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A Computational Study of Hemodynamic Resistance in a Symmetrical Arterial Stenosis Under Magnetic Influence

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Abstract: This study presents a comprehensive computational analysis of blood flow resistance in a symmetrically stenosed artery under the influence of an external magnetic field—a scenario of growing clinical relevance due to the rising interest in magnetically assisted therapies. Modeling blood as a viscous, incompressible, and electrically conducting fluid with radially variable viscosity, the problem incorporates both geometric non-uniformity and magnetohydrodynamic effects through the inclusion of a transverse magnetic field. The governing equations, derived in cylindrical coordinates and non-dimensionalized using characteristic parameters, are solved using the Finite Difference Method, offering robust insights into velocity distributions and flow resistance. The results reveal that stenosis height alone significantly elevates resistance, while the presence of a magnetic field amplifies this effect nonlinearly, with higher field strengths causing pronounced suppression of axial velocity. Velocity profiles flatten and shear rates near the arterial wall intensify with increasing stenosis and magnetic influence, underscoring the synergistic impact of these parameters. This work not only advances our understanding of MHD- modulated hemodynamics but also provides a theoretical foundation for future biomedical applications such as targeted drug delivery, vascular diagnostics, and therapeutic flow control.

Keywords: Hemodynamic resistance, Symmetrical stenosis, Magnetohydrodynamics (MHD), Blood flow modeling, Lorentz force, Finite Difference Method (FDM).

I. Introduction

The study of hemodynamic alterations in stenosed arteries has been a subject of significant interest in cardiovascular biomechanics, especially under the influence of external forces such as magnetic fields. Maurya and Kumar [1] recently modeled coronary artery stenosis with an emphasis on magnetic field-induced changes in flow resistance, revealing the potential for magnetic modulation in therapeutic interventions. In a related effort, Karim

[2] explored the impact of magnetic fields on composite arterial geometries, emphasizing the importance of non-uniform vessel anatomy in regulating flow characteristics. Ali et al.

[3] presented a parametric mathematical model that quantified the effects of arterial narrowing on flow dynamics, reinforcing the complex interplay between geometry and hemodynamic parameters. In parallel, Hewlin Jr. et al. [4] conducted a computational assessment of magnetic nanoparticle targeting in carotid bifurcation arteries, highlighting the significance of unsteady flow effects under magnetically active environments. Asha and Srivastava [5], through theoretical investigations, illustrated how variations in stenosis geometry markedly influence pressure and velocity distributions within the artery.

A computational simulation by Khairuzzaman [6] investigated different stenosis shapes, emphasizing their distinct effects on wall shear stress and velocity gradients. Priyadharshini and Ponalagusamy [7] further expanded this understanding by considering radially varying viscosity in pulsatile flow under magnetic field exposure, thereby integrating non-Newtonian blood rheology with magnetohydrodynamic (MHD) modeling. Building on these foundations, Oyelami et al. [8] analyzed magneto-radiative effects in symmetric stenotic arteries, illustrating the temperature-dependence of flow resistance. Varshney et al. [9] offered one of the early numerical studies on magnetic field impacts in arteries with multiple stenoses, affirming the resistive nature of magnetic forces. A more recent computational fluid dynamics (CFD) study by Chen et al. [10] highlighted the influence of mild stenosis morphology in coronary arteries, indicating that even subtle changes in shape could lead to major shifts in pressure gradients. Kumar and Shah [11] supported this with a hemodynamic simulation framework, showcasing the practical utility of CFD in predicting flow behavior in stenotic vessels. Zaman et al. [12] contributed a numerical model for unsteady MHD blood flow in overlapping stenoses, capturing transient behaviors often overlooked in steady-state models. Singh [13] examined how stenosis shape, under a magnetic field, modulates arterial rheology, particularly when considering deformable walls. Abdelsalam and Bhatti [14] investigated nanoparticle dynamics in stenosed arteries, indicating synergistic interactions between flow resistance and particle motion in the presence of field forces. Wahab et al. [15] provided further validation through elliptic artery simulations, confirming the utility of CFD in replicating physiological flow disruptions. Ponalagusamy and Priyadharshini [16] expanded on two- fluid micropolar-Newtonian models, incorporating core-plasma interactions under variable magnetic field conditions. Alshare et al. [17] performed comprehensive MHD simulations of non-Newtonian blood through stenosed geometries, highlighting deviations in shear profiles under external fields.

Magnetic nanoparticle dynamics have also been explored by Hewlin Jr. and Tindall [18], who examined transport efficiency in complex vascular networks such as the circle of Willis. Babatunde and Dada [19] focused on unsteady, tapered, and overlapping stenoses in a magnetic environment, reinforcing the dynamic nature of resistive forces. Abdollahzadeh Jamalabadi et al. [20] introduced heat transfer and biomagnetic interactions into the Carreau blood model, accounting for thermal feedback mechanisms

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during flow. A broader theoretical perspective is offered by Tu et al. [21], whose foundational text on computational hemodynamics provides the underpinning for modern simulations across physiological domains. Hossain [22] proposed control mechanisms for stenosed blood flow using power-law fluids, offering new avenues for non-invasive intervention techniques. Zuberi et al. [23] demonstrated the enhancement of flow stability using copper and alumina nanoparticles in stenosed arteries, showing the efficacy of nanoparticle-assisted flow rectification. Further, Zuberi et al. [24] analyzed the role of viscous dissipation and metallic nanoparticles (gold and silver) in altering flow resistance, unveiling new mechanisms of energy transfer in hemodynamic systems. Lastly, Zainal et al. [25] investigated mixed convection in hybrid nanofluid systems near a shrinking plate, providing insights transferable to the arterial microenvironment, especially where boundary-layer interactions dominate.

These diverse investigations collectively underscore the need for a comprehensive computational study focusing specifically on the hemodynamic resistance in a symmetrically stenosed artery under magnetic influence, which this work endeavors to present. Through the integration of magnetic field modeling, variable viscosity, and geometrically induced resistance, the present study aims to enhance our understanding of flow mechanics within physiologically realistic and clinically relevant arterial structures. A key novelty of this research lies in the implementation of the Finite Difference Method (FDM) to numerically solve the governing nonlinear differential equations with spatially varying coefficients arising from both radial viscosity variation and complex arterial geometry. The use of FDM provides a robust, stable, and computationally efficient framework that captures the intricate interplay between magnetic damping and geometric constriction, making it particularly well-suited for analyzing magnetohydrodynamic blood flow in stenosed vessels. This methodological advancement strengthens the model's capability to simulate realistic physiological conditions and offers valuable insights for biomedical applications involving magnetic field-based interventions.

Mathematical Modeling

In this section, we develop a mathematical framework to describe the steady, incompressible, laminar flow of blood through an axially symmetric artery containing a mild, symmetric stenosis. Blood is treated as a viscous, electrically conducting fluid subjected to a transverse external magnetic field. The governing model accounts for the variation in viscosity across the radial coordinate and incorporates the influence of magnetic body forces through the Lorentz force term.

Geometric Configuration

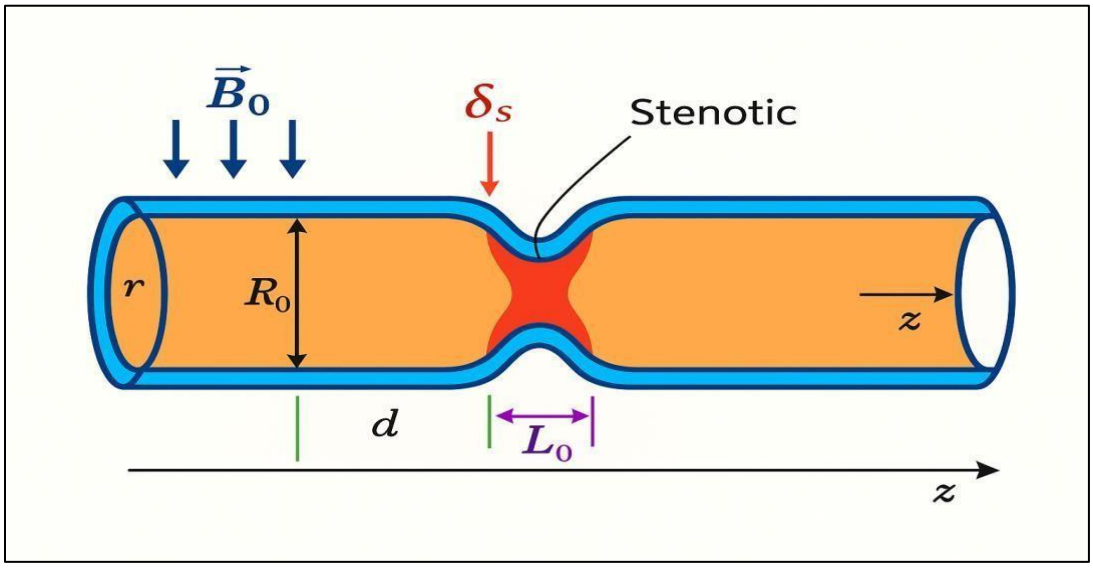

Fig. 1: Geometry of symmetrical stenosis in an artery under the effect of the magnetic field

We consider an artery modeled as a rigid cylindrical tube with a localized symmetric stenosis situated along the axial direction, as shown in Figure 1. The radius of the arterial wall varies axially and is defined piecewise as:

where

- R_0: radius of the normal (unstenosed) artery,
- δs: maximum stenosis height,
- L_0: axial length of the stenotic region,
- d: axial location of stenosis center,
- z: axial coordinate.

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Solution Strategy

The governing equations developed in Section 2 describe the axial velocity distribution of blood flow through a stenosed artery under the influence of a transverse magnetic field. The primary equation is a second-order, nonlinear, ordinary differential equation with spatially variable coefficients due to radial viscosity variation and geometric non- uniformity introduced by the stenosis shape. To solve this boundary value problem numerically, we employed the Finite Difference Method (FDM) owing to its simplicity, stability, and effectiveness in handling one-dimensional spatial discretization in cylindrical coordinates. The artery's radial domain [0, (z)] is discretized into a uniform mesh comprising N nodes, where central difference schemes are applied to approximate the second-order derivative of the velocity profile.

0 at the

centerline $r = 0$.

The resulting system of algebraic equations is solved iteratively using matrix operations in MATLAB, with appropriate convergence criteria based on residual norms. Due to the dependence of the viscosity and geometric terms on the radial coordinate, all variable coefficients are evaluated at each discrete point, and the stiffness of the system is managed through implicit formulation where necessary. Once the velocity profile u(r)u(r)u(r) is obtained at various axial locations, it is used to compute the volumetric flow rate and subsequently the resistance to flow using numerical integration technique. These computations are automated and visualized within the MATLAB environment to ensure consistency and reproducibility. The results, presented in the form of parametric plots for velocity profiles and resistance variation, are analyzed and discussed in detail in Section 4.

II. Results and Discussion

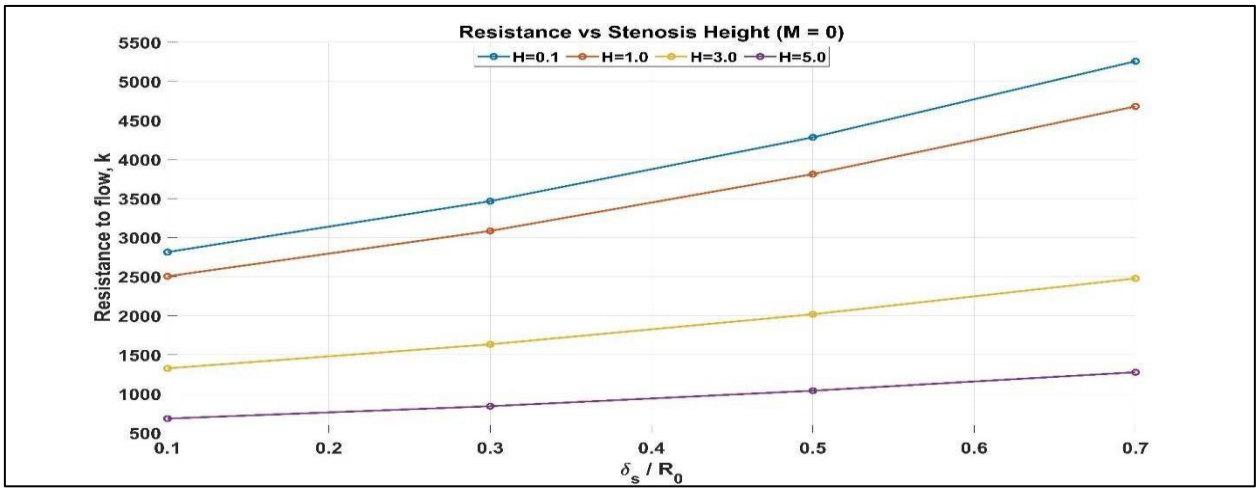

Fig. 2: Resistance vs Stenosis Height for $M = 0$.

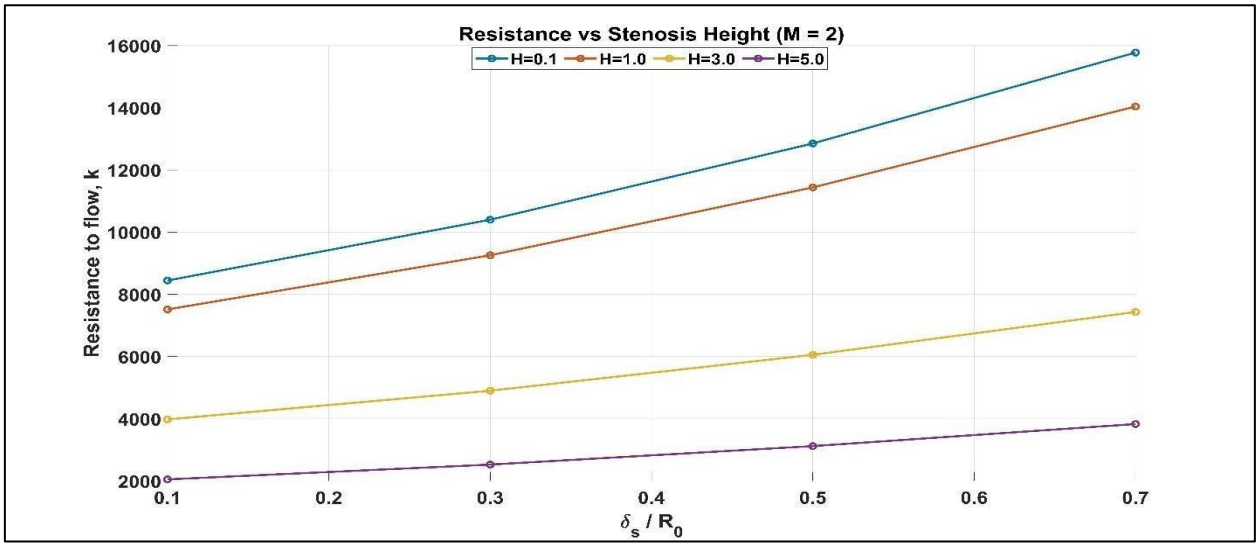

Fig. 3: Resistance vs Stenosis Height for $M = 2$.

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Figure 2 illustrates the relationship between non-dimensional hemodynamic resistance and stenosis height in the absence of a magnetic field, emphasizing the purely geometric and viscous influences on blood flow. As the stenosis height increases, the resistance to flow rises consistently, indicating that the narrowing of the arterial passage significantly obstructs fluid movement. This trend is physically intuitive, as a more pronounced stenosis reduces the effective cross-sectional area available for flow, thereby enhancing the velocity gradient and increasing viscous shear forces. With no magnetic field applied, the Lorentz force is absent, and thus the resistance is solely attributed to the constrictive geometry and the viscosity of the blood. This result underscores the critical impact of stenosis severity on hemodynamic parameters, even without magnetic modulation. It reflects a fundamental characteristic of arterial flow—higher degrees of stenosis inevitably lead to elevated pressure drops and greater flow resistance, which can compromise circulatory efficiency and elevate cardiovascular risk.

Figure 3 depicts the variation of non-dimensional hemodynamic resistance with stenosis height in the presence of a magnetic field, representing a case where magnetic effects are active during blood flow. Compared to the zero-field scenario, the resistance curve in this figure exhibits a steeper rise as the stenosis height increases, indicating that the magnetic field amplifies the opposition to flow. This enhancement in resistance is primarily due to the Lorentz force generated by the interaction between the induced electric currents in the conducting blood and the applied transverse magnetic field. The Lorentz force acts as a damping mechanism, further suppressing axial velocity and increasing the overall resistance beyond what is caused by geometry alone. Consequently, the combined effects of reduced cross-sectional area from the stenosis and the magnetic braking force significantly elevate the hemodynamic resistance. This behavior demonstrates the important role of magnetohydrodynamic effects in modulating blood flow and suggests that external magnetic fields could be strategically used to influence vascular resistance in therapeutic or diagnostic contexts.

Figure 4 presents the variation of non-dimensional hemodynamic resistance with stenosis height for a stronger magnetic field compared to the previous case, highlighting the compounded effect of increasing magnetic field strength on blood flow resistance. As the stenosis height increases, the resistance shows a more pronounced escalation than observed in Figures 2 and 3, confirming that both geometric constriction and magnetic influence synergistically contribute to flow opposition. The presence of a stronger transverse magnetic field intensifies the Lorentz force acting against the motion of the electrically conducting blood, further reducing axial velocity and increasing pressure losses across the stenosed segment. This results in significantly higher resistance values for the same stenosis height when compared to lower magnetic field strengths. The trend underlines the nonlinear amplification of resistance with respect to both magnetic field intensity and geometric narrowing, suggesting that in clinical scenarios where magnetic fields are applied, even mild stenoses may lead to substantial hemodynamic alterations. This reinforces the importance of accounting for magnetic field effects when assessing blood flow in magnetically influenced biomedical applications.

Figure 5 displays the axial velocity profiles of blood flow across the radial coordinate for different stenosis heights in the absence of a magnetic field. The profiles exhibit a parabolic nature typical of laminar flow, with maximum velocity occurring at the centerline and gradually decreasing to zero at the arterial wall due to the no-slip condition. As the stenosis height increases, the peak velocity decreases, and the velocity gradient near the wall becomes steeper. This reduction in central velocity is a direct consequence of increased flow resistance caused by the narrowing of the artery, which restricts the available flow area and enhances viscous effects. The steeper gradients indicate higher shear stresses, which can have physiological implications such as increased risk of endothelial damage. The results clearly demonstrate how geometric constriction alone, even without magnetic influence, significantly alters the velocity distribution within the artery, affecting overall hemodynamic performance.

Figure 6 shows the axial velocity profiles of blood flow for varying stenosis heights in the presence of a magnetic field. Compared to the non-magnetic case, the velocity profiles here are noticeably flattened, indicating the suppressive effect of the magnetic field on flow velocity due to the action of the Lorentz force. As stenosis height increases, the peak velocity further diminishes and the flow becomes more restricted across the entire radial domain. The presence of the magnetic field adds an additional resistive force that acts against the motion of the conducting blood, leading to reduced axial momentum and more pronounced velocity suppression near the centerline. The velocity gradient near the wall remains steep, reflecting high shear rates, especially in the stenosed region. These profiles illustrate how the combined influence of stenosis and magnetic field significantly modifies the internal velocity structure, reducing the overall transport efficiency and potentially impacting shear-sensitive physiological processes within the arterial wall.

Figure 7 illustrates the axial velocity profiles for different stenosis heights under the influence of a stronger magnetic field compared to the previous case. The velocity suppression is more significant, with the profiles becoming increasingly flattened as both stenosis height and magnetic field intensity increase. The peak velocity at the centerline reduces markedly, and the flow near the wall remains sharply tapered due to the no-slip boundarycondition. This behavior is attributed to the enhanced Lorentz force, which exerts a greater resistive influence on the motion of the electrically conducting blood. As the stenosis height rises, the available flow area decreases, and the combined effect of geometric constriction and strong magnetic damping further impedes the flow. The result is a substantial decrease in overall velocity magnitude across the arterial cross-section. These findings highlight that in the presence of a strong magnetic field, even moderate stenoses can lead to significantly altered flow profiles, emphasizing the critical role of magnetohydrodynamic effects in vascular flow dynamics.

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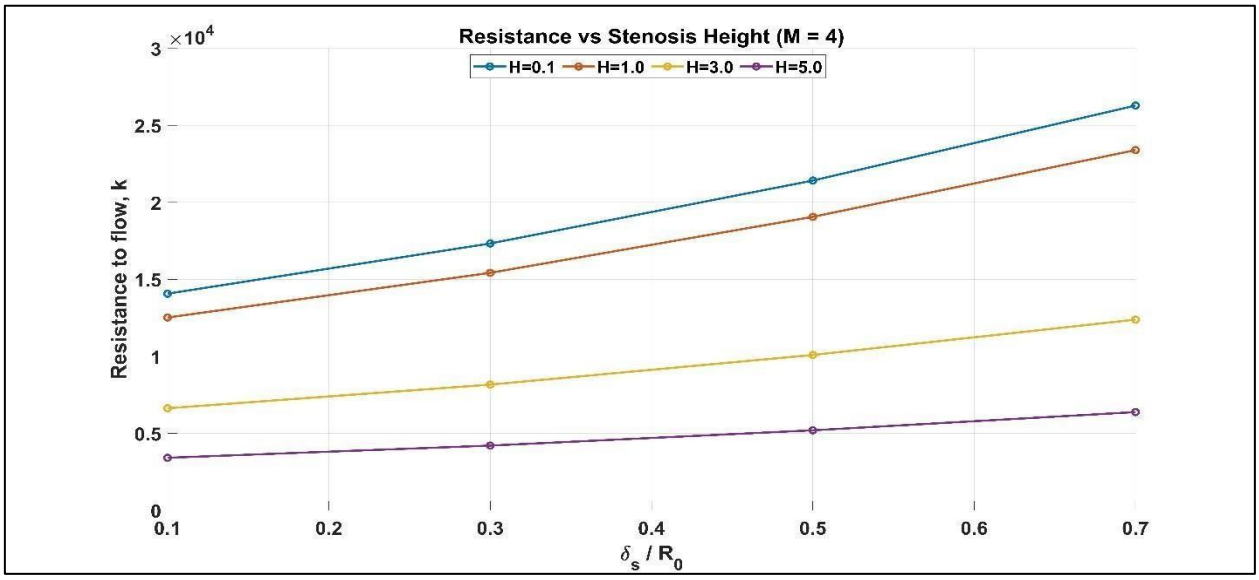

Fig. 4: Resistance vs Stenosis Height for $M = 4$.

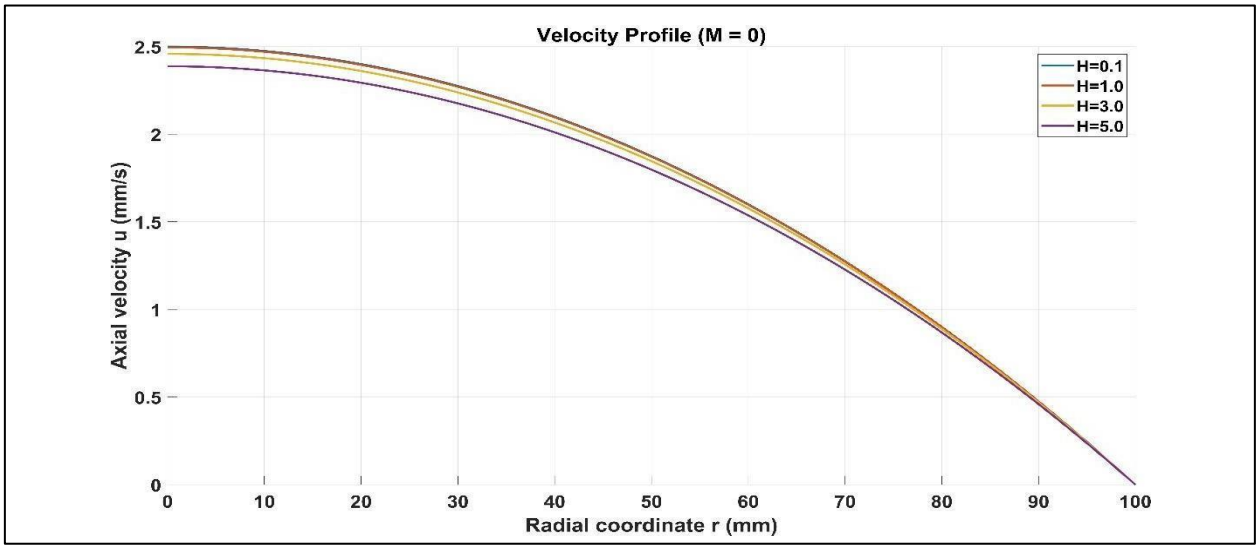

Fig. 5: Velocity profiles for $M = 0$.

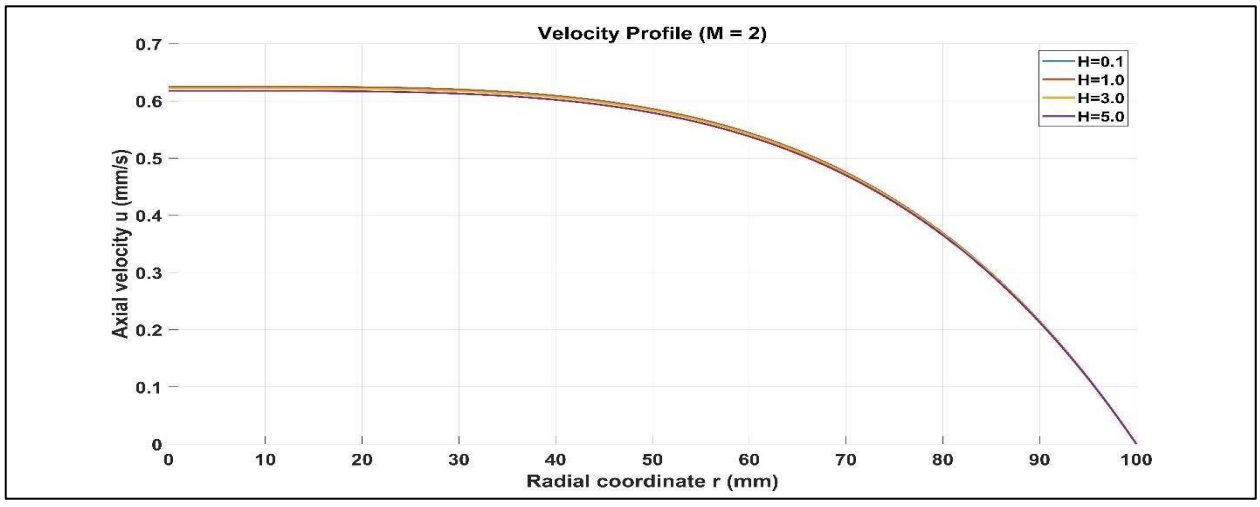

Fig. 6: Velocity profiles for $M = 2$.

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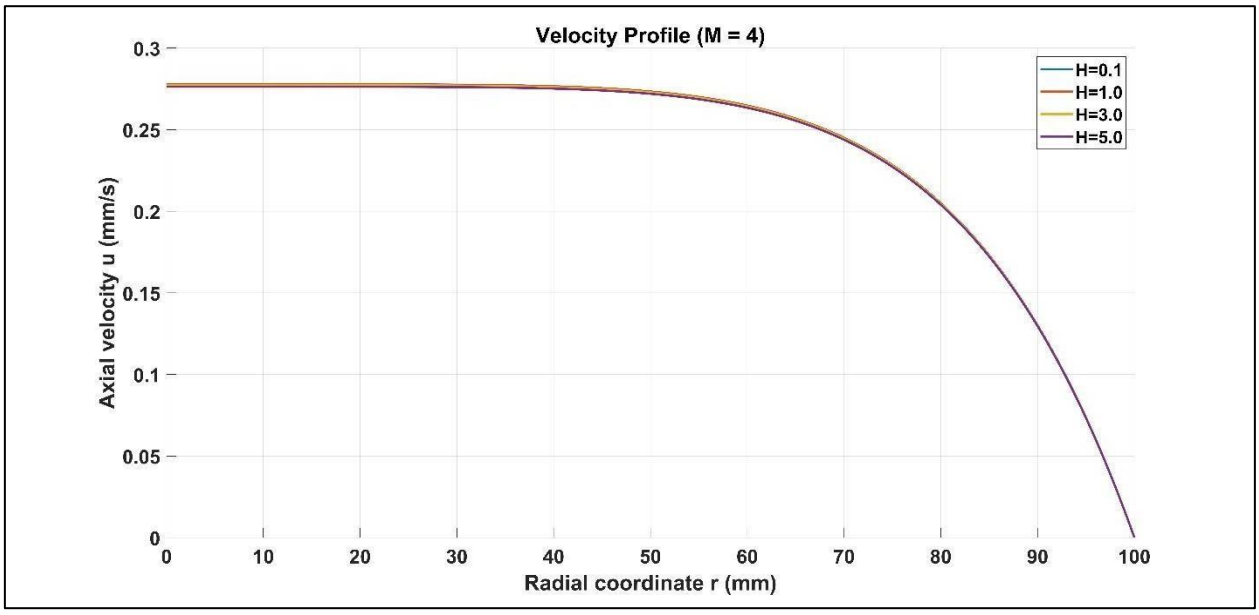

Fig. 7: Velocity profiles for $M = 4$.

III. Conclusions

This study investigates the hemodynamic resistance in a blood vessel affected by symmetrical arterial stenosis under the influence of an external magnetic field. The flow is modeled as steady, incompressible, and laminar, assuming the artery to be a rigid cylindrical tube with a localized symmetric stenosis. Blood is considered an electrically conducting, viscous fluid with variable viscosity dependent on the radial position, capturing the effect of hematocrit variation. A transverse magnetic field is applied, introducing a Lorentz force that interacts with the flow. The governing equations are derived in cylindrical coordinates and non-dimensionalized using characteristic scales, incorporating the Hartmann number to represent magnetic field strength. The resulting boundary value problem, characterized by a second-order nonlinear differential equation with spatially varying coefficients, is solved numerically using the Finite Difference Method (FDM). The axial velocity profiles and flow resistance are computed across varying degrees of stenosis and magnetic field strengths. The conclusions drawn from the numerical analysis are as follows:

- Resistance to flow increases significantly with stenosis height, even in the absence of a magnetic field, due to reduced cross-sectional area and enhanced viscous effects.
- The presence of a magnetic field amplifies resistance further, as the Lorentz force suppresses the axial velocity and increases energy dissipation.
- A stronger magnetic field results in a steeper increase in resistance for the same stenosis height, demonstrating nonlinear interaction between magnetic effects and geometric narrowing.
- Velocity profiles become increasingly flattened and suppressed at the centerline with higher stenosis and magnetic field strength, indicating reduced transport efficiency and elevated shear near arterial walls.
- Even mild stenoses can cause substantial hemodynamic alterations in the presence of strong magnetic fields, highlighting the importance of magnetohydrodynamic considerations in medical diagnostics and targeted therapy applications.

Future Scope

The present study opens several avenues for future exploration in the field of magnetohydrodynamic blood flow through stenosed arteries. One promising direction involves extending the model to incorporate unsteady or pulsatile flow conditions, which more accurately represent the physiological nature of blood circulation. Additionally, integrating non-Newtonian rheological models, such as the Carreau or Casson fluid models, would provide a more realistic depiction of blood behavior, especially under varying shear rates. The influence of thermal effects and nanoparticle-based drug delivery systems in the presence of magnetic fields could also be investigated to enhance the model's biomedical relevance. Furthermore, analyzing asymmetric or multiple stenoses and accounting for arterial wall elasticity would allow for a more comprehensive understanding of real-life vascular dynamics. Coupling this model with patient-specific geometries obtained from medical imaging could aid in the development of predictive tools for clinical diagnosis and personalized treatment planning.

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