Computational analysis of patient-specific pulsatile blood flow: The influence of non-Newtonian models on wall shear stress assessment

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Computational analysis of patient-specific pulsatile blood flow: The influence of non-Newtonian models on wall shear stress assessment

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ABSTRACT
Blood is a sophisticated biological fluid with components like erythrocytes that give it non-Newtonian behavior. Hemodynamic factors such as velocity magnitude, pressure, and wall shear stress descriptors are the most important factors in the development of atherosclerosis. The wall shear stress descriptors are regulated not only by flow geometry but also by blood rheological properties. In the current study, we carried out a numerical analysis of the non-Newtonian pulsatile blood flow while taking into account a patient-specific geometry and transient boundary conditions. Non-Newtonian blood flow is modeled using the four non-Newtonian models: the power-law model, the Carreau model, the Casson model, and the Quemada model, and compared with the Newtonian model. Streamline analysis vividly illustrates velocity patterns, revealing the presence of recirculation zones near sinus regions. The study suggests the significance of selecting appropriate viscosity models for accurate assessments, particularly in regions with low time-average wall shear stress values, such as those associated with atherosclerotic plaques. The differences in the time-averaged wall shear stress between the four non-Newtonian models were found to be the highest in the Quemada model. The study concluded that the non-Newtonian model is required when the focus is on the low-time-averaged wall shear stress area.

I. INTRODUCTION
Concurrently, exploring mechanical and biomechanical processes is required to identify and predict the area affected by atherogenesis in the carotid arterial network. Atherosclerosis is a major cardiovascular disease (CVD) that causes endothelial dysfunction and plaque formation. As a result, it creates a framework for research into the detailed knowledge of flow patterns, particularly in bifurcation, which is of great clinical concern. A stroke happens when the flow of blood to a portion of the brain is interrupted, depriving it of oxygen and quickly...
leading to brain cell death. This type of condition is caused by the development of fat plaques, cholesterol, and other compounds on the walls of arteries. Understanding the pathophysiology of the carotid artery has benefited from the findings of CFD research. Atherosclerosis has been shown to grow or evolve under conditions like flow separation or flow recirculation in regions with low or high shear stress. The place where CCA splits into ICA and ECA, as well as the carotid sinus, is where the areas prone to atherosclerosis at the carotid bifurcation point are located.

Minerals, plasma, and red blood cells makeup blood. The almost Newtonian behavior of plasma is altered to a non-Newtonian behavior by the existence of red blood cells. The chemical composition of blood under different circumstances has been the subject of extensive study in recent years. The findings demonstrated that there is little variation among various non-Newtonian theories, and there are two examples of such metrics. Van Wyck et al. contended that ignoring the non-Newtonian impacts of blood flow can result in incorrect forecasts of mean WSS and OSI hemodynamic factors. Numerous numerical studies show that the blood’s shear-thinning characteristics have minimal impact on how blood flows through the major vessels. While examining Newtonian and shear-thinning fluid models, other research works have found a substantial impact of scalability approaches. According to numerous researchers, low wall shear stress is a vital factor in plaque formation, but additionally, time-averaged wall shear stress and oscillatory shear stress index factors are used as indicators in the prediction of the location of future plaque formations.

Gijsen et al. studied the effect of non-Newtonian characteristics of blood on steady flow in a carotid bifurcation model using the Carreau–Yasuda model. They concluded that minor differences in shear rate have a significant impact on how the shear thinning characteristics affect the velocity distribution in bigger arteries. Using Quemada and power-law models, Razavi et al. investigated the hemodynamic factors, including mean WSS and OSI, in pulsatile blood flow through a stenosed tube. Quemada has greater agreement with experimental findings according to their results comparing non-Newtonian models to Newtonian models. Additionally, maximal WSS and maximum OSI are shown by the power-law and Newtonian models, respectively.

Xiang et al. (2011) concluded that, in a hemodynamic analysis of three internal carotid artery saccular aneurysms, the Casson and Herschel–Bulkley blood viscosity models were tested against the Newtonian model. It was discovered that the Newtonian model can overstate WSS in areas of sluggish recirculation. Jahangiri et al. (2017) used the five non-Newtonian models to analyze plaque development, forecast endothelial cell injury, and determine the size of disease-prone regions with single and consecutive stenosis. Additionally, the Newtonian model was used to research how the blood’s non-Newtonian character affected the factors under consideration. They stated that the modified Casson model and the power-law model, when compared to the other models, generated forecasts for the most extensive endothelial cell damage and the most disease-prone regions, respectively, when examining the mean WSS and OSI. Moradicheghamahi et al. numerically studied the mathematical solution of patient-specific elastic carotid bifurcation with physiological pulses that are chaotic, pulsatile, and non-Newtonian in circulation. Their results suggested that as a result of the low rate of shear stress on the wall, the velocity drops, and significant particle sedimentation happens there; hence, the likelihood of atherosclerosis plaque development in places like the posterior wall of the inner carotid artery sinus is greater.

Mendieta et al. (2019) investigated the overall flow pattern or the mean values of the WSS descriptors using a Newtonian and five non-Newtonian models. The research indicates that the premise of a Newtonian model is plausible for the mean values of the WSS descriptors. However, when the low TAWSS area is the emphasis, the non-Newtonian model is required, particularly in arteries with significant stenosis. Thondapu et al. (2019) conducted patient-specific coronary endothelial shear stress calculations, comparing Newtonian and non-Newtonian rheological models. Their study revealed that the common assumption of Newtonian blood behavior yields different results from
TABLE I. Parameters of the Non-Newtonian models.

<table>
<thead>
<tr>
<th>Non-Newtonian Models</th>
<th>Formula</th>
<th>Parameters</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newtonian</td>
<td>$\mu = k\dot{\gamma}^{n-1} &lt; \mu_{\text{max}}$</td>
<td>$\mu = 0.00345 \text{ Pa s}$, $k = 0.35$, $n = 0.7755$, $\mu_{\text{min}} = 0.00345 \text{ Pa s}$, $\mu_{\text{max}} = 0.25$</td>
</tr>
<tr>
<td>Power-law</td>
<td>$\mu = \mu_{\infty} + (\mu_0 - \mu_{\infty})(1 + (\dot{\gamma})^2)^{(n-1)/2}$</td>
<td>$\mu_0 = 0.056 \text{ Pa s}$, $\mu_{\infty} = 0.0035 \text{ Pa s}$, $i = 3.313$, $n = 0.3568$</td>
</tr>
<tr>
<td>Carreau</td>
<td>$\mu = \mu_{\infty} + (\mu_0 - \mu_{\infty})(1 + (\dot{\gamma})^2)^{(n-1)/2}$</td>
<td>$\mu_0 = 0.0012 \text{ Hct}$, $\mu_{\infty} = 0.7755$, $\mu_{\text{min}} = 0.00345 \text{ Pa s}$, $\mu_{\text{max}} = 0.25$</td>
</tr>
<tr>
<td>Casson</td>
<td>$\mu = (\mu_{\infty} + N_{\infty})^2$, $N_{\infty} = \sqrt{\mu_{p}(1 - \text{Hct})^{2.5}}$, $\mu_{\infty} = \sqrt{0.625 \times \text{Hct}}$</td>
<td>$\mu_{\infty} = 0.00345 \text{ Pa s}$, $K_0 = 4.33$, $K_{\infty} = 2.07$, $\dot{\gamma} = 1.88 \text{ s}^{-1}$, $\phi = 0.45$</td>
</tr>
<tr>
<td>Quemada</td>
<td>$\mu = \mu_p \left[ 1 - \frac{K_0 + K_{\infty} \left( \frac{|\nabla|}{\gamma_c} \right)^{5 \phi}}{1 + \left( \frac{|\nabla|}{\gamma_c} \right)^{5 \phi}} \right]^{-2}$</td>
<td>$\mu_p = 0.0012 \text{ Hct}$, $K_0 = 4.33$, $K_{\infty} = 2.07$, $\dot{\gamma} = 1.88 \text{ s}^{-1}$, $\phi = 0.45$</td>
</tr>
</tbody>
</table>

The more realistic non-Newtonian model. Specifically, the non-Newtonian blood model produced higher quantitative ESS values than the Newtonian model. This suggests that incorporating non-Newtonian blood behavior may enhance the accuracy of ESS measurements. Abhugattas et al. (2020) studied numerically the flow dynamics of bifurcated flows using power-law, Cross, and Carreau–Yasuda models. Their results predicted that the results of the calculations computed using the Cross and Carreau–Yasuda models differed slightly from those obtained with power-law fluids, which suggests lower WSS values for the equivalent conditions. The research by Amir Faraji et al. (2022) explored the influence of four non-Newtonian blood viscosity models: Carreau, Casson, Herschel–Bulkley, power-law, and the Newtonian model on the distribution of wall shear stress, shear rate, and oscillatory shear index within a patient-specific 3D model of a thoracic aortic aneurysm (TAA). Simulation findings indicate that, in general, both non-Newtonian and Newtonian models predict comparable patterns for blood flow and shear rate. Nonetheless, during high flow rates in the cardiac cycle, WSS values deviate, particularly for the power-law model due to its shear-thinning characteristics. Giuseppe De Nisco et al. (2023) conducted 288 angiography-based computational fluid dynamics simulations using 144 right coronary artery models exhibiting varying degrees of stenosis. The study indicates that assumptions regarding blood rheology, whether Newtonian or non-Newtonian, have minimal impact on both wall shear stress and the helical flow profiles associated with the coronary artery disease.

The intricacy of physiological systems and the significance of inherited variables make it demanding, essential, and difficult to simulate these systems. As a result, any model that closely mimics the behavior of the system is useful, even though it does not appear to be feasible to model such a complicated system precisely. Numerical modeling can correctly approximate velocity and wall shear stress fields that are very challenging to measure and provide for the evaluation of the system’s reaction under various circumstances. It is also relatively cheap. Through the use of simulation techniques, it is possible to research atherosclerosis specifically for each patient and create a stent that is unique to them. Additionally, a more thorough understanding and depiction of the movement are crucial for the early detection of atherosclerosis.
II. METHODOLOGY

A. Image acquisition and reconstruction of realistic patient-specific carotid artery model from CT images

The process for creating the most accurate 3D model of the patient-specific carotid artery from CT scans is described here. The computed tomography output files comprise a sequence of 2D images of the patient’s neck. Therefore, this process covers picture pre-processing, basic image-processing operations, and CAD operations for creating surfaces and solids. Materialise MIMICS, a medical image processing software, converts the DICOM libraries into STL files.\textsuperscript{5,7,8,25} It is simpler to manage the geometry when it is concentrated on a specific area of the neck, so the first step in extracting the 3D geometry is to limit the region of interest (ROI) by employing a suitable threshold between $-150$ and $450$ HU and identifying the carotid artery as displayed in Fig. 1.\textsuperscript{23}

Where the artery used to be attached to the bone will be ruptured along its boundary surface during the detachment process. In ANSYS SpaceClaim, a 3D solid model of the carotid artery gets generated using this STL file. The creation of geometry followed established procedures.\textsuperscript{17} All openings must be filled and then the surfaces must be smoothed, which further includes the extraction of volume for the CFD study.

B. Modeling and computation blood flow setup

The momentum and mass balance equations are supposed to regulate blood flow. The flow of blood in the human carotid artery was considered to be laminar and incompressible in all numerical models. As governing equations, the time-dependent Navier–Stokes equations for incompressible flow were utilized. The equation system for the filtered Navier–Stokes equations of motion for non-Newtonian time-dependent incompressible fluid flow may be written as

\[
\rho \frac{\partial u_i}{\partial t} + \rho u_i \frac{\partial u_i}{\partial x_j} = -\frac{\partial p}{\partial x_i} + \frac{\partial}{\partial x_j} \left( \eta \frac{\partial u_i}{\partial x_j} \right),
\]

\[
\frac{\partial u_i}{\partial x_j} = 0, \quad i, j = 1, 2, 3,
\]

where $u_i = \{u, v, w\}$ are velocity components of flow in three dimensions, and $x_i = \{x, y, z\}$ are the three coordinate systems, respectively, for $i = 1, 2, 3$; $t$ denotes time; the volume force vector operating on the fluid is denoted by $f_B$; $\rho = 1060\text{kg/m}^3$ is defined as the fluid density.

FIG. 3. Flow profile used as boundary condition at inlet.

FIG. 4. Anterior and posterior view showing the velocity magnitude and streamline distribution for the (a) Newtonian, (b) power-law, (c) Carreau, (d) Casson, and e Quemada models.
which is considered as constant. Equations (1) and (2) govern the momentum and continuity fluid for an incompressible flow, respectively. $\sigma_{ij}$ is the stress tensor component, which takes the form of the following equation:

$$\sigma_{ij} = -p\delta_{ij} + \mu(\dot{\gamma}) \left( \frac{\partial u_i}{\partial x_j} + \frac{\partial u_j}{\partial x_i} \right),$$

(3)

where $p$ is the pressure, $\delta_{ij}$ denotes the Kronecker delta, which is $(\delta_{ij} = 1$ for $i = j$, otherwise, $\delta_{ij} = 0)$, and the blood viscosity is $\mu$, which depends on the shear rate $\dot{\gamma}$. Thus, the equation system for the filtered Navier–Stokes equations of motion for non-Newtonian time-dependent incompressible fluid flow becomes

$$\rho \frac{\partial u_i}{\partial t} + \rho u_i \frac{\partial u_i}{\partial x_j} + \frac{\partial p}{\partial x_j} - \mu(\nabla^2 u_i) - \left( \frac{\partial \mu}{\partial x_j} \left( \frac{\partial u_i}{\partial x_j} + \frac{\partial u_j}{\partial x_i} \right) \right) = f_B;$$

(4)

$$\frac{\partial u_i}{\partial x_i} = 0, \quad i, j = 1, 2, 3.$$  

(5)

When considering the blood viscosity as a Newtonian fluid, the value of $\mu(\dot{\gamma}) = \mu_0$ approaches a constant value and is free from strain rate. While nonlinear models represent the viscosity of a non-Newtonian model, and they are provided in Sec. II B 1.

1. Rheological non-Newtonian models

A Newtonian fluid has a constant viscosity at a given pressure and temperature because there is a linear connection between shear stress and the rate of deformation.9,29,39 Long-chain molecular fluids are replaced with fluids whose viscosity has a nonlinear relationship with shear stresses due to the collaboration of molecular arrangements during the flow process. This is a brief definition of non-Newtonian behavior from a rheological perspective. Shear thinning occurs in normal human blood vessels. In healthy arteries, the shear rate varies from $1 \text{s}^{-1}$ to $1200 \text{s}^{-1}$ over the cardiac cycle, and blood behaves as either Newtonian or non-Newtonian in different phases of the cardiac cycle.26 If the shear stress acting on the blood is greater than $100 \text{s}^{-1}$, it is assumed to follow the behavior of a Newtonian fluid. Conversely, if
the shear stress is less than $100\text{s}^{-1}$, the blood is believed to exhibit non-Newtonian fluid characteristics. In this research paper, we used four non-Newtonian models to model the fluid behavior. The power-law, Carreau, Casson, and Quemada models, four models that take into account shear thinning effects and have appropriate constants, were found to be consistent with the expected blood viscosity. The table includes a detailed presentation of the formulas and constants. Nonetheless, these models are homogenized and cannot accurately describe the non-uniform viscoelastic behavior of red blood cells.

When comparing non-Newtonian blood models, the power-law model is the easiest to characterize the nonlinear relationship between shear stresses and the rate of deformation in a fluid. The description of this model is expressed as follows:

$$l_{\text{min}} > \mu = k|\dot{\gamma}|^{n-1} < l_{\text{max}}.$$  

This model describes the variables involved in characterizing fluid behavior, specifically for non-Newtonian fluids. The parameters include the flow consistency index, i.e., resistance to flow of the blood, $k$, the shear rate or velocity gradient perpendicular to the plane of shear, $\dot{\gamma}$, and the power-law index $n$, which determines the degree of non-Newtonian behavior. The variables $l_{\text{min}}$ and $l_{\text{max}}$ refer to the minimum and maximum limits of the fluid’s viscosity, respectively.21

The Carreau model shares similarities with the power-law model, as it is a broader form of a Newtonian model and can be represented by the subsequent equation,$^{13,18,20}$

$$\mu = \mu_\infty + (\mu_0 - \mu_\infty)(1 + (\dot{\gamma}^2)\lambda^2)^{(n-1)/2}.$$  

The equation includes five variables: $\mu_0$, which represents the viscosity at zero shear rate in Pa s; $\mu_\infty$, which is the viscosity at an infinite shear rate in Pa s; $\lambda$, which denotes the relaxation time in s; $\dot{\gamma}$, which represents the shear rate in s$^{-1}$; and $n$ is the power index.$^{6,10,13,24,37}$

Casson fluid$^1$ is a non-Newtonian fluid that exhibits shear-thinning behavior, implying that its viscosity decreases as the shear rate increases. Casson fluid is unique in that it is said to have an infinite viscosity at a zero shear rate, meaning that it will not flow unless a certain threshold of shear stress is applied. This threshold is known as the...
yield stress, and below this value, the fluid will not flow. At an infinite shear rate, Casson fluid is said to have zero viscosity, meaning that it will flow easily under high shear conditions,\(^13\)

\[
\mu = \left( \frac{\mu_\infty}{\sqrt{7}} + N_\infty \right)^2,
\]

where

\[
N_\infty = \sqrt{\mu_p (1 - \text{Hct})^{-2.5}}
\]

and

\[
\mu_\infty = \sqrt{0.625 \times \text{Hct}}.
\]

Regarding the blood flow in large arteries, it should be noted that the yield stress is denoted as \(\tau_y\) and is equal to \(\mu_\infty^2\). The consistency index is defined as \(K_c = N_\infty\).

Quemada created a model for calculating the viscosity of concentrated dispersion systems that takes into account the shear rate and hematocrit level of blood. The model determines the shear rate as follows:\(^4,27,32\)

\[
\mu = \mu_1 \left[ 1 - \frac{K_0 + K_\infty \left( \frac{\phi}{\phi_c} \right)^{1/2}}{1 + \left( \frac{\phi}{\phi_c} \right)^{1/2}} \right]^{-2},
\]

This model includes two key parameters: \(\mu_1\) and \(\phi\), which correspond to the viscosity of suspended fluid, i.e., blood plasma and hematocrit level, respectively. Additionally, there are two other parameters: \(k_0\), which represents the maximum volume fraction at zero shear rate, and \(k_{\infty}\), which represents the maximum volume fraction at infinite shear rate. The parameters of these viscosity models are summarized in Table I.

2. Boundary Conditions

The iterative solution process involves solving the governing Eqs. (4) and (5) while also considering the constitutive Eqs. (6)–(11) and (12) as applying the relevant boundary conditions. Since blood flow is characterized by pulses, the velocity at the inlet is defined as a function of time. Additionally, it is assumed that the velocity is constant across the cross-sectional area at any given moment. The velocity waveforms observed by Holdsworth and Bloch\(^19\) were obtained experimentally at the entrance of the CCA, as illustrated in Fig. 2. In order to verify the accuracy of the computational simulations, the Holdsworth waveform at the inlet was utilized in the current analysis. This cyclic function is provided to the model as a user-defined function (UDF) written in the C programming language,

\[
f(t) = a_0 + \sum_{n=1}^{\infty} \left( a_n \cos \frac{n\pi x}{T} + b_n \sin \frac{n\pi x}{T} \right).
\]

The blood flow in the arteries is pulsating, which causes the pressure at the outlet of the ICA and ECA to be a periodic function of time. Due to this pulsatility, the velocity of blood at the inlet is also pulsatile. Although the pressure at the outlet varies with time, the problem becomes convoluted and leads to a solution that does not converge when both the time-varying velocity inlet condition and the time-varying pressure outlet condition are applied together. To overcome this issue, the boundary condition is set to atmospheric pressure, a simpler gauge...
pressure of 0 or 332 Pa with natural outflow, while the wall is assumed to be inflexible, which means that a no-slip boundary condition is enforced at the wall. This modification allows the problem to be solved effectively and helps achieve a convergent solution.

3. Numerical method procedure

To determine the blood flow in a specific section of the circularity system, the area is segmented into numerous small units, referred to as control volumes or cells, ranging from tens of thousands to a few thousand.
The finite volume method calculates the values of variables at the center of each control volume by performing volume integrals and surface integrals. These values are also referred to as node values. To calculate node values, the method integrates the fluxes through each face of the control volume. Therefore, the finite volume method involves the computation of node values using both volume and surface integrals. In this paper, the simulations for the governing equations are formulated using a commercial ANSYS Fluent finite volume program. To discretize the equations in space, the QUICK approach is utilized, and the velocity–pressure interaction is modeled.

![Graphs showing pressure plots and pressure gradients for different blood viscosity models.](image)

**FIG. 9.** Pressure plot at the (a) inner wall and (b) outer wall of ICA for the different blood viscosity models.

<table>
<thead>
<tr>
<th>Model</th>
<th>Power-law (%)</th>
<th>Carreau (%)</th>
<th>Casson (%)</th>
<th>Quemada (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carotid Artery</td>
<td>1</td>
<td>3</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td>ICA</td>
<td>1</td>
<td>2</td>
<td>8</td>
<td>10</td>
</tr>
<tr>
<td>ECA</td>
<td>2</td>
<td>4</td>
<td>10</td>
<td>11</td>
</tr>
</tbody>
</table>

**FIG. 10.** (a) Pressure gradient for the carotid artery, (b) for ICA, (c) for ECA, and (d) percentage differences in mean pressure gradient between the non-Newtonian models and the Newtonian model.
using the SIMPLEC algorithm, which is a modified version of the original SIMPLE algorithm proposed by Van Doormal and Raithby. The variables are computed at the cell center, and input is provided by the four neighboring cells. An algebraic approximation of the integral balance for the discrete value of \( \phi \) is used to represent the equation for the \( P \) depending on neighboring cells,

\[
a_P \Phi_P \equiv \sum_{nb} a_{nb} \phi_{nb} + F_{\phi}.
\]  

The subscript \( P \) denotes the cell center, and the superscript \( nb \) represents the neighboring cells, and the coefficients denoted by \( \alpha' \) are the numerical values that correspond to the various terms in the conservation equation. A source term, \( F_{\phi} \), can include the pressure gradient, boundary conditions, or any other impact, in addition to convection and diffusion. The solution is obtained through a process involving various steps:

1. To obtain accurate pressure and velocity, corrections are added to the current pressure and velocity,

\[
u^{n+1} = u^* + u'; p^{n+1} = p^* + p',
\]  

where \( u^* \) denotes the guessed velocity field and \( u' \) denotes the velocity correctly.

2. To establish a connection between the velocity and pressure corrections, the momentum equations and the correction relation were utilized,

\[
u + D(\Delta p').
\]  

3. The continuity equation for the control volume is discretized as

\[
(pAu)_{W} - (pAu)_{E} + (pAv)_{S} - (pAv)_{N} = 0,
\]  

where \( A \) represents the cell face area in the equation.

4. When the velocities \( u \) and \( v \) are substituted in Eq. (16), the result is

\[
a_P p^{n+1} = a_V p^{n+1}_{E} + a_W p^{n+1}_{W} + a_S p^{n+1}_{S} + a_N p^{n+1}_{N} + F,
\]  

where \( S = (pAu^*)_{W} - (pAu^*)_{E} + (pAv^*)_{S} - (pAv^*)_{N} = 0.\)

**TABLE II. Maximum–Minimum WSS values (Pa).**

<table>
<thead>
<tr>
<th></th>
<th>Maximum WSS (Pa)</th>
<th>Minimum WSS (Pa)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Newtonian</td>
<td>Power-law</td>
</tr>
<tr>
<td>Carotid artery</td>
<td>31.3646</td>
<td>31.3496</td>
</tr>
<tr>
<td>ICA</td>
<td>12.6434</td>
<td>13.3931</td>
</tr>
<tr>
<td>ECA</td>
<td>26.2308</td>
<td>27.439</td>
</tr>
<tr>
<td>Carotid artery</td>
<td>0.00428964</td>
<td>0.0131225</td>
</tr>
<tr>
<td>ICA</td>
<td>0.212918</td>
<td>0.201552</td>
</tr>
<tr>
<td>ECA</td>
<td>0.0731309</td>
<td>0.0246451</td>
</tr>
</tbody>
</table>

Obtaining the velocity correction is possible when momentum calculations are performed in their corrected form. A time step of 0.01 was used, and the solution convergence criteria for all variables were set to \( 10^{-6} \). The inlet boundary condition discussed in Sec. II B 2 is initially...
written in the C programming language and then interpreted as a user-defined function within the Fluent software. It involves defining how fluid flows into a simulation using a specific programming language and then incorporating this definition into a software program.

4. WSS-based hemodynamic descriptors (post-processing)

The wall shear stress (WSS), which is the force exerted by blood on the artery walls, is a critical parameter to monitor as extremes in its magnitude or rapid changes can increase the risk of cardiovascular diseases. To determine the time-averaged wall shear stress (TAWSS), the WSS at each instant was computed and then averaged over all elements within the region of interest (ROI) across the cardiac cycle, either spatially or temporally. The TAWSS magnitude is obtained as follows:

$$\text{TAWSS} = \frac{1}{T} \int_0^T \left| \text{WSS} \right| dt,$$

where WSS represents the instantaneous shear stress vector and T is the pulse period.

The oscillatory shear index (OSI) is a significant factor that indicates the changes in WSS over time and shows the difference between AWSS and AWSSV. It measures the level of fluctuation in the shear stress during a pulsating cycle. In other words, OSI represents the extent to which the shear stress varies back and forth during each cycle of pulsation. The formula used to calculate OSI is as follows:

$$\text{OSI} = 0.5 \times \left( 1 - \frac{\int_0^T \left| \text{WSS} \right| dt}{\int_0^T \left[ \text{WSS} \right] dt} \right).$$

WSS would fluctuate periodically due to the repetitive boundary conditions. This oscillation in WSS could have a significant impact on the dynamics of the system under consideration. OSI varies from zero (for no changes in the direction of the WSS vector with respect to the dominant direction of the flow) to 0.5 (for a 180° variation in the direction of the WSS vector with respect to the dominant direction of the flow). Even though the mechanism of formation of plaques depends on many different biological parameters, it was shown that the oscillatory shear stress is an appropriate measure when dealing with the oscillatory flow of the blood.

To address the effects of initialization in the low, mid, and high WSS metric regions, calculations for TAWSS and OSI are performed.

C. Results

1. Flow Distribution

The aim of this activity is to establish and confirm all the parameters required for conducting the simulation. Flow rates are determined through simulations at the outlet, while wall shear stresses are calculated at the inner and outer walls of the ICA. For the provided flow rate in the common carotid artery, Fig. 3 illustrates the volume of flow rate through the internal carotid artery and external carotid artery. The flow rate in the ICA is noted to be roughly two-thirds of the flow in the CCA. This disparity is due to the fact that, in a healthy carotid artery, the ICA possesses a larger diameter compared to the ECA. These recorded outlet flow rates also align with measurements taken in vivo by Bharadwaj et al. [3]

2. Velocity

Streamline plots serve as visual representations of fluid flow by tracing curves that align with the instantaneous vector field. They complement fluid flow analysis, providing insights into the motion of fluids. The streamline plots in Fig. 4 vividly depict the velocity magnitude patterns for different viscosity models. The streamline plots revealed the presence of atherosclerotic plaques within the ICA as a re-circulation zone that occurs at the stenosis regions, showing the average flow patterns using both Newtonian and various non-Newtonian models. In the non-Newtonian cases, there is an observed expansion of the re-circulation zone into the post-stenotic area. The orientation of the streamlines provides evidence of fluid recirculation within this region. Importantly, the velocity values observed align with existing literature in this field, affirming the accuracy of the simulation results.

This situation raises concerns from a medical perspective, as it indicates prolonged and stagnant blood flow in the post-stenosis region. This stasis has the potential to trigger the formation of plaques.
clots or thrombosis, ultimately increasing the risk of stroke and heart attacks. Furthermore, we examined the magnitude of velocity at six distinct positions along the carotid artery: the middle of the CCA, the bifurcation point, three specific locations on both the ECA and internal carotid artery (ICA), and the distal region of the ICA and ECA. Within the CCA, the fluid occupies the entire lumen, as shown in Fig. 5(a), characterized by velocity values that are maximum at the center of the geometry and diminish to zero at the walls. However, as the flow reaches the bifurcation, a significant distortion occurs, diverting much of the flow toward the inner walls of the outlet arteries as shown in Fig. 5(b). This phenomenon results in lower velocity values within the outer zones. Across all instances, the Quemada non-Newtonian model consistently yielded the highest peak velocity magnitude; however, velocity magnitudes demonstrate minimal distinctions across the Newtonian, power-law, Carreau, and Casson models, Figs. 5(c)–5(e), which align with Amir Faraji et al.’s investigation on blood flow in Newtonian and non-Newtonian models. In the first line of our findings, the highest recorded value for velocity magnitude was 0.38 m/s using the Quemada model. The vector velocity distribution and axial velocity distribution at various planes of the artery are depicted in Fig. 6 and 7, respectively.

3. Pressure gradient

Figure 8 displays contour plots illustrating the pressure across the geometry. The outcomes were derived from Newtonian models to prevent repetition, given that the highest pressure values and their positions were nearly identical for all presumed viscosity models. The apex exhibited the highest pressure, and then the stenotic region of the ICA experienced the highest pressure. Corresponding to the TAWSS pattern, pressure levels escalated by about tenfold. Moreover, we compare the mean pressure at different positions: the inner wall and the outer wall. In terms of viscosity models, the Quemada model generated higher pressure values, as demonstrated in Fig. 9 both at the inner and outer walls of the artery. The sites of maximum pressure corresponded with locations displaying peak TAWSS in all viscosity models. The anticipated blood pressure from all viscosity models display a sudden decline at the center of the stenosis on the ICA and ECA, which could potentially contribute to hypotension in a patient with artery stenosis. Although differences in mean pressure gradient between the Newtonian and non-Newtonian models are clearly discernible, variations among the power-law and Carreau are quite minor, Figs. 10(a)–10(c). The most notable percentage differences in maximum pressure were 9% for the Quemada model, 8% for the Casson model, 3% for the Carreau model, and a minimum of 1% for power-law for the carotid artery depicted in Table II.

4. WSS

The magnitude of the wall shear stress was analyzed for each constitutive of the viscosity models, providing a comprehensive relation of the rheology of blood impact relative to the healthy carotid artery, as shown in Fig. 11. Regions characterized by low WSS consistently coincide with locations where blood flow separates from the vessel walls. Specifically, these areas include the outer walls of the carotid sinus in the ICA and the outer walls of the initial segment of the ECA. Conversely, regions exhibiting high wall shear stress are situated at the carotid apex as well as the inner walls of both the ICA and ECA segments. The prediction process remains generally consistent when employing the power-law, Carreau, Casson, and Quemada models.

However, variations in the resulting WSS values are attributed to the distinct models utilized. Differences between the non-Newtonian and Newtonian models in shear distribution are also evident in the turbulent region after the stenosis.

FIG. 13. Anterior and posterior view showing TAWSS for the (a) Newtonian, (b) power-law (c) Carreau, (d) Casson, and (e) Quemada models.
Table II provides a quantitative analysis of the dissimilarities among the constitutive models by revealing the minimum and maximum WSS values obtained for each flow condition and model. It is significant to highlight that the maximum value is located at the carotid apex, while the minimum value is identified along the outer walls of the carotid sinus in each constitutive velocity model. Carreau and power-law models yielded prominent maximum and minimum WSS values with the Newtonian model. Whereas, the Casson model has a significantly higher maximum and minimum WSS value. The Quemada model produced the highest WSS values, both in maximum and minimum WSS.

In Fig. 12, a comparison of the distributions of WSS at different locations, the outer wall and the inner wall, for various viscosity models is shown. Examining the outer wall, it is notable that the stress drop is higher in the non-Newtonian model compared to the Newtonian model. Specifically, the power-law model forecasts the greatest stress drop at the leading edge of stenosis. In terms of magnitude, the Quemada model exhibits a WSS drop of approximately 0.274256, which is approximately 49% lower than the drop in the Newtonian model, i.e., 0.541411. However, on the inner wall, the WSS is higher in the Quemada model than in the Newtonian model.

5. WSS hemodynamics descriptors

The total maximum TAWSS values exhibited a comparable pattern to the WSS values across the models. Figure 13 depicts the profiles of TAWSS, which represent the average shear stress experienced by the arterial walls throughout a complete cardiac cycle. It has been confirmed that the highest TAWSS values are observed at the carotid apex. This can be attributed to the significant deceleration of fluid flow in this specific area. When comparing the TAWSS values obtained from the five models (excluding the carotid bulb), the order of higher TAWSS values is as follows: ECA, ICA, and CCA. These findings align with the velocity profiles shown in Fig. 4, where the outlet arteries exhibited higher velocity values.

This observed trend can be explained by the fact that the cross section area of the ECA is relatively smaller compared to the ICA, leading to greater velocity gradients in the former. In contrast, the carotid sinus consistently displays the lowest TAWSS values across these viscosity models. Interestingly, unlike the velocity profiles, the TAWSS profiles generally exhibit higher values when utilizing the Quemada viscosity model. Moreover, Figs. 14(a)–14(c) illustrate a comparison of spatially averaged TAWSS for five different viscosity models, both in the complete carotid artery and a specific region of interest in ICA and ECA. Across the complete carotid artery (ICA and the ECA), minor differences in TAWSS among the Newtonian, Carreau, Cross, and power-law models have been found. However, notably higher TAWSS values were observed in the Quemada model.

In the case of the complete carotid artery, the maximum TAWSS obtained was 51.2571 (Pa) for the Quemada model. The percentage
variations in TAWSS for the non-Newtonian models, in comparison to the Newtonian model used as a reference, are summarized in Table II for the complete carotid artery, ICA, and ECA. Among these non-Newtonian models, the Quemada model exhibited the greatest percentage deviations, followed by the Casson model, which displayed a maximum variation of 25%. The Carreau and power-law models yielded the lowest percentage differences, with a maximum variation of 8% and 3%, respectively. In ICA, the maximum TAWSS measured about 20.6287 (Pa) for the Quemada model. When comparing the average TAWSS values between the complete carotid artery and the ICA, there was an approximately threefold increase observed. Conversely, the TAWSS values were diminished in the ICA compared to those found in the complete geometries. Figure 15 displays the OSI contours for five cases, within the range of 0 to 0.5. A low OSI value indicates minimal changes in WSS vectors aligned with the primary flow direction, while a high OSI signifies deviations of WSS vectors in multiple directions during the cardiac cycle. The locations with maximum OSI were identified at the inner wall of the ICA, the bifurcation point, and the distal regions of both the ICA and the ECA. Distinct viscosity models captured similar disturbance locations across various geometries. Notably, the Quemada model exhibited more pronounced and extensive oscillation regions. Intriguingly, comparing the maximum OSI values for all geometries and viscosity models revealed a modest difference of only 10.5% between the Newtonian and non-Newtonian models. Despite this relatively small difference, the Newtonian and power-law models demonstrated the least oscillation. Similarly, for the average OSI value, the maximum percentage difference was also approximately 10%.

D. Conclusion

In the present study, we conducted a comprehensive investigation into the flow distribution, velocity profiles, pressure, wall shear stress, and WSS descriptor characteristics within the carotid artery under various viscosity models. Our findings shed light on the intricate interplay between fluid dynamics and arterial geometry, contributing to a deeper understanding of the potential implications for vascular health. Four distinct viscosity models—power-law, Carreau, Casson, and Quemada—were used to anticipate the outcome of the non-Newtonian behavior exhibited by blood. Conventional Newtonian models were utilized as reference points. These models incorporate diverse assumptions pertaining to intrinsic viscosity properties. Through simulations, we established that the ICA consistently exhibited flow rates approximately two-thirds of those in the CCA owing to the natural variation in vessel diameters. The velocity distributions, portrayed through streamline plots, further elucidated the flow patterns across different viscosity models, revealing the formation of recirculation zones near sinus regions. We extensively analyzed the WSS distributions along the arterial walls. Low-WSS regions coincided with zones of flow separation, predominantly observed at the outer walls of the carotid sinus and the initial segment of the ECA. Conversely, regions of high WSS were identified at the carotid apex and inner walls of both ICA and ECA. The Quemada viscosity model consistently exhibited higher WSS values, particularly in regions with flow separation, indicating a potential influence of blood rheology on the hemodynamic environment. Moreover, the WSS magnitude analysis on the inner and outer walls of ICA suggested that the excessive acute shear stress of around 26 – 33 Pa has the potential to cause damage to the endothelial layer on the inner surface of the blood vessel downstream of the stenosis. The research also suggested that the highest TAWSS values were concentrated at the carotid apex, suggesting areas of decelerated flow. Furthermore, the TAWSS values were elevated in
the ECA, ICA, and CCA in the Quemada viscosity model. For regions with low TAWSS values, particularly in the presence of atherosclerotic plaques, the differences between viscosity models become more pronounced. In such cases, the use of a non-Newtonian model is essential for accurately capturing the complex flow behavior. Notably, the OSI values depicted regions of flow disturbances, with the Quemada model revealing more pronounced oscillations, emphasizing the impact of viscosity models on hemodynamic patterns. Specifically, our research suggests that in the context of the present geometric and boundary condition uncertainties, the assumption of a Newtonian model is reasonable for assessing OSI and pressure gradient. This implies that the Newtonian model can effectively capture flow disturbances and changes in pressure gradients within the carotid artery despite the complex non-Newtonian behavior of blood. Moreover, this study contributes to refining the computational modeling approaches to enhance the accuracy of their simulations, leading to better-informed decisions regarding cardiovascular risk assessment and potential interventions.

Furthermore, the present research is limited to the CFD analysis of the effect of non-Newtonian blood rheology on carotid artery hemodynamics with a rigid arterial wall. However, the effects of wall elasticity are an inevitable factor influencing non-Newtonian blood rheology on hemodynamics. Thus, the research can be expanded to include the effect of uniform magnetic fields on pulsatile non-Newtonian blood flow within an elastic artery. Moreover, for future research, validating numerical results by comparing them with clinical data will be crucial to enhance the robustness of the findings.

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AUTHOR DECLARATIONS

Conflict of Interest

The authors have no conflicts to disclose.

Author Contributions

Damini Singh: Conceptualization (lead); Data curation (lead); Formal analysis (lead); Investigation (lead); Methodology (equal); Software (lead); Validation (equal); Writing – original draft (equal). Sarita Singh: Conceptualization (equal); Methodology (equal); Validation (equal); Writing – review & editing (equal).

DATA AVAILABILITY

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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