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Research Article

Optimizing Cardiovascular Performance Following Myocardial Infarction: The Significance of Nitroglycerin in Regulating Blood Flow

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ABSTRACT

Background & Objectives: Nitroglycerin, a potent vasodilator, is crucial in managing blood flow dynamics in patients recovering from Myocardial Infarction (MI). This research aims to comprehensively review the role of nitroglycerin in regulating blood flow and enhancing cardiovascular performance post-MI.

Materials and Methods: Blood flow was modeled as a Herschek-Bulkley fluid. The study derives expressions for various blood flow characteristics, including pressure, viscosity, resistance to flow, and wall shear stress. Pharmacological mechanisms of nitroglycerin, its effects on vascular function, and its role in mitigating ischemic events are explored.

Results: The findings indicated that blood flow increases with the height of stenosis. Nitroglycerin effectively dilates blood vessels, reducing peripheral vascular resistance and easing blood flow. Clinical applications, including administration routes, dosage regimens, and potential adverse effects, are discussed.

Conclusion: Nitroglycerin plays a pivotal role in managing blood flow post-MI by dilating blood vessels and reducing peripheral vascular resistance. This mechanism contributes to improved cardiovascular outcomes and symptom relief in patients recovering from MI. Understanding its multifaceted role is crucial for optimizing treatment strategies and enhancing patient care.

Keywords: Herschek-Bulkley fluid, Myocardial infarction, Nitroglycerin, Radially non-symmetric, Resistance to flow.

INTRODUCTION

Myocardial Infarction (MI) commonly known as a heart attack, is a critical medical condition characterized by the sudden interruption of blood flow to a part of heart muscle, leading to tissue damage or cell death due to lack of oxygen and nutrients [1]. This interruption typically occurs when a coronary artery, responsible for supplying oxygen-rich blood to heart, becomes blocked or narrowed due to the formation of plaque or a blood clot [2]. The impact of myocardial infarction on cardiovascular health is profound and multifaceted. Firstly, Myocardial Infarction (MI) can result in significant damage to heart muscle, leading to impaired cardiac function and potentially life-threatening complications such as heart failure, arrhythmias, or cardiac arrest [3]. Additionally, the occurrence of a heart attack often serves as a warning sign of underlying cardiovascular disease, indicating the presence of risk factors such as hypertension, high cholesterol, diabetes, or smoking [4]. Furthermore, myocardial infarction can have far-reaching consequences on the overall health and well-being of affected individuals, impacting their quality of life, physical capabilities, and emotional health [5]. Post-MI rehabilitation and lifestyle modifications are essential components of managing the condition and reducing the risk of future cardiovascular events [6]. Myocardial infarction represents a significant public health concern due to its high morbidity and mortality rates and its profound implications for cardiovascular health and overall well-being [7]. Effective prevention, early recognition, and prompt treatment are crucial in mitigating the impact of heart attacks and improving outcomes for affected individuals. Atherosclerosis, the leading cause of heart attacks and strokes, results from the progressive narrowing and hardening of arteries due to lipid accumulation in the arterial wall [8]. This build-up of deposits, known as plaques, can protrude into the arterial lumen, restricting flow. When coronary artery is damaged, it can lead to heart attack [9].

Additionally, there are alterations in the forces exerted by blood flow on the plaque surface, contributing to disease progression [10]. Stenosis, depicted in [Figure 1] refers to the abnormal narrowing of a blood vessel, which is often a consequence of atherosclerosis. The insightful studies conducted by [11] have highlighted the physiological significance of variations in flow and stress with distance. Theoretical investigations by [12] have provided insights into velocity, pressure, shear stress, and separation phenomena in specific diseased conditions. Building upon this work, a series of papers [13-15] have explored the effects of blood flow on the cardiovascular system, considering blood behavior as that of a Newtonian fluid. It is acknowledged that blood flow does not always exhibit Newtonian behavior, particularly at low shear rates, as it is a suspension of cells [16]. Studies by [17] have demonstrated that blood flow in small-diameter tubes (less than 0.2 mm) and at shear rates below 20 (1/sec) can be modeled using a power-law fluid model. Despite these advancements, the previously discussed models by [18] have not addressed radially non-symmetric stenosis. A mathematical model has been developed to study blood flow through radially non-symmetric stenosis, featuring an improved generalized geometry of multiple stenosis spaced equidistantly [19]. Simplified graphical analyses have been performed for a single loop of stenosis with varying degrees of

depression at different points [20]. Myocardial Infarction has significant implications for cardiovascular health, often resulting in complications such as heart failure, arrhythmias, and even death if not promptly managed. Effective management strategies are crucial in optimizing post-Myocardial Infarction outcomes, including reducing morbidity and mortality rates and improving overall quality of life for affected individuals [21]. Nitroglycerin, a vasodilator medication, plays a pivotal role in regulating blood flow and managing symptoms associated with Myocardial Infarction and other ischemic heart conditions [22]. By inducing vasodilation, nitroglycerin helps to dilate blood vessels, particularly coronary arteries, thereby improving myocardial perfusion and reducing myocardial oxygen demand. Understanding the pharmacological mechanisms of nitroglycerin is essential for its effective utilization in post- Myocardial Infarction care and ischemic heart disease management [23]. Nitroglycerin undergoes rapid metabolism in the body, leading to the release of nitric oxide, which serves as the primary mediator of its pharmacological effects. Nitric oxide activates guanylate cyclase, resulting in the formation of Cyclic Guanosine Monophosphate from guanosine triphosphate. Elevated Cyclic Guanosine Monophosphate levels lead to vasodilation and relaxation of vascular smooth muscle, contributing to decreased vascular resistance and improved blood flow [24].

The vasodilatory effects of nitroglycerin extend to coronary arteries, where it enhances coronary blood flow and myocardial oxygen supply. By reducing cardiac preload and afterload, nitroglycerin helps to alleviate symptoms of angina pectoris and improve left ventricular function in individuals recovering from Myocardial Infarction [25]. Its vasodilatory properties are particularly beneficial in the management of acute coronary syndromes, unstable angina, and during percutaneous coronary intervention (PCI) procedures [26].

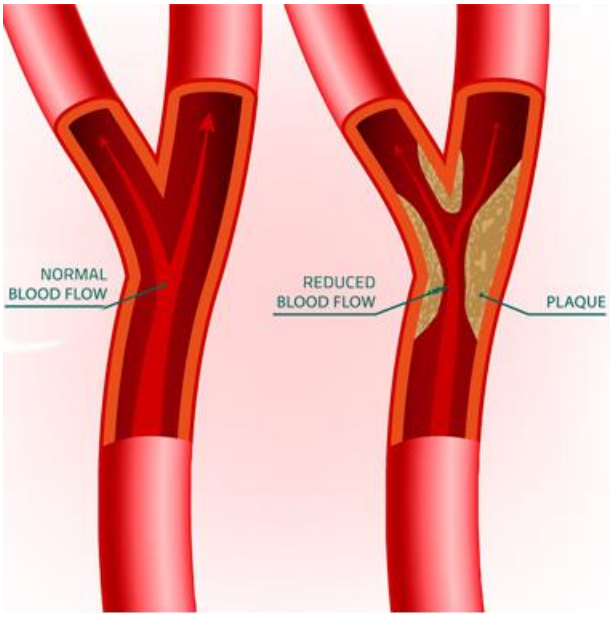

Figure 1: Normal artery and artery with multiple stenosis

Nitroglycerin plays a crucial role in alleviating symptoms of angina pectoris, such as chest pain and discomfort, by promoting vasodilation and increasing coronary blood flow. In the acute phase of Myocardial Infarction, nitroglycerin helps to reduce myocardial ischemia and improve myocardial perfusion, thereby minimizing tissue damage and preserving cardiac function [27]. Additionally, nitroglycerin's hemodynamic effects contribute to the stabilization of individuals with acute coronary syndromes and facilitate optimal outcomes following PCI procedures. In individuals recovering from Myocardial Infarction, nitroglycerin's vasodilatory properties help to reduce cardiac preload and afterload, thereby improving cardiac performance and reducing

myocardial workload [28]. By enhancing coronary blood flow and myocardial oxygen supply, nitroglycerin supports myocardial recovery and promotes left ventricular function. Its administration in the post-MI period is associated with improved clinical outcomes and reduced risk of recurrent ischemic events [29]. Dosage regimens and titration strategies should be tailored to individual patient requirements to achieve optimal therapeutic effects while minimizing adverse reactions. Adverse effects associated with nitroglycerin use include headache, hypotension, and reflex tachycardia, necessitating careful monitoring and dose adjustments as needed [30]. Emerging therapies and novel formulations of nitroglycerin hold promise for enhancing its efficacy and improving patient outcomes in ischemic heart disease management [31]. Future research efforts should focus on elucidating the potential synergistic effects of nitroglycerin with other cardiovascular medications and exploring innovative treatment modalities to address unmet clinical needs in post- Myocardial Infarction care [32]. Clinical investigation into personalized treatment approaches and multidisciplinary care models is warranted to optimize patient outcomes and advance cardiovascular care. Nitroglycerin remains a cornerstone therapy in optimizing cardiovascular performance following myocardial infarction, offering significant benefits in terms of symptom relief, myocardial protection, and clinical outcomes [33]. Its vasodilatory effects and hemodynamic properties play a crucial role in improving myocardial perfusion, reducing myocardial ischemia, and enhancing left ventricular function post- Myocardial Infarction [34]. Moving forward, individualized treatment approaches and collaborative care efforts are essential in maximizing the therapeutic potential of nitroglycerin and improving long-term cardiovascular health outcomes.

MATERIALS AND METHODS

In this current analysis, the assumption is made that stenosis occurs within arterial wall, exhibiting symmetry about axis but non-symmetry concerning radial coordinates and it is represented by [Figure 2]. Under this condition, radius of artery, denoted as R(z), and expressed as below:

$$\frac{R(z)}{R_0} = [1 - A(L_0^{m-1}(\alpha z - kd - (k-1))L_0]\text{-}$$
$$(\alpha z - kd - (k-1))L_0^{m-1}]. \quad (1)$$

Where, $A = \frac{\delta}{R_0 L_0^m} \frac{m^{m/(m-1)}}{(m-1)}$,

In the provided parameters:

- R(z) represents radius of artery with growth of stenosis at a specific axial position z.
- L denotes total length of the artery.
- d indicates is distance.
- k signifies stenoses number present in the artery.
- m is stenosis shape parameter in artery.
- δ is maximum thickness of stenosis.

$$z = \left[\frac{kd + (k-1)L_0 + L_0 / m^{1/(m-1)}}{\alpha}\right]. \quad (2)$$

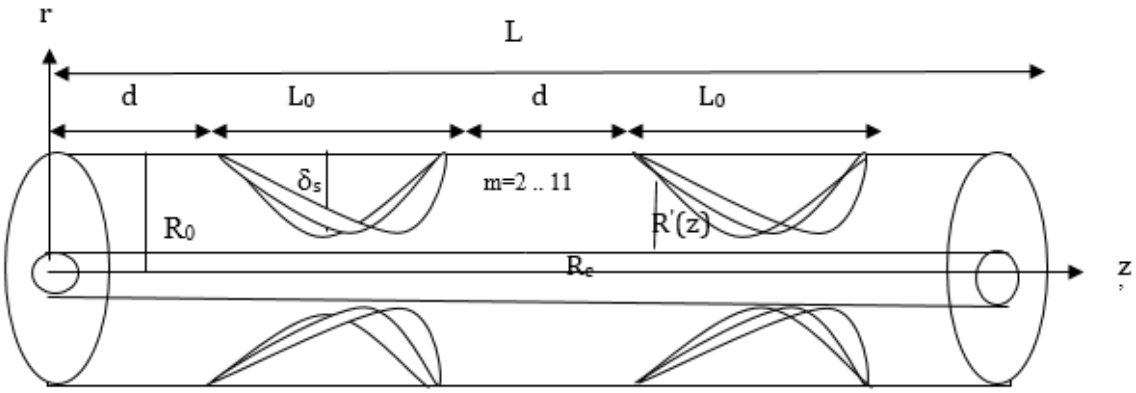

Figure 2: Geometry of Stenosed artery

Conservation Equation and boundary conditions:

In this analysis of blood flow through a stenosed artery, conservation equations and boundary conditions play a crucial role in describing behavior of fluid and its interaction with the arterial walls. These equations and conditions help establish a mathematical framework for studying the flow dynamics [35]. These equations govern flow of blood and its properties such as velocity, pressure, and shear stress distribution within artery. Boundary conditions are necessary to solve conservation set of equations and describe how the flow behaves at the boundaries of the system [36]. They specify the values of flow variables such as velocity, pressure, and shear stress at inlet and outlet boundaries, as well as any solid boundaries representing the arterial walls. Conservation equations and boundary conditions provide a fundamental basis for analyzing blood flow through stenosed arteries and understanding the effects of stenosis on flow characteristics [37]. The equation describes the movement of blood in a steady, fully developed manner through an artery, assuming it is laminar and incompressible. This equation considers the viscosity of blood changing along the radial direction within the artery [38].

This equation describes how changes in pressure and viscosity influence rate of change of velocity with respect to axial distance within the artery. It provides insights into dynamics of blood flow under varying conditions, including stenosis and changes in viscosity along the radial direction [39].

$$0=-(\partial P/\partial r)+(1/r)*(\partial(r\tau)/\partial z)$$
$$0=-(\partial P/\partial r) \quad (3)$$

In the given context, the coordinates (z,r) represent the spatial dimensions of the artery:

- z is measured along axis of the artery.
- r is measured normal (perpendicular) to axis of artery.

These coordinates allow for the description of positions and distances within the artery, enabling the analysis of blood flow characteristics and the effects of stenosis or variations in viscosity along both the axial and radial directions.

1. No-slip condition at the artery wall:
   - $\partial u/\partial r = 0$ at $r = 0$
   - This condition implies that there is no relative motion between the blood and the arterial wall at the boundary.
2. **Velocity at the artery wall:**
   - $u = 0$ at $r = R(z)$.
   - It indicates that the velocity of blood at the boundary of the artery wall is zero.
3. **Finite wall shear stress at the artery wall:**
   - Wall shear stress, (τ) is finite at r=0

This condition ensures that there is a finite shear stress exerted by the blood on the arterial wall.

4. **Pressure boundary conditions:**
   - $P = P_o$ at $z = 0$
   - $P = P_L$ at $z = L$ (4)
   - These conditions specify pressure in the inlet (z=0), outlet (z=L) of the artery, representing the upstream and downstream boundaries, respectively.

Solution of the problem: The Herschel-Bulkley fluid model describes stress-strain relation of certain fluids.

$$f(\tau) = \left(-\frac{du}{dr}\right) = \frac{1}{\mu}(\tau - \tau_0)^n, \qquad \tau \geq \tau_0$$

$$f(\tau) = \left(-\frac{du}{dr}\right) = 0, \qquad \tau \leq \tau_0 \qquad (5)$$

$$\text{where} \quad \tau = \left(-\frac{dp}{dz}\frac{r}{2}\right), \qquad \tau_0 = \left(-\frac{dp}{dz}\frac{R_c}{2}\right),$$

In the equation provided, various parameters are defined:

The equation describes stress-strain relationship of Herschel-Bulkley fluid. If $\tau \leq \tau o$, the fluid exhibits plug flow behaviour. Conversely, when τ exceeds the yield stress ($\tau \geq \tau o$), the fluid behaviour follows a power-law model. This distinction highlights the non-Newtonian behaviour of Herschel-Bulkley fluids under different shear conditions.

From equations (2) and (3), we obtain the following expression:

$$\left(\frac{du}{dr}\right) = -\left(\frac{p}{2\mu}\right)^{1/n}\left[(r - R_c)^{1/n}\right], \qquad (6)$$

Flow of flux is obtained as,

$$Q=\int_0^K 2\,p\,u\,r\;dr = p\int_0^K r^2\frac{du}{dr}dr \qquad (7)$$

$$Q=\frac{\pi}{2}\left(\frac{P}{2\mu}\right)^{1/n}\frac{R^{(3+\frac{1}{n})}}{(1+\frac{1}{n})}f(y), \qquad (8)$$

where $f(y)=[2(Rc/R)^{1+1/n} -[4/(1/n+2)*(1-(Rc/R)^{1+1/n})] + (3/(2/n+2))*(1/2n+3)+ (1-2Rc/R)^{3+1/n} -(1-Rc/2R)^{1+1/n}]$

$$P=\left(-\frac{dp}{dz}\right)=\frac{2\mu}{R^{(1+3n)}}\left(\frac{2Q}{\pi f(\bar{y})}(1+\frac{1}{n})\right)^n \qquad (9)$$

$$P=P_L-P_0=\frac{2\mu}{\pi R_0^{1+3n}}(2Q(1+(1/n)^n\int_0^L\frac{dz}{\frac{R(z)}{R_0}^{1+3n}}\frac{dz}{f(y)^n} \qquad (10)$$

Coefficient λ, defining resistance to flow, is determined as follows:

$$\lambda=(P_L-P_0)/Q. \qquad (11)$$

$$\lambda_0=\frac{2\mu}{R_0^{1+3n}}\left(\frac{2Q(1+\frac{1}{n})}{\pi}\right)^n(M) \qquad (12)$$

$$M=\left(\int_0^d\frac{dz}{(f_0)^n}+\int_d^{d+L_0}\frac{dz}{\left(\frac{R(z)}{R_0}\right)^{1+3n}(f(\bar{y}))^n}+\int_{d+L_0}^{L}\frac{dz}{(f_0)^n}\right)$$

$$f_0 = \left[2\left(1 - \bar{y}_1\right)^{\left(1+\frac{1}{n}\right)} - \frac{4}{\left(\frac{1}{n}+2\right)}\left(1 - \bar{y}_1\right)^{\left(2+\frac{1}{n}\right)} + \frac{4}{\left(2+\frac{1}{n}\right)\left(3+\frac{1}{n}\right)}\left(\left(1 - \bar{y}_1\right)^{\left(3+\frac{1}{n}\right)} - \left((-1)^{\left(3+\frac{1}{n}\right)}\bar{y}_1\right)\right)\right],$$

where $\bar{y}_1 = \frac{R_c}{R_0}$

when there is no stenosis present in the artery, resistance to flow is represented as R_0.

$$\lambda_N = \frac{2\mu}{R_0^{1+sn}}\left[\frac{2Q(1+(1/n))}{\pi}\right]^n \left(\frac{L}{f_0^n}\right). \quad (13)$$

$$\lambda = \frac{\lambda_0}{\lambda_N} = 1 - \frac{L_0}{L} + \frac{(f_0)^n}{L} \int_d^{d+L_0} \frac{dz}{\left(\frac{R(z)}{R_o}\right)^{1+3n} f(\bar{y})^n} \quad (14)$$

RESULTS

The study conducted computational simulations to investigate effect of some factors, namely resistance to flow, stenosis shape parameter, wall shear stress, viscosity, stenosis size, blood flow in diseased systems characterized by arterial stenosis due to local lipid deposition. Stenosis shape parameter refers to a numerical value (ranging from 2 to 11) used to represent shape of the stenosis in the arterial wall, with each value indicating a different degree or type of stenosis shape [40]. The study utilized computer codes or computational models to simulate and analyze blood flow dynamics in arteries affected by stenosis. By varying the stenosis shape parameter and size, researchers could quantitatively assess how these factors influence λ and τ [41]. Resistance to flow refers to the opposition encountered by blood as it moves through narrowed arteries, while wall shear stress refers to the frictional force exerted by the flowing blood on the walls of the blood vessels. Through numerical experiments, the researchers obtained analytical results that quantified the relationship between stenosis characteristics (shape and size) and the associated resistance to flow and wall shear stress. This analysis provided valuable insights into the hemodynamic changes occurring in arteries affected by stenosis, contributing to a better understanding of the pathophysiology of vascular diseases and potentially informing the development of diagnostic and therapeutic strategies. Results are depicted in [Figures 3-6], utilizing parameter values.

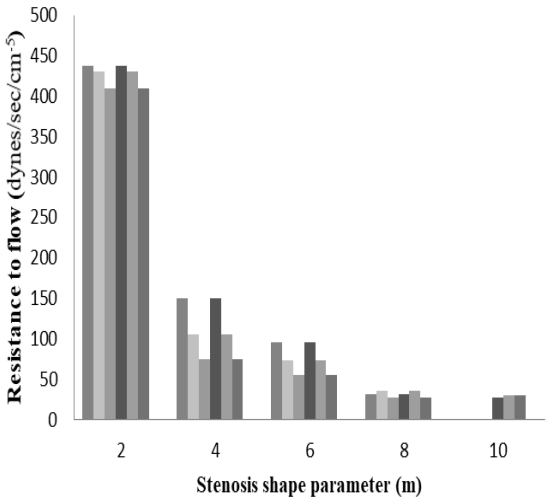

Figure : 3 Resistance to flow with Stenosis shape parameter (m)

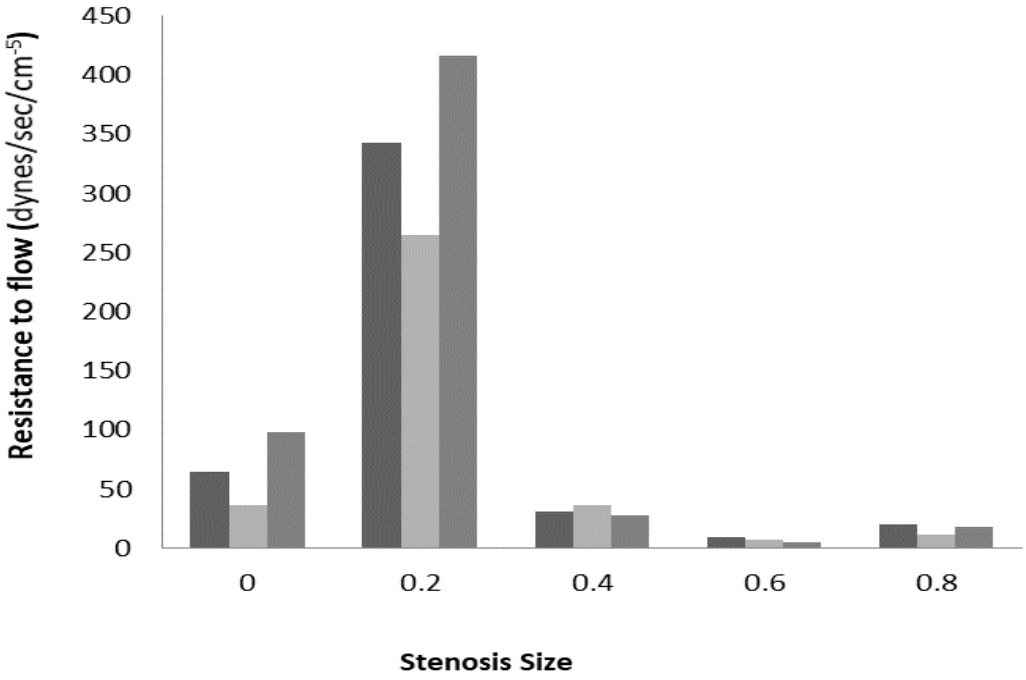

Figure : 4 Variation of Resistance to flow with stenosis size

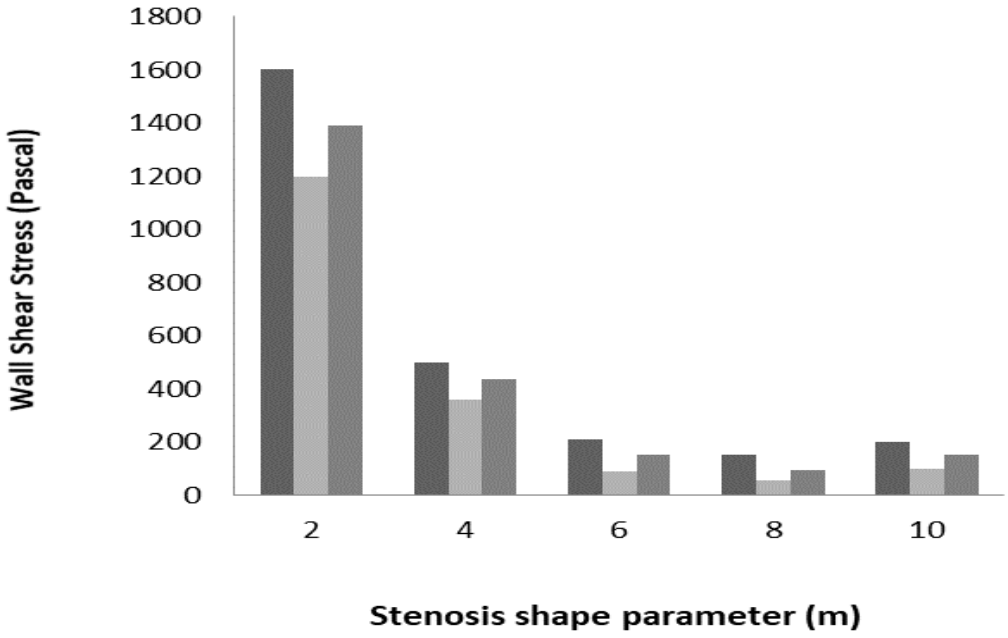

Figure : 5 Wall Shear Stress for different stenosis shape parameter (m)

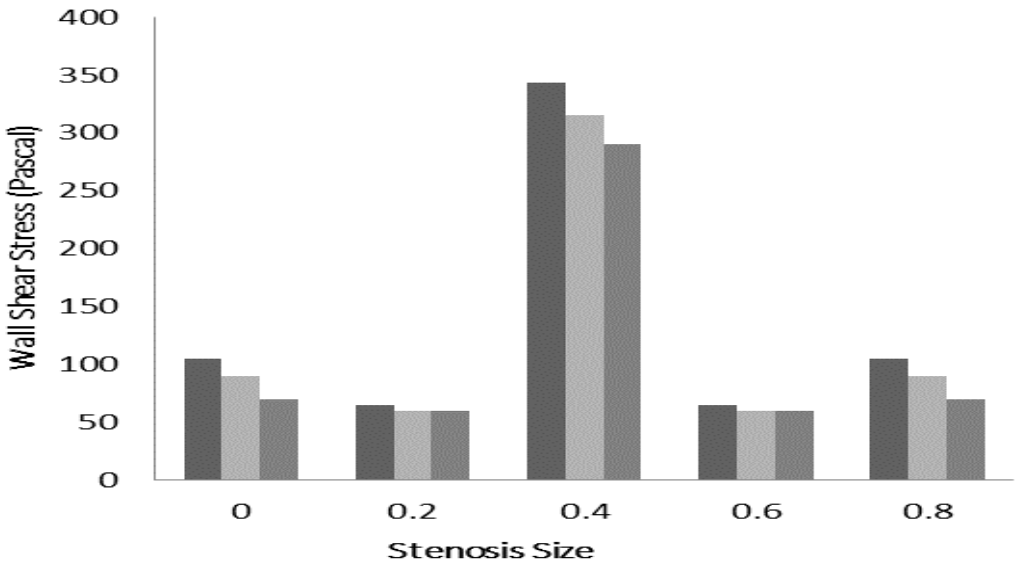

Figure: 6 Wall shear stress with stenosis size

DISCUSSION

Figure 3 provides insight into the relationship between Resistance to flow (λ) and stenosis shape parameter (m). As depicted, resistance to flow (λ) decreases as the stenosis shape parameter (m) rises. This trend is consistent with the results of [38] and indicates that the maximum resistance occurs at m = 2, corresponding to symmetric stenosis. This finding reinforces existing knowledge regarding the impact of stenosis shape on flow dynamics and highlights the significance of considering asymmetry in stenosed arteries [42].

Figure 4 show cases the relationship between resistance to blood flow (λ) and stenosis size (δ/R_0). Notably, resistance to flow increases with larger stenosis sizes, which is consistent with the Fahraeus-Lindquist effect observed in very thin tubes [43]. This phenomenon underscores impact of stenosis size on flow dynamics and highlights the importance of considering stenosis dimensions in assessing vascular health [44].

Figure 5 depicts how shear stress (τ) varies with the stenosis shape parameter. Interestingly, the graph illustrates a decrease in shear stress as the m increases [45]. These finding suggests that the changes in stenosis shape can influence wall shear stress, emphasizing the intricate relationship between arterial geometry and flow dynamics [46].

We observed in Figure 6 the relationship between shear stress and stenosis size. As size of stenosis increases, the graph demonstrates a corresponding increase in wall shear stress (τ). This finding aligns with previous studies and underscores the

influence of stenosis size on wall shear stress within the arterial system [47]. Diabetic patients are more prone to heart disease due to various factors such as insulin resistance, high blood sugar levels, and other metabolic abnormalities that can damage blood vessels and increase the risk of cardiovascular complications [48]. Nitroglycerin is often prescribed to diabetic patients to help manage their heart disease. It works by dilating blood vessels, which can improve blood flow to the heart and reduce the workload on the heart muscle [49]. This helps to alleviate symptoms such as chest pain (angina) and may also lower the risk of more serious cardiovascular events like heart attacks. The findings and derived expressions are based on a Herschek-Bulkley fluid model, which may oversimplify the complex nature of blood flow dynamics in real-world scenarios [50].

This model's applicability in clinical settings could vary due to individual patient characteristics, disease severity, and treatment variations not fully explored in this study [51]. Moreover, the study primarily focuses on short-term effects of nitroglycerin, leaving out considerations of long-term outcomes, potential tolerance development, and broader cardiovascular impacts [52]. Clinical translation of these findings also requires careful consideration of patient specific factors, comorbidities, and adherence to treatment regimens, which were not extensively addressed here [53]. Additionally, while the study discusses potential adverse effects, a comprehensive analysis of all adverse events and their management strategies falls beyond the study's specific scope. Future research should aim to address these limitations to better inform clinical practice and optimize cardiovascular care strategies for patients recovering from MI [54].

CONCLUSION

This study illuminates the intricate dynamics of blood flow in arteries with multi-shape stenosis. Through theoretical and analytical investigations, valuable insights were gained into the effects of non-symmetric multiple stenosis on blood flow dynamics, emphasizing the significance of artery geometry in vascular research. Shifting focus to the pharmacological aspect, nitroglycerin emerges as a critical component in cardiovascular management. As a potent vasodilator, nitroglycerin effectively reduces blood pressure by relaxing smooth muscle cells in blood vessel walls, thereby inducing vasodilation. This mechanism not only decreases peripheral vascular resistance but also enhances coronary blood flow, contributing to improved cardiovascular function. Moreover, nitroglycerin's impact on blood flow within the cardiovascular system is profound, facilitating easier passage of blood and enhancing both coronary and peripheral blood flow. These vasodilatory effects are particularly beneficial in conditions like angina pectoris and acute coronary syndromes, where inadequate blood flow can precipitate adverse cardiac events.

Nitroglycerin further reduces resistance to flow in blood vessels, promoting smoother blood circulation, especially during myocardial infarction and similar conditions characterized by increased vascular resistance. Additionally, nitroglycerin's ability to decrease wall shear stress suggests potential implications for mitigating vascular damage and preventing the progression of vascular diseases. Nitroglycerin's

multifaceted effects on blood pressure, blood flow dynamics, and vascular resistance underscore its pivotal role in cardiovascular management.

The clinical application of nitroglycerin therapy requires careful consideration of individual patient factors and vascular bed specificity to optimize treatment outcomes and ensure patient safety. In summary, nitroglycerin remains a cornerstone in the therapeutic arsenal against cardiovascular conditions, contributing significantly to improved patient outcomes and enhanced cardiovascular health.

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REFERENCES

[1] stenosed arteries. E-Journal of Science and Technology 2015; 109(11), 27-41.

[2] Siddiqui SU, Shah SR. A Physiologic Model for the problem of blood flow through Diseases blood vessels. Int J of Adv. in Appl Sci. 2016; 5(2): 58-64.

[3] Chaturvedi P, Shah SR. Mathematical Analysis for the Flow of Sickle Red Blood Cells in Microvessels for Bio Medical Application. The Yale J. of Bio. and Medi 2023; 96(1): 13.

[4] Shah SR. The effect of Saline Water on viscosity of blood through stenosed blood vessels using Casson's fluid model. J. of Biom. Biomat. & Tiss. Eng 2010; 9: 37-45.

[5] Gupta P, Alshehri Mo, Sharma SK, Shah SR. Empowering the visually impaired: Translating Handwritten Digits into Spoken Language with HRNN-GOA and Haralick Features. J. of Disability Research 2024; 3:1-21.

[6] Chaturvedi P, Shah SR. Assessing the Clinical Outcomes of Voxelotor Treatment in Patients with Sickle Cell Disease. Int J of Appl Sci and Biotech 2024; 12(1): 46-53.

[7] Shah SR. A Biomechanical Approach for the Study of Deformation of Red Cells in Narrow Capillaries. Int. J. of Eng 2012; 25(4), 309-314.

[8] Chaturvedi P, Kumar R and Shah SR. Bio-Mechanical & Bio-Rheological Aspects of Sickle Red Cells in Microcirculation: A Mathematical Modelling Approach.Fluids 2021;6:322 01-15.

[9] Geeta, Siddiqui SU, Sapna. Mathematical Modelling of blood flow through catheterized artery under the influence of body acceleration with slip velocity Application and applied Mathematics An international journal. 2013; 8(2):481-494.

[10] Geeta, Siddiqui SU, Shah SR. Effect of body acceleration and slip velocity on the pulsatile flow of Casson fluid through stenosed artery. Adv. Appl Sci. Res 2014;5(3): 231-225.

[11] Geeta, Siddiqui SU, Shah SR. A Biomechanical approach to the effect of body acceleration through stenotic artery. Appl. Math. and Comput 2016; 109(1): 27-41.

[12] Islam SM, Sadique M, Shah SR, Sharma SK. Effect of significant parameters on squeeze film characteristics in pathological synovial joints. Mathematics 2023; 11(6): 1468.

[13] Jaiswal KM, Akbar S, Sadique M, Shah SR. Exploring capillary-tissue fluid exchange: Insights into red cell deformation in narrow vessels and its clinical implications. Int. J. of Fauna and Biol. Stud 2024; 11(3): 4-14.

[14] Kasturia P, Sharma RK, Chaturvedi P, Dohre, R, Shah SR. Efficacy of venetoclax and azacitidine for targeting leukemic stem cell in acute myeloid leukemia. Int. J. of Biol. Pharm. and Allied Sci 2024; 13(6):3072-3090.

[15] Kumar P, Shah SR. A Hydromechanical Perspective to Study the Effect of Body Acceleration through Stenosed Artery. International journal of mathematical engineering and management sciences 2021; 6 (5): 1381-1390.

[16] Shah SR. Clinical influence of hydroxychloroquine with azithromycin on blood flow through blood vessels for the prevention and Treatment of covid-19. Int J of biology, pharmacy and allied science 2021; 10(7): 2195-2204.

[17] Kumar R, Shah SR. A mathematical approach to study the blood flow through tapered stenosed artery with the suspension of nanoparticles Destech Transactions on Engineering and Technology Research 2017; 01:1-6 10.

[18] Kumar R, Shah SR. A Mathematical Approach to Study the Blood Flow Through Tapered Stenosed Artery with the Suspension of Nanoparticles. Destech Transactions on Engineering and Technology Research 2017; 1: 1-6.

[19] Kumar V, Shah SR. A Mathematical study for heat transfer phenomenological processes in human skin. Int J of Mechanical Engineering 2022; 7(6): 683-692.

[20] Kumar V, Shah SR. Thermobiological Mathematical Model for the study of temperature response after cooling effects. SSRG Int J of Applied physics. 2022; 9(2): 7-11.

[21] Kumar V, Shah SR. Mathematical modelling to study the heat transfer between core and skin. SRMS J. of Mathematical Sci 2024; 7:7-12.

[22] Lenin JS, Shah SR. Mathematical Analysis of Stem Cell Dynamics in Acute Myeloid Leukemia: Towards Precision Medicine Strategies. Int J of Sci and Res 2024; 13(05): 528-535.

[23] Shah SR. Clinical significance of aspirin on blood flow through stenotic blood vessels. J. of Biom. Biomat. & Tiss. Eng. 2011; 10: 17-24.

[24] Sadique M, Shah SR. Mathematical model to study the effect of PRG4, hyaluronic acid and lubricin on squeeze film characteristics of diseased synovial joint. Int. J. of Mech. Eng. 2022; 7(6), 832-848.

[25] Sadique M, Shah SR. Mathematical study for the synovial fluid flow in Osteoarthritic knee joint. J of Eng and Appl Sci 2022; 17(2), 15-21.

[26] Shah SR. Effects of Acetylsalicylic Acid on blood flow through an artery under Atherosclerotic condition. Int. J. of Mol. Medi. & Adv. Sci 2011. 7 (6): 19-24.

[27] Majhi L, Arya S, Shah SR. Exploring Shilajatu's Therapeutic Potential in Diabetes Management: A Comprehensive Study Integrating Ayurvedic Wisdom and Modern Science Int J of Sci and Res. 2024; 13(5): 1374-1380.

[28] Shah SR. Non-Newtonian flow of blood through an atherosclerotic artery. Res. J. of Appl. Sci 2011; 6(1), 76-80.

[29] Geeta, Siddiqui SU, Shah SR. A Computational Analysis of a Two-Fluid non-Linear Mathematical Model of Pulsatile Blood Flow Through Constricted Artery. e-Journal of Science & Technology 2015; 10(4) 65-78.

[30] Shah SR. A Biomechanical Approach for the Study of Two-Phase Blood Flow Through Stenosed Artery. Int. J. of Res. 2013; 1(2): 24-32.

[31] Singh S. Effects of shape of stenosis on arterial rheology under the influence of applied magnetic field. Int J of Biomed. Eng. & Tech 2011; 6(3): 286-294.

[32] Shah SR. An innovative solution for the problem of blood flow through stenosed artery using generalized Bingham plastic fluid model. Int. J. of Res. in Appl. & Nat. Soc. Sci 2013; 1(3): 97-98.

[33] Shah SR. An innovative study for non-Newtonian behaviour of blood flow in stenosed artery using Herschel-Bulkley fluid model. International Journal of Bio-Science and Bio-Technology 2013; 5(5): 233-240.

[34] Anamika, Kumar R, Shah SR: Mathematical Modelling of blood flow through tapered stenosed artery with the suspension of nanoparticles using Jeffrey fluid model Int J of development research 2017; 07: 13494-13500.

[35] Shah SR. Effects of antiplatelet drugs on blood flow through stenosed blood vessels. J. of Biomi., Biomat. and Tissue Eng. 2013; 18: 21-27.

[36] Sadique M, Shah SR. Mathematical Model to Study the Squeeze Film Characteristics of Diseased Human Synovial Knee Joint. World Scient. Ann. Rev. of Biom 2023; 1: 2330004.

[37] Shah SR. Effect of clopidogrel on blood flow through stenosed artery under diseased condition. Int J of Experim. Pharmaco 2014; 4(1): 887-893.

[38] Shah SR. Performance Modelling and Analysis of Magnetic Field on Nutritional Transport Capillary Tissues System Using Modified Herschel-Bulkley Fluid. Int J. Adv Res in Phy Sci 2014; 1(1), 33-41.

[39] Shah SR. Significance of Aspirin on Blood Flow to Prevent Blood Clotting through Inclined Multi-Stenosed Artery.Letters In Health and Bio Sci 2017; 2(2): 97-100.

[40] Shah SR. Study of dispersion of drug in blood flow with the impact of chemical reaction through stenosed artery. Int. J. of Bioscie 2022; 21(3): 21-29.

[41] Kumar R, Shah SR. Study of blood flow with suspension of nanoparticles through tapered artery. Global J. of Pure and Appl. Math 2017; 13(10): 7387-7399.

[42] Shah SR and Kumar R. Mathematical modeling of blood flow with the suspension of nanoparticles through a tapered artery with a blood clot. Front. in Nanotech 2020; 2: 596475.

[43] Singh S, Shah RR. A numerical model for the effect of stenosis shape on blood flow through an artery using power-law fluid. Adv. in AppL. Sci. Res 2010; 1(1), 66-73.

[44] Shah SR, Siddiqui S U (2011) Two-phase model for the study of blood flow through stenosed artery. Int. J. of Pharm. and Bio. Sci 1(3) 246-254.

[45] Singh A, Siddiqui SU, Shah SR. Mathematical Modeling of peristaltic blood flow through a vertical blood vessel using prandtl fluid model. Int. J. of Math and Comput. Res 2016; 4(9): 710-717.

[46] Shah SR, Siddiqui SU. Achievement of Pentoxifylline for Blood Flow through Stenosed Artery. J of Biomim. Biomat. and Tissue Eng 2012; 13: 81-89.

[47] Siddiqui SU, Shah SR. A Physiologic Model for the problem of blood flow through Diseases blood vessels. Int. J of Adv. in Appl. Sci 2016; 5(2), 58-64.

[48] Shah SR and Kumar R. Performance of Blood Flow with Suspension of Nanoparticles Through Tapered Stenosed Artery for Jeferey Fluid Model. Int J. of Nanosci 2018; 17:(06).1850004.

[49] Siddiqui SU, Shah SR. A biomechanical approach to study the effect of body acceleration & slip velocity through stenotic artery. Appl. Math. & Compu 2015;261:148-155.

[50] Singh S. Influence of magnetic field on blood flow through stenosed artery using casson's fluid model. Int J of BioEngineering, CardioPulmonary Sci. & Tech 2010; 1:1-7.

[51] Singh S. Numerical modeling of two-layered micropolar fluid through a normal and stenosed artery. Int J Eng 2011; 24 (2): 177-187.

[52] Akbar S, Shah SR. Mathematical Modeling of Blood Flow Dynamics in the Cardiovascular System: Assumptions, Considerations, and Simulation Results. Journal of Current Medical Research and Opinion 2024; 7(4):2216-2225.

[53] Stiehl T, Kumar R, Shah SR. Understanding the impact of feedback regulations on blood cell production and leukemia dynamics using model analysis and simulation of clinically relevant scenarios. Appl. Math. Mod 2024;129: 340-389.

[54] Tasneem A, Suri S, Kaur H, Solanki R, Shah SR, Singh P, Dohare R. Screening of miRNAs as prognostic biomarkers and their associated hub targets across Hepatocellular carcinoma using survival-based bioinformatics approach. J. of Genetic Eng. and Biotech 2024; 22 (1): 1-10.