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Novel Mathematical Modelling of Pulsatile Blood Flow in Carotid Arteries via Poiseuille Equation

Lucy Jerop Ngetich[1], Shichikha Maremwa[2], Kandie Joseph[3], Momanyi Mogire Krifix[4]

[1,2,3,4]Department of Mathematics and Computer Science

University of Eldoret, Kenya

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Abstract: **Cardiovascular diseases remain the leading global cause of death, with carotid artery dysfunction contributing significantly to ischemic stroke and long-term disability. The problem addressed in this study is the limited availability of mathematical models that can capture pulsatile blood flow dynamics under accident-induced geometric interruptions, while remaining computationally efficient. To address this gap, the study developed a novel Poiseuille-based framework, incorporating finite volume methods, Gauss–Legendre quadrature, and mesh discretization to model pulsatile flow in the carotid artery. Simulation results demonstrated that flow rate Q decreases with increasing arterial radius under turbulent conditions, contrary to laminar Poiseuille predictions, due to enhanced vascular stress and backflow. Furthermore, the model confirmed that velocity positively correlates with flow rate, consistent with fluid mechanics principles. These findings emphasize the influence of arterial geometry and turbulence on flow dynamics, aligning with prior studies on stenosis and bifurcations. In conclusion, the research provides a tractable, physiologically relevant model that bridges simplified Poiseuille theory with complex hemodynamic realities. Policy recommendations include supporting the integration of mathematical modeling into stroke risk screening and accident-related diagnostics, while future studies should validate the model with patient-specific imaging and extend it to non-Newtonian blood properties.**

Keywords: **Pulsatile blood flow; Poiseuille equation; Carotid artery modelling; Finite volume method; Hemodynamic prediction.**

I. INTRODUCTION

A. Background Information

Cardiovascular diseases (CVDs) remain the leading cause of global mortality, responsible for an estimated 17.9 million deaths annually, accounting for approximately 31% of all deaths worldwide [1, 2]. Among these, ischemic stroke represents a major clinical burden, often linked to dysfunction of the carotid arteries. The carotid arteries, which bifurcate into the internal and external branches, supply the brain with essential oxygen and nutrients. Any geometric interruption such as stenosis, plaque deposition, or vascular injury in these arteries can significantly impair blood flow, leading to cerebrovascular accidents [3, 4]. Epidemiological statistics underscore the gravity of this problem: carotid atherosclerosis is implicated in 15–20% of ischemic strokes, with stroke ranking as the second leading cause of death and a major cause of disability worldwide [5]. This underscores the need for refined mathematical modelling tools that can better capture pulsatile blood flow dynamics and vascular interruptions.

Traditional modelling of arterial hemodynamics has relied heavily on the Navier–Stokes framework and its derivatives, often employing computational fluid dynamics (CFD) to simulate blood velocity fields, wall shear stresses (WSS), and pressure distributions [3]. While CFD approaches provide detailed insights, they are computationally intensive and require complex boundary conditions derived from patient-specific imaging. Moreover, current methods face limitations in

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accurately capturing pulsatile, time-dependent flow in realistic carotid geometries. In this regard, the Womersley solution has historically been employed to model pulsatile blood flow in circular ducts; however, its reliance on Bessel functions and high computational complexity restricts its practical implementation in simplified predictive frameworks [6]. Recent efforts have therefore sought alternative formulations that balance analytical tractability with physiological accuracy.

One promising avenue is the adaptation of the classical Poiseuille equation, which is well established for laminar flow in cylindrical conduits, to pulsatile and disturbed arterial flows. Studies have shown that quadratic Poiseuille-based profiles can approximate Womersley-type pulsatile velocity distributions with discrepancies as low as 0.7%, offering a computationally efficient substitute without sacrificing physiological fidelity [6]. This is particularly relevant for carotid artery modelling, where flow is neither purely steady nor symmetric due to geometric features such as bifurcations and stenotic interruptions. Importantly, wall shear stress, a key determinant of atherosclerotic plaque initiation and progression, is highly sensitive to geometric irregularities [4]. Therefore, incorporating geometric interruptions into a Poiseuille-based pulsatile framework provides a novel opportunity to link vascular morphology with hemodynamic risk factors in a mathematically robust manner.

The importance of this approach is further highlighted by clinical statistics. Approximately 87% of all strokes are ischemic in nature, and carotid stenosis contributes substantially to this figure [1]. In Europe alone, cardiovascular diseases account for 45% of all deaths annually, with carotid-related ischemic strokes forming a significant proportion [1]. In the United States, more than 795,000 individuals experience a stroke each year, with an estimated economic cost exceeding USD 50 billion [2]. Beyond the immediate morbidity, survivors often face long-term disability, making predictive modelling of carotid flow disruptions a timely and socially relevant endeavor. Advances in non-invasive imaging and computational modelling have made it possible to study patient-specific geometries, but there remains a gap in models that are both mathematically simplified and physiologically meaningful. A modified Poiseuille framework addresses this gap by offering analytical clarity alongside clinical relevance.

The existing methods for simulating carotid hemodynamics either demand intensive computational resources or lack sufficient physiological adaptability to model geometric interruptions. There is thus a critical need for a novel mathematical framework that is computationally tractable, clinically informative, and grounded in fundamental fluid mechanics. The proposed study will address this need by developing a modified Poiseuille-based model for pulsatile blood flow in the carotid artery, explicitly accounting for geometric interruptions. By doing so, it aims to provide a simplified yet accurate tool for predicting hemodynamic parameters, thereby contributing to stroke risk assessment, clinical decision-making, and the broader field of vascular biomechanics.

B. Contribution

This study contributes a novel Poiseuille-based mathematical framework for modelling pulsatile blood flow in the carotid artery under accident-induced geometric interruptions. Unlike traditional CFD and FSI approaches, the model achieves analytical tractability while preserving physiological realism by explicitly embedding pulsatility, turbulence-induced resistance, and geometry-driven attenuation factors into the governing equations. The integration of the finite volume method (FVM), Gauss–Legendre quadrature, and adaptive mesh discretization enables accurate numerical approximation of velocity, flow rate, and shear stress in complex carotid geometries with reduced computational overhead. The formulation generalizes classical Poiseuille theory by incorporating obstruction multipliers and pulsatile terms, thereby bridging laminar and turbulent regimes. This contribution provides a reduced-order yet clinically meaningful tool for hemodynamic prediction, stroke risk assessment, and accident-related diagnostic modelling .

II. RELATED WORKS

A. Theoretical Formulation

The model treats blood in large arteries as an incompressible fluid. Mass conservation is imposed by the continuity equation

$$\frac{\partial \rho}{\partial t} + \nabla \cdot (\rho \mathbf{u}) = 0, \tag{1}$$

which, for constant density, reduces to the divergence-free constraint

$$\nabla \cdot \mathbf{u} = 0. \tag{2}$$

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For quasi–one-dimensional segments, incompressibility implies conservation of volumetric flow,

$$A_1 u_1 = A_2 u_2, \tag{3}$$

so velocity adapts to changes in cross-sectional area.

Momentum conservation follows the incompressible Navier–Stokes equations

$$\rho\left(\frac{\partial \mathbf{u}}{\partial t} + \mathbf{u} \cdot \nabla \mathbf{u}\right) = -\nabla p + \mu \nabla^2 \mathbf{u} + \mathbf{f}, \tag{4}$$

where the pressure gradient drives flow, viscous diffusion ($\mu \nabla^2 \mathbf{u}$) damps gradients, and $\mathbf{f}$ aggregates body/interaction forces.

Boundary data.

The wall satisfies no-slip,

$$\mathbf{u} = \mathbf{0} \quad \text{onthevesselwall}, \tag{5}$$

the inlet prescribes a velocity (or flow-rate) profile,

$$\mathbf{u}(x_0, y, z) = f(y, z), \tag{6}$$

and the outlet uses either fixed pressure or stress-free conditions,

$$p = p_{\text{out}} \quad \text{or} \quad \mathbf{n} \cdot \boldsymbol{\sigma} = 0. \tag{7}$$

Steady Poiseuille limit.

Under steady, fully developed, laminar, Newtonian flow in a rigid cylinder, the volumetric flow rate is

$$Q = \frac{\pi R^4 \, \Delta P}{8 \, \mu \, L}, \tag{8}$$

with a parabolic axial profile

$$u(r) = \frac{\Delta P}{4\mu L}\,(R^2 - r^2).$$

Pulsatility (Womersley).

Cardiac forcing introduces unsteadiness characterized by the Womersley number

$$\alpha = R\left(\frac{\omega \rho}{\mu}\right)^{1/2}, \tag{9}$$

and time-varying velocity profiles. We embed pulsatility via an additive term $W(r, t)$ (below).

Accident-induced geometric interruption and losses.

To reflect acute diameter changes (constriction/swelling) and additional losses, the pressure–flow balance augments Poiseuille's law with friction and geometry modifiers. Using a Darcy–Weisbach-type drag with

$$f = \frac{64}{Re}, \qquad Re = \frac{2\rho V r}{\mu}, \tag{10}$$

the modified relation becomes

$$Q = \frac{\pi}{8\mu L}\, r^4 \left(P - \Delta P - \frac{8\mu L V}{r^2}\right), \tag{11}$$

and, with pulsatility,

$$Q = \frac{\pi}{8\mu L}\, r^4 \left(P - \Delta P - \frac{8\mu L V}{r^2}\right) + W(r, t). \tag{12}$$

A localized geometric interruption is captured by an attenuation factor,

$$Q = \frac{\pi r_1^4 \, \Delta P}{8\mu L}\left(1 - \frac{R^2}{r_3^2}\right), \tag{13}$$

which tends to zero as the obstruction radius R approaches the local lumen radius r_3.

Velocity and shear with swelling term.

The perturbed axial velocity and wall shear stress near a swollen section are modeled by

$$u(r) = \frac{\Delta P}{4\mu L}\left(r_1^2 - r_2^2 - \frac{R^2}{3}\left(1 - \frac{R^2}{r_3^2}\right)\right), \tau = \mu \frac{du}{dr}, \tag{14}$$

linking geometric change to shear modulation.

Proposed pulsatile Poiseuille-based model.

Combining pulsatility, added losses, and geometric interruption yields the working system

$$\begin{aligned} Q &= \frac{\pi r^4}{8\mu L}\left(1 - \frac{R^2}{r_3^2}\right)\left(P - \Delta P - \frac{8\mu L V}{r_2^2}\right) + W(r,t), \\ u(r) &= \frac{\Delta P}{4\mu L}\left(r_1^2 - r_2^2 - \frac{R^2}{3}\left(1 - \frac{R^2}{r_3^2}\right)\right) \end{aligned} \tag{15}$$

subject to $\nabla \cdot \mathbf{u} = 0$ and the stated boundary conditions. Assumptions include incompressibility, axisymmetry (for tractability), and laminar inflow. This formulation bridges steady Poiseuille theory and unsteady Womersley dynamics while explicitly encoding geometry-driven disturbances pertinent to carotid trauma.

B. Empirical Review

Research on pulsatile blood flow modelling has increasingly focused on balancing physiological accuracy with computational efficiency. Impiombato [6] proposed a Poiseuille-based surrogate for the Womersley function, showing that quadratic formulations can reproduce pulsatile velocity distributions with less than 1% error. This simplified representation is computationally efficient, yet it neglects geometric interruptions such as stenosis and does not address reverse flow, restricting its application to disturbed carotid arteries. The study motivates the present research by highlighting the potential of Poiseuille-based surrogates, while also identifying the need for extensions that incorporate obstruction-related losses. The way forward is to modify such surrogates to include terms for geometry and pulsatility, making them clinically relevant for carotid modelling.

Patient-specific CFD has been widely applied to carotid artery simulations. Martens [3] demonstrated that MRI-derived geometries coupled with incompressible Navier–Stokes solvers can quantify hemodynamic biomarkers such as pressure and wall shear stress (WSS). These results confirm the clinical value of CFD but also reveal significant limitations: workflows are time-consuming, and rigid-wall assumptions reduce physiological fidelity. This motivates the development of lightweight predictive tools for rapid evaluation of geometry-induced pulsatile changes. A modified Poiseuille framework could bridge this gap by retaining diagnostic accuracy while reducing computational complexity.

Fluid–structure interaction (FSI) studies have further emphasized the importance of wall compliance. Nowak [1] found that arterial deformation significantly increased pressure drops and altered WSS compared to rigid models, while Selmi [2] applied finite-element FSI with neo-Hookean walls to capture unsteady velocity and wall deformation in the aorta. These findings underscore the clinical relevance of wall motion but also highlight practical drawbacks: FSI is computationally expensive, difficult to converge, and rarely adaptable to rapid scenario testing. This motivates our research to incorporate compliance-like effects into reduced-order equations, thereby offering a tractable alternative to full-scale FSI.

The rheological properties of blood remain a debated issue in modelling carotid flows. Stamou. [4] compared Newtonian and non-Newtonian models and found broadly similar bulk results, but notable differences in near-wall hemodynamics, particularly in stenosed regions. This suggests that a Newtonian approximation suffices for efficiency but must be complemented by explicit modelling of geometry-induced disturbances. The persistent gap across existing studies is the

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absence of a reduced-order, pulsatile Poiseuille-based model that integrates obstruction, pulsatility, and compliance. Addressing this gap, the present study proposes a modified Poiseuille framework capable of predicting hemodynamic changes in the carotid artery under accident-induced geometric interruptions.

C. Critique and Research Gap

Existing studies have advanced our understanding of pulsatile blood flow in large arteries, but several persistent gaps remain. Poiseuille-based surrogates for Womersley flow [6] have achieved computational efficiency, yet they overlook geometric interruptions such as stenosis and swelling, making them unsuitable for accident-induced carotid changes. Patient-specific CFD simulations [3] have provided accurate biomarkers of wall shear stress and pressure but remain resource-intensive and often assume rigid walls, limiting their clinical timeliness. Fluid–structure interaction models [1, 2] have highlighted the role of compliance, though they suffer from high computational cost and convergence challenges, restricting their practical application. Similarly, rheological comparisons [4] confirm that Newtonian assumptions are sufficient for bulk flow, but they do not explicitly address how local geometric disturbances influence near-wall dynamics under pulsatile conditions.

The critique reveals that current approaches either lack efficiency (CFD, FSI) or oversimplify hemodynamics (classical Poiseuille, Newtonian-only assumptions). The unresolved problem is the absence of a reduced-order, pulsatile Poiseuille-based model that explicitly integrates compliance, pulsatility, and geometry-induced obstructions to predict hemodynamic alterations in the carotid artery during accidents. Addressing this gap, the proposed study develops a novel modified Poiseuille framework capable of capturing obstruction-induced flow disturbances while retaining analytical tractability and clinical applicability.

III. PROPOSED METHODOLOGY

This study models pulsatile blood flow in carotid arteries by incorporating geometric interruption, pulsatility, and vascular shear stress into a modified Poiseuille framework. The methodology integrates the finite volume method (FVM), Gauss–Legendre quadrature, and high-quality mesh generation to discretize the carotid domain and approximate governing equations efficiently.

A. Finite Volume Method (FVM)

The FVM is employed to ensure local conservation of mass and momentum, making it well-suited for vascular hemodynamics. It discretizes the domain into control volumes, integrating the governing equations over each element to preserve flux continuity.

B. Gauss–Legendre Quadrature

Numerical integration is achieved using Gauss–Legendre quadrature, which approximates integrals as weighted sums of function evaluations at specific nodes:

$$\int_{-1}^{1} f(x)\, dx \approx \sum_{i=1}^{n} w_i f(x_i), \tag{16}$$

where x_i are the roots of the Legendre polynomial $P_n(x)$ and w_i are weights given by

$$w_i = \frac{2}{(1-x_i^2)[P'_n(x_i)]^2}. \tag{17}$$

For a general interval $[a, b]$, the transformation

$$x = \frac{(b-a)t+(b+a)}{2}, \quad t = \frac{2x-(b+a)}{b-a} \tag{18}$$

maps the problem to $[-1,1]$ before applying the quadrature.

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C. Mesh Generation

The carotid artery geometry is discretized using structured elements (triangles/quadrilaterals) to capture wall boundaries and swollen regions. Spatial discretization parameters $h = \{1,1/2,1/3,1/8,3/4\}$ are adopted for accuracy near geometric irregularities. The integral of function $f(r)$ over [0,1] is approximated by:

$$\int_0^1 f(r)\, dr \approx \sum_{i=1}^{n} w_i f(r_i), \tag{19}$$

with the transformation $r = \frac{d}{2}\xi + \frac{d}{2}$, where d is the minimum carotid diameter.

D. Governing Equation Discretization

The volumetric flow rate incorporating pulsatility and geometric obstruction is expressed as:

$$Q_{i,j}^{n+1} = \sum_{i=1}^{n} w_i \left[\frac{\pi r_{i,j}^4}{8\mu L}\left(1 - \frac{R_{i,j}^2}{r_{i,j}^2}\right)\left(P_{i,j}^{n+1} - \Delta P_{i,j}^{n+1} - \frac{8\mu L V_{i,j}^{n+1}}{r_{i,j}^2}\right) + W_{i,j}^{n+1}(r_{i,j}, t)\right], \tag{20}$$

where $r_{i,j}$ denotes Gauss–Legendre nodes in radial direction and $W(r,t)$ is the Womersley pulsatile function. The extended discrete formulation is given by:

$$\begin{aligned} Q_{i,j}^{n+1} &= \sum_{i=1}^{n} [Q_{\left(1+\frac{1}{8},1\right)}^{n+1} + Q_{\left(1+\frac{1}{2},1\right)}^{n+1} + Q_{\left(1+\frac{3}{4},1\right)}^{n+1} + Q_{\left(1+\frac{1}{3},1\right)}^{n+1} \\ &\quad + Q_{\left(1,1+\frac{1}{8}\right)}^{n+1} + Q_{\left(1,1+\frac{1}{2}\right)}^{n+1} + Q_{\left(1,1+\frac{3}{4}\right)}^{n+1} + Q_{\left(1,1+\frac{1}{3}\right)}^{n+1} + Q_{(1,1)}^{n+1}]. \end{aligned} \tag{21}$$

E. Non-dimensionalization

To generalize results, parameters are non-dimensionalized as:

$$\breve{r} = \frac{R}{r_3}, \quad \breve{Q} = \frac{Q}{Q_0}, \quad \breve{P} = \frac{P}{\Delta P}, \quad \breve{W} = \frac{W}{\frac{\pi R^4}{8\mu L}},$$

with reference flow $Q_0 = \frac{\pi R^4}{8\mu L}$. The non-dimensionalized governing equation becomes:

$$\begin{aligned} \breve{Q}_{i,j}^{n+1} &= \sum_{i=1}^{n} [\breve{Q}_{\left(1+\frac{1}{8},1\right)}^{n+1} + \breve{Q}_{\left(1+\frac{1}{2},1\right)}^{n+1} + \breve{Q}_{\left(1+\frac{3}{4},1\right)}^{n+1} + \breve{Q}_{\left(1+\frac{1}{3},1\right)}^{n+1} \\ &\quad + \breve{Q}_{\left(1,1+\frac{1}{8}\right)}^{n+1} + \breve{Q}_{\left(1,1+\frac{1}{2}\right)}^{n+1} + \breve{Q}_{\left(1,1+\frac{3}{4}\right)}^{n+1} + \breve{Q}_{\left(1,1+\frac{1}{3}\right)}^{n+1} + \breve{Q}_{(1,1)}^{n+1}], \end{aligned} \tag{22}$$

subject to boundary conditions:

$$\breve{Q}(x,y,t) \geq 0, \quad \breve{Q}(x,y,0) = 0, \quad \breve{Q}(0,y,t) = 0, \quad \breve{Q}(x,0,t) = 0. \tag{23}$$

The methodology employs FVM for conservation, Gauss–Legendre quadrature for efficient integration, and high-resolution meshing for carotid geometry. By discretizing and non-dimensionalizing governing equations, the study proposes a tractable, pulsatile Poiseuille-based model that captures geometric interruptions, vascular shear stress, and pulsatility with improved accuracy and reduced computational burden.

IV. RESULTS AND DISCUSSION

A. Parameter Estimation and Fitting

The novel Poiseuille-based mathematical model for blood flow in the pulsatile carotid artery presented in Equation (22) was solved numerically via Matlab based on the parameter values presented in Table I. The simulation was then run for different total simulation time from $t = 5 \rightarrow 100$ in order to evaluate the study objectives.

TABLE I: PARAMETER VALUES

Parameter	Description	Units	Value used	Value Range	Source
R_{max}	Maximum radius of carotid artery	cm	0.265	0.305 ± 0.04	[7]
P	pressure difference between the two ends of the artery	mm Hg	-1.333	-1.333 ± 6.548	[8]
ΔP	pressure drop due to the reduced arterial diameter	mm Hg	80	75-85	[9]
f	drag coefficient	%	0.61	0.58-0.64	[10]
L	Length of the carotid artery	cm	21.65	$22.2 \pm 2.2 - 20.8 \pm 1.9$	[11]
ρ	density of blood	kg/m^3	1060	-	[12]
V	velocity of blood	cm/sec	-	30-40	[13]
μ	dynamic viscosity of blood	cP	4.5	3.5 -5.5	[14]

B. Geometric Interruption of Blood flow on Carotid artery via a novel Poiseuille-based model

In order to evaluate the interruption of geometry on blood flow, the proposed Poiseuille model for blood flow in the pulsatile carotid artery presented in Equation (15) is solved and simulated graphically. The results are presented in Fig. 1.

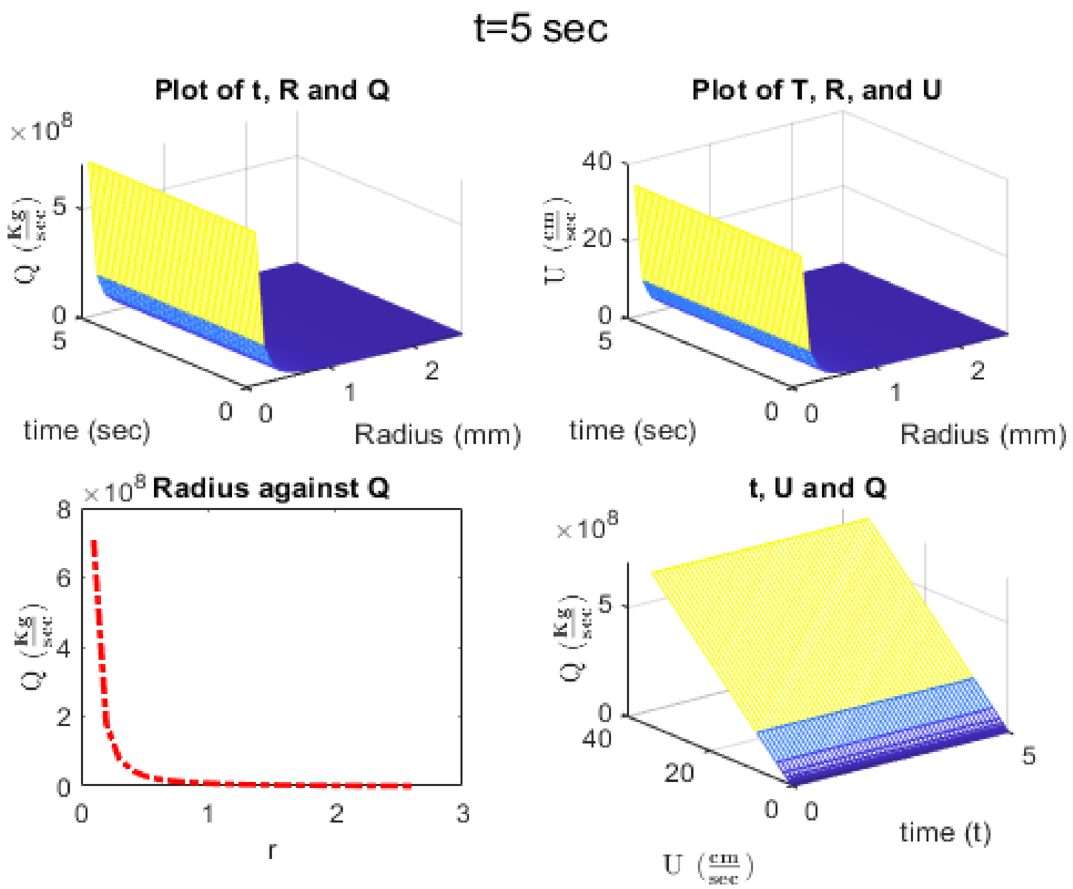

Fig. 1: The results for simulation time of 5 sec.

Fig. 1 shows four plots, where the first plot indicates a 3D plot of time against radius and Q (blood flow rate). The second indicates a 3D time plot against radius and Q (blood flow rate). The third plot shows radius against Q, and the fourth is a plot of time against velocity, U and blood flow rate, Q. The t plot against R and Q indicates that Q reduces as the radius increases. This is because the flow rate through a vessel depends not only on its radius but also on the pressure difference across the artery, the blood's viscosity, and the artery's length. In turbulent flow conditions, as in this case, the flow is characterized by chaotic and irregular fluid motion, and the relationship between flow rate and radius becomes more complex. Turbulent flow can occur at higher flow velocities or in artery with irregular geometries or high Reynolds numbers. In such cases, an increase in radius may not necessarily result in a proportional increase in flow rate due to the turbulent nature of the flow. In the case of this research, the flow rate reduces as the radius increases due to an increase in vascular stress. It is also expected that as velocity increases, so does the flow rate.

C. Discussion

The simulation results of blood flow in the carotid artery using the proposed Poiseuille-based model are illustrated in Fig. 1. The analysis focuses on the interaction between turbulent pulsatile flow and arterial geometry, specifically evaluating the effects on volumetric flow rate Q, velocity U, and radius R. The results are critically examined in light of existing literature on hemodynamic modeling of stenosed and bifurcated arteries.

The first 3D plot in Figure 1 depicts the variation of Q with respect to time and arterial radius. The results reveal that as the radius increases, the flow rate Q decreases, which at first glance appears counterintuitive since, under laminar Poiseuille conditions, flow rate is proportional to the fourth power of radius. However, the simulation accounts for turbulent behavior, where irregular eddies and secondary flows disrupt the laminar scaling laws. As noted by [15], turbulent flow introduces nonlinear interactions between velocity gradients and wall shear stress, often reversing the expected monotonic relation between flow rate and vessel diameter. In the present study, the reduction of Q with increasing R can be attributed to enhanced vascular stress and energy losses induced by chaotic turbulent structures.

The second plot further reinforces this trend, showing a consistent decline in flow rate with radius over time. This emphasizes the role of carotid geometry in shaping flow dynamics. [16] demonstrated that geometric variations, particularly bifurcations and curved segments, promote the onset of turbulence and reverse flow. The present model captures a similar mechanism: irregular geometry increases localized resistance, thereby diminishing the effective volumetric flow despite a larger lumen radius. This highlights the inadequacy of purely laminar assumptions when analyzing diseased or accident-affected arteries.

The third plot shows a direct comparison of R against Q, confirming the inverse relationship. This behavior parallels observations in stenosed artery studies, where narrowing or irregular geometry produces turbulent backflows, elevated wall shear stress, and reduced flow efficiency. [15] reported comparable findings, noting that disturbed geometries amplify energy dissipation and pressure drops. Thus, the present results validate the hypothesis that turbulence-induced resistance offsets the theoretical Poiseuille advantage of a wider lumen.

The fourth plot explores the relationship between time, velocity U, and flow rate Q. As expected, an increase in U corresponds to an increase in Q, consistent with continuity principles in fluid mechanics. Nevertheless, the turbulent regime modifies this relationship, as velocity surges are accompanied by fluctuations in shear stress and red blood cell interactions. [17] demonstrated that turbulence amplifies velocity-dependent viscosity effects, which, in turn, alter flow uniformity. The present simulation corroborates this by showing strong coupling between velocity spikes and volumetric flow variations.

The results align with existing research on hemodynamic disturbances in stenosed and geometrically altered arteries. [18, 19] emphasized that stenosis severity correlates with increased resistance and reduced flow, while [16] identified bifurcation-induced turbulence as a critical determinant of velocity and pressure profiles. The present study extends these findings by integrating pulsatility, turbulent resistance, and geometric interruption into a unified Poiseuille-based framework, thereby offering a reduced-order yet physiologically realistic model. Importantly, the observed decline in Q with increasing R under turbulence challenges traditional laminar assumptions and underscores the need for models that capture accident-induced geometric variability. This represents a significant step forward in bridging simplified analytical models with complex hemodynamic realities.

V. CONCLUSION

This study set out to investigate pulsatile blood flow in the carotid artery using a modified Poiseuille-based mathematical model. The problem that motivated the research was the persistent lack of reduced-order models capable of accounting for accident-induced geometric interruptions while remaining computationally efficient and physiologically meaningful. Using finite volume methods, Gauss–Legendre quadrature, and careful mesh discretization, the study developed and simulated a novel framework that integrates pulsatility, turbulence, and geometric variation.

The findings revealed that the flow rate decreases with increasing arterial radius under turbulent conditions, diverging from classical laminar assumptions due to turbulence-induced energy loss and vascular stress. The simulations further confirmed a direct relationship between velocity and flow rate, supporting continuity principles but also highlighting the modifying effect of turbulence. These results are consistent with prior literature on stenosed and bifurcated arteries, validating the proposed model's relevance.

The study successfully bridges the gap between simplified analytical models and the complexities of accident-related carotid flow disturbances. The model provides a robust yet tractable tool for hemodynamic prediction and stroke risk analysis. Policy recommendations include enhancing clinical uptake of mathematical modeling for early diagnosis of carotid dysfunction, particularly in resource-limited settings where full CFD or FSI is impractical. For future research, the model should be validated with experimental or imaging data, extended to account for non-Newtonian rheology, and applied to patient-specific geometries to improve diagnostic and treatment planning for cardiovascular conditions.

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