

Dual-Rheological Model and Finite Element Simulation of Hemodynamics Through a Multiple Stenotic Artery with Nanodrug-Eluting Stent

B. Vasu^{1,*}, Ankita Dubey², Rama Subba Reddy Gorla³, Mustaque H. Borbora¹, Shabih-ul-Hasan⁴

¹Department of Mathematics, Motilal Nehru National Institute of Technology Allahabad, Prayagraj, Uttar Pradesh- 211004, India

²Department of Mathematics, GLA University, Mathura – 281406, U.P, India

³Department of Aeronautics and Astronautics, Air Force Institute of Technology, Wright Patterson Air Force Base, Dayton, Ohio 45433, USA

⁴Department of Chemical Engineering, Motilal Nehru National Institute of Technology Allahabad, Prayagraj, Uttar Pradesh- 211004, India

*Correspondence should be addressed to Vasu B, bvasu@mnit.ac.in

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Abstract

A two-dimensional rheological study of hemodynamics through a diseased artery with multiple stenosis and drug-eluting stent is simulated computationally. The homogeneous suspension of metallic gold nanoparticles in the blood is considered motivated by pharmacology applications. The Casson (viscoplastic) and Sisko (viscoelastic) fluid models are employed, to mimic non-Newtonian characteristics of the blood flow in the core and in the peripheral arterial region respectively. The revised Buongiorno two-component nanofluid model is utilized for nanoscale effects and natural double-diffusive convection to simulate the dual influence of thermal and solutal buoyancy forces. The transformed governing equations with appropriate non-dimensional variables are solved numerically subject to physical boundary conditions using the finite element method. The effect of significant thermo-physical parameters on velocity, hemodynamic pressure, temperature, and nanoparticle concentration have been calculated for two clinically essential cases of arteries with multiple stenosis (without stent) in the presence of a drug eluting stent. Regarding the color contours and graphs, it is observed that when increasing the thermophoresis parameter, the Nusselt number increases initially but decreases gradually with higher value of thermophoresis parameter. Whereas the pressure decreases with the increase in thermophoresis parameter. The simulations are relevant to transport phenomena in pharmacology and to nano-drug targeted delivery in cardiovascular rheology and vascular sciences.

Keywords: Hemodynamics, Multiple stenosis, Drug-eluting stent, Nanoparticle transport, Finite element method

Introduction

Reports of numerous physiological studies states that the cardiovascular diseases are the primary cause of mortality worldwide including heart attacks and strokes. According to the data of the Global Health Estimates [1], the major percentage of deaths is due to coronary diseases. Cardiovascular diseases include coronary heart disease (CHD), cerebrovascular disease, rheumatic heart disease, and numerous other ailments. The

formation of plaque in the lumen, known as *atherosclerosis*, which manifests as a *stenosis*, can grow and block the artery and hence prevent blood supply to the distant body cells. Calcium prone plaque can also rupture and initiate the formation of blood clotting [2]. Through a complex network of arteries, veins, and capillaries, blood flows to the body organs and body cells. To maintain cell-level metabolism, blood flow ensures the transportation of nutrients, hormones, metabolic wastes, O_2 , and CO_2 throughout the body. It regulates the

pH, osmotic pressure, and temperature of the whole body and protects the body from microbial and mechanical harm [3]. Reddy *et al.* [4] studied blood flow by treating blood as a high-impedance couple stress fluid, showing that significant deviations in flow characteristics arise compared to the classical Newtonian model. Several investigators have analyzed theoretically in detail the contribution of blood rheology to coronary artery disease and cerebral aneurysms. Agrawal *et al.* [5] studied the shear-thinning characteristics of blood using the Carreau–Yasuda model in the context of coil embolization, a minimally invasive endovascular method for treating cerebral aneurysms. They observed that blood rheology plays a prominent role in the performance of the coil, which is aimed at reducing fluid loading on the blood vessel and delaying subsequent wall deformation. Riahi *et al.* [6] studied the blood flow in an artery with overlapping stenosis. Dubey *et al.* [7] discussed the rheological effects of stenosis and aneurysm in a porous walled artery containing a nanoparticle suspension. Vasu *et al.* [8] also investigated hemodynamic blood flow in a time-variant, tapered stenotic artery and discussed the effects of different metallic nanoparticles on flow behavior. Vasu *et al.* [3] implemented a second grade fluid model to study magneto-hemodynamics in a stenosed coronary artery and simulated the results using the finite element method. Razavi *et al.* [9] compared various viscosity models to the Newtonian model for pulsatile hemodynamics in a stenosed carotid artery, noting that a greater degree of stenosis induces increased disturbances in the downstream flow and significantly affects wall shear stress (WSS) at the stenosis throat. Karimi *et al.* [10] implemented the Carreau and modified power-law non-Newtonian models to simulate blood flow in commonly stenosed carotid arteries and compared the results with experimental data to highlight the influence of rheology on hemodynamic characteristics.

An alternative model is the viscoplastic Casson non-Newtonian fluid model, which predicts infinite viscosity at zero shear rate and a constant viscosity at infinite shear rate. Casson [11] initially investigated the validity of the model by studying the flow characteristics of blood observed at low shear rates, where the yield stress for blood is non-zero. Sarifuddin *et al.* [12] presented a mathematical model of blood flow through an asymmetric arterial constriction, where the blood is represented as the suspension of erythrocytes in plasma with the Casson fluid model. Copley [13] established the modest shear behavior of blood in narrow arteries, noting that the Casson fluid model is arguably the most appropriate since it included a yield stress criterion. Blood is also known to exhibit *viscoelasticity* under certain conditions. A popular and robust model for viscoelasticity in hemodynamics is the Sisko model. Haghighi and Chalak [14] used the Sisko rheological model to numerically simulate blood flow in a stenotic artery with body acceleration effects using the finite difference method. Zaman *et al.* [15] presented a non-linear, unsteady blood flow model for a stenosed elastic vessel using the Sisko model. An additional feature of real arteries is the permeability of the vessel wall,

which allows the diffusion of oxygen and other nutrients into the vessel. Several computational studies of hemodynamic flow in permeable arteries have been communicated. Bali and Awasthi [16], and Mishra *et al.* [17], studied blood flow through a composite stenosis in an artery with a porous wall, although blood was simplified to a Newtonian fluid. Further studies of permeable wall and porosity effects on blood flow include Jain *et al.* [18] (for stenosed arteries), and Tripathi [19] (magnetic blood flow in an inclined artery).

Interesting applications of nanofluids in medicine include blood purification systems [20], smart bio-nano-polymer coatings for medical devices [21], nano-drug delivery (pharmacological systems) in cardiovascular treatment [22], biodegradable nano liquids for cerebral pharmacodynamics [23], membrane oxygenator bioreactors [24], orthopedic lubrication with nano-films (superlubricated poly(3-sulfopropyl methacrylate potassium salt)-grafted mesoporous silica nanoparticles suspended in starch base liquids) [25], pulsed laser ablation (PLA) of ultra-pure silicon nanofluid fabrication for cancer therapy [26], smart biomimetic electro-osmotic nanofluid pumps in ocular diagnosis [27], cryopreservation, bone reinforcement via super-paramagnetic nanofluids etc. In hemodynamic therapies, the base liquid is typically blood, which can be doped with a variety of nanoparticles, including gold. To prevent in-stent restenosis and improve the performance of current stents, various nanomaterial coatings and controlled-release nano-carriers are used [28]. Nano-carriers have the potential to deliver imaging and diagnostic agents to precisely targeted destinations. Current therapies focus on decreasing the burden of atherosclerotic plaques and stabilizing vulnerable plaques, defined as those plaques, which have a tendency to rupture and cause thrombosis [29]. A new type of stent has been designed in recent years, known as the drug-eluting stent (DES), which reduces the risk of in-stent thrombosis and restenosis caused by bare-metal stents. Various classes of drugs, including anti-cancer and anti-inflammatory agents, have been tested with DES to prevent smooth muscle cell (SMC) growth and proliferation.

Bio-Rheological Nano-Pharmacodynamic Model

Consider the two-dimensional, laminar, incompressible, steady (non-pulsating) hemodynamic flow in an artery, consisting of the core region (lumen) and peripheral region (porous arterial wall). Two cases of blood flow simulation are also considered, i.e., blood flow through an artery with multiple stenosis and blood flow through the artery when a drug eluted stent has been placed at stenotic part of the artery. In the first case, blood is simulated as a non-homogeneous fluid, while in the second case, it is simulated with uniformly dispersed nanoparticles when a drug-eluting stent is used. Non-Newtonian characteristics are modeled using a dual rheological formulation- namely the Casson fluid model in the core region and the Sisko fluid model in the peripheral region. A cylindrical coordinate system (r, ϕ, z) is employed, where

r is the radial coordinate, z is the axial coordinate and φ is the azimuthal coordinate. Since the axisymmetric flow is studied the contribution in the azimuthal (φ) direction is ignored i.e., the flow occurs only in the radial (r) and axial (z) direction as depicted in **Figure 1**. A finite length (L_0) arterial geometry is

studied which contains a sinusoidal-shaped multiple stenosis, as shown in **Figure 1a**. Additionally, a separate arterial geometry is studied where a stent is placed at the stenotic region of the artery, illustrated in **Figure 1b**.

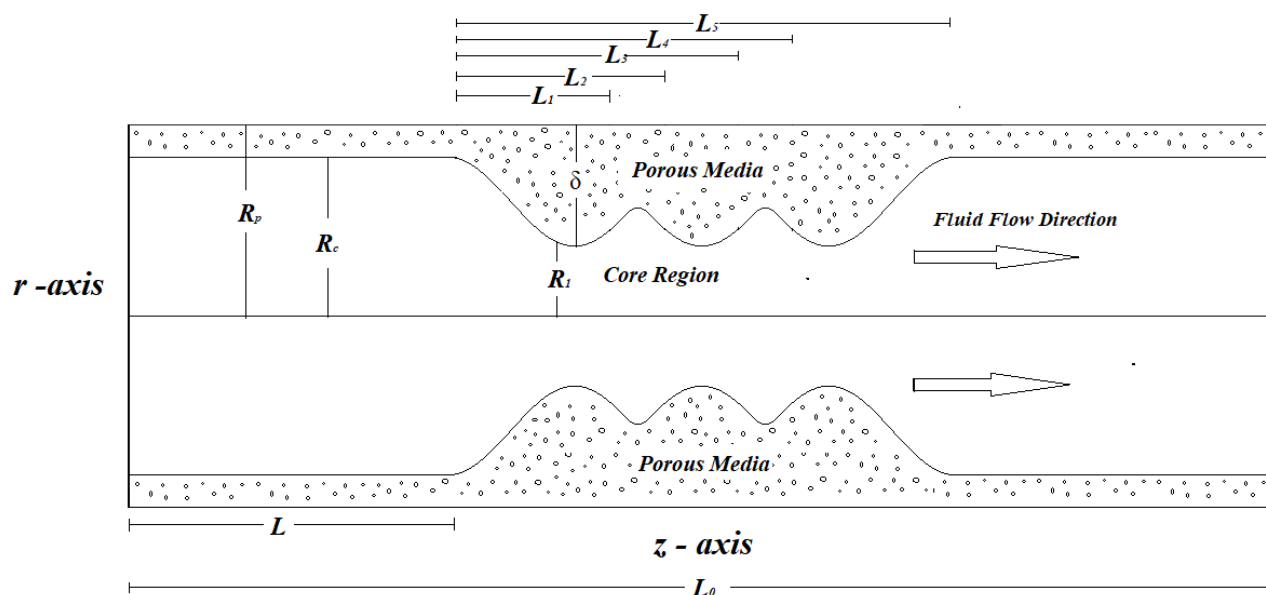


Figure 1a. Physical model for bio-rheological flow in an artery with multiple stenoses.

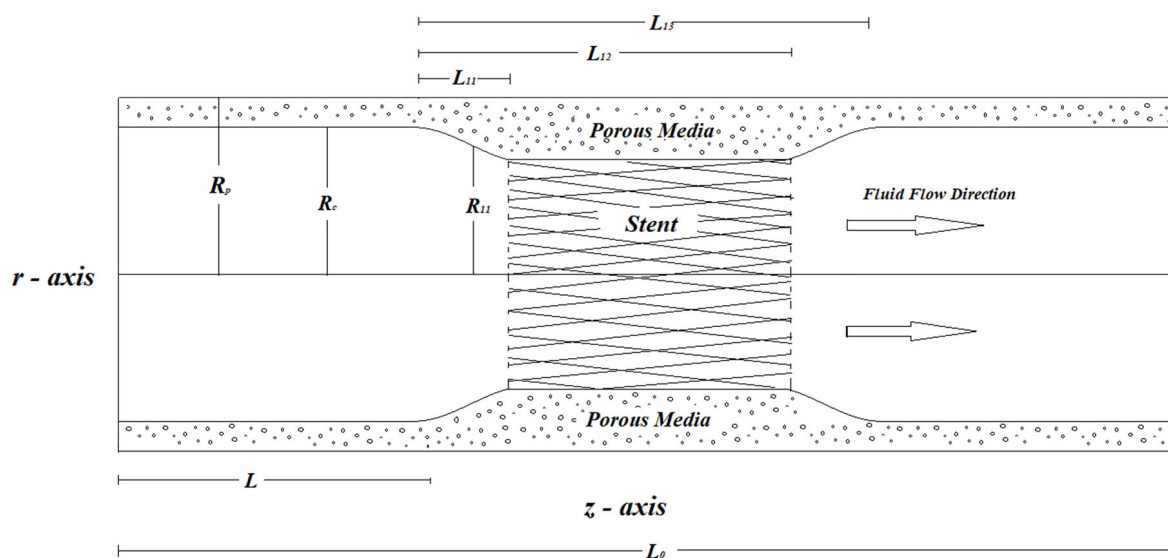


Figure 1b. Physical model for bio-rheological flow in a stenosed artery with a stent.

In the first case, the geometry of the inner layer of the multiple stenosis is assumed to be described by the following mathematical equation:

$$R_i(z) = \begin{cases} -\sin(\pi * z) * 0.35 + 0.65 + 0.5; & L \leq z \leq L + L_1 \\ -\cos(\pi * z) * 1.2 + 0.5994237 - 0.5; & L + L_1 \leq z \leq L + L_2 \\ -\sin(\pi * z) * 0.35 + 0.65 + 0.5; & L + L_2 \leq z \leq L + L_3 \\ -\cos(\pi * z) * 1.2 + 0.5994237 - 0.5; & L + L_3 \leq z \leq L + L_4 \\ -\sin(\pi * z) * 0.35 + 0.65 + 0.5; & L + L_4 \leq z \leq L + L_5 \\ R_c & ; \text{ otherwise} \end{cases} \quad (1)$$

The geometry of the inner layer of the multiple stenosis with drug eluting stent is simulated with the following Mathematical relations:

$$R_{11}(z) = \begin{cases} -\sin(\pi * z) * 0.15 - 0.65 - 0.5 + 0.05; & L \leq z \leq L + L_{11} \\ -0.97864745; & L + L_{11} \leq z \leq L + L_{12} \\ -\sin(\pi * z) * 0.15 - 0.65 - 0.5 + 0.05; & L + L_{12} \leq z \leq L + L_{13} \\ R_c & ; \text{ otherwise} \end{cases} \quad (2)$$

The following notations are adopted: radius of the artery, radius of the core region of the non-stenotic section, and length of the diseased parts of the artery are $R_p, R_c, L_1, L_2, L_3, L_4, L_5, L_{11}, L_{12}$ and L_{13} respectively. The radius of the inner layer of the artery is taken as $R_{11}(z)$ while the radius of the inner layer of the artery with stent is denoted by $R_{11}(z)$. The z-axis is orientated along the direction of blood flow and is perpendicular to the r-axis. The stenosis is symmetrical and has a maximum height δ which is more than 60% of the radius.

For steady, axisymmetric flow of blood in the arterial vessel, the velocity vector V is assumed to take the following forms:

In the core region (Casson fluid model [11])

$$V_c = [u_c(r, z), 0, w_c(r, z)] \quad (3)$$

In the peripheral (porous) region (Sisko fluid model [15]):

$$V_p = [u_p(r, z), 0, w_p(r, z)] \quad (4)$$

Here u_c, u_p and w_c, w_p represent the velocity components in the radial and axial directions, respectively, for both the core and porous regions. Blood is considered to be an incompressible, non-Newtonian fluid (Casson fluid in the core region and Sisko fluid in the peripheral region) in both cases. But in later cases the blood flow contains a homogenous distribution of nanoparticles since we have considered a drug eluting stent.

The two cases of the bio-rheological model and the hemodynamic transport equations in the core region are as follows:

$$\frac{\partial u_c}{\partial r} + \frac{u_c}{r} + \frac{\partial w_c}{\partial z} = 0 \quad (5)$$

$$\rho_f \left[u_c \frac{\partial u_c}{\partial r} + w_c \frac{\partial u_c}{\partial z} \right] = -\frac{\partial p_c}{\partial r} + \frac{1}{r} \frac{\partial}{\partial r} (r S_{rr}) + \frac{\partial}{\partial z} (S_{rz}) \quad (6)$$

$$\rho_f \left[u_c \frac{\partial w_c}{\partial r} + w_c \frac{\partial w_c}{\partial z} \right] = -\frac{\partial p_c}{\partial z} + \frac{1}{r} \frac{\partial}{\partial r} (r S_{rz}) + \frac{\partial}{\partial z} (S_{zz}) + \rho_f g \quad (7)$$

In later case:

$$\frac{\partial u_c}{\partial r} + \frac{u_c}{r} + \frac{\partial w_c}{\partial z} = 0 \quad (8)$$

$$\rho_f \left[u_c \frac{\partial u_c}{\partial r} + w_c \frac{\partial u_c}{\partial z} \right] = -\frac{\partial p_c}{\partial r} + \frac{1}{r} \frac{\partial}{\partial r} (r S_{rr}) + \frac{\partial}{\partial z} (S_{rz}) \quad (9)$$

$$\rho_f \left[u_c \frac{\partial w_c}{\partial r} + w_c \frac{\partial w_c}{\partial z} \right] = -\frac{\partial p_c}{\partial z} + \frac{1}{r} \frac{\partial}{\partial r} (r S_{rz}) + \frac{\partial}{\partial z} (S_{zz}) + (\rho_f (1 - \beta_T (T - T_L) - \beta_S (\phi - \phi_L))) g \quad (10)$$

$$(c\rho)_p \left(u_c \frac{\partial T}{\partial r} + w_c \frac{\partial T}{\partial z} \right) = \kappa \left(\frac{\partial^2 T}{\partial r^2} + \frac{1}{r} \frac{\partial T}{\partial r} + \frac{\partial^2 T}{\partial z^2} \right) + (c\rho)_f \left(D_b \left[\frac{\partial \phi}{\partial r} \frac{\partial T}{\partial r} + \frac{\partial \phi}{\partial z} \frac{\partial T}{\partial z} \right] + \frac{D_T}{T_L} \left[\frac{\partial T}{\partial r} \frac{\partial T}{\partial r} + \frac{\partial T}{\partial z} \frac{\partial T}{\partial z} \right] \right) \quad (11)$$

$$\left(u_c \frac{\partial \phi}{\partial r} + w_c \frac{\partial \phi}{\partial z} \right) = D_b \left(\frac{\partial^2 \phi}{\partial r^2} + \frac{1}{r} \frac{\partial \phi}{\partial r} + \frac{\partial^2 \phi}{\partial z^2} \right) + \frac{D_f}{T_L} \left(\frac{\partial^2 T}{\partial r^2} + \frac{1}{r} \frac{\partial T}{\partial r} + \frac{\partial^2 T}{\partial z^2} \right) \quad (12)$$

The mass and momentum equations in the peripheral region (p), for both the cases, take the following form:

$$\frac{\partial u_p}{\partial r} + \frac{u_p}{r} + \frac{\partial w_p}{\partial z} = 0 \quad (13)$$

$$\rho_f \left[u_p \frac{\partial u_p}{\partial r} + w_p \frac{\partial u_p}{\partial z} \right] = -\frac{\partial p_p}{\partial r} + \frac{1}{r} \frac{\partial}{\partial r} (r S_{rr}) + \frac{\partial}{\partial z} (S_{rz}) + \frac{\mu}{k'} u_p \quad (14)$$

$$\rho_f \left[u_p \frac{\partial w_p}{\partial r} + w_p \frac{\partial w_p}{\partial z} \right] = -\frac{\partial p_p}{\partial z} + \frac{1}{r} \frac{\partial}{\partial r} (r S_{rz}) + \frac{\partial}{\partial z} (S_{zz}) + \frac{\mu}{k'} w_p \quad (15)$$

The Cauchy stress tensors are defined as follows:

$$S_{rr} = -2 \left[\mu_1 + \mu_0 \left\{ \left[\left(\frac{\partial u_p}{\partial r} \right)^2 + \left(\frac{u_p}{r} \right)^2 + \left(\frac{\partial w_p}{\partial z} \right)^2 + \left(\frac{\partial w_p}{\partial r} + \frac{\partial u_p}{\partial z} \right)^2 \right]^{\frac{1}{2}} \right]^{\frac{1}{2}n-1} \right] \left(\frac{\partial u_p}{\partial r} \right) \quad (16)$$

$$S_{zz} = -2 \left[\mu_1 + \mu_0 \left\{ \left[\left(\frac{\partial u_p}{\partial r} \right)^2 + \left(\frac{u_p}{r} \right)^2 + \left(\frac{\partial w_p}{\partial z} \right)^2 + \left(\frac{\partial w_p}{\partial r} + \frac{\partial u_p}{\partial z} \right)^2 \right]^{\frac{1}{2}} \right]^{\frac{1}{2}n-1} \right] \left(\frac{\partial w_p}{\partial z} \right) \quad (17)$$

$$S_{rz} = - \left[\mu_1 + \mu_0 \left\{ \left[\left(\frac{\partial u_p}{\partial r} \right)^2 + \left(\frac{u_p}{r} \right)^2 + \left(\frac{\partial w_p}{\partial z} \right)^2 + \left(\frac{\partial w_p}{\partial r} + \frac{\partial u_p}{\partial z} \right)^2 \right]^{\frac{1}{2}} \right]^{\frac{1}{2}n-1} \right] \left(\frac{\partial w_p}{\partial r} + \frac{\partial u_p}{\partial z} \right) \quad (18)$$

The associated boundary conditions are given as follows:

In the first case:

$$\begin{aligned} u_c = u_p = 0, w_c = U_0, \frac{\partial w_p}{\partial r} = 0, & \quad \text{at } r = 0 \\ w_c = w_p & \quad \text{at } r = R(z) \\ w_p = 0 & \quad \text{at } r = R_1(z) \text{ (no-slip condition)} \end{aligned} \quad (19)$$

In the second case, the mathematical expression for blood flow is as follows:

$$\begin{aligned} u_c = u_p = 0, w_c = U_0, \frac{\partial T}{\partial r} = 0, \frac{\partial \phi}{\partial r} = 0 & \quad \text{at } r = 0 \\ w_c = w_p & \quad \text{at } r = R(z) \\ w_p = 0 & \quad \text{at } r = R_1(z) \text{ (no-slip condition)} \\ \frac{D_T}{T_L} \frac{\partial T}{\partial r} + D_b \frac{\partial \phi}{\partial r} = 0 & \quad \text{at } r = R(z) \\ T = T_w & \quad \text{at } r = R(z) \end{aligned} \quad (20)$$

The wall shear stress at the inner and the outer walls of the artery are defined as:

$$WSS_{inner} = - \left[\sqrt{\tau_c} + \left(\mu \frac{\partial w_p}{\partial r} \right)^{\frac{1}{2}} \right] \quad (21a)$$

$$WSS_{outer} = - \left[\mu_1 + \mu_0 \left\{ \left[\left(\frac{\partial u_p}{\partial r} \right)^2 + \left(\frac{u_p}{r} \right)^2 + \left(\frac{\partial w_p}{\partial z} \right)^2 + \left(\frac{\partial w_p}{\partial r} + \frac{\partial u_p}{\partial z} \right)^2 \right]^{\frac{1}{2}} \right\}^{\frac{1}{n-1}} \right] \left(\frac{\partial w_p}{\partial r} + \frac{\partial u_p}{\partial z} \right) \quad (21b)$$

The heat flux is:

$$q_c = -k \left(\frac{\partial T}{\partial r} \right) \quad (22)$$

Hence the skin friction coefficient and Nusselt number (wall heat transfer rate) are:

$$C_f = \frac{2\tau_w}{\rho_f U_0^2} \quad \text{and} \quad Nu = \frac{z q_c}{k(T_w - T_L)} \quad (23)$$

The volumetric flow rate is expressed as:

$$Q = \left[\int_0^{R_c} r w_c dr + \int_{R_c}^{R_p} r w_p dr \right] \quad (24)$$

The flow resistance λ (impedance) is expressed as:

$$\lambda = \int_0^{L_1} \frac{\left(-\frac{\partial p}{\partial z} \right)}{Q} dz \quad (25)$$

To facilitate the numerical solution of the boundary value problem, it is pertinent to render the derived model as

dimensionless. The following non-dimensional variables and parameters are introduced:

$$\begin{aligned} \bar{u} = \frac{u L_0}{U_0 \delta}, \bar{w} = \frac{w}{U_0}, \bar{z} = \frac{z}{L_0}, \bar{r} = \frac{r}{R_p}, \bar{\mu} = \frac{\mu}{\mu_0}, \bar{\mu}_0 = \mu_0 \left(\frac{U_0}{R_p} \right)^{n-1}, \bar{\tau} = \frac{R_p}{\mu U_0} \tau \\ \bar{\theta} = \frac{T - T_L}{T_w - T_L}, \bar{\phi} = \frac{\phi - \phi_L}{\phi_w - \phi_L}, \bar{p} = \frac{R_p^2 p}{U_0 \mu_0 L_0}, \bar{m} = \frac{\mu_1}{\mu_0}, \bar{R}(z) = \frac{R(z)}{R_c}, \bar{\lambda} = \frac{\lambda R_p^4}{\mu_0 L_0}, \bar{\tau}_c = \frac{R_p}{\mu U_0} \tau_c \\ \bar{R}_1(z) = \frac{R_1(z)}{R_p}, \bar{S}_{rr} = \frac{S_{rr} L_0}{U_0 \mu_0}, \bar{S}_{zz} = \frac{S_{zz} L_0}{U_0 \mu_0}, \bar{S}_{rz} = \frac{S_{rz} R_p}{U_0 \mu_0}, \bar{r}_0 = \frac{R_p}{R_c}, \bar{Q} = \frac{Q}{R_p^2 U_0} \end{aligned} \quad (26)$$

Where $U_0, L_0, R_p, \mu_0, \delta, T_L, T_w, \phi_L$ and ϕ_w denote the reference velocity, reference length of the blood vessel, reference radius, reference dynamic viscosity, stenosis depth, reference fluid temperature, vessel wall temperature, reference mass concentration, and vessel wall mass concentration in the arterial tube model, respectively. By implementing equation (26) in equation (5)-(15) and (21)-(25) the following system of dimensionless conservation equations are produced: Hemodynamic simulations are to be explored for the case of a mild stenotic artery. The nondimensional geometric parameters appearing in the expressions above are the stenosis height parameter ($\delta^* = \delta/R_p \ll 1$) and the vessel aspect ratio ($\varepsilon = R_p/L_0$). For the subsequent analysis, we assume $\delta^* \ll 1$ and $\varepsilon = O(1)$, i.e., the maximum height of the stenosis is small in comparison to the radius of the artery, while the radius of the artery and the length of the stenotic region are of comparable magnitude. After applying the above assumptions in the normalized system, we got the following transformed equations for the core region (Casson viscoplastic fluid):

For the first case:

$$\frac{\partial \bar{p}_c}{\partial \bar{r}} = 0 \quad (27)$$

$$\frac{\partial \bar{p}_c}{\partial \bar{z}} = \frac{1}{\bar{r}} \tau_c + \frac{1}{\bar{r}} \frac{\partial \bar{w}_c}{\partial \bar{r}} + \sqrt{\tau_c} \left(\frac{\partial \bar{w}_c}{\partial \bar{r}} \right)^{\frac{1}{2}} \left(\frac{2}{\bar{r}} + \left(\frac{\partial \bar{w}_c}{\partial \bar{r}} \right) \frac{\partial^2 \bar{w}_c}{\partial \bar{r}^2} \right) + \left(\frac{\partial^2 \bar{w}_c}{\partial \bar{r}^2} \right) + G \quad (28)$$

For the second case:

$$\frac{\partial \bar{p}_c}{\partial \bar{r}} = 0 \quad (29)$$

$$\begin{aligned} \frac{\partial \bar{p}_c}{\partial \bar{z}} = \frac{1}{\bar{r}} \tau_c + \frac{1}{\bar{r}} \frac{\partial \bar{w}_c}{\partial \bar{r}} + \sqrt{\tau_c} \left(\frac{\partial \bar{w}_c}{\partial \bar{r}} \right)^{\frac{1}{2}} \left(\frac{2}{\bar{r}} + \left(\frac{\partial \bar{w}_c}{\partial \bar{r}} \right) \frac{\partial^2 \bar{w}_c}{\partial \bar{r}^2} \right) \\ + \left(\frac{\partial^2 \bar{w}_c}{\partial \bar{r}^2} \right) + Gr(\bar{\theta} + N\bar{\phi}) \end{aligned} \quad (30)$$

$$\left(\frac{\partial^2 \bar{\theta}}{\partial \bar{r}^2} + \frac{1}{\bar{r}} \frac{\partial \bar{\theta}}{\partial \bar{r}} \right) + \left(Nb \left[\frac{\partial \bar{\phi}}{\partial \bar{r}} \frac{\partial \bar{\theta}}{\partial \bar{r}} \right] + Nt \left[\frac{\partial \bar{\theta}}{\partial \bar{r}} \frac{\partial \bar{\theta}}{\partial \bar{r}} \right] \right) = 0 \quad (31)$$

$$\left(\frac{\partial^2 \bar{\phi}}{\partial \bar{r}^2} + \frac{1}{\bar{r}} \frac{\partial \bar{\phi}}{\partial \bar{r}} \right) + \frac{Nt}{Nb} \left(\frac{\partial^2 \bar{\theta}}{\partial \bar{r}^2} + \frac{1}{\bar{r}} \frac{\partial \bar{\theta}}{\partial \bar{r}} \right) = 0 \quad (32)$$

After applying the above assumptions in the normalized system, we got the following transformed equations for the peripheral region (Sisko viscoelastic fluid):

$$\frac{\partial \bar{p}_p}{\partial \bar{r}} = 0 \quad (33)$$

$$\frac{\partial \bar{p}_p}{\partial \bar{z}} = \left(\frac{1}{\bar{r}} \frac{\partial \bar{w}_p}{\partial \bar{r}} + \frac{\partial^2 \bar{w}_p}{\partial \bar{r}^2} \right) \left(m + \left(\frac{\partial \bar{w}_p}{\partial \bar{r}} \right)^{n-1} \right) + \frac{\partial^2 \bar{w}_p}{\partial \bar{r}^2} \left((n-1) \left(\frac{\partial \bar{w}_p}{\partial \bar{r}} \right)^{n-2} \right) \frac{\partial \bar{w}_p}{\partial \bar{r}} + \bar{\mu}^* \bar{w}_p \quad (34)$$

The associated non-dimensional boundary conditions are derived as follows:

For the first case (without stent):

$$\begin{aligned} \bar{u}_c = \bar{u}_p = 0, \bar{w}_c = 1, \frac{\partial \bar{w}_p}{\partial \bar{r}} = 0, & \quad \text{at } \bar{r} = 0 \\ \bar{w}_c = \bar{w}_p & \quad \text{at } \bar{r} = r_0 \bar{R}(z) \\ \bar{w}_p = 0 & \quad \text{at } \bar{r} = \bar{R}_1(z) \end{aligned} \quad (35)$$

For the second case (with stent):

$$\begin{aligned} \bar{u}_c = \bar{u}_p = 0, \bar{w}_c = 1, \frac{\partial \bar{w}_p}{\partial \bar{r}} = 0, \frac{\partial \bar{\theta}}{\partial \bar{r}} = 0, \frac{\partial \bar{\phi}}{\partial \bar{r}} = 0 & \quad \text{at } \bar{r} = 0 \\ \bar{w}_c = \bar{w}_p & \quad \text{at } \bar{r} = r_0 \bar{R}(z) \\ \bar{w}_p = 0 & \quad \text{at } \bar{r} = \bar{R}_1(z) \\ N_t \frac{\partial \bar{\theta}}{\partial \bar{r}} + N_b \frac{\partial \bar{\phi}}{\partial \bar{r}} = 0 & \quad \text{at } \bar{r} = r_0 \bar{R}(z) \\ \bar{\theta} = 1 & \quad \text{at } \bar{r} = r_0 \bar{R}(z) \end{aligned} \quad (36)$$

After non-dimensionalizing the skin friction coefficient and the Nusselt number, the following expressions are obtained:

Skin friction coefficient at the inner wall:

$$\frac{1}{2} C'_f \text{Re} = - \left[\sqrt{\tau'_c} + \left(\bar{\mu} \frac{\partial \bar{w}_p}{\partial \bar{r}} \right)^{\frac{1}{2}} \right]^2 \quad (37)$$

Skin friction coefficient at the outer wall:

$$\frac{1}{2} C'_f \text{Re} = - \left[m + \left(\frac{\partial \bar{w}_p}{\partial \bar{r}} \right)^{n-1} \right] \left(\frac{\partial \bar{w}_p}{\partial \bar{r}} \right) \quad (38)$$

Nusselt number at any wall:

$$Nu' \text{Re} = - \bar{z} \left(\frac{\partial \bar{\theta}}{\partial \bar{r}} \right) \quad (39)$$

Volumetric flow rate:

$$\bar{Q} = \int_0^{R_c} \bar{r} \bar{w}_c d\bar{r} + \int_{R_c}^{R_p} \bar{r} \bar{w}_p d\bar{r} \quad (40)$$

Hemodynamic resistance (impedance):

$$\bar{\lambda} = \int_0^{L_1} \left(\frac{-\partial \bar{p}}{\partial \bar{z}} \right) \frac{d\bar{z}}{\bar{Q}} \quad (41)$$

In equations (54)–(61), the following parameters are involved: the Brownian motion parameter, $Nb = \frac{D_b(\phi_w - \phi_L)(\rho c)_f}{\kappa}$,

thermophoresis parameter $Nt = \frac{D_T(T_w - T_L)(\rho c)_f}{T_L \kappa}$, the

Grashof number $Gr = \frac{(T_w - T_L)\rho_{f,w} g R_p^2 \beta_T}{U_0 \bar{\mu}_0}$, the solutal to

thermal buoyancy ratio $N = \frac{\beta_s(\phi_w - \phi_L)}{\beta_T(T_w - T_L)}$, the Reynolds

number $Re = \frac{\rho_f U_0 L_0}{\bar{\mu}_0}$, Prandtl number $Pr = \frac{c_p \bar{\mu}_0}{k}$, and

Schmidt number $Sc = \frac{\bar{\mu}_0}{\rho_f D_b}$, peripheral viscosity

$\bar{\mu}^* = \frac{\bar{\mu} R_p^2}{k'}$, and also $C'_f = C_f \left(\frac{U_0}{L_0} \right)$, $Nu' = Nu \frac{\bar{\mu}_0 R_p}{\rho_f U_0}$, $G = \frac{\rho_f g R_p^2}{U_0 \bar{\mu}_0}$

Numerical Computations with Freefem++ Code

The non-dimensional boundary value problem derived—namely, equations (27)–(34) with boundary conditions (35)–(36)—remains quite formidable due to strong nonlinearity, the coupling of different variables and two space variables. A robust computational scheme is therefore essential to obtain fast and rapidly convergent solutions. To this end, the finite element method (FEM), based on the variational approach and implemented using FreeFEM++, is employed. Extensive details on the mathematical and algorithmic foundations of FREEFEM++ are provided by Hecht [30]. A weak formulation of the partial differential equation system (27)–(34) is derived by first defining the function spaces:

$$\begin{aligned} X &= \left\{ \bar{u} \in (H_1(\Omega)) \mid \bar{u} = a \text{ on } \Gamma_{in}, \bar{u} = 0 \text{ on } \Gamma_{wall} \right\} \\ Q &= \left\{ \bar{u} \in (H_1(\Omega)) \mid \bar{u} = 0 \text{ on } \Gamma_{in} \cup \Gamma_{wall} \right\} \end{aligned} \quad (42)$$

The weak form of equation (54) – (61) is obtained by determining $w \in X$ and $\phi, \theta \in P$ such that for all test functions $u_1, u_2 \in Q$ and $p_1, p_2, v, q \in P$ the following conditions are satisfied, where $P = L^2(\Omega)$. Therefore, the weak formulation of equation (54) – (61) is given as follows:

For the Core Region (Casson fluid):

For first case (without stent):

$$\int_{\Omega} \frac{\partial \bar{p}_c}{\partial \bar{r}} \cdot p_1 \, d\bar{r} = 0 \quad (43)$$

$$\int_{\Omega} \frac{\partial \bar{p}_c}{\partial \bar{z}} \cdot p_1 \, d\bar{r} - \int_{\Omega} \frac{1}{\bar{r}} \tau_c \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} \frac{1}{\bar{r}} \frac{\partial \bar{w}_c}{\partial \bar{r}} \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} \left(\frac{2}{\bar{r}} + \frac{\partial \bar{w}_c}{\partial \bar{r}} \frac{\partial^2 \bar{w}_c}{\partial \bar{r}^2} \right) \sqrt{\tau_c} \left(\frac{\partial \bar{w}_c}{\partial \bar{r}} \right)^{\frac{1}{2}} \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} \left(\frac{\partial^2 \bar{w}_c}{\partial \bar{r}^2} \right) \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} G \cdot u_{1,h} \, d\bar{r} = 0 \quad (44)$$

For second case (with drug eluting stent):

$$\int_{\Omega} \frac{\partial \bar{p}_c}{\partial \bar{r}} \cdot p_1 \, d\bar{r} = 0 \quad (45)$$

$$\int_{\Omega} \frac{\partial \bar{p}_c}{\partial \bar{z}} \cdot p_1 \, d\bar{r} - \int_{\Omega} \frac{1}{\bar{r}} \tau_c \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} \frac{1}{\bar{r}} \frac{\partial \bar{w}_c}{\partial \bar{r}} \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} \left(\frac{2}{\bar{r}} + \frac{\partial \bar{w}_c}{\partial \bar{r}} \frac{\partial^2 \bar{w}_c}{\partial \bar{r}^2} \right) \sqrt{\tau_c} \left(\frac{\partial \bar{w}_c}{\partial \bar{r}} \right)^{\frac{1}{2}} \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} \left(\frac{\partial^2 \bar{w}_c}{\partial \bar{r}^2} \right) \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} Gr \bar{\theta} \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} Gr N \bar{\phi} \cdot u_{1,h} \, d\bar{r} = 0 \quad (46)$$

$$\int_{\Omega} \frac{\partial^2 \bar{\theta}}{\partial \bar{r}^2} \cdot v \, d\bar{r} + \int_{\Omega} \frac{1}{\bar{r}} \frac{\partial \bar{\theta}}{\partial \bar{r}} \cdot v \, d\bar{r} + \int_{\Omega} Nb \left[\frac{\partial \bar{\phi}}{\partial \bar{r}} \frac{\partial \bar{\theta}}{\partial \bar{r}} \right] \cdot v \, d\bar{r} + \int_{\Omega} Nt \left[\frac{\partial \bar{\theta}}{\partial \bar{r}} \frac{\partial \bar{\theta}}{\partial \bar{r}} \right] \cdot v \, d\bar{r} = 0 \quad (47)$$

$$\int_{\Omega} \frac{\partial^2 \bar{\phi}}{\partial \bar{r}^2} \cdot q \, d\bar{r} + \int_{\Omega} \frac{1}{\bar{r}} \frac{\partial \bar{\phi}}{\partial \bar{r}} \cdot q \, d\bar{r} + \int_{\Omega} \frac{Nt}{Nb} \left(\frac{\partial^2 \bar{\theta}}{\partial \bar{r}^2} + \frac{1}{\bar{r}} \frac{\partial \bar{\theta}}{\partial \bar{r}} \right) \cdot q \, d\bar{r} = 0 \quad (48)$$

For the Peripheral region (Sisko fluid):

$$\int_{\Omega} \frac{\partial \bar{p}_p}{\partial \bar{r}} \cdot p_2 \, d\bar{r} = 0 \quad (49)$$

$$\int_{\Omega} \frac{\partial \bar{p}_p}{\partial \bar{z}} \cdot p_2 \, d\bar{r} - \int_{\Omega} \left(\frac{1}{\bar{r}} \frac{\partial \bar{w}_p}{\partial \bar{r}} + \frac{\partial^2 \bar{w}_p}{\partial \bar{r}^2} \right) \left(m + \left(\frac{\partial \bar{w}_p}{\partial \bar{r}} \right)^{n-1} \right) \cdot u_{2,h} \, d\bar{r} - \int_{\Omega} \frac{\partial^2 \bar{w}_p}{\partial \bar{r}^2} \left((n-1) \left(\frac{\partial \bar{w}_p}{\partial \bar{r}} \right)^{n-2} \right) \frac{\partial \bar{w}_p}{\partial \bar{r}} \cdot u_{2,h} \, d\bar{r} - \int_{\Omega} \bar{\mu}^* \bar{w}_p \cdot u_{2,h} \, d\bar{r} = 0 \quad (50)$$

To attain the requisite smoothness of the solutions, which is bounded due to the weaker restriction, these differential equations cannot be solved directly. Therefore, the finite dimensional subspaces must be defined as $Q_h \subset Q$ and $P_h \subset P$. Consider the finite dimensional approximations as $u_{1,h}, u_{2,h} \subset Q_h$ and $p_{1,h}, p_{2,h}, v_h, q_h \subset P_h$. In view of the finite dimensional approximation, the set of equations (43)-(50) becomes:

For the Core Region (Casson fluid):

For the first case (without stent):

$$\int_{\Omega} \frac{\partial \bar{p}_c}{\partial \bar{r}} \cdot p_{1,h} \, d\bar{r} = 0 \quad (51)$$

$$\int_{\Omega} \frac{\partial \bar{p}_c}{\partial \bar{z}} \cdot p_{1,h} \, d\bar{r} - \int_{\Omega} \frac{1}{\bar{r}} \tau_c \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} \frac{1}{\bar{r}} \frac{\partial \bar{w}_c}{\partial \bar{r}} \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} \left(\frac{2}{\bar{r}} + \frac{\partial \bar{w}_c}{\partial \bar{r}} \frac{\partial^2 \bar{w}_c}{\partial \bar{r}^2} \right) \sqrt{\tau_c} \left(\frac{\partial \bar{w}_c}{\partial \bar{r}} \right)^{\frac{1}{2}} \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} \left(\frac{\partial^2 \bar{w}_c}{\partial \bar{r}^2} \right) \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} G \cdot u_{1,h} \, d\bar{r} = 0 \quad (52)$$

For the second case (with drug eluting stent):

$$\int_{\Omega} \frac{\partial \bar{p}_c}{\partial \bar{r}} \cdot p_{1,h} \, d\bar{r} = 0 \quad (53)$$

$$\int_{\Omega} \frac{\partial \bar{p}_c}{\partial \bar{z}} \cdot p_{1,h} \, d\bar{r} - \int_{\Omega} \frac{1}{\bar{r}} \tau_c \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} \frac{1}{\bar{r}} \frac{\partial \bar{w}_c}{\partial \bar{r}} \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} \left(\frac{2}{\bar{r}} + \frac{\partial \bar{w}_c}{\partial \bar{r}} \frac{\partial^2 \bar{w}_c}{\partial \bar{r}^2} \right) \sqrt{\tau_c} \left(\frac{\partial \bar{w}_c}{\partial \bar{r}} \right)^{\frac{1}{2}} \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} \left(\frac{\partial^2 \bar{w}_c}{\partial \bar{r}^2} \right) \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} Gr \bar{\theta} \cdot u_{1,h} \, d\bar{r} - \int_{\Omega} Gr N \bar{\phi} \cdot u_{1,h} \, d\bar{r} = 0 \quad (54)$$

$$\int_{\Omega} \frac{\partial^2 \bar{\theta}}{\partial \bar{r}^2} \cdot v_h \, d\bar{r} + \int_{\Omega} \frac{1}{\bar{r}} \frac{\partial \bar{\theta}}{\partial \bar{r}} \cdot v_h \, d\bar{r} + \int_{\Omega} Nb \left[\frac{\partial \bar{\phi}}{\partial \bar{r}} \frac{\partial \bar{\theta}}{\partial \bar{r}} \right] \cdot v_h \, d\bar{r} + \int_{\Omega} Nt \left[\frac{\partial \bar{\theta}}{\partial \bar{r}} \frac{\partial \bar{\theta}}{\partial \bar{r}} \right] \cdot v_h \, d\bar{r} = 0 \quad (55)$$

$$\int_{\Omega} \frac{\partial^2 \bar{\phi}}{\partial \bar{r}^2} \cdot q_h \, d\bar{r} + \int_{\Omega} \frac{1}{\bar{r}} \frac{\partial \bar{\phi}}{\partial \bar{r}} \cdot q_h \, d\bar{r} + \int_{\Omega} \frac{Nt}{Nb} \left(\frac{\partial^2 \bar{\theta}}{\partial \bar{r}^2} + \frac{1}{\bar{r}} \frac{\partial \bar{\theta}}{\partial \bar{r}} \right) \cdot q_h \, d\bar{r} = 0 \quad (56)$$

For the Peripheral region (Sisko fluid):

$$\int_{\Omega} \frac{\partial \bar{p}_p}{\partial \bar{r}} \cdot p_{2,h} \, d\bar{r} = 0 \quad (57)$$

$$\int_{\Omega} \frac{\partial \bar{p}_p}{\partial \bar{z}} \cdot p_{2,h} \, d\bar{r} - \int_{\Omega} \left(\frac{1}{\bar{r}} \frac{\partial \bar{w}_p}{\partial \bar{r}} + \frac{\partial^2 \bar{w}_p}{\partial \bar{r}^2} \right) \left(m + \left(\frac{\partial \bar{w}_p}{\partial \bar{r}} \right)^{n-1} \right) \cdot u_{2,h} \, d\bar{r} - \int_{\Omega} \frac{\partial^2 \bar{w}_p}{\partial \bar{r}^2} \left((n-1) \left(\frac{\partial \bar{w}_p}{\partial \bar{r}} \right)^{n-2} \right) \frac{\partial \bar{w}_p}{\partial \bar{r}} \cdot u_{2,h} \, d\bar{r} - \int_{\Omega} \bar{\mu}^* \bar{w}_p \cdot u_{2,h} \, d\bar{r} = 0 \quad (58)$$

The transformed equations (51)-(58) along with boundary conditions (35) and (36), are solved numerically using the finite element method through the variational formulation scheme implemented with FreeFEM++. An unstructured, fixed mesh (grid) comprising piecewise linear triangular elements with bubble functions (P1b), is implemented for the discretization of the arterial flow domain, as depicted in **Figure 2**. The mesh is constructed using the automatic FreeFEM++ mesh generator, which is based on the popular Delaunay-Voronoi algorithm. The non-linear system of governing equations is solved by employing the Generalized Minimal Residual (GMRES) iteration method.

Mesh Independence Analysis

The numerical values of the Nusselt Number and skin-friction coefficient at the stenosis, both without and with a stent, for various designs comprising unstructured fixed mesh elements with nodes and triangular elements, are provided in **Figures 3a** and **3b** respectively. Seven different mesh distributions have been tested to ensure the simulated numerical results are mesh independent for stenosed arterial geometry, both without and with a stent respectively. Therefore, the selected mesh for the present calculations consisted of 75,766 triangular elements and 114,090 nodes for the arterial geometry without a stent, and 116,568 triangular elements and 175,372 nodes for the arterial geometry with a stent, respectively. As shown in **Tables 1a** and **1b** and **Figures 3a** and **3b**, increasing the mesh element density beyond the selected design does not significantly modify the numerical values of the non-dimensional Nusselt Number and skin-friction coefficient

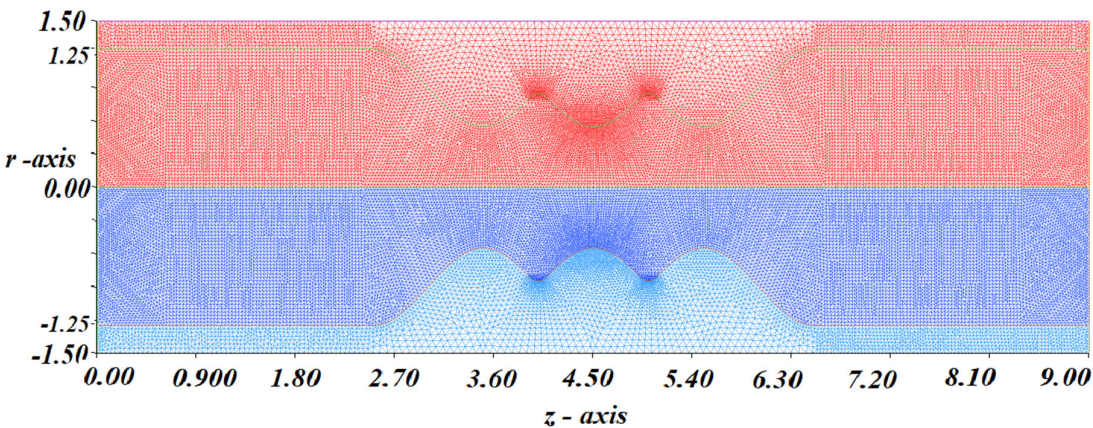


Figure 2a. Unstructured fixed mesh of triangular elements (without stent).

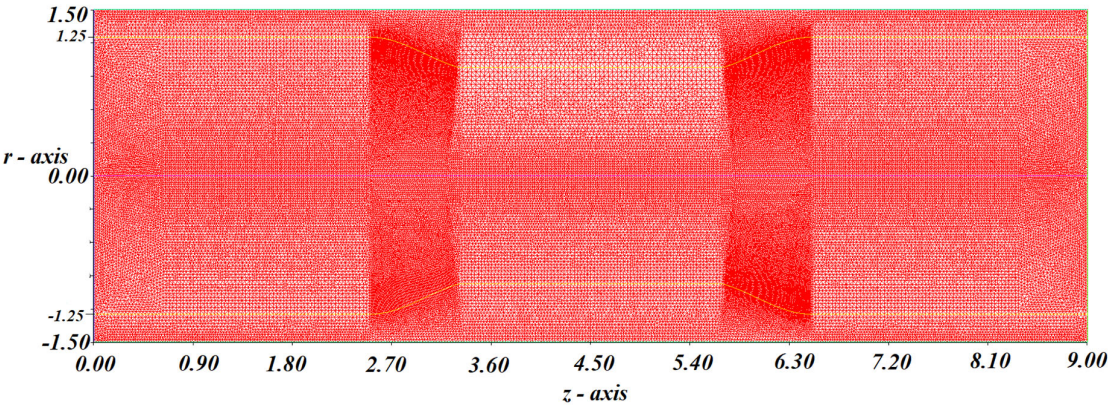


Figure 2b. Unstructured fixed mesh of triangular elements (with stent).

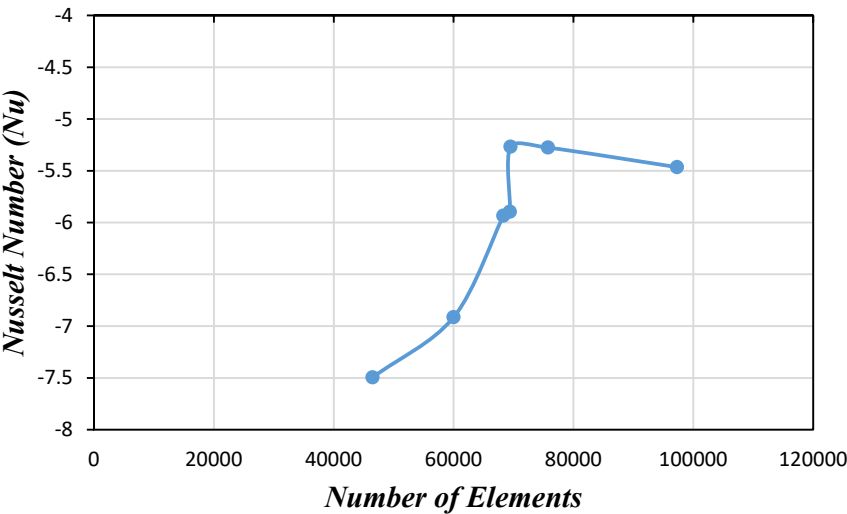


Figure 3a. Grid Independence study for stenosed artery without stent (Nusselt Number).

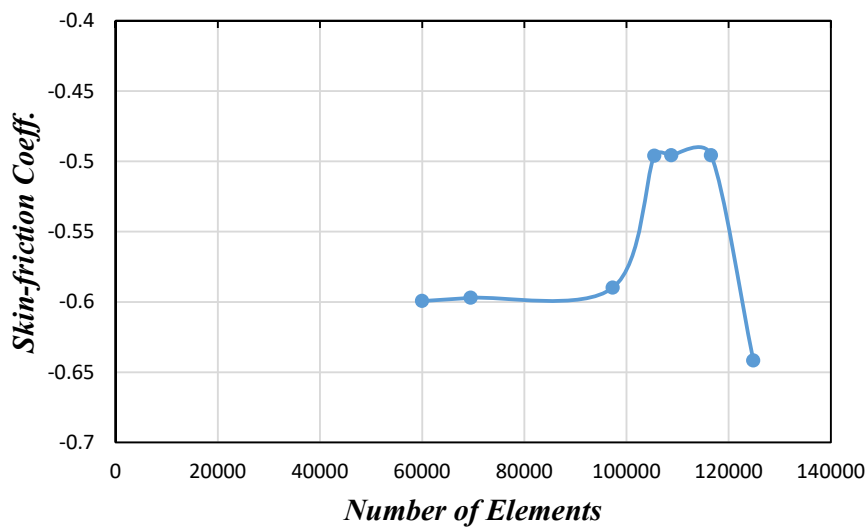


Figure 3b. Grid Independence study for stenosed artery with stent (Skin-friction Coeff.).

Table 1a. Grid Independency analysis without a stent at $m = 0.657$, $N_b = 0.3$, $N_t = 0.3$, $Gr = 4.0$, $N = 0.2$ at $r = 0.450$, $z = 4.50$.			
S. No.	Number of Elements	Number of Nodes	Nusselt Number (Stenosis)
1.	46,494	70,012	-7.4948
2.	60,008	90,313	-6.914
3.	68,316	102,825	-5.9341
4.	69,408	104,443	-5.896
5.	69,522	104,614	-5.2673
6.	75,766	114,090	-5.2756
7.	97,306	146,480	-5.4652

Table 1b. Grid Independency analysis with a stent at $m = 0.657$, $N_b = 0.3$, $N_t = 0.3$, $Gr = 4.0$, $N = 0.2$ at $r = 0.450$, $z = 4.50$.			
S. No.	Number of Elements	Number of Nodes	Skin-friction coeff. (Stenosis)
1.	60,008	90,313	-0.59934
2.	69,522	104,614	-0.59708
3.	94,242	141,884	-0.58993
4.	105,470	158,725	-0.496
5.	108,786	163,699	-0.4957
6.	116,568	175,372	-0.49567
7.	124,860	187,810	-0.64173

within the domain, for the prescribed parametric values i.e. $m = 0.657$, $N_b = 0.3$, $N_t = 0.3$, $Gr = 4.0$, $N = 0.2$ at $r = 0.450$, $z = 4.50$. Mesh independent results are therefore ensured with the mesh design comprising 114,090 nodes and 75,766 triangular elements for the artery without a stent and 175,372 nodes and 116,568 triangular elements for the artery with a stent (simulation number 6 in **Tables 1a** and **1b**).

Results and Discussion

An extensive investigation of the impact of the key multi-physical parameters such as the Buoyancy ratio parameter (N), Sisko rheological material parameter ratio (m) —on the velocity in core and porous (peripheral) regions, and also the influence of thermophoresis parameter (N_t), Brownian

motion parameter (N_b), the principal hemodynamic transport characteristics i.e. velocity, temperature and nanoparticle concentration is presented graphically as contour plots in **Figures 4-13** for both regions. The FREEFEM++ computations are performed using the default values of various parameters values listed in **Table 2**.

Effect of stent on the arterial stenosis

Figure 4 describes the effect of stent on the skin-friction coefficient $\frac{1}{2}ReC_f^*$ on the stenotic part of the arterial wall. The graph presented in **Figure 4** clearly depicts the minimum skin-friction coefficient occurs at the midpoint of the stenotic wall. Moreover, the use of a drug-eluting stent results in a lower skin-friction coefficient along the arterial wall compared to the case without a stent.

Effect of Brownian motion parameter (N_b) and thermophoresis parameter (N_t)

The impact of Brownian motion parameter (N_b) and thermophoresis parameter (N_t) on the temperature profile, Nusselt number, nanoparticle concentration, and pressure distribution are shown in the different colored contours, graphs, and tables, showing variations along the radial and axial coordinates. **Figures 5** and **6** depicted the influence of thermophoresis parameter (N_t) and Brownian motion parameter (N_b) on the temperature distribution (θ) and nanoparticle concentration (ϕ) in the treated stenotic part with a stent and also the whole arterial segment. These results are

presented with a fixed value of Grashof number ($Gr=2.0$), and solutal to thermal buoyancy ratio ($N=0.4$) in the core region. In **Figures 5b** and **5c**, an increase in the thermophoresis parameter (N_t) from 0.3 to 0.5 results in an approximate 0.51% increase in the temperature distribution across all radial coordinate values. Furthermore, in **Figures 5c** and **5d**, increasing N_t from 0.5 to 0.7 leads to a significant rise—approximately 32%—in temperature distribution values within the core region and near the inner arterial wall. **Figure 6** illustrates the variation in nanoparticle concentration distribution with the maximum concentration (volume fraction) occurring at the stenotic arterial segment which then decreases substantially in the arterial region. There is an elevation in the magnitudes of nano-particle concentration across all axial coordinates, with an ~1.22% increment when the Brownian motion parameter is increased from 0.3 to 0.5 and then a marked ~34% increment when increasing the Brownian motion parameter from 0.5 to 0.7 respectively at the stent treated stenotic part and near the inner wall of the arterial segment. Both nanoscale effects generally promote the diffusion of nanoparticle species across all radial coordinates, with the effect being most pronounced at intermediate distances from the arterial midline. Brownian motion, in particular, helps distribute the nanoparticles as uniformly as possible throughout the blood flow regime. It reduces the nanoparticle concentration gradient and diminishes regional variations in fluid characteristics, while simultaneously promoting species diffusion, which leads to an increase in nanoparticle concentration magnitudes.

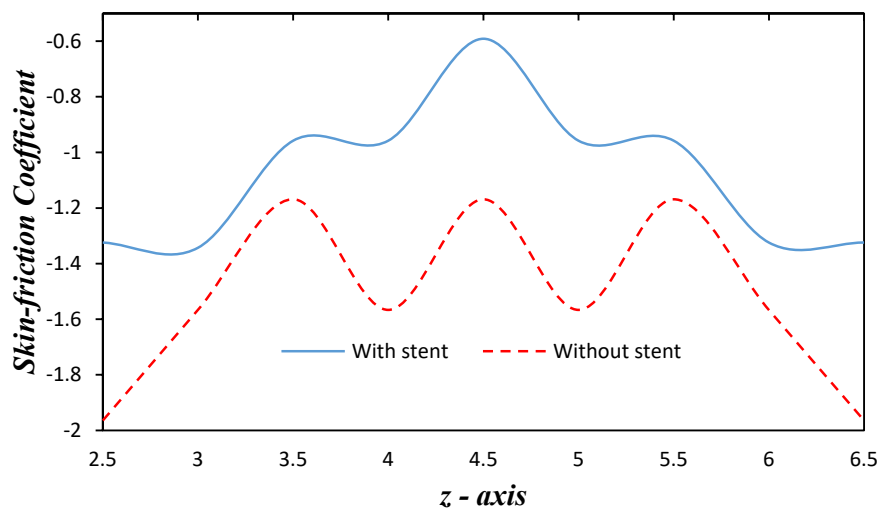


Figure 4. Effect of the stent on the skin-friction coefficient $\frac{1}{2}ReC_f^*$ at the stenotic wall when $m = 0.657$, $Gr = 1.0$, $N = 0.2$, $N_t = 0.3$, and $N_b = 0.3$.

Table 2. Data employed in FREEFEM++ computations (unless otherwise indicated).											
Parameter	n	M	Gr	μ^*	L_{13}	L_0	N	Nb	Nt	τ_c^*	L_s
Values	1.4	0.657	1.0	2.4	4.0	9.0	0.2	0.3	0.3	0.3	4.0

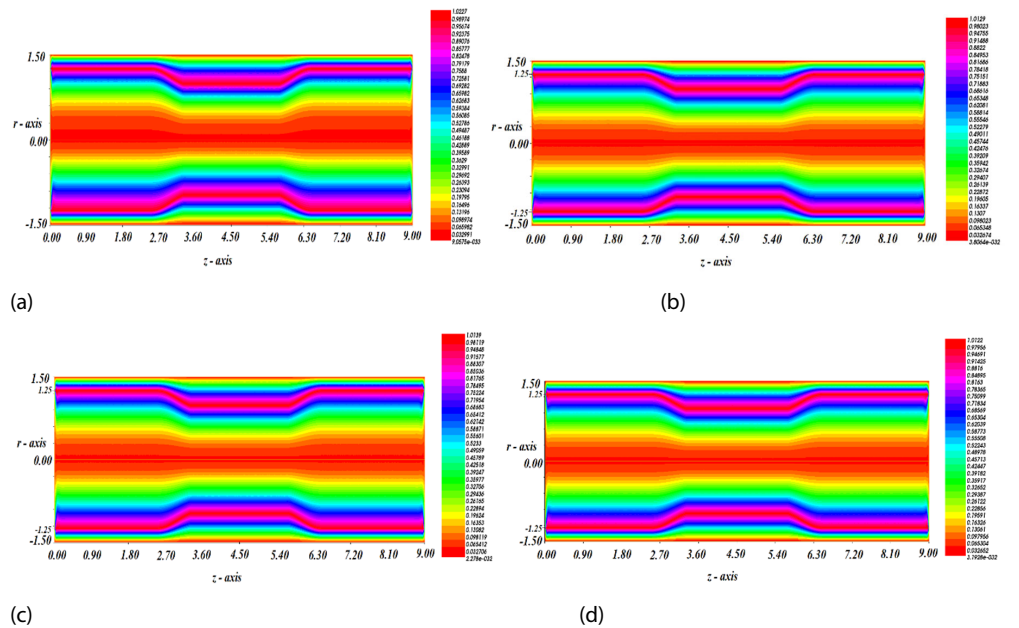


Figure 5. Effect of the thermophoresis parameter (N_t) on temperature when (a) $m = 0.657$, $Gr = 2.0$, $N = 0.4$, $N_b = 0.5$, and $N_t = 0.2$. (b) $m = 0.657$, $Gr = 2.0$, $N = 0.4$, $N_b = 0.5$, and $N_t = 0.3$. (c) $m = 0.657$, $Gr = 2.0$, $N = 0.4$, $N_b = 0.5$, and $N_t = 0.5$. (d) $m = 0.657$, $Gr = 2.0$, $N = 0.4$, $N_b = 0.5$, and $N_t = 0.7$.

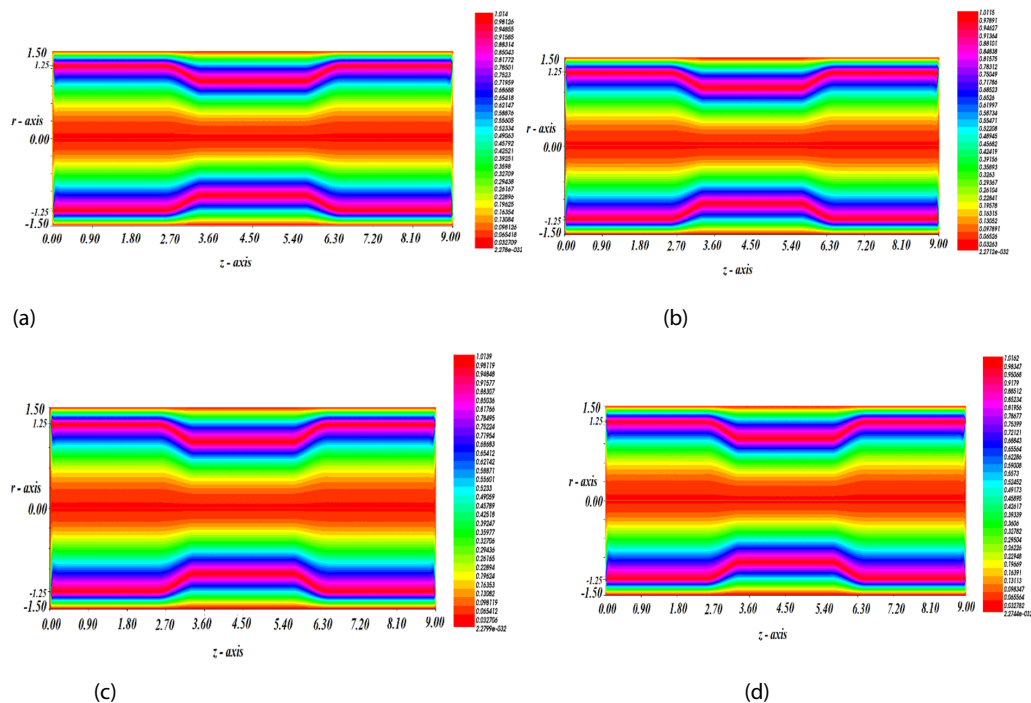


Figure 6. Effect of the Brownian motion parameter (N_b) on concentration when (a) $m = 0.657$, $Gr = 2.0$, $N = 0.4$, $N_t = 0.5$, and $N_b = 0.2$. (b) $m = 0.657$, $Gr = 2.0$, $N = 0.4$, $N_t = 0.5$, and $N_b = 0.3$. (c) $m = 0.657$, $Gr = 2.0$, $N = 0.4$, $N_t = 0.5$, and $N_b = 0.5$. (d) $m = 0.657$, $Gr = 2.0$, $N = 0.4$, $N_t = 0.5$, and $N_b = 0.7$.

Figures 7 and 8 are the graphical representation of the effect of thermophoresis parameter on the Nusselt number, showing variations along the radial and axial axes, respectively. The graphs in **Figure 7** shows an elevation in the Nusselt number as N_t increased from 0.2 to 0.3 followed by a gradual decrease as N_t increases from 0.3 to 0.5, and from 0.5 to 0.7, at the stent-treated stenotic arterial region. The Nusselt number decreases monotonically from the centerline to the artery, but there is a sudden increase near the inner arterial wall. **Figure 8** shows a similar trend for the Nusselt number, which decreases as the thermophoresis parameter increases. The minimum value can be attained at the stent treated stenotic part of the artery.

Figure 9 is a graphical representation of the velocity field in the porous region of the stent treated stenotic part. The velocity shows a slight decrement with the increasing Brownian motion parameter (N_b). The velocity field exhibits a noticeable difference near the inner wall of the stenosed segment but shows an equal decrement towards the outer wall of the artery for both values of N_b . A Higher value of the Brownian motion parameter enhances the mobility of the nanoparticles which promotes the ballistic collisions and momentum diffusion in the hemodynamic flow, resulting in flow acceleration. However, due to the increased resistance to blood flow as it moves from the inner wall to the outer wall in

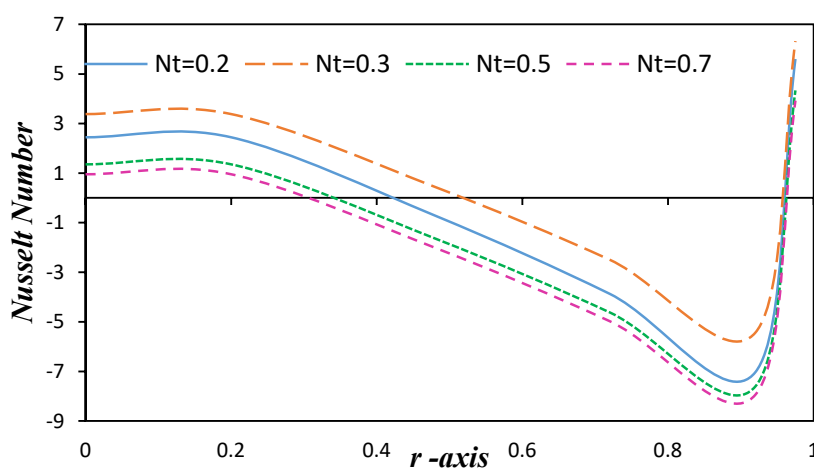


Figure 7. Effect of the thermophoresis parameter (N_t) on the Nusselt number $Nu' Re$ at the stenosis when $m = 0.657$, $Gr = 2.0$, $N = 0.4$, $N_b = 0.5$, and $z = 4.50$.

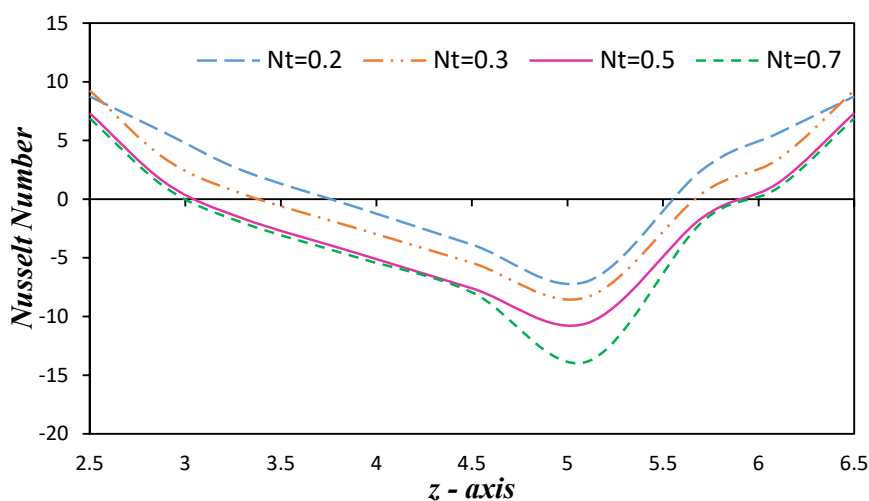


Figure 8. Effect of the thermophoresis parameter (N_t) on the Nusselt number $Nu' Re$ at the stenotic arterial segment when $m = 0.657$, $Gr = 2.0$, $N = 0.4$, and $N_b = 0.5$.

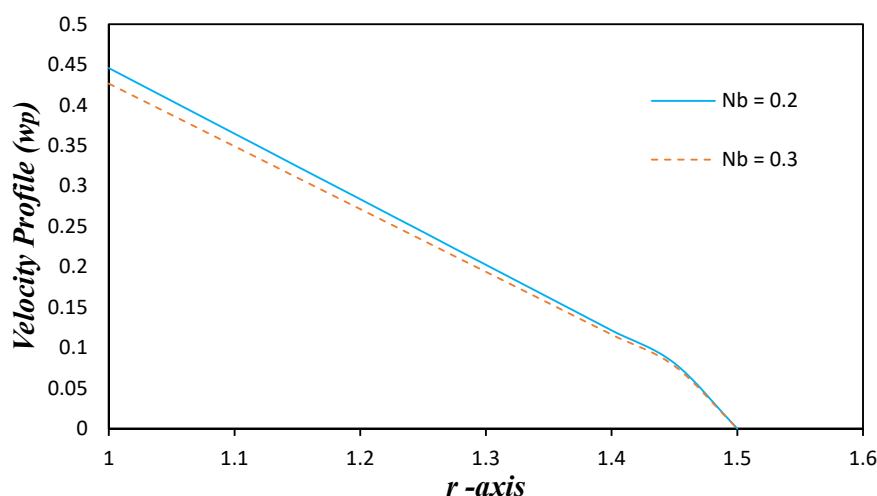


Figure 9. Effect of N_b on the velocity field at the stent treated stenotic arterial segment in the porous region for $m = 0.657$, $Gr = 2.0$, $N = 0.4$, $N_t = 0.3$, and $z = 4.50$.

the peripheral region, there is a pronounced velocity variation near the inner wall.

Effect of Sisko parameter (m)

Figure 10 provides a graphical representation of blood velocity field with respect to radial coordinate for various values of the Sisko parameter (m). **Figure 10** is plotted for the fixed values of the parameters: $Gr = 1.0$, $N = 0.2$, $N_t = 0.3$ and $N_b = 0.3$. The graph presented does not show a significant elevation in the velocity profile within the porous arterial region as the Sisko parameter (m) increases. However, a noticeable rise in velocity is observed near the inner wall of the artery as the Sisko parameter (m) increases from 0.357 to 0.957.

Effect of buoyancy ratio parameter (N)

The effect of solutal-to-thermal buoyancy ratio (N) on the hemodynamic velocity field in the core region is illustrated in **Figure 11**.

Figure 11 illustrates the evolution of the velocity field along the axial coordinate at a fixed radial coordinate ($r = 0.830$) within the core region (viscoplastic Casson fluid) under varying values of the thermal buoyancy ratio (N). It is evident at the stent treated stenosis segment that the velocity is elevated (lower z value), however, due to the presence of the stent, the velocity becomes more uniformly distributed, and the difference in velocity elevation is less pronounced compared to

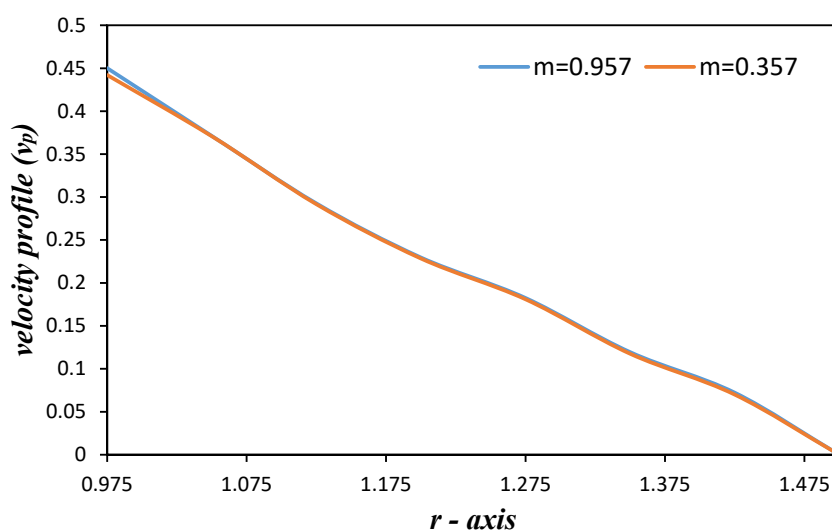


Figure 10. Effect of Sisko parameter (m) on the velocity field in the porous wall of the artery when $Gr = 1.0$, $N = 0.2$, $N_t = 0.3$, $N_b = 0.3$, and $z = 4.50$.

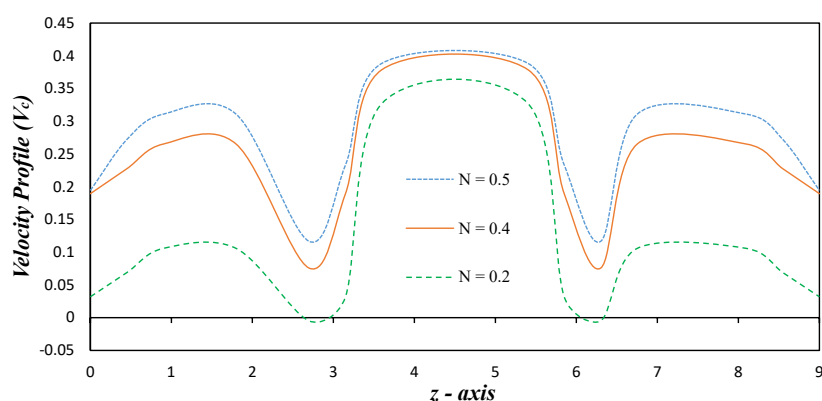


Figure 11. Effect of N on the velocity field in the core region when $N_b = 0.3$, $N_t = 0.3$, $Gr = 1.0$ and $r = 0.830$.

the non-stenotic arterial region. Increasing thermal buoyancy accelerates the flow only in the vicinity of the stenosis while causing significant deceleration elsewhere. The impact of thermal buoyancy is not consistent in the hemodynamic regime; therefore, care must be taken in deploying nanodrugs to ensure the desired effect is achieved whether at the entry zone, stenosis, or indeed any other location in the target zone for the treatment.

Finally, in **Figures 12** and **13**, to provide two-dimensional visualizations of the circulation hemodynamics in obstructed blood flow, contour plots have been presented, specifically for trapping. The results describe an interesting phenomenon for the blood flow pattern that is more closely associated with vorticity patterns than with conventional velocity profiles. In particular, they provide valuable insight into occlusion effects in hemodynamics, as represented by stenotic regions with

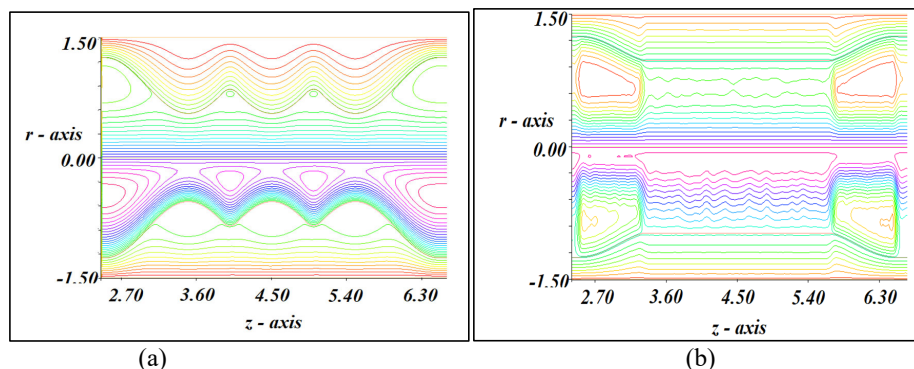


Figure 12. Streamlines in the stenotic region with $m = 0.657$, $Gr = 1.0$, $N = 0.2$, $N_t = 0.3$, and $N_b = 0.3$ for **(a)** without stent and **(b)** with stent.

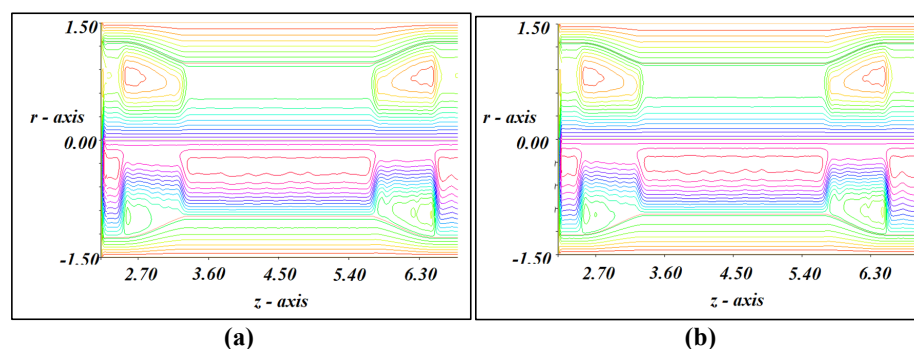


Figure 13. Streamlines in the stenotic region with stent when **(a)** $Gr = 1.0$, $N = 0.4$, $N_t = 0.3$, and $N_b = 0.3$ and **(b)** $Gr = 2.0$, $N = 0.4$, $N_t = 0.3$, and $N_b = 0.3$

and without stent features. **Figures 12a** and **12b** represents the streamlines of blood flow in the arterial domain at the stenosis without and with a stent respectively, while **Figures 13a** and **13b** illustrate the streamlines of blood flow at the stenotic region specifically within the core region of the artery. In **Figures 12a** and **12b**, it is observed that both the quantity and magnitude of the trapped bolus decrease with the use of a stent. However, in **Figures 13a** and **13b**, increasing the Grashof number (Gr) has a significant effect on the bolus structure, as the size of the bolus increases with higher Gr values at the stenosis when a stent is used.

Table 3 shows the effect of the Brownian motion parameter on the pressure distribution in the arterial segment. A noticeable decrease in pressure distribution is observed at the wall of the stent-treated stenotic region of the artery as the Brownian motion parameter increases, with the Grashof number ($Gr=2.0$) and buoyancy ratio ($N=0.4$) held constant.

Conclusion

The present study describes computational fluid dynamics modelling of steady-state, two-dimensional blood flow conveying spherical Gold (Au) nanoparticles in an arterial geometry with a porous wall featuring both multiple stenoses, with and without stent. The Casson fluid model and Sisko fluid model have been adopted for the core and porous regions, respectively, to mimic non-Newtonian effects and analyze the effects of Brownian motion and thermophoresis, with a natural convection model employed. The non-dimensional conservation equations for momentum, heat, and nanoparticle species, along with the appropriate boundary conditions, are solved numerically using the finite element method based on the variational approach and simulated with the FreeFEM++ code. Modifications in heat and mass transfer characteristics, as well as hemodynamic velocity in the porous region, are studied for the effects of geometric, nanoscale, rheological,

Table 3. Effect of Brownian motion parameter (N_b) on pressure distribution at inner wall of the stenotic artery segment for fixed values of the thermophoresis parameter, Grashof number and Buoyancy parameter.			
z - axis	$Gr = 2.0, N = 0.4, \text{ and } N_t = 0.3.$		
	$N_b = 0.2$	$N_b = 0.3$	$N_b = 0.5$
2.50	0.56396	0.48954	0.48793
2.70	0.5225	0.52081	0.51918
2.90	0.5225	0.48954	0.51918
3.10	0.48104	0.52081	0.1918
3.30	0.43968	0.45827	0.45668
3.50	0.60542	0.48954	0.48793
3.70	0.60542	0.48954	0.48793
3.90	0.60542	0.48954	0.48793
4.10	0.60542	0.48954	0.48793
4.30	0.60542	0.48954	0.48793
4.50	0.60542	0.48954	0.48793
4.70	0.60542	0.48954	0.48793
4.90	0.60542	0.48954	0.48793
5.10	0.60542	0.48954	0.48793
5.30	0.60542	0.48954	0.48793
5.50	0.60542	0.48954	0.48793
5.70	0.5225	0.52081	0.45668
5.90	0.5225	0.52081	0.51918
6.10	0.48104	0.45827	0.51918
6.30	0.64688	0.52081	0.51918
6.50	0.72981	0.55207	0.55043

and viscosity parameters, including arterial geometry with a stent, the thermophoresis parameter (N_t), Brownian motion parameter (N_b), Sisko parameter (m) and Buoyancy ratio parameter (N) at the throat of the stenosis and throughout the domain. The main outcomes from the present investigation are as follows:

- The study observed that in the porous region, as the Sisko parameter (m) increases, the hemodynamic velocity field shows a slight increment near the inner wall of the artery at the throat of the stenosis.
- At the stent-treated stenotic arterial segment, the Nusselt number initially increases with an increase in the thermophoresis parameter but gradually decreases at higher values of thermophoresis parameter.
- Moving from the centerline to boundary of the arterial segment, the Nusselt number shows decrement while increasing the thermophoresis parameter but shows sudden increment near the inner wall of the artery.
- The pressure distribution decreases in the core region as the Brownian motion parameter increases at the wall of the stent-treated stenotic part of the arterial segment. It also shows that the pressure is evenly distributed along the arterial wall when using the stent to treat multiple stenoses.
- The velocity field does not show significant variation as the Brownian motion parameter increases in the porous region of the stent-treated stenosis.
- The velocity field shows different behavior in the core region as the Buoyancy ratio parameter (N) increases, with minimal variation observed when moving from the non-stenotic arterial segment toward the stent-treated stenosis region.
- The Bolus structure and magnitude exhibit greater sensitivity to the application of stent. The stent may provide a more useful mechanism for treatment than extracorporeal magnetic fields in cardiovascular diagnostics and clinical treatments.

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