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COMPUTATIONAL FLUID DYNAMIC SIMULATION OF TWO-FLUID NON-NEWTONIAN NANO-HEMODYNAMICS THROUGH A DISEASED ARTERY WITH A STENOSIS AND ANEURYSM

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ABSTRACT

This article presents a two-dimensional theoretical study of hemodynamics through a diseased permeable artery with a mild stenosis and an aneurysm present. The effect of metallic nanoparticles on the blood flow is considered, motivated by drug delivery (pharmacology) applications. Two different models are adopted to mimic non-Newtonian characteristics of the blood flow; the Casson (viscoplastic) fluid model is deployed in the core region and the Sisko (viscoelastic) fluid model employed in the peripheral (porous) region. The revised Buongiorno two-component nanofluid model is utilized for nanoscale effects. The blood is considered to contain a homogenous suspension of nanoparticles. The governing equations are derived by extending the Navier-Stokes equations with linear Boussinesq approximation (which simulates both heat and mass transfer). Natural (free) double-diffusive convection is considered to simulate the dual influence of thermal and solutal buoyancy forces. The conservation equations are normalized by employing appropriate non-dimensional variables. The transformed equations are solved numerically using the finite element method with the variational formulation scheme available in the FreeFEM++ code. A comprehensive mesh-independence study is included. The effect of selected parameters (thermophoresis, Brownian motion, Grashof number, thermo-solutal buoyancy ratio, Sisko parameter ratio and permeability parameter) on velocity, temperature, nanoparticle concentration
and hemodynamic pressure have been calculated for two clinically important cases of arteries with a stenosis and an aneurysm. Skin-friction coefficient, Nusselt number, volumetric flow rate and resistance impedance of blood flow are also computed. Colour contours and graphs are employed to visualize the simulated blood flow characteristics. It is observed that by increasing thermal buoyancy parameter i.e. Grashof number \((Gr)\), the nanoparticle concentration and temperature decrease whereas velocity increases with an increment in Brownian motion parameter \((Nb)\). Furthermore, velocity decreases in the peripheral porous region with elevation in the Sisko material ratio \((m)\) and permeability parameter \((k^\prime)\). The simulations are relevant to transport phenomena in pharmacology and nano-drug targeted delivery in haematology.

**Keywords:** Arterial stenosis, aneurysm, Casson fluid model, Sisko fluid model, Nano-drugs, Porous walls, Thermophoresis, Finite Element Method, FreeFEM++.

**Nomenclature:**

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
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<tbody>
<tr>
<td>(D_b)</td>
<td>Brownian diffusion coefficient</td>
</tr>
<tr>
<td>(D_r)</td>
<td>Thermophoretic diffusion coefficient</td>
</tr>
<tr>
<td>(g)</td>
<td>Gravitational vector</td>
</tr>
<tr>
<td>(G_r)</td>
<td>Grashof Number</td>
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<tr>
<td>(Re)</td>
<td>Reynolds Number</td>
</tr>
<tr>
<td>(Pr)</td>
<td>Prandtl Number</td>
</tr>
<tr>
<td>(S_c)</td>
<td>Schmidt Number</td>
</tr>
<tr>
<td>(R(z))</td>
<td>Radius of artery</td>
</tr>
<tr>
<td>(r)</td>
<td>Radial coordinate</td>
</tr>
<tr>
<td>(z)</td>
<td>Axial coordinate</td>
</tr>
<tr>
<td>(u)</td>
<td>Velocity in radial direction</td>
</tr>
<tr>
<td>(w)</td>
<td>Velocity in axial direction</td>
</tr>
<tr>
<td>(N)</td>
<td>Ratio of thermal to buoyancy forces</td>
</tr>
<tr>
<td>(Nb)</td>
<td>Brownian motion parameter</td>
</tr>
<tr>
<td>(Nt)</td>
<td>Thermophoresis parameter</td>
</tr>
<tr>
<td>(p)</td>
<td>Pressure</td>
</tr>
<tr>
<td>(Q)</td>
<td>Volumetric flow rate</td>
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<tr>
<td>(\lambda)</td>
<td>Resistance Impedance</td>
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<tr>
<td>(Nu)</td>
<td>Nusselt Number</td>
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<tr>
<td>(C_f)</td>
<td>Skin-friction Coefficient</td>
</tr>
<tr>
<td>(m)</td>
<td>Sisko material Parameter</td>
</tr>
<tr>
<td>(T)</td>
<td>Temperature</td>
</tr>
<tr>
<td>(T_w)</td>
<td>Temperature at wall</td>
</tr>
<tr>
<td>(T_{ref})</td>
<td>Reference temperature</td>
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**Greek Letters:**

<table>
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<tr>
<th>Symbol</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>(\mu_1)</td>
<td>Asymptotic value of viscosity at very high rate</td>
</tr>
<tr>
<td>(\phi)</td>
<td>Nanoparticle volume fraction</td>
</tr>
<tr>
<td>(\mu_0)</td>
<td>Consistency</td>
</tr>
<tr>
<td>(\rho_f)</td>
<td>Density of the base fluid</td>
</tr>
<tr>
<td>(\delta)</td>
<td>Stenosis depth</td>
</tr>
<tr>
<td>(\kappa)</td>
<td>Thermal conductivity</td>
</tr>
<tr>
<td>(\rho_p)</td>
<td>Density of the nanoparticles</td>
</tr>
<tr>
<td>(\phi_w)</td>
<td>Concentration at wall</td>
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1. INTRODUCTION

Based on the reports of numerous physiological studies, the primary cause of mortality worldwide is due to diseases in the blood vessels and the heart including heart attacks and strokes. According to the data of the World Health Organization (WHO), the percentage of deaths due to non-communicable diseases is 68% from which one-third are attributable to cardiovascular diseases [1]. Cardiovascular disease includes coronary heart disease (CHD), cerebrovascular disease, rheumatic heart disease and numerous other ailments. The cause of these events is the formation of plaque in the lumen, known as atherosclerosis, and manifests as a stenosis which can grow and block the artery and hence prevent blood supply to the distant body cells. Plaques with calcium in them can also rupture and initiate the formation of blood clotting [2]. Blood is an immensely complex, aqueous polymeric and ionic solution (concentrated suspension) containing several cellular elements and these elements include erythrocytes, leukocytes, and platelets (thrombocytes) in the plasma. Plasma which generally behaves as a Newtonian fluid [3], is composed of water (93%) and electrolytes, organic molecules, numerous proteins (3%), and waste products. The non-Newtonian properties of blood can be seen in the small arteries due to low shear rates. At high shear rate blood usually behaves like a Newtonian fluid as observed in large arteries [4, 5]. A study done by Schmidt-Schonbein et al. [6], shows that blood component interaction (e.g. erythrocytes and leukocytes) in the capillaries is the primary cause of non-Newtonian behaviour of blood (biorheology). Through a complex network of arteries, veins, and capillaries, blood flows to the body organs and body cells. The motion of blood is due to continuous pumping by the heart; deoxygenated blood is transported to the heart from the organs through veins and the heart pumps oxygenated blood to the whole body through the arteries. 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 [7]. A significant number of studies have been reported by considering blood as a Newtonian fluid. Relevant examples include Bluestein et al. [8] and Young and Tsai [9] studied the hydrodynamic characteristics in a healthy blood vessel by considering either laminar or turbulent behaviour of the flow. More recently non-Newtonian hemodynamics has been addressed by a number of investigators. Reddy et al. [10]
studied the blood flow by treating the blood as a high impedance couple stress fluid, showing that significant deviation in flow characteristics arise compared with the classical Newtonian model. Several investigators have however analysed theoretically in detail the contribution of blood rheology to coronary artery disease and cerebral aneurysms. Agrawal et al. [11] studied the shear-thinning characteristics of blood with a Carreau–Yasuda Model, for coil embolization as a mildly invasive endovascular method for treatment of a cerebral aneurysm. They observed that the blood rheology exerts a prominent role in the performance of the coil which is aimed at reducing fluid loading of the blood vessel and delaying subsequent wall deformation. Priyadharshini and Ponalagusamy [12] used a finite difference computational method to simulate time-dependent magneto-hemodynamics in a tapered arterial stenosis with variable viscosity, considering blood in the core region as viscoelastic Jeffrey fluid and plasma in the peripheral layer as Newtonian. They confirmed that the results with blood rheology included better correlate with experimental data.

Arterial blood flow is fundamental to the human circulatory system, and the presence of arterial stenosis adversely influences the health of the cardiovascular system [13]. The essential contributions related to heart diseases (stenosis) via diagnostic tools and simulations of chirurgical treatments such as stents and by-passes, has mobilized strong interest in biologists, medical engineers and scientists in this area [14]. Riahi et al. [15] studied the blood flow in an artery containing an overlapping stenosis. Chakravarty et al. [16] and Mekheimer and El Kot, [17] each considered hemodynamics in a time-variant tapered stenotic artery. Akbar et al. [18] employed the Jeffrey rheological model for blood flow through a tapered artery with a stenosis to study non-Newtonian fluid characteristics. Ellahi et al. [19] simulated the arterial blood flow through a mild composite stenosis with Eringen’s micropolar model. Razavi et al. [20] compared various viscosity models to the Newtonian model for pulsatile hemodynamics in a stenosed carotid artery, noting that with greater degree of stenosis there is increased disturbance induced in the downstream flow of the stenosis and wall shear stress (WSS) which develops at the stenosis throat noticeably. Karimietal [21] implemented the Carreau and modified power-law non-Newtonian models in blood flow via common stenosed carotid arteries and compared the simulation results with the experimental data to highlight the influence of rheology on hemodynamic characteristics.

An aneurysm is a balloon-like dilation usually found on the walls of blood arteries including the abdominal artery, cerebral artery, carotid artery, thoracic artery, femoral artery, etc. It develops gradually and grows faster as time passes [22]. The role of hemodynamics in the growth of aneurysms has been the subject of several studies. Mukhopadhyay et al. [23] modelled blood flow in an artery featuring a local aneurysm in the presence of haematocrit. Kumar et al. [24],
investigated the pulsatile blood flow characteristics using a Saffman particle-fluid suspension two phase model and determined the flow, pressure and shear stress distributions in an artery with an aneurysm. Bluestein et al. [25] employed both computational fluid dynamics and digital particle Image Velocimetry (DPIV) to study both laminar and turbulent hemodynamics in an artery with aneurysm and identified the formation of a recirculation zone inside the aneurysm cavity which induces thrombus formation and increases the chances of rupture. As noted above a variety of different non-Newtonian models have been applied to stenotic and aneurysm-modified hemodynamics. An alternative model is the viscoplastic Casson fluid model. This model predicts an infinite viscosity at zero shear rate, and a constant viscosity at an infinite rate of shear. It is a robust shear-thinning fluid model and has been explored in several areas of hemodynamics such as blood flow in regular and constricted tubes. Casson [26] initially investigated the validity of this model by studying the flow characteristics of blood observed that at low shear rates, the yield stress for blood is non-zero. Sarifuddin et al. [27] 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. Blair [28] and Copley [29] established in their studies that for representation of the simple shear behaviour of blood in narrow arteries, 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. It is a relatively simple non-Newtonian model which provides robust predictions for high shear rate flows and is a modification of the power-law model. Originally introduced for lubricants, the Sisko model has been adopted in a number of hemodynamic investigations. Haghighi and Chalak [30] used the Sisko rheological model to simulate blood flow in a stenotic artery with body acceleration effects and a finite difference numerical method. Zaman et al. [31] presented a non-linear unsteady blood flow model for a stenosed elastic vessel with the Sisko model. Mekheimer et al. [32] developed an unsteady blood flow model for anisotropic elastic tapered arteries with time-variant overlapping stenosis. Ali et al. [33] showed that the Sisko fluid model is appropriate for medium and high shear rate scenarios and quite accurately mimics several hemo-rheological characteristics.

An additional feature of real arteries is the permeability of the artery wall. This 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 [34], and Mishra et al. [35], studied the 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 in
blood flow include Jain et al. [36] (for stenosed arteries), Tripathi [37] (magnetic blood flow in an inclined artery), Eldesoky [38] (who included slip effects and body acceleration) and Srivastav [39] (for Newtonian blood in a catheterized stenosed artery). The expansion of nanotechnology has revolutionized biomedicine in the 21st century. Many new nanomaterials have also been implemented in a variety of areas in biomechanics and biofluid mechanics. A sub-set of nanomaterials (which include carbon nanotubes, nanoshells, nanorods and nanowires) is nanofluids. Pioneered by Choi and Eastman [40] at Argonne Energy Laboratory in the USA, nanofluids are engineered colloids which are synthesized by suspending metallic or non-metallic nanoparticles in a base fluid. The resulting suspension achieves improved thermal characteristics compared with conventional working fluids in engineering. Although initially developed for automotive and thermal power applications, nanofluids have been successfully utilized in an astonishing range of clinical applications. These have included both theoretical and experimental studies, largely based on the Buongiorno [41] and revised Buongiorno [42,43] model which is a two-component nanoscale formulation or the Tiwari-Das model [44] which is a volume fraction-based doping model (based on the Maxwell-Garnett theory). Improvement in the thermal conductivity of conventional fluids (e.g. water, refrigerant, ethyl glycol and engine oil) has become a prominent area of intensive activity with the objective of enhancing heat transfer characteristics (thermal conductivity, heat transfer coefficient, etc.) and nanoparticles playing an essential role in improving the heat transfer. Buongiorno [41] has presented a comprehensive study of convective transport in nanofluids. Nanofluids display substantial enhancement of thermal conductivity, which is boosted with increasing volumetric fraction of nanoparticles as reported by Masuda et al. [45]. Farooq et al. [46] and Hayat et al. [47] studied stagnation point heat and mass transfer in nanofluid flow over a non-linear stretching sheet. Other heat and mass transfer investigations have considered different nanoparticles and studied temperature and nanoparticle concentration [48]. Thermophoresis and Brownian motion aspects have also been addressed in thermosolutal nanoparticle diffusion from different geometries [49-50]. The classical models are not applicable for the enhancement of thermal conductivity of nanofluids. In recent years, most developed models only include one or two postulated mechanisms of nanofluid thermophysics. For example, there has been a sparsity in fundamental work reported for the determination of the effective thermal diffusivity or heat transfer coefficients for nanofluids in natural convection [51].

Interesting examples of the deployment of nanofluids in medicine include blood purification systems [52], smart bio-nano-polymer coatings for medical devices [53], nano-drug delivery (pharmacological systems) in cardiovascular treatment [54, 55], biodegradable nanoliquids for
cerebral pharmaco-dynamics [56], membrane oxygenator bioreactors [57], orthopaedic lubrication with nano-films (superlubricated poly (3-sulfopropyl methacrylate potassium salt)-grafted mesoporous silica nanoparticles suspended in starch base liquids) [58], pulsed laser ablation (PLA) ultra-pure silicon nanofluid fabrication for cancer therapy [59], smart biomimetic electro-osmotic nanofluid pumps in ocular diagnosis [60], cryopreservation, bone reinforcement via super-paramagnetic nanofluids etc. In hemodynamic therapies, the base liquid is clearly blood and can be doped with a variety of nanoparticles including gold. To prevent in-stent restenosis and to improve the performance of current stents, different nanomaterial coatings, and controlled-release nano-carriers are used [61]. Nano-carriers have the potential for delivery of imaging and diagnostic agents to precisely targeted destinations. Current therapies focus on decreasing the burden of atherosclerotic plaque and stabilizing vulnerable plaques defined as those plaques, which have a tendency to rupture and cause thrombosis [62]. A new type of stent has been designed in recent years and is known as the drug-eluting stent (DES), which reduces the risk of in-stent thrombosis and restenosis caused by bare-metal stents. Different classes with a wide range of drugs have been tested with DES to prevent smooth muscle cell (SMC) growth and proliferation, anti-cancer, and anti-inflammatory agents. Giljohann et al. [63], and Kumar et al. [64], investigated the effect of gold nanoparticles suspension in blood. Ali et al. [65] presented a comprehensive computational model for studied unsteady heat and mass transfer in nanoparticle doped streaming blood flow via a tapered stenotic artery using the Buongiorno model.

A close inspection of the scientific literature has shown that relatively few works have been conducted for hemodynamic transport in porous-walled arteries featuring either a constriction (stenosis) and/or dilation (aneurysm). Furthermore, very little research has appeared considering thermophysical/mass diffusion aspects (e.g. thermo-solutal buoyancy) of obstructed hemodynamics with nanoscale models. Also, the modification in blood pressure distribution in nano-pharmacodynamics has not been extensively studied. Hence the novelty of the present study lies in combining the effect of geometric complexities, non-Newtonian (hemo-rheological behaviour), thermophysics and nanoscale parameters on hemodynamic transport characteristics in obstructed nano-doped blood flow in an artery with porous walls. A mathematical model is developed for steady rheological blood flow, heat transfer and mass (nanoparticle) diffusion in an artery with porous wall featuring both a stenosis and aneurysm (fusiform). The Casson viscoplastic fluid model [66] is deployed in the core part of the artery and a Sisko viscoelastic fluid model [67] is employed in the peripheral region to mimic non-Newtonian characteristics. With appropriate boundary conditions, the non-dimensional transformed conservation equations are solved with the
finite element method using a variational approach based on the FreeFem++ code [68]. Mesh-independence is included. The present simulations are relevant to nano-pharmaco-dynamics and provide a deeper understanding of the complex mechanisms inherent to nano-particle drug treatments for cardiovascular diseases.

2. BIO-RHEOLOGICAL NANO-PHARMACODYNAMIC MODEL

Consider the two-dimensional, laminar, incompressible steady (non-pulsating) hemodynamic flow in an artery with a porous wall. Here the core and peripheral regions are the lumen and porous arterial wall respectively and for convenience the following convention is used throughout the paper - core region (lumen) and peripheral region (porous arterial wall). Blood is simulated as a non-homogeneous fluid with dispersed uniform nanoparticles. Non-Newtonian characteristics are treated with a dual rheological formulation- namely a Casson fluid model in the core region and a Sisko fluid model in the peripheral region. Thermosolutal flow is considered i.e. both heat and mass (nanoparticle) diffusion are incorporated with associated buoyancy effects. A cylindrical coordinate system \((r, \varphi, z)\) is employed, where \(r\) is the radial coordinate, \(z\) is axial coordinate and \(\varphi\) is azimuthal coordinate. Axisymmetric flow is studied and therefore contribution in the azimuthal \((\varphi)\) direction is ignored i.e., the flow is only in the radial \((r)\) and axial \((z)\) direction as depicted in Fig. (1). A finite length arterial geometry is studied which contains a sinusoidal-shaped stenosis with a fusiform aneurysm. In the arterial segment, the geometry of the outer layer of the mild stenosis and aneurysm is assumed to be described by:

\[
R_a(z) = \begin{cases} 
-R_p + a \exp\left(-\frac{1}{2a^2}(z - \frac{l}{2} + 0.5)^2\right); & L \leq z \leq L + L_0 \text{ (Stenosis)} \\
-R_p - (a - 0.3) \exp\left(-\frac{1}{2a^2}(z - \frac{l}{2} + 0.5)^2\right); & L + L_2 \leq z \leq L + L_2 + L_0 \text{ (Aneurysm)} \\
R_p & \text{otherwise}
\end{cases}
\]  

The geometry of inner layer of the mild stenosis and aneurysm are simulated with the following Mathematical relations:

\[
R(z) = \begin{cases} 
-R_c + a \exp\left(-\frac{1}{2a^2}(z - \frac{l}{2} + 0.5)^2\right); & L \leq z \leq L + L_0 \text{ (Stenosis)} \\
-R_c - (a - 0.3) \exp\left(-\frac{1}{2a^2}(z - \frac{l}{2} + 0.5)^2\right); & L + L_2 \leq z \leq L + L_2 + L_0 \text{ (Aneurysm)} \\
R_c & \text{otherwise}
\end{cases}
\]
Here the following notation applies: radius of the artery, radius of the core region of the nonstenotic section and length of the diseased part of the artery are $R_p, R_c$ and $L$, respectively. The radius of the inner layer of the artery is taken as $R(z)$ and radius for the outer layer of the artery is taken as $R_i(z)$. The $z$-axis is orientated in the blood flow direction and normal to the $r$-axis. The stenosis is symmetrical and has a maximum height $\delta$ with $\alpha = \frac{9}{10}\delta$. Fig. 1 also visualizes the erythrocytes (red cells) and spherical nanoparticles (drug) suspended in the blood flow in the artery.

**Figure 1.** Physical model for biorheological nano-pharmacodynamic flow in an artery with stenosis and aneurysm

For steady axisymmetric flow of blood in the arterial vessel, the velocity vector $V$ is assumed to be of the form:

For the core region (Casson fluid model)

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

(3)

For the peripheral (porous) region (Sisko fluid model):

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

(4)

Here $u_c, u_p$ and $w_c, w_p$ are the velocity components in radial and axial directions for both core and porous region. Blood is considered to be an incompressible non-Newtonian fluid (Casson fluid in the core region and Sisko fluid in the peripheral region) containing a homogenous distribution of nano-particles.
Since natural (free) convection is considered, which mobilizes buoyancy forces, the appropriate expression under the Boussinesq’s approximation, the nanofluid density is:

\[
\rho = \rho_f \left[1 - \beta_f (T - T_L) - \beta_s (\phi - \phi_L)\right]
\]  

Here \( \rho_f \) is the base fluid’s density at the reference fluid temperature \( T_L \) and reference concentration \( \phi_L \), \( \beta_f \) and \( \beta_s \) are the thermal and solutal expansion coefficients respectively.

The governing equations for conservation of mass, momentum, thermal energy and nanoparticle volume fraction (species) for the nano-pharmacodynamic flow in vectorial form are as follows:

**For core region:**

\[
\nabla \cdot V_c(r, z) = 0
\]

\[
\rho_f \left( \frac{\partial V_c}{\partial t} + (V_c \cdot \nabla) V_c \right) = \text{div} \tau_c + \left[ \rho_f (1 - \beta_f (T - T_L) - \beta_s (\phi - \phi_L)) \right] g
\]

\[
(r \rho_c) \left( \frac{\partial T}{\partial t} + (V_c \cdot \nabla) T \right) = k \nabla^2 T + (r \rho_c) \left[ D_b (\nabla \phi \cdot \nabla T) + \frac{D_L}{T_L} (\nabla T \cdot \nabla T) \right]
\]

\[
\frac{\partial \phi}{\partial t} + (V_c \cdot \nabla) \phi = D_b \nabla^2 \phi + \frac{D_L}{T_L} \nabla^2 T
\]

Here \( \phi \) is the nanoparticle volume fraction (e. g. gold).

The constitutive equation for the Cauchy stress tensor in a Casson viscoplastic fluid is given by [66]:

\[
\sqrt{\tau_i} = \left\{ \begin{array}{ll}
\sqrt{\tau_i} & \text{when } \tau > \tau_c \\
\left| \mu \left( \frac{\partial w_c}{\partial r} \right) \right|^{1/2} & \text{when } \tau < \tau_c \\
\end{array} \right.
\]

Where \( \tau_c \) is yield stress and \( \mu \) is the viscosity of blood.

**For peripheral region:**

\[
\nabla \cdot V_p(r, z) = 0
\]

\[
\rho_f \left( \frac{\partial V_p}{\partial t} + (V_p \cdot \nabla) V_p \right) = \text{div} \tau_c + \frac{\mu V_p}{k'}
\]

Where \( k' \) is the permeability parameter, and the Cauchy stress tensor for the Sisko fluid is:

\[
\tau_i = -pI + S
\]
In Eq. (11), due to the restraint of incompressibility, \(-pI\) represents the spherical stress and \(S\) will be defined as [58]:

\[
S = \left( \mu + \mu_0 \right) \sqrt{\gamma^{-1}} A_1
\]  

(14)

Where \(n, \mu, \mu_0\) are material parameters (which vary for different fluids). The kinematical tensor \(A_1\) can be defined by the following equation

\[
A_i = \nabla V_r + \left( \nabla V_r \right)^T
\]  

(15)

And

\[
\dot{\gamma} = \frac{1}{2} \text{tr}(A_i)^2
\]  

(16)

Eqns. (6) - (9) i.e. the hemodynamic transport equations in the core region \((-c\)) effectively emerge in the following form:

\[
\frac{\partial u_r}{\partial r} + \frac{u_r}{r} + \frac{\partial w_z}{\partial z} = 0
\]  

(17)

\[
\rho_f \left[ u_r \frac{\partial u_r}{\partial r} + w_z \frac{\partial u_r}{\partial z} \right] = - \frac{\partial p}{\partial r} + \frac{1}{r} \frac{\partial}{\partial r} \left(rS_{rr}\right) + \frac{\partial}{\partial z} \left(S_{rz}\right)
\]  

(18)

\[
\rho_f \left[ u_z \frac{\partial w_z}{\partial r} + w_z \frac{\partial w_z}{\partial z} \right] = - \frac{\partial p}{\partial r} + \frac{1}{r} \frac{\partial}{\partial r} \left(rS_{zz}\right) + \frac{\partial}{\partial z} \left(S_{zz}\right)
\]  

(19)

\[
(c\rho) \left[ u_r \frac{\partial T}{\partial r} + w_z \frac{\partial T}{\partial z} \right] = \kappa \left( \frac{\partial^2 T}{\partial r^2} + \frac{1}{r} \frac{\partial T}{\partial r} + \frac{\partial^2 T}{\partial z^2} \right) + (c\rho) \left[ \frac{\partial \phi}{\partial r} \frac{\partial T}{\partial r} + \frac{\partial \phi}{\partial z} \frac{\partial T}{\partial z} \right]
\]  

(20)

\[
\left[ u_r \frac{\partial \phi}{\partial r} + w_z \frac{\partial \phi}{\partial z} \right] = D_b \left( \frac{\partial^2 \phi}{\partial r^2} + \frac{1}{r} \frac{\partial \phi}{\partial r} + \frac{\partial^2 \phi}{\partial z^2} \right) + D_b \left( \frac{\partial^2 T}{\partial r^2} + \frac{1}{r} \frac{\partial T}{\partial r} + \frac{\partial^2 T}{\partial z^2} \right)
\]  

(21)

The mass and momentum Eqns. in the peripheral region \((-p\)), assume the form:

\[
\frac{\partial u_r}{\partial r} + \frac{u_r}{r} + \frac{\partial w_p}{\partial z} = 0
\]  

(22)

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

(23)

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

(24)
Here the Cauchy stress tensors are defined as follows:

\[
S_{rr} = -2 \left( \mu_1 + \mu_0 \right) \left\{ \left( \frac{\partial u_\rho}{\partial r} \right)^2 + \left( \frac{u_\rho}{r} \right)^2 + \left( \frac{\partial w_\rho}{\partial z} \right)^2 + \left( \frac{\partial w_\rho}{r} + \frac{\partial u_\rho}{\partial z} \right)^2 \right\} \left( \frac{\partial u_\rho}{\partial r} \right) \] (25)

\[
S_{zz} = -2 \left( \mu_1 + \mu_0 \right) \left\{ \left( \frac{\partial u_\rho}{\partial r} \right)^2 + \left( \frac{u_\rho}{r} \right)^2 + \left( \frac{\partial w_\rho}{\partial z} \right)^2 + \left( \frac{\partial w_\rho}{r} + \frac{\partial u_\rho}{\partial z} \right)^2 \right\} \left( \frac{\partial w_\rho}{\partial z} \right) \] (26)

\[
S_{rz} = - \left( \mu_1 + \mu_0 \right) \left\{ \left( \frac{\partial u_\rho}{\partial r} \right)^2 + \left( \frac{u_\rho}{r} \right)^2 + \left( \frac{\partial w_\rho}{\partial z} \right)^2 + \left( \frac{\partial w_\rho}{r} + \frac{\partial u_\rho}{\partial z} \right)^2 \right\} \left( \frac{\partial w_\rho}{r} + \frac{\partial u_\rho}{\partial z} \right) \] (27)

The associated boundary conditions are prescribed as follows:

\[
u_e = u_\rho = 0, \quad w_e = U_0, \quad \frac{\partial w_\rho}{\partial r} = 0, \quad \frac{\partial T}{\partial r} = 0, \quad \frac{\partial \phi}{\partial r} = 0 \quad \text{at} \ r = 0
\]

\[
w_e = w_\rho \quad \text{at} \ r = R(z)
\]

\[w_\rho = 0 \quad \text{at} \ r = R_s(z) \quad \text{(no-slip condition)}
\]

\[
\frac{D_e}{T_e} \frac{\partial T}{\partial r} + D_b \frac{\partial \phi}{\partial r} = 0 \quad \text{at} \ r = R(z)
\]

\[T = T_w \quad \text{at} \ r = R(z)
\]

The wall shear stress at the \textit{inner} and the \textit{outer} wall of the artery are defined as:

\[
WSS_{\text{inner}} = - \sqrt{\tau_e + \left( \mu \frac{\partial w_\rho}{\partial r} \right)^{\frac{1}{2}}} \] (29)

\[
WSS_{\text{outer}} = - \left( \mu_1 + \mu_0 \right) \left\{ \left( \frac{\partial u_\rho}{\partial r} \right)^2 + \left( \frac{u_\rho}{r} \right)^2 + \left( \frac{\partial w_\rho}{\partial z} \right)^2 + \left( \frac{\partial w_\rho}{r} + \frac{\partial u_\rho}{\partial z} \right)^2 \right\} \left( \frac{\partial w_\rho}{r} + \frac{\partial u_\rho}{\partial z} \right) \] (30)

The heat flux is:

\[
q_e = -k \left( \frac{\partial T}{\partial r} \right)
\] (31)

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

\[
C_f = \frac{2\tau_w}{\rho_f U_0^2} \quad \text{and} \quad Nu = \frac{zq_e}{k(T_w - T_L)}
\] (32)
The volumetric flow rate takes the form:

\[
Q = \left[ \int_0^R rw_c \, dr + \int_r^p rw_p \, dr \right]
\]  

(33)

The flow resistance \( \lambda \) (impedance) will be expressed as:

\[
\lambda = \frac{L}{Q} \int_0^R \left( \frac{\partial p}{\partial z} \right) \, dz
\]  

(34)

To facilitate a numerical solution of the boundary value problem, it is pertinent to render the derived model as dimensionless. In this spirit, the following non-dimensional variables and parameters are introduced:

\[
\bar{w} = \frac{uL_0}{U_0 \delta}, \quad \bar{w} = \frac{w}{U_0}, \quad \bar{z} = \frac{z}{L_0}, \quad \bar{r} = \frac{r}{R_p}, \quad \bar{\mu} = \frac{\mu}{\mu_0}, \quad \bar{\mu}_0 = \mu_0 \left( \frac{U_0}{R_p} \right)^{n-1}, \quad \bar{r} = \frac{U_0}{\mu U_0} \tau
\]

\[
\bar{\theta} = \frac{T - T_w}{T_u - T_L}, \quad \bar{\phi} = \frac{\phi - \phi_L}{\phi_w - \phi_L}, \quad \bar{\varphi} = \frac{R_p}{U_0 \bar{\mu}_0 L_0}, \quad \bar{R}(z) = \frac{R(z)}{R_p}, \quad \bar{\lambda} = \frac{\lambda R_p^4}{\bar{\mu}_0 L_0}, \quad \tau = \frac{R_p}{\mu U_0} \tau' \quad \text{(35)}
\]

\[
\bar{R}_1(z) = \frac{R(z)}{R_p}, \quad \bar{S}_\gamma = \frac{S_{\alpha} L_0}{U_0 \bar{\mu}_0}, \quad \bar{S}_\iota = \frac{S_{\iota} L_0}{U_0 \bar{\mu}_0}, \quad \bar{S}_\iota' = \frac{S_{\iota'} R_p}{U_0 \bar{\mu}_0}, \quad \bar{r}_0 = \frac{R_p}{R_c}, \quad \bar{Q} = \frac{Q}{R_p^2 U_0}
\]

Where \( U_0, L_0, R_p, \mu_0, \delta, T_u, T_L, \phi_L \) and \( \phi_w \) 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. Implementing Eqns. (35) in eqns. (17)-(21), (22)-(24) and (29)-(34) the following system of dimensionless conservation equations are produced:

For Core region (Casson viscoplastic Fluid):

\[
\begin{align*}
\text{Re} \left[ \delta' \epsilon^4 \bar{u}_c \frac{\partial \bar{u}_c}{\partial \bar{r}} + \epsilon' \epsilon^4 \bar{w}_c \frac{\partial \bar{u}_c}{\partial \bar{z}} \right] &= - \frac{\bar{\varphi}}{\bar{r}} + \epsilon^2 \frac{1}{\bar{r}} \frac{\partial}{\partial \bar{r}} \left( \bar{S}_\iota' \right) + \epsilon^2 \frac{\partial}{\partial \bar{z}} \left( \bar{S}_\iota \right) \\
\text{Re} \left[ \delta' \epsilon^2 \bar{u}_c \frac{\partial \bar{w}_c}{\partial \bar{r}} + \epsilon' \epsilon^2 \bar{w}_c \frac{\partial \bar{w}_c}{\partial \bar{z}} \right] &= - \frac{\bar{\varphi}}{\bar{z}} + 1 \frac{\partial}{\partial \bar{r}} \left( \bar{S}_\iota' \right) + \epsilon^2 \frac{\partial}{\partial \bar{z}} \left( \bar{S}_\iota \right) + Gr \left( \bar{\theta} + N \bar{\phi} \right)
\end{align*}
\]  

(36) 

(37)
Re Pr \left( \delta^* \varepsilon \hat{u}_c \frac{\partial \hat{p}}{\partial \tau} + \varepsilon^2 \hat{w}_c \frac{\partial \hat{p}}{\partial \zeta} \right) = \left( \frac{\partial^2 \hat{p}}{\partial \tau^2} + \frac{1}{\tau} \frac{\partial \hat{p}}{\partial \tau} + \varepsilon^2 \frac{\partial^2 \hat{q}}{\partial \zeta^2} \right) + N_p \left[ \frac{\partial^2 \hat{p}}{\partial \tau^2} + \varepsilon^2 \frac{\partial^2 \hat{q}}{\partial \zeta^2} \right] + N_t \left[ \frac{\partial^2 \hat{p}}{\partial \tau^2} + \varepsilon^2 \frac{\partial^2 \hat{q}}{\partial \zeta^2} \right] 

(38)

Re Sc \left( \delta^* \varepsilon \hat{u}_c \frac{\partial \hat{q}}{\partial \tau} + \varepsilon \hat{w}_c \frac{\partial \hat{q}}{\partial \zeta} \right) = \left( \frac{\partial^2 \hat{q}}{\partial \tau^2} + \frac{1}{\tau} \frac{\partial \hat{q}}{\partial \tau} + \varepsilon^2 \frac{\partial^2 \hat{q}}{\partial \zeta^2} \right) + N_t \left( \frac{\partial^2 \hat{q}}{\partial \tau^2} + \frac{1}{\tau} \frac{\partial \hat{q}}{\partial \tau} + \varepsilon^2 \frac{\partial^2 \hat{q}}{\partial \zeta^2} \right) 

(39)

The Cauchy stress tensor for the Casson fluid in non-dimensional form becomes:

\[ \sqrt{\tau} = \sqrt{\tau_c} + \left( \frac{\partial \tilde{w}}{\partial \tau} \right)^{\frac{1}{2}} \]

(40)

For the Peripheral region (Sisko viscoplastic fluid):

\[ \text{Re} \left[ \delta^* \varepsilon^2 \frac{\partial \tilde{u}_p}{\partial \tau} + \delta^* \varepsilon \frac{\partial \tilde{w}_p}{\partial \zeta} \right] = -\frac{\partial \tilde{p}_p}{\partial \tau} + \varepsilon^2 \frac{\partial \tilde{q}}{\partial \zeta} + \delta^* \varepsilon^2 \frac{R_p^2 \tilde{u}}{k} \tilde{w}_p \]

(41)

The Cauchy stress tensors i.e. eqns. (25)-(27) in dimensionless form become:

\[ \bar{S}_{\tau} = -2 \left[ m + \left[ \left( \delta^* \varepsilon \frac{\partial \tilde{u}_p}{\partial \tau} \right)^2 + \left( \delta^* \varepsilon \frac{\partial \tilde{w}_p}{\partial \zeta} \right)^2 \right] \left[ \left( \varepsilon \frac{\partial \tilde{q}}{\partial \zeta} \right)^2 + \delta^* \varepsilon^2 \frac{R_p^2 \tilde{u}}{k} \tilde{w}_p \right] \right]^{-1} \left( \delta^* \frac{\partial \tilde{u}_p}{\partial \tau} \right) \]

(43)

\[ \bar{S}_{\zeta} = -2 \left[ m + \left[ \left( \delta^* \varepsilon \frac{\partial \tilde{u}_p}{\partial \tau} \right)^2 + \left( \delta^* \varepsilon \frac{\partial \tilde{w}_p}{\partial \zeta} \right)^2 \right] \left[ \left( \varepsilon \frac{\partial \tilde{q}}{\partial \zeta} \right)^2 + \delta^* \varepsilon^2 \frac{R_p^2 \tilde{u}}{k} \tilde{w}_p \right] \right]^{-1} \left( \frac{\partial \tilde{w}_p}{\partial \zeta} \right) \]

(44)

\[ \bar{S}_{\tau} = -2 \left[ m + \left[ \left( \delta^* \varepsilon \frac{\partial \tilde{u}_p}{\partial \tau} \right)^2 + \left( \delta^* \varepsilon \frac{\partial \tilde{w}_p}{\partial \zeta} \right)^2 \right] \left[ \left( \varepsilon \frac{\partial \tilde{q}}{\partial \zeta} \right)^2 + \delta^* \varepsilon^2 \frac{R_p^2 \tilde{u}}{k} \tilde{w}_p \right] \right]^{-1} \left( \frac{\partial \tilde{w}_p}{\partial \tau} + \delta^* \varepsilon^2 \frac{\partial \tilde{u}_p}{\partial \tau} \right) \]

(45)

Hemodynamic simulations are to be explored for the case of a mild stenotic artery. The nondimensional geometric parameters featured in the preceding terms defined above are stenosis height parameter (\( \delta^* = \delta/R_p \ll 1 \)) and the vessel aspect ratio (\( \varepsilon = R_p/L_0 \)). For the subsequent analysis, we shall assume that \( \delta^* \ll 1 \) and \( \varepsilon = O(1) \), i.e., the maximum height of the stenosis is small in comparison with the radius of the artery and also that the radius of the artery and length of
the stenotic region are of comparable magnitude [69]. After applying the above assumptions in the normalized system of Eqns. (36) – (39), we obtain for the core region (Casson viscoplastic fluid):

\[
\frac{\partial \tau_c}{\partial \tau} = 0 \quad (46)
\]

\[
\frac{\partial \tau_c}{\partial \tau} = \frac{1}{\tau_c} \tau_c' + \frac{1}{\tau_c} \frac{\partial \bar{w}_c}{\partial \tau} + \sqrt{\tau_c} \left( \frac{\partial \bar{w}_c}{\partial \tau} \right)^{\frac{1}{2}} \left( \frac{2}{\sqrt{\tau_c}} + \left( \frac{\partial \bar{w}_c}{\partial \tau} \right) \left( \frac{\partial \bar{w}_c}{\partial \tau} \right)^{-\frac{1}{2}} \frac{\partial \bar{w}_c}{\partial \tau} \right)
\]

\[
+ \left( \frac{\partial^2 \bar{w}_c}{\partial \tau^2} \right) + Gr \left( \tilde{\theta} + N\tilde{\phi} \right) \quad (47)
\]

\[
\left( \frac{\partial^2 \bar{\theta}}{\partial \tau^2} + \frac{1}{\tau_c} \frac{\partial \bar{\theta}}{\partial \tau} \right) + \left( Nb \left[ \frac{\partial \phi}{\partial \tau} \frac{\partial \bar{\theta}}{\partial \tau} \right] + Nt \left[ \frac{\partial \bar{\theta}}{\partial \tau} \frac{\partial \bar{\theta}}{\partial \tau} \right] \right) = 0 \quad (48)
\]

\[
\left( \frac{\partial^2 \bar{\theta}}{\partial \tau^2} + \frac{1}{\tau_c} \frac{\partial \bar{\theta}}{\partial \tau} \right) + \frac{Nt}{Nb} \left( \frac{\partial^2 \bar{\theta}}{\partial \tau^2} + \frac{1}{\tau_c} \frac{\partial \bar{\theta}}{\partial \tau} \right) = 0 \quad (49)
\]

After applying the above assumptions in the normalized system of Eqns. (41) – (45), we get for the peripheral region (Sisko viscoelastic fluid):

\[
\frac{\partial \tau_p}{\partial \tau} = 0 \quad (50)
\]

\[
\frac{\partial \tau_p}{\partial \tau} = \left( \frac{1}{\tau_c} \frac{\partial \bar{w}_p}{\partial \tau} + \frac{\partial^{2} \bar{w}_p}{\partial \tau^2} \right) \left( m + \left( \frac{\partial \bar{w}_p}{\partial \tau} \right)^{n-1} \right) \left( n-1 \left( \frac{\partial \bar{w}_p}{\partial \tau} \right)^{n-2} \frac{\partial \bar{w}_p}{\partial \tau} \right) \frac{\partial \bar{w}_p}{\partial \tau} + \mu \frac{\partial \bar{w}_p}{\partial \tau} \quad (51)
\]

The associated non-dimensional boundary conditions emerges as:

\[
\bar{u}_c = \bar{u}_p = 0, \quad \bar{w}_c = 1, \quad \frac{\partial \bar{w}_c}{\partial \tau} = 0, \quad \frac{\partial \bar{\theta}}{\partial \tau} = 0, \quad \frac{\partial \tilde{\phi}}{\partial \tau} = 0 \quad \text{at} \quad \bar{\tau} = 0
\]

\[
\bar{w}_c = \bar{w}_p \quad \text{at} \quad \bar{\tau} = r_c \tilde{R}(z)
\]

\[
\bar{w}_p = 0 \quad \text{at} \quad \bar{\tau} = \tilde{R}(z)
\]

\[
N_t \frac{\partial \bar{\theta}}{\partial \tau} + N_b \frac{\partial \tilde{\phi}}{\partial \tau} = 0 \quad \text{at} \quad \bar{\tau} = r_c \tilde{R}(z)
\]

\[
\bar{\theta} = 1 \quad \text{at} \quad \bar{\tau} = r_c \tilde{R}(z)
\]

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

Skin friction coefficient at the inner wall:

\[
\frac{1}{2} C_f \text{Re} = - \left[ \sqrt{\tau_c} + \left( \mu \frac{\partial \bar{w}_p}{\partial \tau} \right)^{\frac{1}{2}} \right]^2 \quad (53)
\]
Skin friction coefficient at the outer wall:

\[
\frac{1}{2} \frac{C_r}{Re} = -\left[ m + \left( \frac{\partial \bar{W}_p}{\partial \bar{r}} \right)^{-1} \right] \left( \frac{\partial \bar{W}_p}{\partial \bar{r}} \right)
\]  

(54)

Nusselt number at any wall:

\[
Nu' \ Re = -\bar{z} \left( \frac{\partial \bar{\theta}}{\partial \bar{r}} \right)
\]  

(55)

Volumetric flow rate:

\[
\bar{Q} = \int_0^k \bar{\rho} \bar{c} \bar{r} d\bar{r} + \int_k^R \bar{\rho} \bar{c} \bar{r} d\bar{r}
\]  

(56)

Hemodynamic resistance (impedance):

\[
\bar{X} = \int_0^k \left( \frac{\partial \bar{p}}{\partial \bar{z}} \right) \frac{d\bar{z}}{\bar{Q}}
\]  

(57)

In Eqns. (46)-(57) the Brownian motion parameter, \( Nb = \frac{D_b(\phi_w - \phi_h)(\rho c)_{f}}{\kappa} \), thermophoresis parameter \( Nt = \frac{D_t(T_u - T_i)(\rho c)_{f}}{\kappa T_i} \), the Grashof number \( Gr = \frac{(T_u - T_i)\rho_f \gamma g R^2 \beta_f}{U_o \bar{\rho}_b} \), the solutal to thermal buoyancy ratio \( N = \frac{\beta_s(\phi_w - \phi_h)}{\beta_f(T_u - T_i)} \), the Reynolds number \( Re = \frac{\rho_f U_0 L_0}{\bar{\mu}_b} \), Prandtl number \( Pr = \frac{c_{\mu} \bar{\rho}_b \bar{L}_0}{k} \), and Schmidt number \( S_c = \frac{\bar{\rho}_b}{\rho_f D_b} \). Peripheral viscosity \( \bar{\mu}_c = \bar{\mu}_p R^2 / k \), and also

\[
C_r' = C_f \left( \frac{U_o}{L_0} \right), \quad Nu' = Nu \frac{\bar{\rho}_b R_p}{\rho_f U_o}
\]

3. NUMERICAL COMPUTATIONS WITH FreeFEM++ CODE

The non-dimensional boundary value problem derived i.e. Eqns. (46)-(51) with boundary conditions (52) is still quite formidable owing to strong nonlinearity, coupling of different variables and two space variables. A powerful computational scheme is therefore essential to obtain fast and rapidly convergent solutions. Here the finite element method (FEM) with the variational approach as available in the FreeFEM++ software is selected. The finite element method is the most versatile numerical method both for accommodating complex geometries and also material nonlinearity, two key features of the current nano-pharmacodynamic biorheological
problem under investigation. Discretization of the flow domain is central to finite element simulation and involves division of domain of the problem into multiple subdomains. A set of element equations represents each subdomain. In the final solution, re-assembly of all sets of element equations into a global system of equations is executed systematically in the finite element code. Extensive details of the mathematical and algorithmic foundations of FREEFEM++ are given by Hecht [68]. The impressive versatility of FEM in engineering mechanics and multi-physics is provided in the treatise of Bathe [70]. Some other applications of FEM in hemodynamics (e.g. biomagnetics, pulsatile flows, tissue diffusion etc) have been reviewed by Bég [71]. FREEFEM++ has been employed successfully and very recently in simulating cardiovascular stenotic flows by Dubey and Vasu [72]. Here we provide extensive details of the finite element formulation employed via FREEFEM++ in solving the problem at hand. A weak formulation of the partial differential equation system (46)-(51) is derived by first defining the function spaces:

\[ X = \left\{ \vec{u} \in (H_1(\Omega)) \left| \vec{u} = a \ on \ \Gamma_{in}, \vec{u} = 0 \ on \ \Gamma_{wall} \right. \right\} \]

\[ Q = \left\{ \vec{u} \in (H_1(\Omega)) \left| \vec{u} = 0 \ on \ \Gamma_{in} \cup \Gamma_{wall} \right. \right\} \]  

(58)

The weak form of Eqns. (46) - (51) is obtained by determining \( w \in X \) and \( \phi, \Theta \in P \) such that every \( u_1, u_2 \in Q \) and \( p_1, p_2, \nu, q \in P \) where \( P = L^2(\Omega) \). Therefore, the weak formulation of Eqns. (46) – (51) will emerges as:

**For the Core Region (Casson fluid):**

\[
\int_\Omega \frac{\partial p_c}{\partial \vec{r}} \cdot p_1 \ d\vec{r} = 0
\]

(59)

\[
\int_\Omega \frac{\partial \vec{u}}{\partial \vec{r}} \cdot p_1 \ d\vec{r} - \frac{1}{\Omega} \tau_e \cdot u_1 \ d\vec{r} - \int_\Omega \left[ \frac{1}{\rho} \frac{\partial \vec{w}}{\partial \vec{r}} \cdot \vec{u}_1 \ d\vec{r} - \int_\Omega \left( \frac{2}{\rho} \frac{\partial \vec{w}}{\partial \vec{r}} \cdot \frac{\partial^2 \vec{w}}{\partial \vec{r}^2} \right) \sqrt{\tau_e} \left( \frac{\partial \vec{w}}{\partial \vec{r}} \right)^2 \cdot u_1 \ d\vec{r} \right.
\]

\[
- \int_\Omega \left( \frac{\partial^2 \vec{w}}{\partial \vec{r}^2} \right) \cdot u_1 \ d\vec{r} - \int_\Omega Gr \vec{\Theta} \cdot u_1 \ d\vec{r} - \int_\Omega Gr N \vec{\Theta} \cdot u_1 \ d\vec{r} = 0
\]

(60)

\[
\int_\Omega \frac{\partial^2 \vec{\Theta}}{\partial \vec{r}^2} \cdot \nu \ d\vec{r} + \int_\Omega \left[ \frac{1}{\rho} \frac{\partial \vec{\Theta}}{\partial \vec{r}} \cdot \nu \ d\vec{r} + \int_\Omega \left[ \frac{\partial \phi}{\partial \vec{r}} \right] \cdot \nu \ d\vec{r} + \int_\Omega \left[ \frac{\partial \vec{\Theta}}{\partial \vec{r}} \right] \cdot \nu \ d\vec{r} \right.
\]

\[
\int_\Omega \left[ \frac{\partial \vec{\Theta}}{\partial \vec{r}} \right] \cdot \nu \ d\vec{r} + \int_\Omega \left[ \frac{1}{\rho} \frac{\partial \vec{\Theta}}{\partial \vec{r}} \cdot q \ d\vec{r} + \int_\Omega \left[ N_t \left( \frac{\partial^2 \vec{\Theta}}{\partial \vec{r}^2} + \frac{1}{\rho} \frac{\partial \vec{\Theta}}{\partial \vec{r}} \right) \right] \cdot q \ d\vec{r} = 0
\]

(61)
For the Peripheral region (Sisko fluid):

\[
\int_{\Omega} \frac{\partial p}{\partial r} \cdot p_{2} \, d\overline{r} = 0 \tag{63}
\]

\[
\int_{\Omega} \frac{\partial p}{\partial r} \cdot p_{2} \, d\overline{r} - \int_{\Omega} \left[ \frac{1}{r} \frac{\partial \overline{w}_{p}}{\partial r} + \frac{\partial^{2} \overline{w}_{p}}{\partial r^{2}} \right] \left( m + \left( \frac{\partial \overline{w}_{p}}{\partial r} \right)^{n-1} \right) \cdot u_{2} \, d\overline{r}
- \int_{\Omega} \frac{\partial^{2} \overline{w}_{p}}{\partial r^{2}} \left( n-1 \right) \left( \frac{\partial \overline{w}_{p}}{\partial r} \right)^{n-2} \frac{\partial \overline{w}_{p}}{\partial r} \cdot u_{2} \, d\overline{r}
- \int_{\Omega} \overline{u} \cdot \overline{w}_{p} \cdot u_{2} \, d\overline{r} = 0 \tag{64}
\]

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 have to be defined as \( Q_{n} \subset Q \) and \( P_{n} \subset P \). Consider the finite dimensional approximations as \( u_{i,h}, u_{i,j} \subset Q_{n} \) and \( p_{i,h}, v_{i,h}, q_{i,h} \subset P_{n} \). In view of the finite dimensional approximation, the set of Eqns. (59)-(64) becomes:

For the Core Region (Casson fluid):

\[
\int_{\Omega} \frac{\partial p_{1}}{\partial r} \cdot p_{1} \, d\overline{r} = 0 \tag{65}
\]

\[
\int_{\Omega} \frac{\partial p_{1}}{\partial r} \cdot p_{1} \, d\overline{r} - \int_{\Omega} \frac{1}{r} \tau_{c} \cdot u_{1,h} \, d\overline{r} - \int_{\Omega} \frac{1}{r} \frac{\partial \overline{w}_{c}}{\partial r} \cdot u_{1,h} \, d\overline{r}
- \int_{\Omega} \left[ \frac{2}{r} + \frac{\partial \overline{w}_{c}}{\partial r} + \frac{\partial \overline{w}_{c}}{\partial r} \right] \sqrt{\tau_{c}} \left( \frac{\partial \overline{w}_{c}}{\partial r} \right)^{1/2} \cdot u_{1,h} \, d\overline{r}
- \int_{\Omega} \frac{\partial^{2} \overline{w}_{c}}{\partial r^{2}} \cdot u_{1,h} \, d\overline{r}
- \int_{\Omega} Gr \cdot u_{1,h} \, d\overline{r}
- \int_{\Omega} Gr N \overline{\phi} \cdot u_{1,h} \, d\overline{r} = 0 \tag{66}
\]

\[
\int_{\Omega} \frac{\partial \overline{\phi}}{\partial r^{2}} \cdot v_{h} \, d\overline{r} + \int_{\Omega} \frac{1}{r} \frac{\partial \overline{\phi}}{\partial r} \cdot v_{h} \, d\overline{r}
+ \int_{\Omega} Nb \left[ \frac{\partial \overline{\phi}}{\partial r} \frac{\partial \overline{\phi}}{\partial r} \right] \cdot v_{h} \, d\overline{r}
+ \int_{\Omega} Nt \left[ \frac{\partial \overline{\phi}}{\partial r} \frac{\partial \overline{\phi}}{\partial r} \right] \cdot v_{h} \, d\overline{r} = 0 \tag{67}
\]

\[
\int_{\Omega} \frac{\partial^{2} \overline{\phi}}{\partial r^{2}} \cdot q_{h} \, d\overline{r} + \int_{\Omega} \frac{1}{r} \frac{\partial \overline{\phi}}{\partial r} \cdot q_{h} \, d\overline{r}
+ \int_{\Omega} Nt \left( \frac{\partial^{2} \overline{\phi}}{\partial r^{2}} + \frac{1}{r} \frac{\partial \overline{\phi}}{\partial r} \right) \cdot q_{h} \, d\overline{r} = 0 \tag{68}
\]

For the Peripheral region (Sisko fluid):

\[
\int_{\Omega} \frac{\partial p_{2}}{\partial r} \cdot p_{2} \, d\overline{r} = 0 \tag{69}
\]
\[
\int_{\Omega} \frac{\partial \vec{p}}{\partial x} \cdot p_{z, h} \, d\vec{r} - \int_{\Omega} \left( \frac{1}{\rho} \frac{\partial \vec{\omega}}{\partial x} + \frac{\partial^2 \vec{\omega}}{\partial x^2} \right) \left( m + \left( \frac{\partial \vec{\omega}}{\partial x} \right)^{n-1} \right) \cdot u_{z, h} \, d\vec{r}
\]

\[
- \int_{\Omega} \frac{\partial^2 \vec{\omega}}{\partial x^2} \left( n-1 \left( \frac{\partial \vec{\omega}}{\partial x} \right)^{n-2} \right) \frac{\partial \vec{\omega}}{\partial x} \cdot u_{z, h} \, d\vec{r} - \int_{\Omega} \mu \frac{\partial \vec{\omega}}{\partial x} \cdot u_{z, h} \, d\vec{r} = 0
\]

(70)

The transformed equations (65)-(70) with boundary conditions (52) are solved numerically using finite element method through the variational formulation scheme with FreeFEM++ [68, 72]. An unstructured fixed mesh (grid) comprising 111,662 piecewise linear triangular elements with bubble (P1b) and 168,194 nodes, is implemented for the discretization of the arterial flow domain, and depicted in Figure 2. The mesh is constructed by utilizing 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. In the fixed mesh, a minimum step size (\(h_{\text{min}}\)), 0.0132355 is considered with the tolerance of computation < 10^{-6} for all the numerical experiments.

![Figure 2. Unstructured fixed mesh of triangular elements](image)

### 4. MESH INDEPENDENCE ANALYSIS

By conducting several different finite element mesh (grid) distribution tests, it may be established whether the calculated numerical results are grid-independent or not. The numerical values for skin-friction coefficients, at the stenosis and aneurysm, for various designs comprising unstructured fixed mesh elements with vertices and triangular elements, are provided in Figure 3. Eight different mesh distributions have been tested to ensure the simulated numerical results are mesh independent. Therefore, the selected mesh for the present calculations consisted of 111662 triangular elements and 168194 nodes, respectively. From Table 1 and figure 2, it is evident that...
increasing the mesh element density beyond this design does not modify the numerical values for non-dimensional skin-friction coefficient significantly in the domain with the parametric values prescribed i.e. \( m = 0.657, \ Nb = 0.6, \ Nt = 0.6, \ Gr = 1.0, \ N = 0.2 \) at \( r = 1.095, \ z = 3.20 \) and \( r = 2.60, \ z = 10.80 \) respectively. Mesh independent results are therefore ensured with the mesh design comprising 168194 nodes and 111662 triangular elements (simulation number 6 in Table 1).

### Table 1. Grid Independency analysis with \( m = 0.657, \ Nb = 0.6, \ Nt = 0.6, \ Gr = 1.0, \ N = 0.2 \) at \( r = 1.095, \ z = 3.20 \) and \( r = 2.60, \ z = 10.80 \)

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Number of Elements</th>
<th>Number of Nodes</th>
<th>Skin-friction coeff. (Stenosis)</th>
<th>Skin-friction coeff. (Aneurysm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>4260</td>
<td>6521</td>
<td>1.3686</td>
<td>1.7108</td>
</tr>
<tr>
<td>2.</td>
<td>10016</td>
<td>15245</td>
<td>1.5318</td>
<td>1.7232</td>
</tr>
<tr>
<td>3.</td>
<td>25324</td>
<td>38287</td>
<td>1.688</td>
<td>1.9791</td>
</tr>
<tr>
<td>4.</td>
<td>58488</td>
<td>88233</td>
<td>1.7003</td>
<td>2.5505</td>
</tr>
<tr>
<td>5.</td>
<td>98774</td>
<td>148762</td>
<td>1.7501</td>
<td>2.5788</td>
</tr>
<tr>
<td>6.</td>
<td>111662</td>
<td>168194</td>
<td>1.7751</td>
<td>2.6251</td>
</tr>
<tr>
<td>7.</td>
<td>150038</td>
<td>225758</td>
<td>1.7758</td>
<td>2.6627</td>
</tr>
<tr>
<td>8.</td>
<td>161558</td>
<td>243138</td>
<td>1.7921</td>
<td>2.6637</td>
</tr>
</tbody>
</table>

**Figure 3**: Grid Independence study (Skin friction coefficient)

5. **RESULTS AND DISCUSSION**

An extensive investigation of the impact of the key multi-physical parameters such as Sisko rheological material parameter ratio \( (m) \), permeability parameter \( (k') \), on the velocity in porous (peripheral) region and also the influence of Grashof number \( (Gr) \), thermophoresis parameter \( (Nt) \),
Brownian motion parameter \((N_b)\) and solutal to thermal buoyancy ratio \((N)\) the principal hemodynamic transport characteristics i.e. velocity, temperature and nanoparticle concentration is presented graphically as contour plots in the core and peripheral (porous) regions in Figs. 4-31.
Numerical results are also given in Tables 4-10. Further, the effects of various parameters on physiological flow properties such as the local skin friction coefficient \((\frac{1}{2} Re C_f^*)\), Nusselt number \((Nu Re)\), Volumetric flow rate \((Q)\) and hemodynamic resistance are also addressed. For FREEFEM++ computations, the default values of various parameters are considered as Table 2.

**Table 2:** Data employed in FREEFEM++ computations (unless otherwise indicated)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>(n)</th>
<th>(M)</th>
<th>(Gr)</th>
<th>(\mu^*)</th>
<th>(L_1)</th>
<th>(L_0)</th>
<th>(N)</th>
<th>(N_b)</th>
<th>(N_t)</th>
<th>(\tau_c^*)</th>
<th>(l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Values</td>
<td>1.4</td>
<td>0.657</td>
<td>1.0</td>
<td>2.4</td>
<td>14.0</td>
<td>3.20</td>
<td>0.2</td>
<td>0.3</td>
<td>0.3</td>
<td>7.0</td>
<td></td>
</tr>
</tbody>
</table>

**Table 3:** Comparison of temperature \((\theta)\) values, using the present scheme with the existing results for fixed values of \(N_b = 0.3, N_t = 0.3, Gr = 1.0\).

<table>
<thead>
<tr>
<th>(r) - axis</th>
<th>Analytical values of temperature ((\theta)) given by Ellahi et al. [73]</th>
<th>Values of temperature ((\theta)) by implementation of FEM on Ellahi et al. [73] modelled problem</th>
<th>Values of temperature ((\theta)) in the present study using FEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.05</td>
<td>0.015202</td>
<td>0.015207</td>
<td>0.015207</td>
</tr>
<tr>
<td>0.25</td>
<td>0.014947</td>
<td>0.014688</td>
<td>0.014688</td>
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<tr>
<td>0.45</td>
<td>0.013755</td>
<td>0.013169</td>
<td>0.013169</td>
</tr>
<tr>
<td>0.55</td>
<td>0.012600</td>
<td>0.012583</td>
<td>0.012583</td>
</tr>
<tr>
<td>0.6</td>
<td>0.011851</td>
<td>0.011661</td>
<td>0.011661</td>
</tr>
<tr>
<td>0.65</td>
<td>0.010977</td>
<td>0.010132</td>
<td>0.010132</td>
</tr>
<tr>
<td>0.75</td>
<td>0.008812</td>
<td>0.008612</td>
<td>0.008612</td>
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<tr>
<td>0.8</td>
<td>0.007495</td>
<td>0.007093</td>
<td>0.007093</td>
</tr>
<tr>
<td>0.85</td>
<td>0.005995</td>
<td>0.005576</td>
<td>0.005576</td>
</tr>
<tr>
<td>0.9</td>
<td>0.004281</td>
<td>0.004055</td>
<td>0.004055</td>
</tr>
<tr>
<td>1.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
</tbody>
</table>
Figure 4 Effect of $Nb$ and $Nt$ on nanoparticle concentration in at stenosis in the core region when $z = 3.20$, $Gr=1.0$, $N=0.5$

Figure 5 Effect of $Nb$ on nanoparticles concentration in core region for $r=0.78$, $Gr=1.0$ $Nt =0.3$ and $N=0.5$
Table 4: Nanoparticles concentration for $Gr = 1.0$ and $N = 0.5$, at stenosis in core region

<table>
<thead>
<tr>
<th>$r$ - axis</th>
<th>$Nb = 0.3$</th>
<th>$Nb = 0.3$</th>
<th>$Nb = 0.6$</th>
<th>$Nb = 0.6$</th>
<th>$Nb = 0.9$</th>
<th>$Nb = 0.9$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$Nt = 0.3$</td>
<td>$Nt = 0.6$</td>
<td>$Nt = 0.3$</td>
<td>$Nt = 0.6$</td>
<td>$Nt = 0.3$</td>
<td>$Nt = 0.6$</td>
</tr>
<tr>
<td>0.0</td>
<td>0.034777</td>
<td>0.034644</td>
<td>0.034762</td>
<td>0.034635</td>
<td>0.034745</td>
<td>0.034619</td>
</tr>
<tr>
<td>0.13</td>
<td>0.069538</td>
<td>0.069287</td>
<td>0.069524</td>
<td>0.06927</td>
<td>0.069491</td>
<td>0.069237</td>
</tr>
<tr>
<td>0.26</td>
<td>0.10431</td>
<td>0.10393</td>
<td>0.13905</td>
<td>0.13854</td>
<td>0.17373</td>
<td>0.17309</td>
</tr>
<tr>
<td>0.39</td>
<td>0.20861</td>
<td>0.20786</td>
<td>0.24334</td>
<td>0.24245</td>
<td>0.27796</td>
<td>0.27695</td>
</tr>
<tr>
<td>0.52</td>
<td>0.34769</td>
<td>0.34644</td>
<td>0.38238</td>
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<td>0.41694</td>
<td>0.41542</td>
</tr>
<tr>
<td>0.65</td>
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<td>0.51965</td>
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<td>0.59067</td>
<td>0.58852</td>
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<tr>
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<td>0.73015</td>
<td>0.72751</td>
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<td>0.79623</td>
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<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

Figure 6 Effect of $Nt$ on temperature distribution at the aneurysm in core region for $Gr = 2.0$, $N = 0.2$, $Nb = 0.6$ and $z = 10.80$
Figure 7 Effect of $Nb$ on temperature distribution at the stenosis in core region for $Gr = 1.0$, $N = 0.2$, $Nt = 0.3$ and $z = 3.20$
Figure 8 Temperature distribution in the core region in arterial segment at (a) $Gr = 1.0$, $N = 0.5$, $Nt = 0.3$ and $Nb = 0.2$ (b) $Gr = 1.0$, $N = 0.5$, $Nt = 0.3$ and $Nb = 0.3$ and (c) $Gr = 1.0$, $N = 0.5$, $Nt = 0.3$ and $Nb = 0.5$. 
Figure 9 Effect of $N_t$ on temperature distribution in core region for $r = 0.78$, $N_t = 0.3$, $Gr = 1.0$ and $N = 0.2$

Figure 10 Effect of $N_b$ on velocity field at stenosis in porous region for $Gr = 1.0$, $N = 0.2$, $N_t = 0.3$ and $z = 3.20$
Figure 11 Effect of \( N_b \) on velocity field at aneurysm in porous region for \( Gr = 1.0, N=0.2, N_t = 0.3 \)

and \( z = 10.80 \)

Figure 12 Effect of \( N_b \) on velocity field at stenosis in core region for \( Gr = 1.0, N = 0.2, N_t = 0.3 \)

and \( z = 3.20 \)
Figure 13 Effect of $k'$ and $Nb$ on velocity field at stenosis in porous region for $Gr = 1.0$, $N=0.2$, $Nt = 0.3$, $m = 0.657$ and $z = 3.20$

Figure 14 Effect of $Nb$ on velocity field at stenosis in porous region for $Gr = 1.0$, $N=0.2$, $Nt = 0.3$, $m = 0.957$, $k' = 0.5$ and $z = 3.20$
**Figure 15** Effect of $Nb$ and $Nt$ on velocity field at stenosis in core region for $z=3.20$, $Gr=2.0$, $N=0.2$

**Table 5**: Volumetric flow rate at aneurysm for core region i.e. $m = 0.657$, $k' = 0.25$, $Gr = 1.0$, $N = 0.2$ and $Nt = 0.3$

<table>
<thead>
<tr>
<th>Parameters (Fixed Values)</th>
<th>Axial Value ($z$)</th>
<th>Volumetric flow Rate (Q)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Parameter Value ($Nb = 0.3$)</td>
</tr>
<tr>
<td></td>
<td>2.4</td>
<td>0.178944</td>
</tr>
<tr>
<td></td>
<td>2.34</td>
<td>0.329797</td>
</tr>
<tr>
<td></td>
<td>2.21</td>
<td>0.44996</td>
</tr>
<tr>
<td></td>
<td>2.08</td>
<td>0.542061</td>
</tr>
<tr>
<td></td>
<td>1.95</td>
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</tr>
<tr>
<td></td>
<td>1.82</td>
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</tr>
<tr>
<td></td>
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<td>0.676228</td>
</tr>
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<td></td>
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<td>0.650828</td>
</tr>
<tr>
<td></td>
<td>1.43</td>
<td>0.611975</td>
</tr>
<tr>
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<td>1.3</td>
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</tr>
<tr>
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<tr>
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<td>0.78</td>
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</tr>
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</tr>
<tr>
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</tbody>
</table>
Table 6: Volumetric flow rate at stenosis in the core region when $m = 0.657$, $k' = 0.25$, $Gr = 1.0$ $N = 0.2$ and $N_t = 0.3$

<table>
<thead>
<tr>
<th>Parameters (Fixed Values)</th>
<th>Axial Value ($z$)</th>
<th>Volumetric flow Rate ($Q$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Parameter Value ($Nb = 0.3$)</td>
</tr>
<tr>
<td>$Gr = 1.0$, $N = 0.2$ and $N_t = 0.3$</td>
<td>0.9015</td>
<td>0.067216</td>
</tr>
<tr>
<td></td>
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<td>0.101754</td>
</tr>
<tr>
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<td>0.110853</td>
</tr>
<tr>
<td></td>
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<td>0.12338</td>
</tr>
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<tr>
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<td>0.168416</td>
</tr>
</tbody>
</table>

**Figure 16** Effect of $Nb$ on the volumetric fluid flow rate at stenosis in porous region for $Gr = 1.0$, $N = 0.2$, $N_t = 0.3$, and $z = 3.20$
**Figure 17** Effect of $Nb$ on the volumetric fluid flow rate at Aneurysm in porous region for $Gr = 1.0$, $N=0.2$, $Nt = 0.3$ and $z = 10.80$

**Figure 18** Effect of $Nt$ on the volumetric fluid flow rate at stenosis in porous region for $N = 0.2$, $Gr = 1.0$, $Nb = 0.3$ and $z = 3.20$
Figure 19 Effect of $Nb$ and $Nt$ on the volumetric fluid flow rate at aneurysm in porous region for $N = 0.2$, $Gr = 1.0$, $Nb = 0.3$ and $z = 10.80$

Figure 20 Effect of $Nt$ on the pressure distribution at stenosis in porous region for $N = 0.2$, $Gr = 1.0$, $Nb = 0.3$ and $z = 3.20$
Figure 21 Effect of $N_t$ on the pressure distribution at aneurysm in porous region for $N = 0.2$, $Gr = 1.0$, $Nb = 0.3$ and $z = 10.80$

Table 7: Numerical values of local skin friction coefficient $\frac{1}{2} \Re C_f$ for various values of $Nb$ and $N_t$ at stenosis (inner wall).

<table>
<thead>
<tr>
<th>Parameters (Fixed values)</th>
<th>Parameter Values ($Nb$)</th>
<th>Parameter values ($N_t$)</th>
<th>Local skin-friction coeff ($C_f$) (Stenosis)</th>
<th>Local skin-friction coeff ($C_f$) (Aneurysm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$m=0.657$, $Gr=5.0$ and $N=0.2$</td>
<td>0.3</td>
<td>0.3</td>
<td>0.46159</td>
<td>1.395</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.6</td>
<td>0.48529</td>
<td>1.4291</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.9</td>
<td>0.49494</td>
<td>1.4683</td>
</tr>
<tr>
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<td>0.6</td>
<td>0.3</td>
<td>0.40502</td>
<td>1.3147</td>
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<td></td>
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<td>0.37089</td>
<td>1.2934</td>
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</table>
Table 8: Numerical values of local skin friction coefficient $\frac{1}{2} \text{Re}C_f$ for various values of Nb and Nt at stenosis (outer wall).

<table>
<thead>
<tr>
<th>Parameters (Fixed values)</th>
<th>Parameter Values (Nb)</th>
<th>Parameter Values (Nt)</th>
<th>Local skin-friction coeff. ($C_f$) (Stenosis)</th>
<th>Local skin-friction coeff. ($C_f$) (Aneurysm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$m=0.657$, $Gr=1.0$ and $N=0.2$</td>
<td>0.3</td>
<td>0.3</td>
<td>1.7619</td>
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<td></td>
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<tr>
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<td>0.3</td>
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<td></td>
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<td>1.4447</td>
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</tr>
</tbody>
</table>

Table 9: Numerical values of Nusselt number $\text{Nu} \text{Re}$ for various values of Gr, Nb and Nt at stenosis (inner wall).

<table>
<thead>
<tr>
<th>Parameters (Fixed Values)</th>
<th>Parameter Values (Nb)</th>
<th>Parameter Values (Nt)</th>
<th>Nusselt Number ($\text{Nu} \text{Re}$) (Stenosis)</th>
<th>Nusselt Number ($\text{Nu} \text{Re}$) (Aneurysm)</th>
</tr>
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<tbody>
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Figure 22 Effect of $Gr$ on temperature distribution at Aneurysm in core region for $Nb = 0.3$, $Nt = 0.3$, $N = 0.2$ and $z = 10.80$

Figure 23 Effect of $Gr$ on temperature distribution at Aneurysm in core region for $Nb = 0.3$, $Nt = 0.6$, $N = 0.2$ and $z = 10.80$
Figure 24 Effect of $Gr$ and $Nt$ on nanoparticles concentration at stenosis in core region for $Nb = 0.3$, $N = 0.2$ and $z = 3.20$

Figure 25 Effect of $Gr$ and $Nb$ on nanoparticles concentration at aneurysm in core region for $Nt = 0.6$, $N = 0.2$ and $z = 10.80$
Figure 26 Effect of $N$ and $Nt$ on nanoparticles concentration at aneurysm in core region for $Nb = 0.6$, $Gr = 2.0$ and $z = 10.80$

Figure 27 Effect of $Gr$ on velocity field in core region for $Nb = 0.3$, $Nt = 0.3$, $N = 0.2$ and $r = 0.9015$
Figure 28 Effect of $Gr$ on volumetric fluid flow rate at stenosis in core region for $Nb = 0.3$, $Nt = 0.3$, $N = 0.2$, $m = 0.657$, $\mu^* = 2.4$ and $r = 0.9015$

Figure 29 Effect of $Gr$ on volumetric fluid flow rate at Aneurysm in core region for $Nb = 0.3$, $Nt = 0.3$, $N = 0.2$, $m = 0.657$, $k' = 0.5$ and $r = 0.9015$
Table 10: Resistance impedance of blood flow in the artery at $Nb = 0.3$, $Nt = 0.3$ and $r = 0.9015$

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<th>Resistance Impedance $(\lambda)$</th>
<th>Parameters $(Gr=1.0, N=0.2)$</th>
<th>Parameters $(Gr=2.0, N=0.2)$</th>
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![r-axis image](30(a)) ![z-axis image](30(b))
Figure 30 Velocity Streamlines (Aneurysm) at (a) $Gr = 5.0, N = 0.2, Nb = 0.3, \text{ and } Nt = 0.3$, (b) $Gr = 5.0, N = 0.2, Nb = 0.6, \text{ and } Nt = 0.3$, (c) $Gr = 5.0, N = 0.2, Nb = 0.6, \text{ and } Nt = 0.6$, (d) $Gr = 5.0, N = 0.2, Nb = 0.6, \text{ and } Nt = 0.9$

Figure 31 Velocity Streamlines (Stenosis) at (a) $Gr = 5.0, N = 0.2, Nb = 0.3, \text{ and } Nt = 0.3$, (b) $Gr = 5.0, N = 0.2, Nb = 0.6, \text{ and } Nt = 0.3$, (c) $Gr = 5.0, N = 0.2, Nb = 0.6, \text{ and } Nt = 0.6$, (d) $Gr = 5.0, N = 0.2, Nb = 0.6, \text{ and } Nt = 0.9$
To validate the scheme which has been adopted to simulate the current problem, the analytical results obtained by Ellahi et al. [73] for temperature of viscous blood flow are compared with the results obtained by using FEM for the model discussed by Ellahi et al. [73] and both are benchmarked with the current FREEFEM++ solutions, as displayed in table 3. It is evident that the results acquired by using the analytical method (HPM) and the FEM (variational approach) match up to three decimal points and both concur with the FREEFEM++ solution. Hence it can be deduced that the FREEFEM++ code is very accurate and suitable for this type of hemodynamic simulation.

5.1 Effect of Brownian motion parameter (Nb) and thermophoresis parameter (Nt)

The impact of Brownian motion parameter \( Nb \) and thermophoresis parameter \( Nt \) on temperature profile and nanoparticle concentration is shown in the different graphs and tables with the variation in radial coordinates. Fig. 4 and 5 depict the influence of thermophoresis parameter \( Nt \) and Brownian motion parameter \( Nb \) on radial distribution of the nano-particle concentration \( \phi \) at the throat of the stenosis and in the whole arterial segment with the fixed value of Grashof number \( (Gr=1.0) \), and solutal to thermal buoyancy ratio \( (N=0.5) \) in the core region. Similarly, with a change in thermophoresis parameter \( Nt \) there is \( \sim 0.36\% \) decrement in magnitudes of nanoparticle concentration for all radial coordinate values when \( Nt \) increases from 0.3 to 0.6. Figure 5 illustrates the axial variation of nanoparticles concentration distribution and maximum concentration (volume fraction) arises at the stenotic arterial segment and decreases substantially in the aneurystic region. There is a marked elevation in the magnitudes of nano-particle concentration for all axial coordinates with \( \sim 5\% \) increment when increasing Brownian motion parameter from 0.3 to 0.6 and then \( \sim 10\% \) increment when increasing the Brownian motion parameter from 0.6 to 0.9 respectively. Both nanoscale effects generally assist in the diffusion of the nano-particle species at all radial coordinates although the effect is maximized at intermediate distances from the arterial mid-line. Brownian motion acts to distribute the nano-particles as uniformly as possible throughout the blood flow regime. This reduces the nanoparticle concentration gradient and diminishes the regional variations in fluid characteristics whereas it mobilizes species diffusion leading to an elevation in nano-particle concentration magnitudes.

Table 4 shows the effect of Brownian motion and thermophoresis parameters on the nanoparticle concentration distribution in the arterial segment. There is a noticeable elevation in nanoparticles concentration at the throat of stenosis induced with increment in Brownian motion parameter whereas the converse effect is generated with increasing thermophoresis parameter, when the
Grashof number ($Gr=1.0$) and buoyancy ratio ($N=0.5$). Relatively low sensitivity in nanoparticles concentration to alteration in thermophoresis parameter ($Nt$) is computed whereas there is a weak enhancement in concentration with larger Brownian motion effect ($Nb$).

Figures 6 and 7 are graphical representations of temperature distribution in blood flow with respect to radial coordinate for various values of thermophoresis parameter ($Nt$) and Brownian motion parameter ($Nb$) respectively. Figure 6 is drawn for the fixed values of $Gr = 2.0$, $N = 0.2$ and $Nb =0.3$ whereas figure 7 is drawn for the fixed values of $Gr = 1.0$, $N = 0.2$ and $Nt =0.3$. Figure 9 visualizes temperature distribution in the blood flow with respect to axial coordinate for various values of thermophoresis parameter and is plotted with $Gr = 2.0$, $N = 0.2$ and $Nb =0.3$. A parabolic distribution is computed in Fig. 6 at the aneurysm and exhibits an increment in temperature magnitudes in the arterial segment with increasing value of thermophoresis parameter from $Nt = 0.3$ to $Nt =0.6$. Fig. 7 shows the comparison of computation done at stenosis and indicates that the temperature increases with an increment in the Brownian motion parameter ($Nb$). Higher values of nanoscale thermophoresis parameter ($Nt$) induces a slight enhancement in temperatures at all radial locations of the arterial vessel; however, the principal elevation is only observed around the central zone and diminishes progressively toward the vessel inner boundaries. Similarly, inspection of the coloured contours presented in Figure 8(a)-8(c), reveals that temperature increases with both an increase in thermophoresis parameter ($Nt$) and Brownian motion parameter ($Nb$). The increment in Brownian motion parameter $Nb$ is from 0.2 to 0.3 and then from $Nb = 0.3$ to $Nb = 0.5$; at $Nt =0.3$, the temperature increases at the wall as well as in the centreline region. The temperature magnitudes are greater near the inner wall of the artery in comparison to the midline which is attributable to the increment in $Nb$ decreasing the thermal conductivity of the base fluid. The thermophoretic force due to temperature gradient, causes a rapid migration of heat from the midline. Therefore, hot fluid is moved away from the centreline, and the thermal boundary layer becomes thicker. Fig. 9 further shows that significant heating of the blood is generated i.e. higher temperatures generated with an increase in the thermophoresis parameter. Again, the deviation from axisymmetric distributions (observed in unobstructed vessels) is clearly captured in this figure (for the core region) confirming the significant influence of the stenosis and aneurysm on distorting temperature distributions in diseased (obstructed) arteries. Considerable elevation in temperature is seen in the vicinity of the stenosis whereas there is depletion in the aneurysm location.

Figs. 10- 12 show the velocity profile distributions with variation of Brownian motion parameter ($Nb$) in both the regions i.e., core region and peripheral region (porous region) for the dual cases
of stenosis and aneurysm respectively. There is a distinct elevation computed in velocity (i.e. flow acceleration is produced) in the porous region with increasing Brownian motion parameter (progressively smaller nanoparticles) in both figures 9 and 10 at the stenosis and aneurysm, respectively (corresponding to the peripheral region). The velocity field exhibits a difference only near the inner wall but shows equal decrement to the outer wall of the artery for all values of \( Nb \) in both the constriction (stenosis) and dilation (aneurysm). Increment in Brownian motion parameter enhances the mobility of the nanoparticles which encourages ballistic collisions and momentum diffusion in the hemodynamic flow and this manifests in flow acceleration. However, owing to the greater resistance to blood flow while moving from inner wall to outer wall in the peripheral region, there is an exaggerated velocity variation near the inner wall. Figure 11 is a graphical representation of the velocity field in the core region for the stenosis. Clearly velocity increases with Brownian motion parameter but it shows some different variations in comparison to the nanoparticle concentration and temperature. The velocity field generally decreases while approaching the artery boundary (wall) and in closer proximity to the wall is observed to increase. The velocity field is maximum along the arterial centre line \((r=0)\) and decays towards the wall. However, the presence of nanoparticles leads to an acceleration near the wall and a reduction in the momentum boundary layer thickness there.

**Figs. 13** presents the evolution in radial velocity distribution in the porous (peripheral) region for different value of permeability parameter \( (k') \). While comparing both the figures it has been found that increment in permeability parameter (drag force) increases resistance to the flow which induces a decrement in velocity in the porous region i.e. blood flow retardation. Clearly porous effects are non-trivial and stronger at smaller values of radial coordinate in the peripheral zone.

**Figs. 14-15** depict the velocity field variation at the peripheral and stenotic part of the arterial segment (core region), respectively, for different values of Brownian motion parameter \( (Nb) \) and thermophoresis parameter \( (Nt) \). A weak acceleration in the flow is induced with increasing \( Nb \), in the peripheral region (Fig. 13) and the effect is most prominent at lower radial coordinate values. Fig. 14 shows that the velocity field increases in the core region with thermophoresis parameter and is less sensitive to Brownian motion parameter in this zone.

**Tables 5 and 6** and **Figs 16-17** document and illustrate variation in volumetric flow rate \( (Q) \) in the arterial segment at stenosis and aneurysm in the porous (peripheral) region respectively for a modification in Brownian motion parameter \( (Nb) \). The flow rate is maximum at the mid-line of the artery and decreases as we move radially to the wall of the artery. The decay is smooth and undisturbed from the centre to the boundary. The flow rate is the summation of variable velocity
with the radial direction i.e., the integration of the velocity with respect to radial coordinate. Hence the flow rate shows similar behavior to the velocity. Lower magnitudes of flow rate are however computed at the stenosis compared with the aneurysm, in particular at lower radial coordinate values.

**Figs. 18-19** illustrate respectively the effect of thermophoresis parameter \((N_t)\) and collective effect of Brownian motion \((N_b)\) and thermophoresis parameters \((N_t)\) on the volumetric flow rate \((Q)\) again in the porous region at the stenosis and aneurysm locations. From the figures, it can be seen that, moving from inner wall to outer wall the flow rate is decreasing towards zero due to the no-slip velocity boundary condition and the flow rate decreases by increasing the values of thermophoresis parameter \(N_t\). However larger magnitudes of flow rate correspond to the aneurysm (Fig. 19) and lower magnitudes to the stenotic region (Fig. 18). These different geometrical obstructions therefore do not exert the same influence on flow rates in the diseased artery.

**Figs. 20-21** describe the influence of thermophoresis parameter \((N_t)\) on pressure distribution in the porous region at the stenosis and aneurysm for fixed values of \(Gr = 1.0 \text{ and } N = 0.2\) respectively. The pressure **decreases** significantly with a boost in thermophoretic body force but does not continuously rise with greater radial coordinate values. Pressure peaks at intermediate radial coordinate and thereafter decays with further increase in radial coordinate. Nanoscale effects (of which thermophoresis is merely one manifestation [41]-[44]) clearly modify the pressure distribution in diseased arterial blood flow and clearly undesirably high blood pressures may be controlled with the judicious deployment of nanoparticles in hemodynamics. This has important implications in promoting nano-pharmacodynamics as an effective technique for controlling hypertension and other ailments associated with stenotic arterial disease.

**Tables 7-10** show the variation in values of local skin-friction coefficient for the stenosis and aneurysm in the core and porous region and Nusselt number in the core region, respectively. In **Table 7** it is evident that with increasing Brownian motion parameter \((N_b)\) there is a marked suppression in local skin friction both at the stenosis and at the aneurysm although the decrement is more prominent for the former. With a boost in thermophoresis parameter \((N_t)\), the reverse response is induced i.e. skin friction is elevated consistently indicating that flow acceleration is produced in the stenosis and aneurysm locations. Thermophoretic body force is therefore the primary mechanism for ensuring nanoparticles improve drug delivery in obstructed hemodynamics since it accelerates the flow (with an associated decrease in blood pressure). Preferential particle deposition is therefore an excellent mechanism for achieving improved delivery of pharmaceutical nanoparticles, achieving longer residence-time and targeting specific zones for mediated therapy
[74, 75]. In Table 8 again the collective influence of Brownian motion parameter \((Nb)\) and thermophoresis parameter \((Nt)\) on local skin friction is given although for a much weaker thermal Grashof number \((Gr = 1.0)\) compared with Table 7 (in which \(Gr = 5.0)\). In both tables the Sisko rheological parameter, \(m = 0.657\) and \(N = 0.2\), implying that the viscoelasticity is weak in the core arterial region and that nano-particle species buoyancy force is one fifth of the thermal buoyancy force). Much greater local skin friction magnitudes are observed in Table 8 indicating that weaker thermal buoyancy force \((Gr = 1.0)\) implies the thermal buoyancy and viscous hemodynamic forces are equal) leads to a strong acceleration in the blood flow, both at the stenosis and the aneurysm. It is further of note that negative skin friction never arises in either Table 7 or 8 implying that despite the presence of a stenosis or aneurysm, *back flow is never generated* in the arterial domain. Table 9 shows the results for Nusselt number in the core region (inner wall) at both the stenosis and aneurysm for two cases of Grashof number \((Gr = 5.0 \text{ and } 2.0)\) again with the influence of Brownian motion parameter \((Nb)\) thermophoresis parameter \((Nt)\). With larger \(Nb\) there is an elevation in local Nusselt numbers at both the stenosis and the aneurysm. Heat transfer to the arterial wall (inner) is therefore enhanced with more intense Brownian dynamics (smaller nanoparticles) since temperatures are decreased in the blood with this effect i.e. the blood is cooled. The contrary effect is generated with greater thermophoresis parameter i.e. \(Nt\), *i.e. increasing \(Nt\) leads to a strong suppression in Nusselt number which is observed both in the stenotic and aneurysm zones*. With greater value of Grashof number there is a clear depression in Nusselt numbers i.e. heat transfer to the inner wall of the artery is significantly decreased. The greater thermal buoyancy effect is known to heat the blood flow and this transfers heat from the boundaries (walls) to the body of the fluid, manifesting in a plummet in heat transfer to the wall i.e. decreasing Nusselt numbers.

5.2 Effect of Grashof Number \((Gr)\) and Buoyancy ratio parameter \((N)\)

The effect of Grashof number \((Gr)\) and solute: thermal buoyancy ratio \((N)\) on the hemodynamic characteristics i.e. temperature, nanoparticle concentration, pressure distribution and velocity field are illustrated in Figs. 22-29.

Figure 22-24 show the influence of Grashof number on profiles for temperature distribution for two different values of thermophoresis parameter \((Nt = 0.3 \text{ and } 0.6)\) at the aneurysm (axial location, \(z = 10.80)\). With elevation in Grashof number (Fig. 22) the temperature decreases near the mid-line (central zone) but it increases near the inner wall of the artery (large values of radial coordinate, \(r)\). Grashof number represents the ratio between the buoyancy forces due to spatial
variation in fluid density (caused by temperature differences) to the shearing force due to the viscosity of the fluid. An accentuation in thermal buoyancy will therefore enhance temperature difference resulting in cooling (decrement in temperature) near the arterial mid-line and heating (increment in temperature) near the inner wall of the artery. Thermal buoyancy therefore exerts some effect on thermal characteristics of the hemodynamics. The contribution from species buoyancy is also present, however it is much weaker than thermal buoyancy for the numerical experiments performed ($N = 0.2$). A more prominent reduction in core temperatures is witnessed at the stenosis (Fig. 22) than at the aneurysm zone (Fig. 23), although the trends are similar at the stenosis and aneurysm both in proximity to the arterial centre line and the wall.

**Figs. 24-25** visualize the nanoparticle concentration distributions with variation of Grashof number ($Gr$) and Brownian motion parameter ($Nb$) and thermophoresis parameter ($Nt$), at the stenosis and aneurysm respectively. In figure 24, for the stenotic zone, the increment in Grashof number weakly reduces the nanoparticle concentration near the wall and in the central zone of the arterial segment. The higher value of Grashof number implies a more dominant thermal buoyancy force which mobilizes stronger thermal convection currents in the blood flow, and this inhibits the diffusion of nanoparticles i.e. decreases concentration of nanoparticles. Increasing thermophoresis effect ($Nt$) also marginally reduces nano-particle concentrations in the blood since it promotes the migration of nanoparticles to the periphery away from the central zone. In fig. 25, for the aneurysm part of the artery, a significant variation in nanoparticle concentration is computed with a rise in Brownian motion parameter. The exacerbation in collisions of nanoparticles with Brownian dynamics (smaller nanoparticles correspond to larger values of $Nb$) leads to enhanced species diffusion and a boost in nanoparticle concentration i.e. volume fraction (~4%). However as noted at the stenotic region (Fig. 24) increasing Grashof number again generally suppresses the nanoparticle concentrations.

**Fig. 26** presents the collective influence of solutal to thermal buoyancy ratio ($N$) and thermophoresis effect ($Nt$) on nanoparticle concentration ($\phi$). Increasing the value of solutal to thermal buoyancy ratio ($N$) generates a reduction in nanoparticle concentration.Stronger species buoyancy force is associated with $N = 0.5$ whereas weaker species (solutal) buoyancy force is represented by $N = 0.2$. At the arterial walls, the species (nanoparticle) boundary layer thickness will also be reduced. There is also a much weaker decrement in nanoparticle concentration ($\phi$) with greater thermophoresis effect.

**Fig. 27** depicts the evolution in velocity field with axial coordinate at a fixed radial coordinate ($r = 0.9015$) in the core region (viscoplastic Casson fluid) with a change in Grashof number ($Gr$). It
is evident that at the stenosis the velocity is elevated (lower $z$ value) whereas it is significantly decreased at the aneurysm location (higher $z$ values) at all values of Grashof number. Increasing thermal buoyancy only accelerates the flow in the vicinity of the stenosis whereas everywhere else it results in strong flow deceleration. Momentum boundary layer thickness will therefore be reduced at the stenosis whereas the boundary layer thickness will be significantly increased downstream at the aneurysm. The impact of thermal buoyancy is therefore not consistent in the hemodynamic regime and care must be taken in deploying nano-drugs to ensure that the desired effect is achieved whether at the entry zone, stenosis, aneurysm or indeed any other location in the target zone for the treatment. Figs 28 and 29 indicate that volumetric flow rate ($Q$) is consistently elevated in the core zone at the stenotic section with increasing Grashof number whereas it is depressed in the aneurysm zone. The $Q$-$z$ profiles in Fig. 28 (stenosis) are clearly inverted parabolas whereas the $Q$-$z$ profiles in Fig 29 (aneurysm) are approximately linear decays indicating that a very different interplay between flow rate and axial coordinate exists at the different arterial locations since the former is associated with a constriction (stenosis) whereas the latter is representing a dilatation (aneurysm) in the vessel geometry. 

Table 9 documents the values of hemodynamic resistance (impedance) in the artery with respect to axial coordinate ($z$-axis) for the variation in Grashof number at fixed value of thermophoresis parameter and Brownian motion parameter i.e. ($Nb = 0.3, Nt = 0.3$). Resistance impedance of the fluid flow is associated with the ratio of negative of pressure gradient and fluid flow rate. Since the flow rate decreases while increasing the Grashof number in the aneurysm ($higher z$-values $s it is found further downstream of the stenosis$) and also the negative pressure gradient increases, this produces an increase in resistance impedance at the aneurysm with higher thermal buoyancy effect i.e. larger Grashof number. However, at the stenosis ($lower z$-values, as it is located further upstream of the aneurysm), the flow rate increases while increasing the Grashof number and also the pressure gradient increases. This effectively manifests in a decrement in resistance (impedance) with Grashof number increment. Therefore, lower hemodynamic impedance is observed at the aneurysm with greater thermal buoyancy effect. The inclusion of heat transfer in obstructed hemodynamic simulations is therefore proven to be a significant factor in predicting more accurately the characteristics of such flows. The heat-conducting nature of blood is fundamental to life-support and is well-documented in physiological and also pharmacological literature and is therefore strongly recommended for inclusion in mathematical/computational fluid dynamic models of stenotic/aneurysmic hemodynamics [76, 77].
In addition with these results it can also be pointed out that the skin–friction shows a decreasing behaviour as it is significantly more at inner boundary than outer boundary at stenosis and aneurysm both.

Finally, in figs. 30-31, to provide two-dimensional visualizations of the circulation hemodynamics in obstructed blood flow, contour plots have been presented for trapping. This describes an interesting phenomenon for the blood flow pattern and relates more to vorticity rather than velocity profiles. In particular it provides a good insight into occlusion effects in hemodynamics as represented by stenosis and aneurysm features. Figs. 30(a)-30(d), represents the streamlines of blood flow in the arterial domain at the aneurysm and figures 31(a)-31(d), represents the streamlines of blood flow in an artery at the stenosis both for the core region. In figures 30(a)-30(b) and 31(a)-31(b), it is observed that the magnitude of trapped boluses grows with an increase in the Brownian motion parameter for both obstruction cases i.e., stenosis and aneurysm. Clearly there are two trapped boluses in the stenotic zone but four in the aneurysm zone, at any Brownian motion number. Figs. 30(c)-30(d) and 31(c)-31(d), however shows that increasing thermophoresis parameter does not exert any tangible effect on bolus structure or quantity in either the stenosis or the aneurysm.

6. Conclusion

In this article, computational fluid dynamics modelling of steady-state, two-dimensional blood flow conveying spherical metallic nanoparticles in an arterial geometry with porous wall and featuring both a stenosis and aneurysm has been described. The objective has been to provide further refinement in simulating nano-pharmacological transport phenomena in the treatment of hemodynamic ailments (obstructed arteries etc). A Casson viscoplastic fluid model has been adopted for hemo-rheology in the core region and a Sisko viscoelastic fluid model in the peripheral (porous) region to represent non-Newtonian behaviour. The revised Buongiorno two component nanofluid model is employed for nanoscale effects. The governing equations have been derived by extending the Navier-Stokes equations with linear Boussinesq approximation (which simulates both heat and mass transfer). Natural (free) double-diffusive convection is considered to simulate the dual influence of thermal and solutal buoyancy forces. The dimensionless, transformed mass, momentum, energy and nano-species diffusion equations have been solved computationally with the powerful variational finite element method in the FreeFEM++ software. A comprehensive mesh-independence (grid sensitivity) study has been included. The effect of selected parameters (thermophoresis, Brownian motion, Grashof number, thermo-solutal buoyancy ratio, and Sisko
parameter ratio and permeability parameter) on velocity, temperature, nanoparticle concentration and hemodynamic pressure have been calculated for two clinically important cases of arteries with a stenosis and an aneurysm. Skin-friction coefficient, Nusselt number, volumetric flow rate and hemodynamic (resistance) impedance of blood flow are also computed. Colour contours and graphs are employed to visualize the simulated blood flow characteristics. The principal findings from the numerical simulations may be summarized as follows:

• It has been observed that in the core region moving from the inlet to the outlet, the nanoparticle concentrations increase with increasing the Brownian motion parameter. Also, the temperature increases noticeably when increasing the thermophoresis parameter.

• Coloured contours describes the enhancement in the temperature as increases in the thermophoresis parameter and Brownian motion parameter at both the stenosis and aneurysm while moving in radial direction.

• The velocity field increases with higher Brownian motion parameter (smaller nanoparticles) in the core region. In the porous (peripheral) region the flow is also accelerated (as in the core region) with increasing Brownian motion parameter at both the stenosis and aneurysm sections.

• The volumetric flow rate increases in the aneurysm region when increasing the Brownian motion parameter whereas it is suppressed near the inner wall. However, it exhibits a different response in the stenotic region since volumetric flow rate decreases here with increasing the Brownian motion parameter.

• Local skin-friction coefficient shows a noticeable increment with increasing thermophoresis parameter whereas it manifests in a strong decrement by increasing the Brownian motion parameter for both obstructed arterial regions i.e. stenosis and aneurysm at inner wall as well as at the outer wall.

• The Nusselt number shows the opposite behaviour to local skin-friction coefficient as it decreases with increasing the thermophoresis parameter and increases with greater values of Brownian motion parameter.

• The pressure distribution increases in the porous region with increasing thermophoresis parameter in both the stenosis and aneurysm section. Pressure also shows noticeable increment in the core region with increasing thermophoresis parameter.

• The nanoparticle concentration shows a decrement with increasing Grashof number (Gr) and also it decreases with an elevation in solutal: thermal buoyancy ratio parameter (N).
- The velocity field increases at the stenotic region with increasing the value of Grashof number $(Gr)$ whereas it is suppressed at the aneurysm location, indicating that thermal buoyancy exerts different effects at different axial locations in the arterial segment.
- The volumetric flow rate rises in the stenotic section while it decreases in the aneurysm section with higher thermal buoyancy forces i.e. with increasing Grashof number $(Gr)$.
- Increasing the value of Grashof number $(Gr)$, induces lower hemodynamic impedance near the inner boundary whereas at the outer boundary (wall) there is a higher resistance in the core region of the diseased.
- The study has been motivated by applications in novel nanofluid drug delivery systems.
- This study also signify the potential of rheological properties of the blood in the blood vessel during the two crucial cardiovascular diseases (i.e., stenosis and aneurysm).

In the current analysis, shape factor effects of the nanoparticles have been ignored. Furthermore, specific nano-particle materials have been neglected since this is not possible with the Buongiorno model and revised Buongiorno model. Future studies will therefore employ the Tiwari-Das nanoscale model [57] which allows specific thermal conductivity, viscosity and heat capacity values to be computed for specific nano-particles (e.g. gold, iron oxide) and also enables different geometrical configurations to be studied via a shape factor model [78]. Additionally, in the present study attention has been confined to rigid wall arterial vessels. The deformability of blood vessels is also an important consideration [79] and this requires modification of the present model to include fluid-structure interaction (FSI) effects. The results of efforts in these directions will be communicated imminently, and it is envisaged that they will provide a deeper understanding of nano-drug fate and impact in the treatment of cardiovascular disease.

The potential application of numerical blood flow simulation is to aid decision-making processes during the treatment of cardiovascular diseases, for example, stenoses and aneurysms. Although a conventional method for treating a stenosis or aneurysm is to deploy a stent or coiling of wire and catheter, the modern trend is increasingly now to deploy nanodrugs within a particular targeted environment. The resulting modification of the flow field triggers the process of blood clotting at the stenosis and inside the aneurysm and the effect of these post-treatment processes can be predicted by computational simulation.
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