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# **Modeling Arterial Blood Flow under Stenosis: A Comparative Study of Newtonian and Carreau-Yasuda Non-Newtonian Approaches**

Erwin<sup>1[\\*](https://orcid.org/0000-0002-2316-4521)</sup><sup>1</sup>, Jonathan Liviera Marpaung<sup>2</sup>

*1\*,1 Mathematics Department, Universitas Sumatera Utara, Medan, 20155, Indonesia* \*Corresponding Author: erwin.mipa@usu.ac.id

### **ARTICLE INFO ABSTRACT**

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This study investigates the simulation of blood flow within arteries experiencing stenosis using both Newtonian and non-Newtonian models. The Newtonian model employs standard fluid dynamics assumptions, while the non-Newtonian Carreau-Yasuda model accounts for the unique viscoelastic properties of blood, providing a more accurate representation of its flow behavior under various shear rates. By utilizing COMSOL Multiphysics, the simulations are conducted with parameters including blood velocity, viscosity, and pressure in normal and narrowed artery conditions. Results reveal that while the Newtonian model predicts general flow patterns, it lacks the precision needed to reflect the complexities of blood behavior in stenosed regions. Conversely, the Carreau-Yasuda model demonstrates enhanced accuracy by capturing viscosity variations and pressure differentials, particularly in the narrowed artery sections, showing the significance of non-Newtonian characteristics in modeling blood flow. These findings underscore the potential of non-Newtonian models in improving diagnostic and therapeutic approaches for vascular conditions.

**Keywords:** arterial blood flow, stenosis, Newtonian model, non-Newtonian model, Carreau-Yasuda model



#### **1. Introduction**

Arterial blood flow is a fundamental aspect of cardiovascular physiology, influencing oxygen and nutrient delivery throughout the body. The study of blood flow in arteries, particularly under pathological conditions such as stenosis, is crucial for understanding cardiovascular health and disease. Stenosis, a narrowing of blood vessels caused by plaque build-up or other obstructions, disrupts normal blood flow and can lead to severe complications, including ischemia and thrombosis. Accurately modelling blood flow within stenosed arteries is essential for predicting how these conditions progress and for developing therapeutic strategies to mitigate their impact. Arterial blood flow dynamics are driven by complex interactions between the heart, blood vessels, and the blood itself, which is a non-Newtonian fluid with properties that change under different flow conditions. Blood is primarily composed of plasma, red blood cells, white blood cells, and platelets, which interact to give it unique rheological characteristics. The viscoelastic nature of blood means that it does not behave like a simple fluid; rather, its viscosity changes in response to shear rates. This non-Newtonian behaviour is particularly significant in small vessels or regions with altered flow, such as stenosed arteries, where shear rates can vary widely. Stenosis imposes physical changes on blood flow dynamics. In healthy arteries, blood flow is typically laminar, with a parabolic velocity profile and minimal resistance. However, in stenosed arteries, the narrowing creates regions of high shear stress and turbulence downstream of the stenosis. These alterations in flow patterns can lead to increased resistance, reduced perfusion downstream, and even reverse flow in certain cases. The body's response to such conditions often exacerbates the problem; for example, platelets are more likely to adhere to the arterial wall under high shear conditions, leading to clot formation and potential blockage.

Given these complexities, accurately modelling blood flow in arteries is critical for several reasons. First, it helps predict the progression of conditions such as atherosclerosis, where plaque buildup gradually narrows arteries, increasing the risk of a critical blockage[1]–[3]. Second, models allow researchers to simulate the hemodynamic effects of interventions, such as stent placement or angioplasty, providing insights into how these treatments might restore normal blood flow. Third, understanding the stress patterns and turbulence induced by stenosis can inform the design of biomedical devices and drugs that better interact with blood flow in compromised vessels. The primary challenge in modelling arterial blood flow is accurately representing the non-Newtonian properties of blood[4], [5]. Traditional Newtonian models assume constant viscosity, which simplifies calculations but fails to capture the complexities of blood's behaviour. In regions of high shear, such as stenosed arteries, blood viscosity decreases, a phenomenon known as shear-thinning. This characteristic is significant in modelling because it influences how blood moves through constricted areas and affects the stresses on vessel walls[6]–[9]. Another key challenge is replicating the pulsatile nature of blood flow. Blood flow in arteries is not steady; it pulsates in sync with the cardiac cycle, creating varying pressure and flow rates. This pulsatility is particularly relevant in the vicinity of stenoses, where the alternating flow patterns can amplify the stresses on vessel walls and potentially accelerate the degradation of compromised tissue. Consequently, an accurate model must account for these timedependent variations to provide realistic predictions[10]–[13].

Over the past decade, advancements in blood flow modelling have significantly improved our understanding of hemodynamic in stenosed and diseased arteries. Traditional Newtonian models, which assume constant viscosity, often fall short in capturing the true dynamics of blood flow, especially under complex conditions such as stenosis and aneurysms. Studies by Nadau and Sequeira (2007) and Jędrzejczak et al. (2023) emphasize the need for non-Newtonian models to reflect blood's unique rheological properties, such as shear-thinning and viscoelasticity, which become particularly pronounced in regions of high shear stress like arterial stenoses[14], [15]. Non-Newtonian models, including Carreau-Yasuda and generalized Oldroyd-B models, have been widely applied to better simulate these conditions. For example, Roy et al. (2017) demonstrated that in stenosed arteries, non-Newtonian models significantly improve accuracy by allowing viscosity to vary with shear rate, which Newtonian models cannot achieve [16]–[18]. Studies like those by Hyde-Linaker et al. (2022) and Assi et al. (2023) also integrate patient-specific data through high-resolution imaging, providing more precise hemodynamic simulations that can predict areas of high wall shear stress (WSS) and oscillatory shear index (OSI), key factors in assessing disease progression and treatment risks[19], [20]. Recent advancements in computational fluid dynamics (CFD) have facilitated these complex simulations, allowing for more accurate analyses of blood flow patterns and vessel wall stresses. For instance, research by Abhilash et al. (2024) focused on bifurcation geometries and identified high-risk areas for plaque accumulation due to altered WSS[21]. Similarly, the work by Otero-Cacho et al. (2024) explored the effects of pressure wires on fractional flow reserve (FFR) measurements, finding that invasive devices significantly impact hemodynamic readings, particularly in severely stenosed vessels[22]. The importance of geometry and device alignment in stented vessels was further underscored by Georgakarakos et al. (2014), who investigated misaligned stent-grafts and found that their orientation could lead to flow disturbances and elevated WSS, which in turn raises the risk of complications like thrombosis[18]. Such findings are complemented by studies focusing on RBC behavior and aggregation, such as those by Jędrzejczak et al. (2023), who used population balance models to examine hemolysis in narrowed arteries, further demonstrating the value of localized non-Newtonian adjustments in high-shear regions[23], [24]. Additionally, hybrid modeling techniques have emerged to capture both fluid and structural dynamics. Roy et al. (2023) demonstrated that fluid-structure interaction models could provide insights into vascular compliance and flow mechanics under pulsatile conditions, crucial for understanding blood flow in diseased arteries[25]. Studies incorporating finite element methods and advanced rheology models, as seen in the work of Nadau and Sequeira (2007), have enabled simulations of shear-thinning and viscoelastic flows that account for variable viscosity, contributing to a deeper understanding of blood flow behavior in complex arterial geometries [15]. Collectively, these studies highlight the need for non-Newtonian and CFD-based approaches in accurately modeling arterial blood flow, particularly under pathological conditions. This research underscores the role of advanced modeling in clinical applications, from designing personalized stent placements to predicting high-risk areas for plaque buildup and thrombosis. By capturing the intricate interactions between blood flow, vessel geometry, and medical devices, these models pave the way for more effective diagnostic and therapeutic strategies in vascular health management.

Many early blood flow models treated blood as a Newtonian fluid, with constant viscosity and no dependency on shear rate. While these models provide a basic understanding, they are limited in their ability to capture the complexities of blood flow in stenosed arteries. Newtonian models fail to represent the shear-thinning behaviour of blood, leading to inaccuracies in predicting flow velocity, wall shear stress, and turbulence in constricted areas. Non-Newtonian models, such as the Carreau-Yasuda model, address these limitations by incorporating blood's rheological properties. The Carreau-Yasuda model describes blood viscosity as a function of shear rate, allowing it to capture the shearthinning behaviour seen in real blood. By adjusting parameters such as zero shear rate viscosity, infinite shear rate viscosity, and relaxation time, the Carreau-Yasuda model can more accurately simulate the changes in blood viscosity as it moves through a stenosis. This enhanced accuracy is essential for predicting how blood flow will behave under pathological conditions, where high shear rates are prevalent. Accurate modelling of arterial blood flow, particularly in stenosed arteries, has significant implications for clinical and biomedical applications. One of the primaries uses of these models is in risk assessment for cardiovascular diseases. For example, understanding the stress patterns on vessel walls can help predict where plaque build-up is likely to occur or where an existing stenosis might worsen. This information is valuable for clinicians in developing preventative strategies and monitoring patients at risk for cardiovascular events. In the field of medical device design, blood flow models play a crucial role in the development of stents, grafts, and other vascular implants. When designing these devices, engineers must consider how they will interact with blood flow, especially in regions prone to turbulence and high shear stress. A stent that disrupts blood flow too much can increase the risk of clot formation, while one that fails to address the stenosis fully may not provide the intended benefit. Accurate modelling allows engineers to simulate these interactions before a device is implanted, reducing the likelihood of complications. Furthermore, blood flow modelling is valuable in drug delivery, particularly for treatments that target specific sites within the vascular system. In cases of severe stenosis, it may be necessary to administer clot-dissolving agents or other medications directly to the affected area. Blood flow models can help determine the optimal delivery method and dosage by predicting how the drug will disperse under the given flow conditions. This targeted approach can enhance the effectiveness of treatment and minimize side effects. As technology advances, there is potential for blood flow models to become even more accurate and useful. One promising area is the integration of particle dynamics into blood flow simulations. For instance, incorporating the behaviour of nanoparticles within the blood can provide insights into how they might be used for targeted drug delivery in stenosed arteries. Additionally, models that incorporate the porosity of arterial walls can offer a more detailed view of how substances move in and out of the bloodstream, which is especially relevant in the study of drug delivery and tissue engineering. Another area for future exploration is the application of machine learning to blood flow modelling. Machine learning algorithms can analyse large datasets from patient imaging and simulations, identifying patterns that may not be immediately apparent to human researchers.

The objective of this research is to compare the efficacy of Newtonian and non-Newtonian (Carreau-Yasuda) models in accurately simulating blood flow dynamics within stenosed arteries. By examining flow characteristics such as velocity, pressure, and shear stress under these two models, the study aims to determine which model better reflects the complex rheological properties of blood, particularly its non-Newtonian behaviour in regions of high shear, such as those caused by stenosis. The findings are expected to contribute to improved modelling techniques for cardiovascular applications, offering insights that could enhance diagnostic accuracy, treatment planning, and medical device design for vascular conditions. By combining traditional fluid dynamics with machine learning insights, researchers can create hybrid models that improve predictive accuracy and adapt to new data. In conclusion, the modelling of arterial blood flow, particularly in the presence of stenosis, is a critical area of research with far-reaching implications for cardiovascular health, medical device design, and drug delivery. Accurate models are essential for understanding how stenoses impact blood flow and for developing strategies to mitigate their effects. While traditional Newtonian models have provided a foundation, the shift toward non-Newtonian models like Carreau-Yasuda represents a significant advancement, offering a closer approximation of real blood behavior. As research continues, these models will become increasingly sophisticated, paving the way for more effective diagnostics, treatments, and preventative measures in cardiovascular care.

#### **2. Methods**

#### *2.1 Model Design*

The model geometry for simulating blood flow is based on a simplified arterial structure that includes a stenosed region, representing a narrowing of the artery. This narrowing is designed to mimic realworld conditions seen in atherosclerosis, where plaque buildup restricts blood flow. The geometry incorporates a cylindrical arterial segment with a localized constriction to replicate the stenosis. Meshing is a critical step for accurate simulations, as it subdivides the geometry into small elements for computational analysis. The meshing technique uses a finer mesh in the stenosis region to capture detailed flow dynamics, such as variations in velocity and pressure, while a coarser mesh is applied to less complex regions of the artery. This approach balances accuracy and computational efficiency, ensuring that key areas are sufficiently resolved without excessive computation times.

#### *2.2 Boundary and Initial Conditions*

Boundary and initial conditions define how blood flow enters, moves through, and exits the artery in the simulation. For the inlet, a fixed velocity is applied to simulate the pulsatile entry of blood into the artery, often set to a physiological average (e.g., 0.45 m/s in the case of normal blood flow). The arterial walls are assigned a no-slip condition, meaning blood flow velocity is zero at the wall surface, which mirrors the interaction of blood with endothelial cells. Finally, a pressure boundary condition is set at the outlet, usually at a fixed value to reflect downstream resistance in the circulatory system. These conditions are essential to replicate the natural flow characteristics and stress distribution within the arterial segment.

## *2.3 Material Properties*

The Newtonian model assumes constant viscosity, meaning that blood's resistance to flow is unaffected by changes in shear rate. This simplification is characterized by basic fluid parameters, including:

1. Density: The mass of blood per unit volume, typically set at 1060 kg/m³.

2. Yield Stress: The stress level at which blood begins to flow, set to a standard physiological value.

3. Standard Viscosity: For Newtonian models, this is a constant value representing blood's resistance to deformation (typically 0.0035 Pa.s for human blood).

This model provides a simplified approach to blood flow, useful for general understanding but limited in reflecting the shear-dependent behavior of real blood.

### *2.4 Carreau-Yasuda Model*

The Carreau-Yasuda model represents blood as a non-Newtonian fluid, where viscosity changes with shear rate. This model accounts for the fact that blood becomes less viscous under higher shear rates, a phenomenon known as shear-thinning. Key parameters include:

1. Zero Shear Rate Viscosity: Viscosity at very low or zero shear rates, when blood flow is minimal.

2. Infinite Shear Rate Viscosity: Viscosity at very high shear rates, representing the lowest viscosity achievable by blood.

3. Relaxation Time: The time it takes for blood to adjust its viscosity in response to changes in shear.

4. Power Index: A parameter that controls how sharply viscosity changes with shear rate.

These parameters allow the model to replicate the complex flow properties of blood, especially in the stenosed region where shear rates are high, making it more accurate for simulating pathological conditions.

## *2.5 COMSOL Multiphysics Configuration*

The simulation environment is set up using COMSOL Multiphysics, a software platform that enables complex fluid flow simulations. Key configurations include selecting the Laminar Flow Module for Newtonian conditions and the Non-Newtonian Fluid Module for Carreau-Yasuda modeling. Solver settings are fine-tuned to handle nonlinearities, particularly in the non-Newtonian model, where changes in viscosity must be recalculated dynamically. Computational resources are optimized based on the complexity of the mesh and model, often requiring high memory and processing capabilities to resolve fine details in the stenosis region. To evaluate the differences between Newtonian and non-Newtonian flow, the study adjusts parameters such as flow velocity and pressure to observe how each model responds under varying conditions. For instance, simulations are run at different inlet velocities to examine how increased flow rate affects shear stress and pressure in the stenosed region. The results provide insights into how blood flow dynamics change with varying physiological and pathological conditions, highlighting the strengths and limitations of each model in capturing these variations.

## **3. Result and Discussion**

This research simulates blood flow in an arterial vessel experiencing a stenosis of *l* mm, with normal velocity and pressure. The simulation applies the Newtonian postulate with several changes in material properties, namely velocity  $(u_{x,y})$  and pressure  $(p_{x,y})$ . The first simulation is conducted using normal data with the following values:

- 1. Normal blood flow velocity =  $0.45 \text{ m/s}$
- 2. Yield stress of normal vessel  $= 0.65$  Pa
- 3. Normal blood density =  $1060 \text{ kg/m}^3$
- 4. Normal blood viscosity =  $0.0035$  Pa·s (cP = centipoise)

In Newtonian conditions, blood flow will behave under the following conditions:

$$
\rho(\mathbf{u}.\nabla)\mathbf{u} = \nabla.[-p\mathbf{l} + \mathbf{k}] + \mathbf{F}
$$

$$
\rho \nabla.\mathbf{u} = 0
$$

$$
\mathbf{k} = \mu(\nabla \mathbf{u} + (\nabla \mathbf{u})^T)
$$

In normal (stationary) conditions, the blood flow velocity (inlet) is 0.45 m/s under the following conditions:

 $u, t = 0$ 



Figure 1 shows a simulation of blood flow velocity through an artery with a stenosed (narrowed) region in the center. The color gradient represents the magnitude of velocity in meters per second (m/s), with blue indicating lower velocities and red indicating higher velocities. As blood flows from left to right, it accelerates as it enters the narrowed region due to the reduced cross-sectional area, reaching peak velocity (in red) at the center of the stenosis. Beyond the stenosed area, the velocity gradually decreases as the artery widens again. This pattern demonstrates how stenosis impacts blood flow dynamics, with increased velocity in constricted regions leading to higher shear stress on the arterial walls.



Figure 2. Blood pressure in artery

Figure 2 illustrates the simulation of blood pressure within an artery containing a stenosed (narrowed) section. The image on the left represents the pressure distribution along the artery, with a color gradient indicating pressure magnitude in Pascals (Pa). Red indicates higher pressure, while blue represents lower pressure. As blood flows from the wider section into the narrowed area, the pressure decreases

due to the increased flow velocity required to pass through the constricted space. This reduction in pressure is most pronounced at the center of the stenosis, reflecting the effect of the narrowed geometry on blood pressure. The chart on the right shows the convergence of the nonlinear solver during the simulation, with "Iteration number" on the x-axis and "Error" on the y-axis. This graph demonstrates how the error decreases across successive iterations, indicating that the simulation reached a stable solution. Lower error values signify increased accuracy in the calculated pressure distribution. This simulation provides insights into the pressure changes in stenosed arteries, which can inform understanding of stress on the arterial walls and potential for damage in regions of narrowing. To observe changes in blood flow behavior, an additional simulation was conducted by altering the viscosity and velocity properties in the arterial blood flow model using the Carreau-Yasuda model. In the non-Newtonian case, the equation was modified as follows:

$$
\rho(\mathbf{u}.\nabla)\mathbf{u} = \nabla. [-p\mathbf{l} + \mathbf{k}] + \mathbf{F}
$$
  
\n
$$
\rho \nabla.\mathbf{u} = 0
$$
  
\n
$$
\mathbf{k} = \mu (\nabla \mathbf{u} + (\nabla \mathbf{u})^T)
$$
  
\n
$$
\mu_{app} = \mu_{inf} + (\mu_0 - \mu_{inf}) [1 + (\lambda \dot{\gamma})^a]^{\frac{n-1}{a}}
$$
  
\n
$$
\dot{\gamma} = \sqrt{2s} : s, s = \frac{1}{2} [\nabla \mathbf{u} + (\nabla \mathbf{u})^T]
$$

The Carreau-Yasuda model is one of the models used to describe the rheological properties of non-Newtonian fluids, including those of blood. The Zero Shear Rate Viscosity value in this model can be calculated from the parameters given in the model. In the Carreau-Yasuda model, the parameters used are:  $n=1$ 

$$
\mu_{app} = \mu_{inf} + (\mu_0 - \mu_{inf}) [1 + (\lambda \dot{\gamma})^a]^{\frac{n-1}{a}}
$$

The basic properties of the material use the general values of the Carreau-Yasuda model



This table lists the properties and parameters used in the Carreau-Yasuda model for simulating non-Newtonian blood flow. Key parameters include Zero shear rate viscosity (mu0), set at 100 Pa·s, which represents the viscosity when shear rate approaches zero, and Infinite shear rate viscosity (mu\_inf), set at 0.1 Pa·s, indicating the viscosity at high shear rates. Relaxation time (lam\_car) of 1 second reflects the time blood takes to adjust its viscosity with changes in shear rate. The Power index (n\_car), set at 2, determines the degree of shear-thinning behavior, while the Transition parameter (a\_car) of 1 controls the smoothness of the transition between low and high shear viscosities. Additionally, basic properties such as Heat capacity (Cp), Density (rho), and Thermal conductivity  $(k$  iso) are provided for completeness in the thermal aspects of the simulation. These parameters enable accurate modeling of blood's non-Newtonian properties in response to varying flow conditions. With the same geometric model, changes in blood flow behavior are produced as shown in Figure 3 and Figure 4.



Figure 3 depicts the simulation of blood flow through an artery with a stenosis, modeled using the Carreau-Yasuda non-Newtonian approach. The color gradient represents blood velocity in meters per second (m/s), with blue indicating lower velocities and red indicating higher velocities. In this non-Newtonian model, as blood flows from left to right, it accelerates significantly when passing through the narrowed region due to the stenosis, with the highest velocities shown in red at the center of the constriction. The Carreau-Yasuda model captures the shear-thinning behavior of blood, where viscosity decreases at higher shear rates, allowing for a more realistic representation of blood flow compared to a Newtonian model. This approach highlights how non-Newtonian properties affect flow dynamics, particularly in stenosed regions, with implications for understanding blood behavior under various physiological conditions.



Figure 4 displays the simulation results of blood pressure using the Carreau-Yasuda non-Newtonian model. The left image shows a contour plot of the pressure distribution along an artery with a stenosis, with the color gradient indicating pressure magnitude in Pascals (Pa). Red represents high pressure, which decreases toward blue as pressure reduces. As blood flows through the artery from left to right, the pressure decreases sharply at the stenosed region, reflecting the changes in blood flow dynamics due to the narrowing. This drop in pressure corresponds with an increase in blood velocity through the constricted area, consistent with the principles of fluid dynamics. The right image is a convergence graph from the nonlinear solver, with the x-axis showing the iteration number and the y-axis showing the error. As iterations progress, the error reduces significantly, indicating that the simulation reached a stable and accurate solution. The use of the Carreau-Yasuda model provides a realistic representation of blood pressure behavior in the presence of arterial narrowing, highlighting the model's ability to simulate non-Newtonian fluid characteristics. The simulation continues by assuming that the blockage of blood vessels will result in a rapid change in flow and pressure that occurs, so that property modifications are carried out with changes in m/s and Pa pressure so that it is produced.  $u_{xy} = 0.95 p =$ 0.1



Figure 5 presents a simulation of blood flow velocity through an artery with a narrowed (stenosed) section, highlighting changes in flow behavior in response to the constriction. The color scale represents the velocity magnitude in meters per second (m/s), with blue indicating lower velocities and red indicating higher velocities. As blood flows from the wider section on the left to the stenosed region, velocity increases sharply, reaching a maximum at the center of the constriction (shown in red). This increase in velocity is due to the reduced cross-sectional area, which requires the blood to move faster to maintain flow continuity. Beyond the stenosed area, the velocity gradually decreases as the artery widens again. This simulation emphasizes the significant impact of arterial narrowing on blood flow dynamics, specifically how stenosis causes localized acceleration and changes in velocity profile.



Figure 6. Changes in pressure in the area of narrowing of blood vessels Figure 6 displays the simulation results of blood pressure in an artery with a stenosed (narrowed) region. The left image shows a contour plot of the pressure distribution along the artery, with the color gradient representing pressure magnitude in Pascals (Pa). Higher pressure is indicated by red, while lower pressure is shown in blue. As blood flows from left to right, the pressure decreases significantly in the stenosed region due to the increased flow velocity required to pass through the constricted area. This drop in pressure is most prominent at the narrowest point of the artery, reflecting the impact of stenosis on blood pressure dynamics. The right image presents a convergence graph for the nonlinear solver used in the simulation, with iteration numbers on the x-axis and error values on the y-axis. The steady decrease in error across iterations indicates that the simulation reached a stable solution. The simulation

highlights how stenosis affects blood pressure, providing insights into the pressure gradient caused by arterial narrowing and its implications for stress on vessel walls and potential vascular complications.

#### **4. Conclusions**

This study has demonstrated the effectiveness of using both Newtonian and non-Newtonian (Carreau-Yasuda) models to simulate blood flow in stenosed arteries. While the Newtonian model provides a basic understanding of blood flow characteristics, it lacks the precision to capture the complex behavior of blood, especially in regions of high shear rates such as those caused by stenosis. The Carreau-Yasuda model, on the other hand, successfully represents the shear-thinning properties of blood, accurately showing changes in viscosity and flow behavior under varying conditions. The results indicate that the non-Newtonian model is better suited for simulating realistic blood dynamics in narrowed arteries, highlighting its importance in understanding hemodynamic changes in pathological conditions. The findings from this research have significant implications for both clinical diagnostics and biomedical engineering. By using a non-Newtonian model, clinicians can gain a more accurate understanding of blood flow patterns in patients with arterial stenosis, allowing for better risk assessment and prediction of complications. This improved accuracy can also inform the design of targeted treatments, such as customized stents or surgical interventions that consider specific flow characteristics in narrowed arteries. Additionally, the insights gained from non-Newtonian simulations can be applied to the development of medical devices and drug delivery systems that interact with blood flow, potentially improving the efficacy of these technologies. Overall, this research underscores the value of advanced modeling techniques in enhancing cardiovascular care and supporting innovation in medical device design.

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