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Research Article

Mathematical Modelling on Functioning of Blood Flow in Arteries and Rheology

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Abstract. Navier-Stokes equations present a comprehensive numerical simulation of arterial flow by integrating fluid dynamics, non-Newtonian blood rheology, and arterial wall mechanics in a coupled framework. Using cylindrical coordinates with axisymmetric assumptions, the flow of blood is presented by the Navier-Stokes equations, while the viscosity and wall dynamics are Carreau-Yasuda non-Newtonian law to reflect the decrease in blood viscosity with increased blood flow. The arterial wall dynamics are represented by a linear elastic model, incorporating radial displacement as a function of internal pressure and wall elasticity. A coupling condition links pressure to wall deformation allowing for accurate simulation of fluid structure interaction. The simulation reveals critical insights into the propagation of pressure and mechanical response of arterial length, the role of shear-dependent viscosity in shaping flow profiles and the transmission and wall compliance effects, validating the physiological relevance of the improved model. The outcomes of this research hold significant potential for applications in cardiovascular diagnostics, treatment strategy, and the design of vascular prosthetics or drug delivery systems.

Keywords. Mathematical modelling, Blood flow, Rheology

Mathematics Subject Classification (2020). 01Axx, 35, 70, 92, 00A71

1. Introduction

The cardiovascular system is a complex network responsible for the transport of blood, nutrients, and gases throughout the human body. Accurate modelling of blood flow dynamics and arterial wall behavior is essential for understanding both healthy physiology and pathological conditions such as atherosclerosis, hypertension, and aneurysms. Over the years, mathematical and computational models have significantly advanced our ability to simulate these biological processes with high precision. Blood flow in large arteries is often modeled using the Navier-Stokes equations in cylindrical coordinates due to their compatibility with the geometry of vessels (Pedley [12], and Formaggia *et al*. [5]). These equations describe the conservation of momentum and mass in fluid dynamics and form the basis for simulations of pulsatile flow in axisymmetric arterial geometries. However, blood exhibits non-Newtonian behavior, with its viscosity depending on the local shear rate. The Carreau-Yasuda model provides a more accurate characterization of the blood viscosity compared to Newtonian approximations (Merrill [9], and Boyd *et al*. [2]). To simulate the mechanical response of arterial walls, researchers often adopt linear or nonlinear elastic models. These models account for wall stiffness, elasticity, and interaction with internal pressure, enabling simulations of radial displacement in response to pulsatile flow (Fung [7], and Holzapfel *et al*. [8]). The coupling between blood pressure and arterial wall deformation is crucial for physiological accuracy, and is often captured using coupling conditions that relate pressure to wall displacement (Formaggia *et al*. [5], and Narasimhudu *et al*. [10]). Advancements in numerical methods and simulation tools, such as finite difference and finite element methods implemented in MATLAB, have enabled the solution of this complex, coupled equations (Chapra and Canale [3], and Baskurt and Meiselman [1]). These integrated models offer valuable insights into cardiovascular dynamics and serve as powerful tools in biomedical research and clinical planning.

2. Mathematical Model Formation

We consider the following assumption for the mathematical model:

(a) Blood is assumed to behave as an incompressible, and non-Newtonian fluid.

(b) Arterial walls exhibit viscoelastic nature and are modelled as deformable elastic tubes

(c) Blood flow is laminar under physiological conditions

(d) Arterial geometry is considered in 2-dimension or axisymmetric 3-dimension for some realism

2.1 Mathematical Model

The base model for Blood Flow in Cylindrical Coordinates for Axisymmetric Arterial Geometry is the incompressible Navier–Stokes equations in cylindrical coordinates under the assumption of axisymmetric and Newtonian or non-Newtonian fluid flow.

Case I: For blood flow

There are three governing equations for blood flow in cylindrical coordinates for axisymmetric arterial geometry, which are given below:

$$\frac{\partial u}{\partial t}+u\frac{\partial u}{\partial t}+v\frac{\partial u}{\partial t}=-\frac{1}{\rho}\frac{\partial p}{\partial r}+v(\Delta^2 u)+F_r,$$

$$\frac{\partial v}{\partial t}+u\frac{\partial v}{\partial t}+v\frac{\partial v}{\partial t}=-\frac{1}{\rho}\frac{\partial p}{\partial z}+v(\Delta^2 v)+F_z,$$

$$\frac{1}{r}\frac{\partial (ru)}{\partial r}+\frac{\partial v}{\partial z}=0.$$

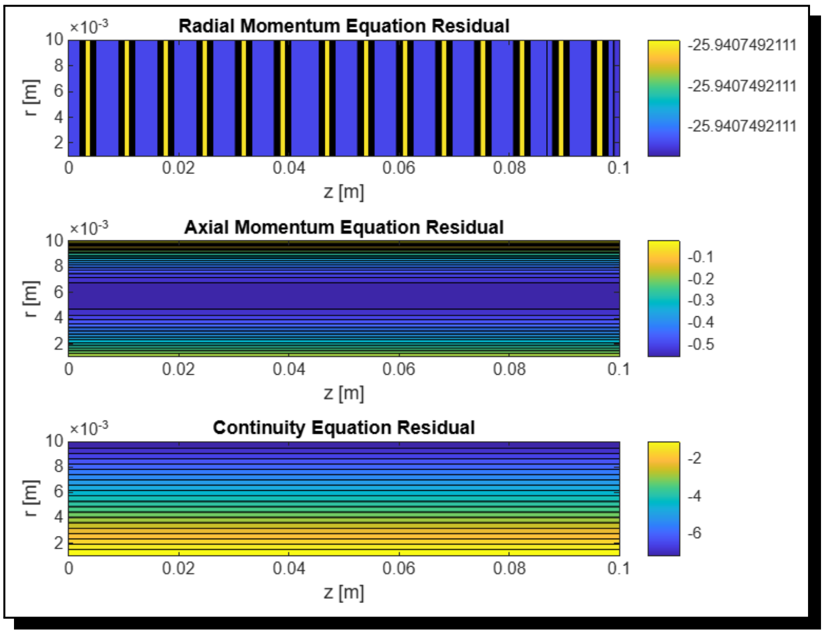

Figure 1. Combined graph for three governing equations

Figure 1 represents the residual distributions of the governing equations in blood flow modelling. The radial momentum residual exhibits oscillatory patterns, while the axial momentum and continuity residuals remain relativity smooth and stable, indicating that numerical convergence is better achieved in the axial and continuity equations compared to the radial momentum equation.

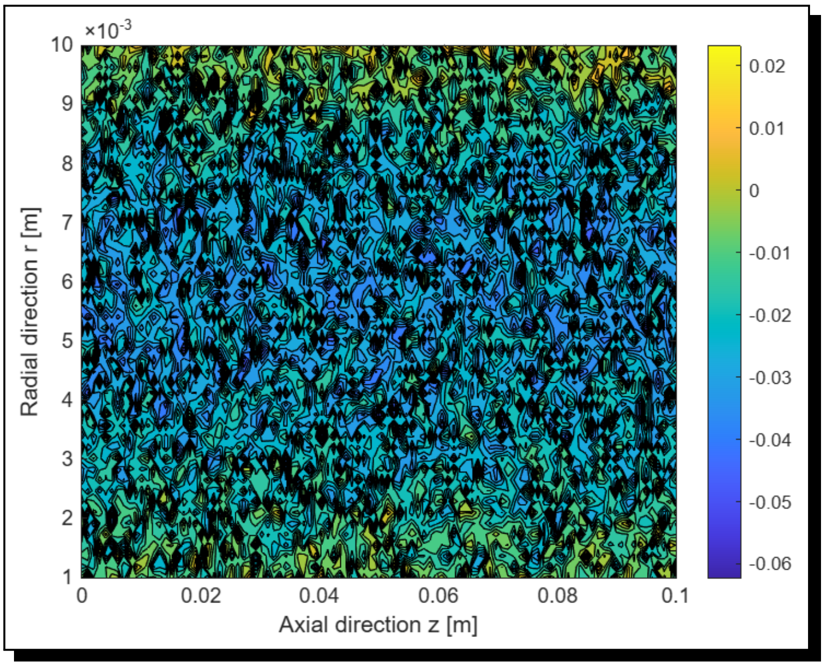

Figure 2. Radial momentum equation residual (Navier-Stokes)

Figure 2 provides the radial momentum equation residuals, showing localized fluctuations with both positive and negative valuers, including non-uniform convergence behaviour across the radial and axial directions.

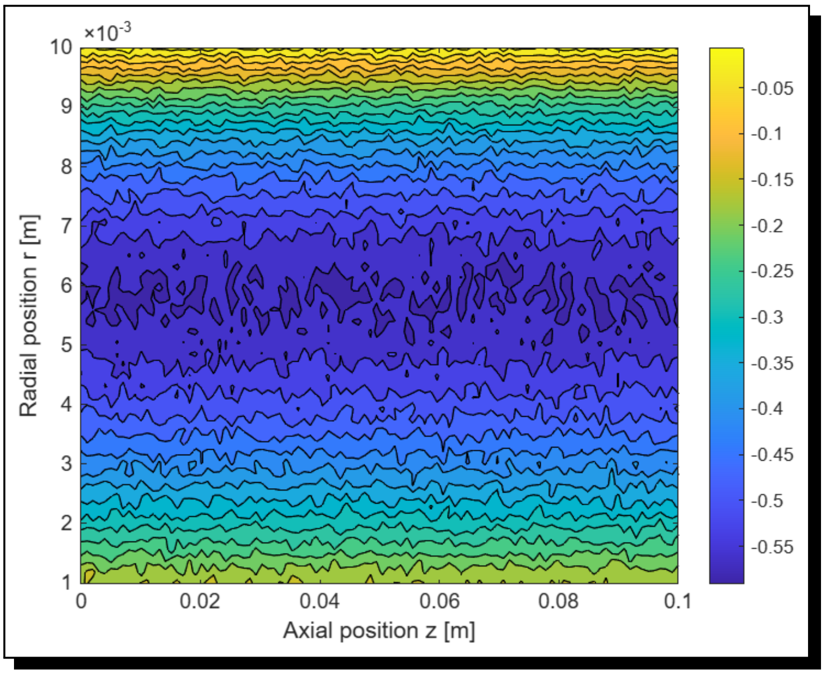

Figure 3. Axial momentum equation residual (Navier-Stokes)

Figure 3 presents the axial momentum equation residuals, which exhibits a smooth gradient along the radial direction, with lower residuals concentrated near the central region, indicating relativity stable convergence.

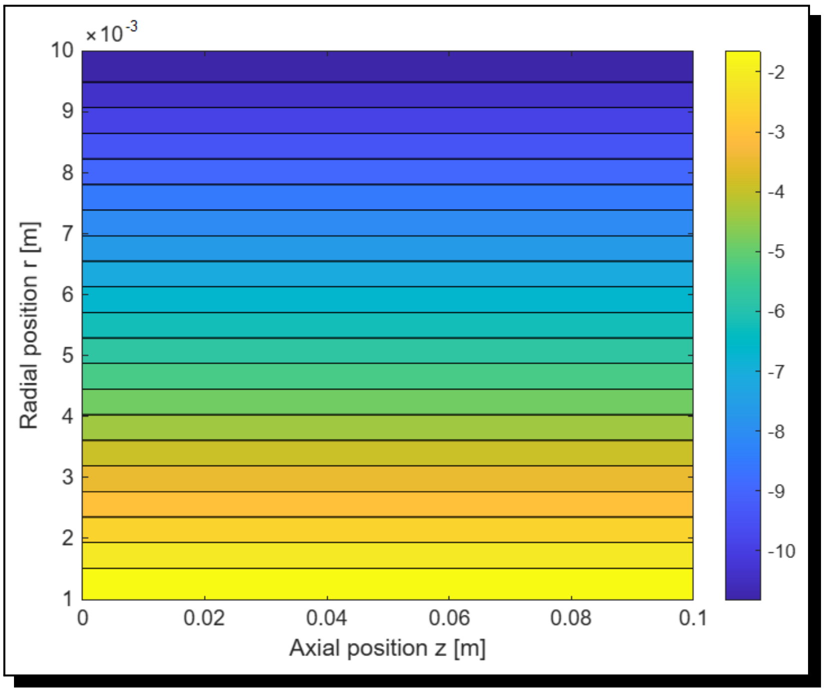

Figure 4. Continuity equation residual: $\frac{1}{r}\frac{\partial(ru)}{\partial r} + \frac{\partial v}{\partial z}$ (incompressibility)

Figure 4 depicts the continuity equation residuals, which showing a smooth and uniform variation along the axial direction, with residuals primarily distributed across the radial axis, indicating consistent mass conservation behaviour.

Case II: Non-Newtonian Rheology

The Carreau-Yasuda model effectively represents the shear thinning behaviour of blood flow. It is denoted by $\mu(\dot{\gamma})$ and defined by

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

where

μ_0, μ_∞	:	viscosities at zero and infinite shear rate
λ	:	time constant
a, n	:	empirical constants
$\dot{\gamma}$	:	shear rate

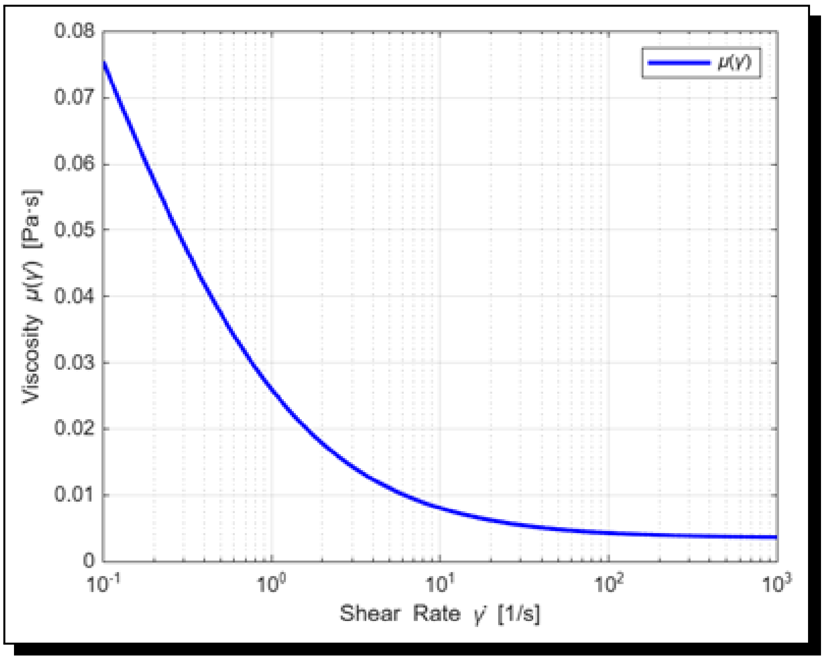

Figure 5. Non-Newtonian Rheology - Carreau-Yasida Model: Blood viscosity vs. Shear rate

Figure 5 demonstrates the Carreau Yasuda model of blood viscosity decreases nonlinearly with increasing shear rate, highlighting the shear thinning behaviour of blood.

Case III: Arterial Wall Dynamics

Consider Radial displacement $R(t,z)$

$$\rho_w \frac{\partial^2 R}{\partial t^2} = E\frac{\partial^2 R}{\partial z^2} - \beta(R(t,z) - R_0) + p - p_{ext},$$

where

R_0	:	reference radius
β	:	wall stiffness coefficient
E	:	Young's modulus of arterial wall
$\dot{p}_{ext}$	:	external pressure

Figure 6 presents the arterial wall experiences a small but stable radial displacement (0.010002 m), which rapidly rises with time and axial position before reaching a plateau indicating a steady state deformation response.

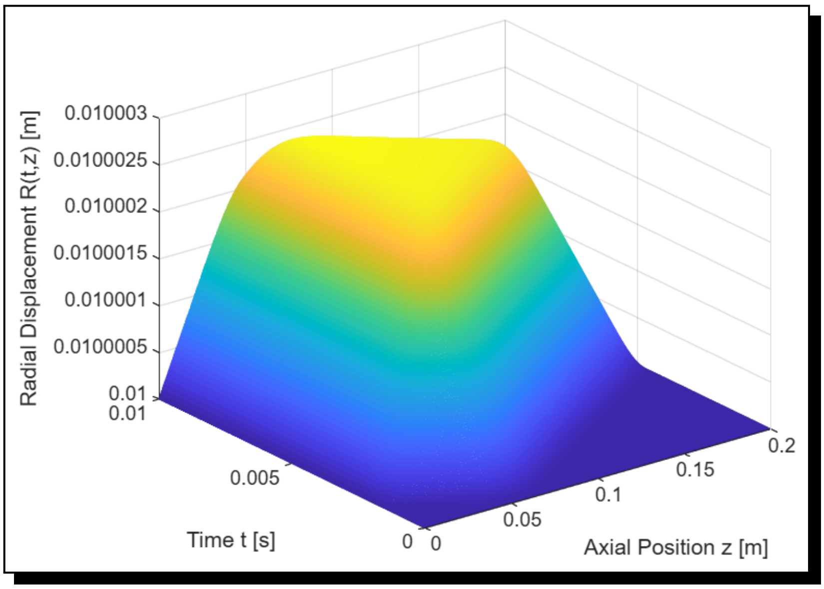

Figure 6. Arterial wall dynamics – Radial displacement

Case IV: Coupling Condition

$$p(t,z) = p_{ext} + \beta(R(t,z) - R_0).$$

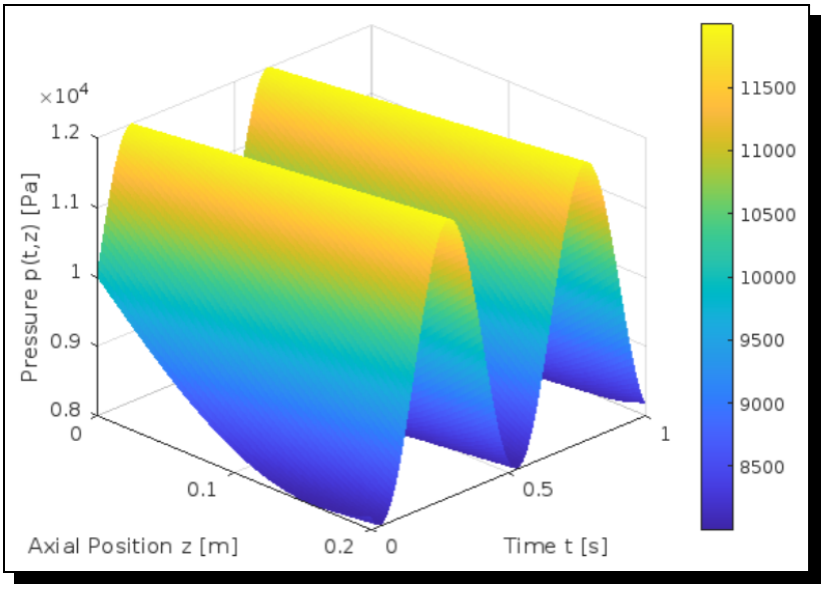

Figure 7. Coupling condition: $p_{ext} + \beta(R(t,z) - R_0)$

Figure 7 presents the pressure radius coupling effect, where arterial pressure rises proportionally with wall deformation, reflecting the elastic response of the vessel.

3. Simulation

The model couples axisymmetric Navier-Stokes flow, shear dependent viscosity via the Carreau Yasuda model, and an elastic arterial wall whose radial displacement feeds back to pressure through the linear coupling $p(t,z) = p_{ext} + \beta(R(t,z) - R_0)$. The outcomes of the study show:

(i) the fluid field drives time and space dependent shear rate that cause pronounced shear thinning,

(ii) the wall responds quickly to internal pressure producing a small radial expansion that approaches a quasi-steady value, and

(iii) residual maps indicate good axial convergence but localized oscillatory behaviour in the radial.

4. Discussion

The comprehensive numerical simulation developed herein integrates three critical physiological components to model blood flow dynamics in human arteries: fluid mechanics governed by the Navier-Stokes equations, non-Newtonian rheology of blood using the Carreau-Yasuda model, and arterial wall mechanics via a linear elastic model. The use of axisymmetric cylindrical coordinates reflects the true anatomical structure of blood vessels, ensuring physiological accuracy.

The simulation reveals that arterial blood flow is intricately influenced by the interaction between pressure gradients, viscosity changes due to shear rate, and the mechanical properties of the arterial wall. The Navier-Stokes equations, adapted for axisymmetric conditions, accurately capture the core dynamics of pulsatile flow along the vessel. The axial velocity evolves over time in response to pressure gradients, which are not static but dynamically linked to the arterial wall's displacement via the coupling condition. This coupling serves as a fundamental interface between fluid and solid mechanics in the model, enhancing the realism of the simulation.

One of the key insights derived from the simulation is the significance of non-Newtonian behavior of blood. The Carreau-Yasuda model successfully represents how blood viscosity changes with shear rate: at low shear rates, the viscosity is high, reflecting red blood cell aggregation, while at high shear rates, it decreases, corresponding to shear-thinning behavior. This variation in viscosity affects velocity profiles, leading to smoother flow transitions and more physiologically accurate representations of hemodynamics, especially in regions with varying flow acceleration such as during cardiac cycles.

The linear elastic model of the arterial wall provides further insight into the mechanical responsiveness of blood vessels. The wall displacement, governed by wave-like dynamics, shows that arterial expansion and contraction are not merely passive responses to internal pressure but involve wave propagation governed by material stiffness and density. The simulation results demonstrate that regions of high wall displacement correspond to zones of elevated pressure, validating the model's physical assumptions. Moreover, the temporal evolution of the wall radius shows how mechanical forces generate pressure waves, which propagate along the vessel length characteristic of real-life pulse wave transmission. The incorporation of a coupling condition, where pressure is a direct function of radial displacement proves essential. It not only ties the fluid and structural domains but also ensures that pressure profiles evolve in a manner consistent with wall compliance. This results in more accurate predictions of how pressure pulses travel and reflect within the circulatory system, critical for applications in cardiovascular diagnostics and treatment planning.

5. Conclusion

The integrated computational model effectively captures the coupled dynamics of blood flow, arterial wall mechanics and non-Newtonian rheological properties. It establishes a robust framework for investigating cardiovascular physiology under both normal and pathological conditions. The modular nature of the simulation makes it adaptable to more complex arterial geometries, disease models (e.g., aneurysms or stenoses), and can be further extended with more advanced *fluid-structure interaction* (FSI) techniques or two- or three-dimension domains. This study provides a solid foundation for future research in biomedical engineering and computational physiology, with the potential to advance clinical insights into hemodynamic processes and vascular health.

Competing Interests

The authors declare that they have no competing interests.

Authors' Contributions

All the authors contributed significantly in writing this article. The authors read and approved the final manuscript.

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