consistent constant-viscosity assumption failing in accounting shear-thinning belongings near the bifurcation.

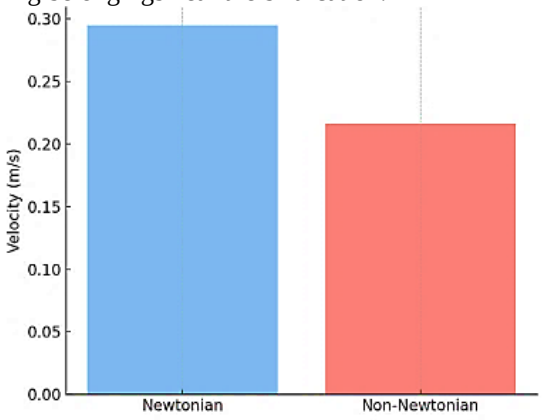

Fig 10. Comparison between Maximum Velocity in the two cases of Non-Newtonian and Newtonian simulation at 0.2 m/s velocity.

For Maximum WSS Comparison as shown in Figure 11 shows that the Non-Newtonian model yields higher peak wall shear stress.

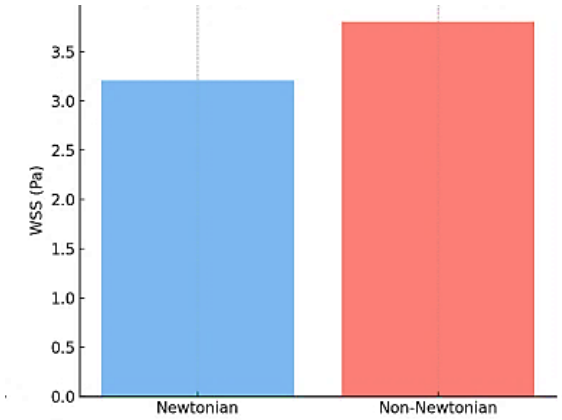

Fig 11. Comparison between Maximum Wall Shear Stress (WSS) in the two cases of Non-Newtonian and Newtonian simulation at 0.2 m/s velocity.

For Wall Shear Stress (WSS) Range as in Figure 12 shows the Non-Newtonian case's higher and wider dispersion of WSS.

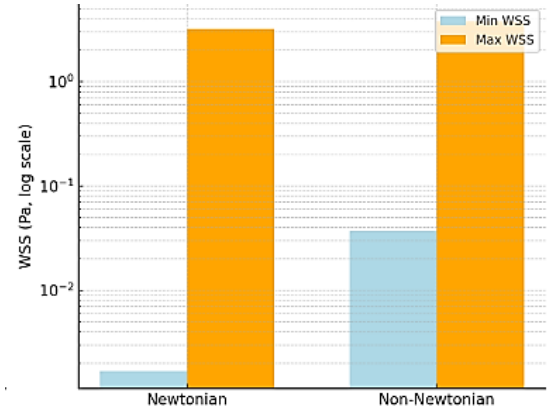

Fig 12. Comparison between WSS Range in the two cases of Non-Newtonian and Newtonian simulation at 0.2 m/s velocity.

In summary, the Non-Newtonian model's greater WSS highlights the significance of employing realistic blood rheological models in computational haemodynamic, particularly in geometrically difficult areas like the carotid artery bifurcation. It guarantees a more precise evaluation of the mechanical forces influencing the development of disease and vascular re-modelling.

VII. CONCLUSION

This study used ANSYS Workbench's Non-Newtonian and Newtonian models to compare the flow of blood inside carotid artery bifurcation. The findings showed notable variations in important haemodynamic factors, particularly velocity magnitude and wall shear stress (WSS).

In contradiction of Non-Newtonian model, Newtonian model, which assumed constant viscosity, underestimated WSS (3.207 Pa) with respect to Non-Newtonian model (3.8009 Pa), however producing greater peak velocities (0.2947 m/s). The Non-Newtonian simulation, on the other hand, which included shear-thinning effects, produced a greater WSS (3.8009 Pa) but a lower maximum velocity (0.2161 m/s), indicating flow behaviour that was more physiologically correct.

These variations demonstrate the drawbacks of applying the Newtonian assumption in intricate artery geometries, where shear-dependent viscosity is crucial for precisely

forecasting blood flow properties. Accurate assessment of vascular disease risk requires careful evaluation of important haemodynamic parameters including velocity gradients and localised shear stress, which are more faithfully captured by the Non-Newtonian model. In conclusion, non-Newtonian modelling is advised for in-depth haemodynamic study, especially in regions susceptible to disease development such as arterial bifurcations, even though Newtonian models may be adequate for simplified or uniformly high-shear regions. The improved accuracy of this modelling approach increases the reliability of CFD simulations in clinical and scientific research. It is also noted that the present study assumes a rigid arterial wall without FSI; future studies incorporating wall compliance and patient-specific geometry would further improve physiological fidelity.

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