Mathematical Modeling of Hemodynamic Flow in Coronary Arteries Under Stenotic Conditions

Ramesh Solanki [1], Dr. G. Radha[2], Dr. S. Sudhakar Reddy[3], Dr.B.Logeshwary[4], R.K. Selvi[5], Pragneshkumar L Thakkar[6]

[1]Government Polytechnic, Vyara, Lecturer in Mathematics, Department of Science and Humanities, mail - rameshsolanki_maths12@yahoo.com, ORCID ID- https://orcid.org/1153-1163-1153-1163

[2]Professor, Department of Humanities and Science, KSRM College of Engineering (Autonomous), KADAPA, ANDHRA PRADESH, INDIA. ORCID ID: 0009 0002 8343 9213, Email ID: radhagajjala79@gmail.com

[3]Professor, Professor, Department of Mathematics, Sri Venkateswara College of Engineering (Autonomous), Tirupati, Andhra Pradesh, India. ORCID ID: 0009 0003 2346 3622
Email ID: drsudhakarreddy.s@svcolleges.edu.in

[4]Assistant Professor, Specialization in graph theory, Department of Mathematics, A.M Jain College, Meenambakkam, Chennai.logeshwary.b@amjaincollege.edu.in, ORCID ID: 1153-1163-1153-1163

[5]Associate Professor, PSNA College of Engineering and Technology, Dindigul (Autonomous), Anna University, E-mail: neelam.rk10@gmail.com, ORCID ID: 0000 0003 0262 4602

[6]Associate Professor of Mathematics, Department of Science and Humanities, L D College of Engineering, Ahmedabad, Gujarat, ORCID ID: 1153-1163-1153-1163X, Email ID: pragnesh83@ldce.ac.in

HOW TO CITE:

Ramesh Solanki,G. Radha,S. Sudhakar Reddy, B.Logeshwary, R.K. Selvi, Pragneshkumar L Thakkar (2026). Mathematical Modeling of Hemodynamic Flow in Coronary Arteries Under Stenotic Conditions

International Journal of Special Education, 41(19s), 1153-1163

ABSTRACT:

Coronary artery stenosis alters vascular geometry and can alter local hemodynamics, in terms of pressure loss, wall shear stress and flow velocity significantly. Quantitative modeling is a constructive tool to examine such effects under controlled conditions, especially when one would like to isolate the contribution of stenosis severity. The present work develops a reduced-order mathematical model of hemodynamic flow in a coronary artery with localized stenosis and evaluates how stenosis severity influences pressure gradient, pressure drop, wall shear stress, and velocity distribution. The artery is represented as a straight axisymmetric vessel with a Gaussian-shaped constriction, and blood flow is modeled as steady, incompressible, laminar, and Newtonian in a rigid domain. A Poiseuille-type formulation with spatially varying radius is used to compute hemodynamic quantities, and the domain is discretized along the axial direction. Simulations are performed for stenosis severities of 0.2, 0.4, 0.6, and 0.8. The results show that increasing stenosis severity produces strongly nonlinear hemodynamic responses. The minimum radius decreases from 2.4 mm to 0.6001 mm, while pressure drop increases from 20.0570 Pa to 541.3338 Pa. Peak wall shear stress rises from 0.4835 Pa to 30.9375 Pa, and peak velocity at the stenosis throat increases from 0.1658 m/s to 2.6521 m/s, whereas upstream velocity remains largely unchanged. These findings indicate that localized geometric constriction significantly amplifies flow resistance and concentrates mechanical stress within the stenosed region. The proposed framework provides a computationally efficient basis for parametric analysis and may support further extensions toward

more physiologically detailed coronary flow models.

Keywords: coronary artery stenosis, hemodynamic modeling, wall shear stress, pressure drop, velocity profile

Introduction

One of the leading causes of morbidity and mortality in the world is coronary artery disease (CAD), which is primarily brought about by the constriction of coronary arteries by the development of atherosclerotic plaque (Malakar et al., 2019). This restrictive or stenosis at the local level causes the effective lumen to be small and changes the physiological blood flow to the heart muscle. The outcome of the stenosis on the clinical outcome directly depends on the disruption of the coronary perfusion, which can eventually result in ischemia and cardiac events (Shao et al., 2020). Geometric obstruction is not the main factor that contributes to the extent of the severity and functional significance of arterial constriction, but is grossly influenced by the hemodynamic environment that ensues. Changes in the pressure gradients, flow velocity, and wall shear stress (WSS) have been reported to take the center stage in the development as well as destabilization of the vascular lesions. It has become evident that clinical and interventional studies provide crucial data on hemodynamic measurements to identify the level of stenosis and guide the decision-making process in therapeutics (Ahmad et al., 2018). The interaction of coronary artery disease with other cardiovascular diseases, such as aortic stenosis, is also not an easy task in terms of the behavior of the flow and needs a deeper understanding of the underlying hemodynamic processes (Androshchuk et al., 2023). Regarding fluid mechanics, any decrease in the radius of the vessel brings about great alterations in resistance to the flow, which in most cases results in nonlinear variations in pressure drop and acceleration. Such effects are especially strong in areas of extreme constriction, where the interactions of viscous forces and geometric constraints control the overall flow dynamics. Consequently, the experimental investigation of the hemodynamic responses in the stenotic state has become a fundamental aspect of cardiovascular investigations.

Mathematical and computational modeling has become an effective way to study the blood flow in coronary arteries, providing a non-invasive model that can be used to investigate various complex hemodynamic processes. Extensive literature has shown that the Navier-Stokes equation-based models are capable of effectively representing the main characteristics of coronary flow behavior, such as velocity distribution and pressure variation (Carvalho et al., 2021). These models are based on high-fidelity computational fluid dynamics (CFD) simulations and simple analytical expressions, each having its merits and demerits. Recent research has applied CFD to the study of blood flow through stenosed arteries, which has detailed information about the spatial distribution of velocity and shear stress. As an example, shear flow computations of symmetric stenotic geometries revealed that the acceleration of flow and pressure drop were highly dependent on the extent of constriction, which is in line with the fundamental principles of fluid mechanics (Akhtar et al., 2023). In the same manner, experiments on the structures of coronary arteries have shown that stenosis and arterial widening can significantly modify the hemodynamic parameters, such as flow separation and distribution of shear stress (Sandeep & Shine, 2021).

Further to the case of coronary arteries, analogous investigations have been conducted in carotid and bifurcated vessels demonstrating the sensitivity of the wall shear stress and velocity field to the geometry. Radial velocity distributions of stenosed carotid arteries have shown that there are significant improvements in peak velocity and local shear stress at the constriction site (Song et al., 2022). Bifurcated arterial studies have demonstrated that various stenotic geometries may result in strongly heterogeneous flow fields, further indicating the importance of geometry in determining hemodynamic responses

(Kanokjaruvijit et al., 2017). In addition to localized flow, hemodynamic measurements have also been applied to clinical situations to assess the functional importance of arterial stenosis. The use of pressure-based indices to establish the need of intervention has been emphasized in systematic reviews, and the connection between vascular constriction and flow resistance (van Brussel et al., 2017). Later modeling studies have generalized these models to include coupled physiological mechanisms, such as cardiac motion and electromechanical interactions, and show that more sophisticated models of the cardiovascular system exist in the modern age (Delestri et al., 2025). The development of computational methodologies has also solved uncertainties that are posed by CFD modeling to enhance the accuracy of predictions made in the simulation (Jewell et al., 2025).

Although there is a significant advancement in computational modeling, most of the current research involves either a high-fidelity model that is computationally costly or patient-specific data that can be restrictive to generalization. Although these methods are very informative, they tend to obscure the underlying connection between the arterial geometry and hemodynamic response. The low order analytically derived models, which can isolate the effect of the severity of stenosis and can be systematically studied in terms of parameters, are still needed. These models play a critical role in comprehending the effect of variations in lumen constriction on pressure drop, wall shear stress and flow velocity alone, without the complicating influence of complex boundary conditions or anatomical variation.

This study aims to establish a mathematical model of hemodynamic flow in a coronary artery with stenosis locally and the effect of the severity of stenosis on the main flow parameters. Quantification of pressure gradients, pressure drop, wall shear stress and velocity profiles in a reduced-order model that takes into account a spatially varying arterial radius is of interest in the analysis. The aim of the study is to explain how the change in geometrical constriction affects the hemodynamic behavior in the conditions of the model.

2. Methodology

2.1 Research Design

In this research, the modeling framework used is deterministic and based on physics to investigate the effect of localized stenosis of arteries on the coronary hemodynamics. The analysis is carried out by means of a reduced-order model of incompressible viscous flow rather than based on empirical data, and it permits the study of the geometric effects on the flow behavior to be controlled.

The study design will be established as a parametric study, the severity of stenosis will be manipulated systematically, and other physical and geometrical parameters will be held unchanged. This approach isolates the influence of lumen constriction, and allows the influence of lumen constriction on pressure fields, wall shear stress and velocity fields to be explicitly evaluated in a standardized mathematical setting.

2.2 Geometric Model

The coronary artery is modeled as a straight axisymmetric conduit that has a constriction. The radius is defined as a smooth function of the axial coordinate:

$$R(x) = R_0\left(1-\delta e^{-x^2/L^2}\right)$$

where R_0denotes the reference radius of the healthy vessel, $\delta$is the dimensionless stenosis severity, and Lcontrols the axial extent of the constriction. The geometry of the formulation is made continuous and differentiable and does not contain discontinuities that would introduce nonphysical gradients.

The domain is defined over a finite interval $x \in [-0.05,0.05]$m, with the stenosis centered at $x = 0$. Four levels of severity ($\delta = 0.2,0.4,0.6,0.8$) are considered to capture the transition from mild to severe narrowing. The localized geometric perturbation is highlighted in this representation, which is in line with observed stenotic lesions.

2.3 Governing Flow Model

The simplification of the equations of motion of the blood flow is done using the following

standard assumptions: steady flow, incompressible flow, laminar flow, axisymmetric flow, and in a rigid vessel. Here, the axial momentum balance is a Poiseuille-type equation with the radius being spatially varying:

$$\frac{dP}{dx} = -\frac{8\mu Q}{\pi R(x)^4}$$

This phrase is used to describe the dominating viscous forces on the opposition to flow in small to medium calibre arteries. The low shear rate (velocity) behavior of blood is non-Newtonian, but in this case, the Newtonian approximation is used to maintain the analytical tractability and to isolate geometrical effects.

The corresponding axial velocity profile is given by:

$$u(r,x) = \frac{1}{4\mu}\left(-\frac{dP}{dx}\right)(R(x)^2 - r^2)$$

which gives a local parabolic distribution that is consistent with full developed laminar flow. The shear stress at the wall is determined by the radial velocity gradient at the vessel boundary:

$$\tau_w(x) = \frac{4\mu Q}{\pi R(x)^3}$$

Together, these relations define a closed system linking geometry to hemodynamic response.

2.4 Numerical Procedure

A one-dimensional discretization of the governing expressions is done with respect to the axial coordinate. The domain is divided into 1000 evenly distributed grid points, which provide a sufficient resolution of the steep gradients in the vicinity of the stenosis throat. The local radius is calculated at every grid point, and then the pressure gradient and wall shear stress are evaluated. Numerical integration of the pressure gradient across the domain gives the total pressure drop across the domain, using the trapezoidal rule, which gives a stable and consistent estimate on smooth integrands. Velocity profiles are calculated at a representative axial location, one of them being an upstream location and the stenosis throat. The radial coordinate is discretized at each point into 300 points, which can be used to recreate the velocity field throughout the lumen. This method does not fully simulate on a multidimensional basis, but has enough detail to describe the redistribution of flows.

2.5 Parameterization

Model parameters are chosen to model typical physiological conditions, but are general. The baseline radius is set to $R_0 = 3 \times 10^{-3}$m, and the stenosis length scale is fixed at $L = 1 \times 10^{-2}$m. Blood viscosity is taken as $\mu = 3.5 \times 10^{-3}$Pa·s, with density $\rho = 1060\text{kg/m}^3$. A constant volumetric flow rate $Q = 1.5 \times 10^{-6}\text{m}^3/\text{s}$ is imposed.

Parameters are kept constant throughout simulations, and the only control variable is stenosis severity δ. This will be necessary so that apparent changes in hemodynamic quantities are due to geometric changes and not due to parameter coupling.

2.6 Quantification of Hemodynamic Metrics

The analysis is directed towards the extraction of the scalar and spatially distributed quantities, which determine the behavior of the flows under stenosis. These are the minimum and average vessel radius, axial pressure gradient, overall pressure drops, distribution of wall shear stress and maximum centerline velocity. Also, derived measures like velocity amplification and peak-to-mean shear ratios are assessed to measure the extent of perturbation on flow. Such a combination of measures will provide a point of comparison, which is the same across cases and the discovery of nonlinear tendencies as the stenosis severity rises.

3. Results

3.1 Geometric Characterization of Stenosis

Figure 1 shows the spatial change in the radius of the arteries at the various levels of stenosis. The smooth and symmetric constriction generated by the Gaussian formulation is at the mid-point of

the artery (x = 0). With the increase in stenosis severity, the extent of constriction progressively becomes more severe, although there is little effect on the upstream and downstream regions.

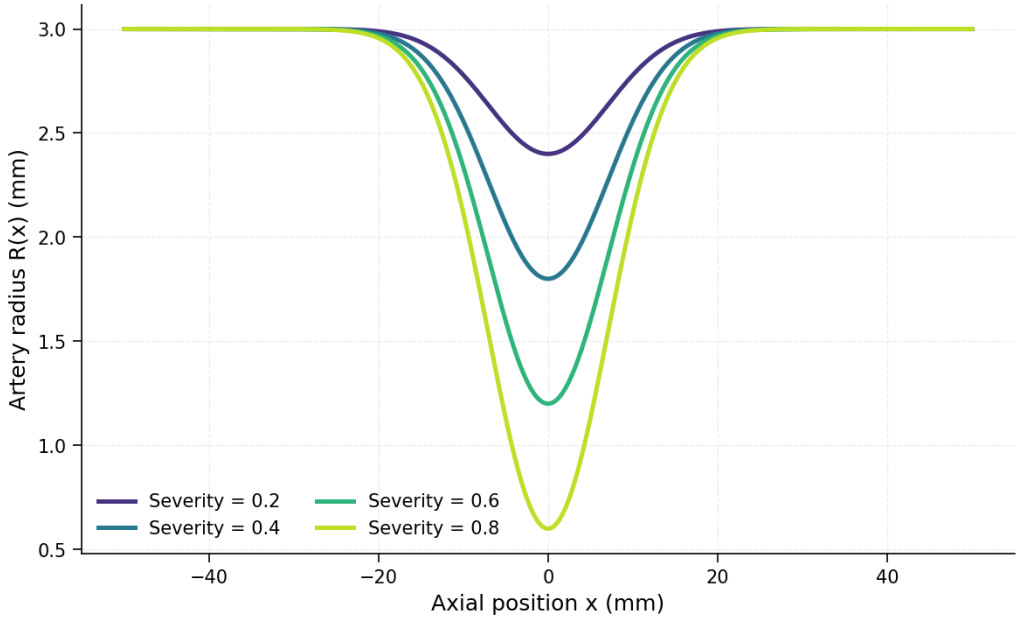

Figure 1. Coronary artery radius profile under varying stenosis severities

Table 1 summarizes the quantitative geometric changes and related pressure-based measures. The minimum radius decreases substantially with increasing stenosis severity, from 2.4 mm at $\delta = 0.2$to approximately 0.6 mm at $\delta = 0.8$, whereas the mean radius exhibits only a modest reduction. This validates the fact that the constriction is very localized and not evenly distributed along the vessel.

Table 1. Geometric and pressure-based hemodynamic metrics for different stenosis severities

Stenosis Severity	**Minimum Radius (mm)**	**Mean Radius (mm)**	**Pressure Drop (Pa)**	**Pressure Drop (mmHg)**	**Peak Pressure Gradient (Pa/m)**
0.2	2.4000	2.8938	20.0570	0.1504	402.9430
0.4	1.8000	2.7875	29.5679	0.2218	1273.4456
0.6	1.2000	2.6813	69.5445	0.5216	6446.2799
0.8	0.6001	2.5750	541.3338	4.0603	103114.6475

3.2 Pressure Gradient and Flow Resistance

The axial pressure gradient is highly spatially localized, and there is a sharp peak of the axial pressure gradient at the stenosis throat. The size of this peak rises sharply with the degree of stenosis and is indicative of the increasing resistance to flow in the constricted area. The resulting total pressure drops along the artery, as reported in Table 1, show a definite nonlinear trend. The increment is moderate between mild and intermediate stenosis, but there is a high increment in the more severe levels. In particular, the pressure drop rises by 69.54 Pa to 541.33 Pa with the increase of lumen, and it indicates that the relationship between the pressure drop and

lumen reduction is highly sensitive. This agrees with the theoretical inverse fourth-power dependence on vessel radius of hydraulic resistance, which suggests a disproportionate effect on hemodynamics of relatively small geometrical changes.

3.3 Wall Shear Stress Distribution

The distribution of wall shear stress (WSS) along the artery is shown in Figure 2. WSS in all instances shows a steep gradient at the stenosis throat, with the vessel radius being minimized. This peak is significantly increased with the severity of stenosis, which implies that mechanical stress is highly localized.

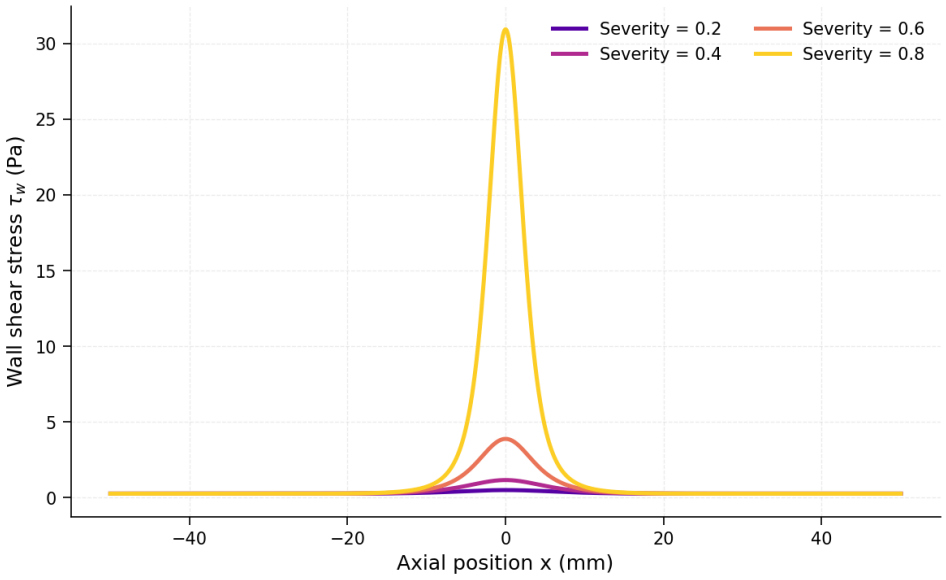

Figure 2. Wall shear stress distribution along the coronary artery

Table 2 provides the quantitative characteristics of WSS. While the mean WSS increases gradually across cases, the peak WSS shows a pronounced nonlinear rise, increasing from 0.48 Pa at $\delta = 0.2$to 30.94 Pa at $\delta = 0.8$. The ratio of peak to mean WSS also rises markedly, indicating that the shear stress progressively becomes localized in a small area as the stenosis severity rises.

Table 2. Wall shear stress characteristics under progressive coronary stenosis

Stenosis Severity	**Mean Wall Shear Stress (Pa)**	**Peak Wall Shear Stress (Pa)**	**Peak-to-Mean WSS Ratio**
0.2	0.2840	0.4835	1.7023
0.4	0.3626	1.1461	3.1605
0.6	0.5992	3.8679	6.4548
0.8	2.1278	30.9375	14.5398

3.4 Velocity Profiles and Flow Acceleration

Figure 3 shows the velocity profiles at the stenosis throat. The profiles all maintain the parabolic form of laminar flow, but the maximum velocity gets much higher with the severity of stenosis. This is a conservation of volumetric flow with cross-sectional areas that are decreasing in size.

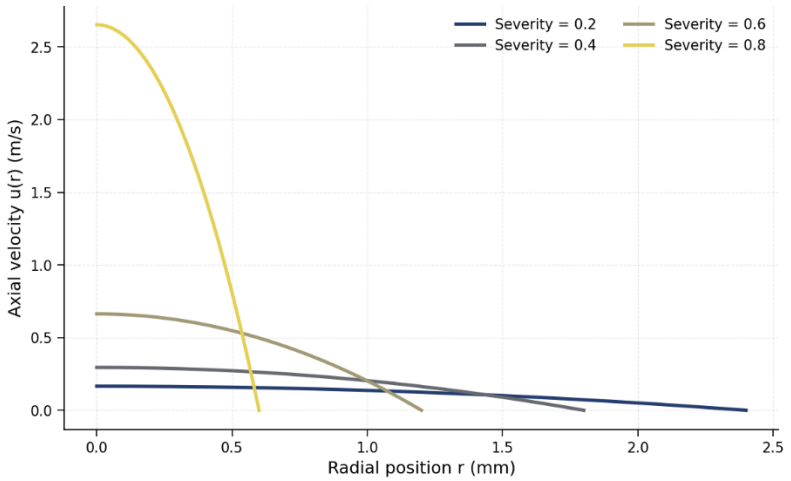

Figure 3. Velocity profiles at the stenosis throat for varying stenosis severities

The corresponding peak centerline velocities and amplification factors are reported in Table 3. Velocity of the healthy upstream part is almost the same in all cases but velocity at the stenosis throat escalates drastically. For the most severe case ($\delta = 0.8$), the peak velocity exceeds 2.6 m/s, representing a significant amplification relative to the upstream flow.

Table 3. Peak centerline velocity and velocity amplification at healthy and stenosed sections

Stenosis Severity	Peak Velocity (Healthy Section) (m/s)	Peak Velocity (Throat) (m/s)	Velocity Amplification Factor
0.2	0.1061	0.1658	1.5625
0.4	0.1061	0.2947	2.7777
0.6	0.1061	0.6631	6.2495
0.8	0.1061	2.6521	24.9950

In all simulations, the severity of stenosis results in a uniform and highly nonlinear change in hemodynamic behavior. Localized narrowing of the vessel radius leads to a high pressure gradient, high resistance to flow and the tremendous amplification of the wall shear stress and flow velocity at the stenosis throat. The results point to the sensitivity of coronary hemodynamics to geometric constriction and give us a coherent picture to understand the consequences of stenosis in the premises of the current model.

4. Discussion

The fundamentals of coronary stenosis are that it changes the balance of the vascular geometry and the viscous flow resistance, and the current findings indicate that such a relationship is clear and internally consistent. The more severe the stenosis, the greater the pressure gradients across the modeled artery, the more pressure was lost through the wall, the greater the wall shear stress and the more pronounced acceleration of axial velocity at the stenosis throat. There was no linearity in these changes through the simulated cases. Rather, the hemodynamic response was amplified more as the constriction increased with the most severe stenosis. This is a trend that coincides with the fact that resistance is extremely dependent on lumen radius in a reduced order laminar flow model. Informative are the pressure results in particular. Where mild narrowing only resulted in small changes in the total pressure drop, severe narrowing resulted in a large change, demonstrating that the system is more sensitive to narrowing, as the vessel approaches the critical

narrowing. The nonlinear behavior was also observed in the distribution of shear stress of the wall. There was a slow increase in mean shear values and a rapid increase in peak wall shear stress, which was highly localized at the stenosis throat. This difference between average and peak performance is significant in that it indicates that geometric narrowing does not simply move the whole hemodynamic field to a higher plane; it concentrates mechanical load into a small area. This can be reinforced by the velocity profiles. The main differences were in the upstream flow, which was almost the same, and in the throat area, the amplification of the centerline velocity was significant, thus the main disturbance was not global along the vessel segment but local.

The measured increase of localized wall shear stress with the severity of stenosis is in agreement with previous hemodynamic measurements of constricted arteries. Strong elevation of wall shear stress and oscillatory flow conditions in the region of constricted area have been linked to increasing stenosis in carotid models, which reinforces the general conclusion that geometric constriction increases mechanical loading on the vessel wall in the area (Basavaraja et al., 2017). Similarly, some numerical studies based on coronary parameters have demonstrated that the severity of stenosis, along with geometric configuration, has a material impact on clinically significant hemodynamic indices, which is in line with the tendencies found here with pressure drop and flow acceleration (Malota et al., 2018). The current findings also concur with lower-order mathematical investigations where the severity of stenosis is a major control parameter of pressure and velocity fields. Nonlinear change of flow disturbance has been reported to increase with the severity of the narrowing of stenosed arteries in parametric studies, a trend that has been consistent with the sudden increase in the pressure loss and throat velocity achieved in the present model (Ali et al., 2021). Geometric sensitivity has also been highlighted by theoretical work demonstrating that the shape and extent of stenosis have a material impact on the resultant flow structure, especially in how they impact local gradients and near-wall stresses (Asha & Srivastava, 2021).

Formulations based on Navier-Stokes have also shown that constriction of the vessel generates considerable spatial variations in the pressure and velocity fields, although at a significantly higher computational cost than reduced-order methods (Gafurjonovich & Ergash o‘g‘li, 2025). The results are also consistent, qualitatively, with more specific artery models, which incorporate more geometric or physical effects. The incorporation of post-stenotic dilatation in studies has demonstrated that the downstream flow fields can be further altered by non-simple constricted geometry, and therefore, the current findings can be considered a baseline against which more complex anatomical states can be analyzed (Dhange et al., 2022). Equally, non-Newtonian models incorporating thrombosis or catheter effects have shown that rheology and intraluminal devices can change the values and distributions of hemodynamic variables, implying that the simplified assumptions made in this case probably underestimate some facets of clinical complexity, rather than exaggerate them (Wajihah et al., 2022). Similar modeling studies with combined stenotic and aneurysmal geometries have also demonstrated that interacting structural irregularities can re-pattern pressure and velocity fields not predicted by single-lesion models (Damno et al., 2021). Computational studies of coronary abnormalities have reinforced the perception that a change in normal vessel geometry is all that is required to cause quantifiable changes in flow organization, which is not inconsistent with the current focus on localized constriction as a key hemodynamic determinant (Hoque et al., 2018).

These findings have a number of implications for biomathematical and cardiovascular modeling. First, they demonstrate that even an analytically simplified model is able to restore the key nonlinear dependence of coronary hemodynamics on the severity of stenosis. Second, the difference in small variations in mean quantities and a strong increase in local peak values implies that the severity should not be evaluated using bulk measures of flow only. Third, the model offers a systematic framework to perform parametric exploration and is applicable to the sensitivity

analysis, benchmarking of methods, and hypothesis testing prior to switching to more realistic simulations.

These findings are to be interpreted within the assumptions of the model. The assumptions made were that blood was a Newtonian fluid, the wall of the arteries was rigid, the vessel was straight and axisymmetric, and the flow was steady and laminar. These selections were suitable in isolating the effect of the degree of stenosis, but fail to reflect pulsatility, compliance of the walls, curvature, branching, and individual anatomy. Moreover, downstream recirculation, turbulence transition, and biological remodeling processes, which could arise in severe disease, are not included in the model.

The current structure can be expanded in a number of ways in the future. It would be a logical extension to include pulsatile inflow and non-Newtonian rheology in order to evaluate the effects of time dependence and shear-dependent viscosity in altering the observed trends. Imaging-based geometries of the patients could be adopted to increase anatomical realism as well. Another possible extension is to combine reduced-order flow models with uncertainty quantification or multiscale models to increase robustness and preserve computational efficiency. This would aid in filling the gap between mathematically analyzable coronary hemodynamics and clinically applicable ones.

5. Conclusion

The focal coronary stenosis leads to a great change in the hemodynamic behavior since it causes increase in resistance to flow and concentration of mechanical stress on the constricted area. The current study revealed that as the severity of stenosis increased, the pressure gradients, overall pressure drop, wall shear stress, and the significant acceleration of flow in the stenosis throat increased. These were highly nonlinear effects, such that severe constriction had disproportionately large effects compared to mild and moderate ones. The reduced-order model employed here gives a straightforward and computationally efficient approach to investigate the arterial geometry-hemodynamic response relationship. Despite the simplifying assumptions that underlie the model, it reflects the prevailing trends that are related to progressive lumen narrowing and provides a convenient starting point for a parametric study. The findings confirm the perception that the severity of stenosis is a key factor in the coronary flow disruption under the model assumptions. The framework can be extended to future applications with pulsatile flow, non-Newtonian rheology, vessel compliance, and geometries that are patient-specific, and thereby enhance the physiological scope of mathematical studies of coronary hemodynamics.

References

Ahmad, Y., Götberg, M., Cook, C., Howard, J. P., Malik, I., Mikhail, G., ... & Sen, S. (2018). Coronary hemodynamics in patients with severe aortic stenosis and coronary artery disease undergoing transcatheter aortic valve replacement: implications for clinical indices of coronary stenosis severity. JACC: Cardiovascular Interventions, 11(20), 1153-1163.

Akhtar, S., Hussain, Z., Nadeem, S., Najjar, I. R., & Sadoun, A. M. (2023). CFD analysis on blood flow inside a symmetric stenosed artery: Physiology of a coronary artery disease. Science progress, 106(2), 00368504231180092.

Ali, A., Hussain, M., Anwar, M. S., & Inc, M. (2021). Mathematical modeling and parametric investigation of blood flow through a stenosis artery. Applied Mathematics and Mechanics, 42(11), 1153-1163.

Androshchuk, V., Patterson, T., & Redwood, S. R. (2023). Management of coronary artery disease in patients with aortic stenosis. Heart, 109(4), 1153-1163.

Asha, K. N., & Srivastava, N. (2021, October). Geometry of stenosis and its effects on the blood flow through an artery-A theoretical study. In AIP conference proceedings (Vol. 2375, No. 1, p. 030006). AIP Publishing LLC.

Basavaraja, P., Surendran, A., Gupta, A., Saba, L., Laird, J. R., Nicolaides, A., ... & Suri, J. S. (2017). Wall shear stress and oscillatory shear index distribution in carotid artery with varying degree of stenosis: a hemodynamic

study. journal of mechanics in medicine and biology, 17(02), 1750037.

Carvalho, V., Pinho, D., Lima, R. A., Teixeira, J. C., & Teixeira, S. (2021). Blood flow modeling in coronary arteries: A review. Fluids, 6(2), 53.

Damno, M. M., Agrawal, B., Kumar, S., & Das, G. (2021). A mathematical model for the blood flow in a modeled artery with a stenosis and an aneurysm. International Journal of Statistics and Applied Mathematics, 6(2), 1153-1163.

Delestri, L. F. U., Al Abed, A., Dokos, S., Mokhtarudin, M. J. M., Kok, F. N., Bressloff, N. W., ... & Bakir, A. A. (2025). Modelling of cardiac biventricular electromechanics with coronary blood flow to investigate the influence of coronary arterial motion on coronary haemodynamic. Computer Methods and Programs in Biomedicine, 267, 108800.

Dhange, M., Sankad, G., Safdar, R., Jamshed, W., Eid, M. R., Bhujakkanavar, U., ... & Chouikh, R. (2022). A mathematical model of blood flow in a stenosed artery with post-stenotic dilatation and a forced field. Plos one, 17(7), e0266727.

Gafurjonovich, M. V., & Ergash o'g'li, O. P. (2025). THE 3D MODELING OF BLOOD FLOW AND PRESSURE BASED ON THE NAVIER–STOKES EQUATIONS. Global Science Review, 11(1), 1153-1163.

Hoque, K. E., Sawall, S., Hoque, M. A., & Hossain, M. S. (2018). Hemodynamic simulations to identify irregularities in coronary artery models. Journal of Advances in Mathematics and Computer Science, 28(5), 1153-1163.

Jewell, E., Farhat, C., & Soize, C. (2025). A tractable nonparametric probabilistic approach for modeling and quantifying model-form uncertainty in cfd computations with turbulence modeling. In AIAA SciTech 2025 Forum (p. 2301).

Kanokjaruvijit, K., Donprai-On, T., Phanthura, N., Noidet, P., & Siripokharattana, J. (2017). Wall shear stress and velocity distributions in different types of stenotic bifurcations. Journal of Mechanical Science and Technology, 31(5), 1153-1163.

Malakar, A. K., Choudhury, D., Halder, B., Paul, P., Uddin, A., & Chakraborty, S. (2019). A review on coronary artery disease, its risk factors, and therapeutics. Journal of cellular physiology, 234(10), 11153-11633.

Malota, Z., Glowacki, J., Sadowski, W., & Kostur, M. (2018). Numerical analysis of the impact of flow rate, heart rate, vessel geometry, and degree of stenosis on coronary hemodynamic indices. BMC cardiovascular disorders, 18(1), 132.

Sandeep, S., & Shine, S. R. (2021). Effect of stenosis and dilatation on the hemodynamic parameters associated with left coronary artery. Computer methods and programs in biomedicine, 204, 106052.

Shao, C., Wang, J., Tian, J., & Tang, Y. D. (2020). Coronary artery disease: from mechanism to clinical practice. Coronary Artery Disease: Therapeutics and Drug Discovery, 1153-1163.

Song, Z., Zhu, P., Yang, L., Liu, Z., Li, H., & Zhu, W. (2022). Study on the radial sectional velocity distribution and wall shear stress associated with carotid artery stenosis. Physics of Fluids, 34(5).

van Brussel, P. M., van de Hoef, T. P., de Winter, R. J., Vogt, L., & van den Born, B. J. (2017). Hemodynamic measurements for the selection

of patients with renal artery stenosis: a systematic review. JACC: Cardiovascular Interventions, 10(10), 1153-1163.

Wajihah, S. A., Sankar, D. S., & Nagar, A. K. (2022). Effects of catheter, stenosis and thrombosis in non-Newtonian blood flow through narrow arteries with clinical applications: a mathematical model. International Journal of Applied and Computational Mathematics, 8(3), 136.