

Analysis of Turbulence and Phase Interaction in Diseased Arterial Blood Flow Systems

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Abstract—In this Chapter we present an analysis on turbulence and plasma-red blood cell (RBC) phase interaction in stenotic arterial blood flow systems with disease. We represent blood as a two-phase fluid consisting of plasma and RBCs, flowing through an axisymmetrical stenosed artery. Continuity and Momentum Equations are formulated by introducing interphase drag, effective turbulent viscosity, and pulse pressures gradient. For both steady and pulsatile flow conditions, analytical solutions for the above equation are presented by making certain assumptions. Additionally, in order to determine how many of the important Hemodynamic Parameters affect the behavior of the fluids in the artery including; velocity profiles, wall shear stresses, pressure drops, turbulence viscosities, hematocrit values, and the amount of "slip" or separation occurring at each phase interface, various parameters were analyzed. These studies indicate that increased stenosis severities, hematocrit levels, and turbulence viscosity values significantly increase resistance to flow through the artery, wall shear stresses, and pressure losses within the system. In addition, these studies have indicated that the interaction between plasma and RBC phases occurs most strongly in proximity to the stenosis throat where velocity gradients are greatest. Therefore, the developed analytical model will provide valuable information regarding the hemodynamics of diseased arteries and can be useful in assisting in diagnosing and treating cardiovascular diseases resulting from arterial stenosis.

Index Terms—Diseased artery, Stenosis, Turbulent blood flow, Plasma–RBC phase interaction, Hematocrit, Wall shear stress, Pressure drop.

I. INTRODUCTION

The study of blood flow through diseased arteries represents one of the main areas of interest in scientific studies because it leads to significant alterations in the normal physiological hemodynamic parameters due to

severe narrowing of the arterial lumen as a result of the development of atherosclerotic plaque, which can lead to dangerous cardiovascular events (myocardial infarctions and strokes). Stenosing the diameter of the artery increases the rate at which blood flows through the stenotic area creating higher velocities that create increased wall shear stress, pressure losses, turbulent behavior and complex forces acting upon the plasma and erythrocytes.

The degree to which these complex forces act upon each other have been shown to be influential factors in oxygen transport; the function of the endothelium; and disease progression. Therefore, a primary method of understanding the fundamental physical processes involved with blood flow through arteries and predicting those flow parameters of clinical relevance, is through the use of mathematical models. In this work, blood will be modeled as a two-phase continuum system composed of plasma and erythrocytes flowing through an axisymmetrically shaped stenosed artery.

The partial differential equations representing the conservation of mass, momentum and energy will be formulated using an effective viscosity representation of turbulence and a plasma-erythrocyte drag model to represent plasma-erythrocyte interaction. Both steady and unsteady solutions will be obtained and used to investigate velocity profiles, wall shear stresses, pressure drops across the stenosis, turbulence viscosity values, hematocrit values and phase slip. This formulation will provide a general analytical methodology for studying the individual influences of turbulence and plasma-erythrocyte interaction on blood flow in diseased arteries, and provide a useful resource for biomedical engineers and researchers in cardiovascular medicine.

Jahangiri et al. (2015) investigated the pulsating blood flow through stenotic arteries as well as the effect of elastic arterial walls at turbulent flow conditions. They identified that the time dependent evolution of the velocity field, pressure gradient and wall shear stress were influenced by the compliant nature of the vessel wall in comparison to models which assume rigid walls. Furthermore, they observed that the interaction between the pulsating blood flow and the deformation of the arterial wall influenced the flow separation and recirculation that occurred downstream of the stenosis. Therefore, these studies provide evidence that coupling the fluid dynamics with structural mechanics is essential to obtain accurate predictions of arterial hemodynamics especially under extreme stenotic conditions. Additionally,

Tripathi and Sharma (2016) analyzed a new type of porous model (porous model), that was designed to represent an inclined porous stenotic artery. In their analysis they accounted for both variable viscosity and heat transfer. The results of this study show that applied magnetic fields decrease blood velocities; however increased arterial permeability enhance fluid transport through the artery. Additionally, the study indicates that variation in viscosity have a strong impact on wall shear stress and thermal properties; therefore, it is important to consider both physiological and externally applied magnetics effects when modeling diseased arteries. Finally,

Tripathi et al. (2017) expanded upon previous research by establishing a two-phase pulsating blood flow model that accounts for both heat and mass transfer under MHD conditions. In addition, their results indicate that there is a correlation between plasma and red blood cells (erythrocytes). Therefore, the hematocrit distribution has a direct relationship with velocity profiles, flow resistances, and shear stress. Their results also demonstrate that a correlation between plasma and erythrocyte phases becomes much stronger in stenotic arteries because of particle migration and non-uniform cell distribution effects on total hemodynamic behavior. As such, two-phase modeling provides a more physiologic representation than single-phase homogeneous representations. Finally,

Gamage et al. (2018) developed a two-way coupled fluid-structure interaction model to investigate pulse wave propagation in stenotic arteries. Their results illustrate how arterial deformations affects pulse wave

propagation, pressure distributions and wall displacements over entire cardiac cycles. The results also show that ignoring arterial deformations would lead to incorrect predictions of hemodynamic variables particularly in regions near stenoses where high mechanical stresses occur. Therefore, their results emphasize the need for coupling structural mechanics into computational fluid dynamics models for improved cardiovascular simulations.

Abbas and Mourad (2020) developed a mathematical framework for simulating blood solidification and rupture within stenotic arteries. Their study highlights the role of abnormal rheological behavior during clot formation and demonstrates that localized changes in viscosity can cause substantial alterations to pressure gradients and blood transport. This study also illustrates how progressive thrombus development can increase resistance to blood flow and mechanical stress causing possible vascular occlusion or rupture.

Athani et al. (2021) investigated left coronary arteries with multiple stenoses using a two-phase blood flow model that is non-Newtonian and pulsatile. Simulations showed distinct velocity distributions especially in severely constricted regions. They report elevated wall shear stress, enhanced secondary flow structures and greater losses of pressure in arteries with multiple stenoses. Results confirmed incorporation of realistic arterial geometry and interaction between phases improves prediction of localized abnormalities related to coronary artery disease.

Zhao et al. (2022) also performed computational investigation of blood flow through arteries with stenosis by looking closely at detailed hemodynamic characteristics varying severities of stenosis. Results show that increasing stenosis significantly raises drop in pressure, wall shear stress and intensity of recirculation and reduces perfusion downstream. They also show viscosity becomes increasingly important at low shear rate regions surrounding stenosis emphasizing limitations of using Newtonian approximations for pathological arterial flow.

Zhao et al. (2024) extended modeling of blood flow phases to narrowed micro vessels and focused on interactions between plasma and suspended blood cells. Numerical analysis showed that phase separation becomes increasingly important in confined vascular geometry producing heterogeneous velocity fields and localized variations in hematocrit concentration.

Results highlighted that multiphase formulations better capture mechanisms of transport at the micro scale and improve resistance prediction in diseased micro circulation.

Haider et al. (2025) compared finite volume and finite element methods for physiological flow simulation. Their investigation showed both numerical approaches reproduce distributions of velocity and pressure accurately but differences in convergence characteristics and computational efficiency were noted. Importance of choosing suitable discretization techniques for reliable cardiovascular simulations was stressed and confirmed that numerical accuracy directly impacts clinically relevant hemodynamic index prediction.

Jouneghani et al. (2025) performed comprehensive biomechanical stress analysis of coronary arteries using a two-phase fluid and structure interaction framework. Their results showed coupling plasma and cell interactions with deformation of arterial walls leads to more accurate estimation of stress and distribution of strain compared to uncoupled models.

Laha et al. (2025) also looked at separation of blood phases in environments with microstructure and found red blood cells and plasma separate differently under complex geometric constraints. Results showed microstructure features strongly influence local distribution of hematocrit, effective viscosity and resistance to flow. Observations improved understanding of transport mechanisms for multiphase flow in confined biological environments and highlighted the physiological importance of segregation of plasma and cells.

Rawat et al. (2025) developed a mathematical model for blood flow through arteries with multiple stenoses under magnetohydrodynamics in porous media and results showed magnetic fields reduce velocity while permeability of porous medium facilitates transport. Presence of multiple stenoses showed much higher losses of pressure and wall shear stress compared to configurations with single stenosis.

Saib et al. (2025) review comprehensively fluid structure interaction in blood flow in arteries and summarize recent advances in computational modeling techniques. They stress that realistic cardiovascular simulations need to simultaneously represent blood rheology, arterial mechanics, specific patient geometry and numerical stability. Their review points out fluid structure interaction is critical for

accurate prediction of stress on arterial walls and deformation and also highlights current challenges in computational efficiency and translation to clinical practice.

Shen et al. (2025) simulated helical flow at high intensity within coronary artery segments. The authors determined that helical motion produces beneficial and harmful effects on hemodynamics. Simulations from Shen et al. indicate that moderate levels of helicity increase mixing and reduce stagnation. Conversely, high levels of helicity result in localized increases in wall shear stress and mechanical loads. Thus, it is possible that some degree of helical flow protects against thrombus formation, but excessive helical rotation can lead to injury of the vasculature.

Computational Fluid Dynamics (CFD) was used by Tina et al. (2025) to investigate blood flow through the aorta under three different sets of physiological conditions; namely, normal, diabetes-induced hyperglycemia, and anemia. Blood properties affect how flow behaves as shown by them simulations. Specifically, the presence of elevated viscosity due to hyperglycemia resulted in higher resistances to flow. Additionally, decreased viscosity associated with anemia caused changes to the overall forces exerted upon the vessel walls. Overall, these studies demonstrate the necessity of incorporating disease-specific rheological characteristics into models designed to evaluate cardiovascular function.

Blood flow past both constricted and expanded portions of diseased arteries was modeled by Hussain et al. (2026). An equation derived from the Sisko non-Newtonian fluid model was employed in their CFD simulation. Results from their simulations indicate that simultaneous constriction and dilation create very complex velocity distributions including areas of high recirculation rates, increased pressure drop, and varying wall shear stresses. Also, Hussain et al. demonstrated that the Sisko rheological model provides improved representations of shear-thinning behaviors over traditional constant-viscosity models, especially in those regions subjected to relatively large shear-rate gradients.

Using a multiphase CFD model, Kumar et al. (2026) studied red blood cell-plasma interactions throughout diseased arterial segments. Their simulations show that mass transfer across interfaces between the plasma and erythrocytes has a strong effect on the

velocity field, hematocrit concentration transport, and local pressure values throughout the diseased segment of the artery. Therefore, they conclude that the use of two distinct phases in blood instead of one homogeneous phase will yield better predictive capabilities for local flow irregularities in narrow diseased arteries.

A novel multiphase computational model for predicting turbulent blood flow in severely narrowed sections of arteries was developed by Patel et al. (2026). Their computational analysis demonstrates that turbulence significantly increases mixing between the plasma and cellular phases leading to larger pressure drops, stronger secondary vortex motions, and higher wall shear stresses. The work also indicates that using a combination of turbulence modeling with multiphase blood rheology would provide more accurate predictive capabilities for severe pathologic states where laminar flow assumption fails. The effects of turbulence on flow through patient-specific stenosed arteries subjected to pulsatile flow conditions were investigated by Wang et al. (2026). Their results demonstrate that turbulence significantly affects velocity fluctuation amplitudes, wall shear stress oscillations and energy dissipation during each cardiac cycle. In addition, they found that spatial variability in flow instability occurs throughout patient-specific geometries indicating the need for individualized computational models for the accurate clinical evaluation of stenotic lesions

II. FORMULATION OF THE MODEL

2.1. Physical model:

Consider blood flow through a diseased artery of length L , where stenosis or plaque deposition reduces the arterial radius. Blood is treated as a two-phase suspension consisting of: plasma phase (p), red blood cell phase (c). The artery is assumed cylindrical, axisymmetric, and mildly stenosed.

The stenosed arterial radius is taken as

$$R(z) = R_0 \left[1 - \delta_s \left(1 - \cos \frac{2\pi z}{L_s} \right) \right]; 0 \leq z \leq L_s \quad (7.1)$$

where R_0 is the normal radius, δ_s is stenosis severity and L_s is stenosis length.

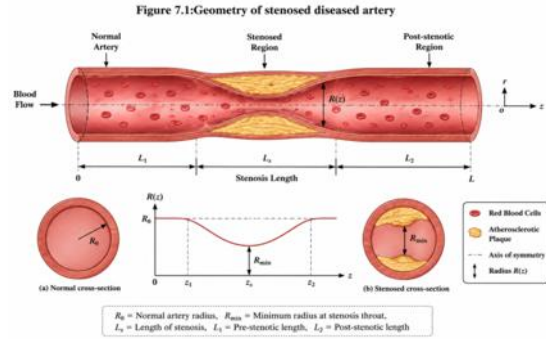


Figure 7.1 illustrates the geometric model of a stenosed diseased artery used for the mathematical analysis of blood flow.

The artery is divided into three regions: the pre-stenotic region, where the artery has its normal radius R_0 ; the stenosed region, where atherosclerotic plaque accumulates and narrows the lumen; and the post-stenotic region, where the artery gradually returns to its original diameter. Blood flows along the axial direction (z -axis), while the radial direction is represented by the r -axis. The local arterial radius is denoted by $R(z)$, which decreases smoothly to the minimum radius R_{min} at the stenosis throat before increasing again downstream.

The lengths of the pre-stenotic, stenosed, and post-stenotic sections are represented by L_1 , L_s and L_2 , respectively. The lower left cross-sectional view shows the normal artery with a circular lumen of radius R_0 , whereas the lower right cross-section illustrates the narrowed lumen caused by plaque deposition, reducing the flow area to R_{min} . The figure (7.1) illustrates a continuous narrowing followed by widening of an artery as one move from top to bottom. An ideal model for this geometry is used to analyze how stenosis affects the velocity profile of fluid flow, wall shear (the frictional force exerted by flowing blood on the endothelium), pressure drop across the area affected by the stenosis, turbulence within the narrowed portion of the artery, and the interaction between plasma and red blood cells during their passage through this segment.

2.2. Governing equations: For each phase $k = p, c$, the continuity equation is

$$\nabla \cdot \mathbf{u}_k = 0 \quad (7.2)$$

The momentum equation for turbulent multiphase blood flow is

$$\rho_k \phi_k \left(\frac{\partial u_k}{\partial t} + u_k \cdot \nabla u_k \right) = -\phi_k \nabla p + \nabla \cdot \tau_k + M_{kj} \quad (7.3)$$

Where $\phi_p + \phi_c = 1$

and $\phi_c = H$ represents hematocrit.

The phase interaction force is modeled by interphase drag:

$$M_{pc} = K(u_c - u_p), M_{cp} = K(u_p - u_c) \quad (7.4)$$

where K is the phase interaction coefficient.

2.3. Turbulence closure: For diseased arteries, disturbed flow near stenosis may become transitional or turbulent. The Reynolds stress is included through an effective turbulent viscosity:

$$\mu_{k,eff} = \mu_k + \mu_{t,k} \quad (7.5)$$

Where $\mu_{t,k} = \rho_k l_m^2 \left| \frac{\partial u_k}{\partial r} \right|$ is the Prandtl mixing-length approximation. For analytical treatment, $\mu_{t,k}$ is locally approximated as constant, so

$$\mu_{k,eff} = \text{constant} \quad (7.6)$$

This gives a solvable closed-form model.

2.4. Axisymmetric reduced model: Assume fully developed axial flow:

$$u_k = (0, 0, u_k(r, t)) \quad (7.7)$$

Then the momentum equations become

$$\rho_p \phi_p \frac{\partial u_p}{\partial t} = -\phi_p \frac{\partial p}{\partial z} + \mu_{p,eff} \left[\frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial u_p}{\partial r} \right) \right] + K(u_c - u_p) \quad (7.8)$$

$$\rho_c \phi_c \frac{\partial u_c}{\partial t} = -\phi_c \frac{\partial p}{\partial z} + \mu_{c,eff} \left[\frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial u_c}{\partial r} \right) \right] + K(u_p - u_c) \quad (7.9)$$

Boundary conditions are

$$\frac{\partial u_p}{\partial r} = 0, \frac{\partial u_c}{\partial r} = 0 \text{ at } r = 0 \quad (7.10)$$

$$u_p = 0, u_c = 0 \text{ at } r = R(z) \quad (7.11)$$

2.5. Pulsatile pressure gradient: Let the pressure gradient be

$$-\frac{\partial p}{\partial z} = G_0 + G_1 e^{i\omega t} \quad (7.12)$$

where G_0 is steady pressure forcing and $G_1 e^{i\omega t}$ is pulsatile forcing.

$$\text{Hence, } u_k(r, t) = u_{k0}(r) + \Re\{U_k(r) e^{i\omega t}\} \quad (7.13)$$

III. SOLUTION OF THE MODEL

3.1. Analytical steady solution:

For the steady case,

$$\frac{\partial u_k}{\partial t} = 0 \quad (7.14)$$

and if phase slip is small, $u_p \approx u_c = u$. The mixture equation becomes

$$0 = -G_0 + \mu_{eff} \left[\frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial u}{\partial r} \right) \right] \quad (7.15)$$

Were

$$\mu_{eff} = (1 - H)\mu_{p,eff} + H\mu_{c,eff} \quad (7.16)$$

$$\text{Integrating twice gives } u(r) = \frac{G_0}{4\mu_{eff}} [R^2(z) - r^2] \quad (7.17)$$

This is the turbulent-modified Poiseuille solution.

$$\text{The volumetric flow rate is } Q(z) = 2\pi \int_0^{R(z)} r u(r) dr \quad (7.18)$$

The wall shear stress is

$$\tau_w = -\mu_{eff} \left. \frac{\partial u}{\partial r} \right|_{r=R(z)} \quad (7.19)$$

$$\tau_w = \frac{G_0 R(z)}{2} \quad (7.20)$$

$$\text{The pressure drop is therefore } \Delta p = \int_0^L \frac{8\mu_{eff} Q}{\pi R^4(z)} dz \quad (7.21)$$

3.2. Analytical pulsatile solution: For pulsatile flow, the reduced mixture equation is

$$\rho_m \frac{\partial u}{\partial t} = -\frac{\partial p}{\partial z} + \mu_{eff} \left[\frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial u}{\partial r} \right) \right]$$

Were

$$\rho_m = (1 - H)\rho_p + H\rho_c$$

Assume

$$u(r, t) = u_0(r) + \Re\{U(r) e^{i\omega t}\}$$

The pulsatile part satisfies $i\omega \rho_m U = G_1 +$

$$\mu_{eff} \left[\frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial U}{\partial r} \right) \right]$$

The solution is

$$U(r) = \frac{G_1}{i\omega \rho_m} \left[1 - \frac{J_0(\lambda r)}{J_0(\lambda R)} \right]$$

were

$$\lambda^2 = \frac{i\omega \rho_m}{\mu_{eff}}$$

and J_0 is the Bessel function of first kind.

Thus, the complete analytical velocity field is

$$u(r, t) = \frac{G_0}{4\mu_{eff}} (R^2 - r^2) + \Re \left\{ \frac{G_1}{i\omega \rho_m} \left[1 - \frac{J_0(\lambda r)}{J_0(\lambda R)} \right] e^{i\omega t} \right\} \quad (7.22)$$

3.3. Phase interaction solution: For plasma and RBC phases, define the slip velocity

$$w = u_c - u_p$$

Subtracting the two-phase momentum equations gives

$$\rho_s \frac{\partial w}{\partial t} = \mu_s \left[\frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial w}{\partial r} \right) \right] - 2Kw$$

where ρ_s and μ_s are effective slip-density and slip-viscosity parameters.

For steady flow,

$$\mu_s \left[\frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial w}{\partial r} \right) \right] - 2Kw = 0$$

$$\text{Let } \beta^2 = \frac{2K}{\mu_s}$$

Then

$$\frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial w}{\partial r} \right) - \beta^2 w = 0$$

The solution is $w(r) = A I_0(\beta r) + B K_0(\beta r)$

Since K_0 is singular at $r = 0$, $B = 0$. Hence,

$$w(r) = A I_0(\beta r)$$

Using prescribed wall slip $w(R) = w_R$,

$$w(r) = w_R \frac{I_0(\beta r)}{I_0(\beta R)}$$

$$\text{Therefore, } u_c(r) = u(r) + \frac{1-H}{1} w(r) \quad (7.23)$$

$$u_p(r) = u(r) - H w(r) \quad (7.24)$$

where $u(r)$ is the mixture velocity.

3.4 non-dimensional parameters: Introduce

$$\bar{r} = \frac{r}{R_0}, \bar{z} = \frac{z}{L}, \bar{u} = \frac{u}{U_0}$$

Important dimensionless parameters are:

$$Re = \frac{\rho_m U_0 R_0}{\mu_{eff}}$$

$$\alpha = R_0 \sqrt{\frac{\omega \rho_m}{\mu_{eff}}}$$

$$Da = \frac{\mu_{eff}}{KR_0^2}$$

$$S = \frac{R_0 - R_{min}}{R_0}$$

where Re is Reynolds number, α is Womersley number, Da is phase interaction parameter, and S is stenosis severity.

IV. RESULTS AND DISCUSSION

Table 7.1. Model Parameters and Physiological Values

Symbol	Parameter	Typical Value	Unit
R_0	Normal artery radius	3	mm
L_s	Stenosis length	20	mm
S	Stenosis severity	20–80	%
ρ	Blood density	1060	kg m ⁻³
μ	Blood viscosity	3.5×10^{-3}	Pa·s
μ_t	Turbulent viscosity	0 – 5 $\times 10^{-3}$	Pa·s
H	Hematocrit	0.45	—
U_0	Mean velocity	0.3	m s ⁻¹

Q	Flow rate	5×10^{-6}	m ³ s ⁻¹
ΔP	Pressure drop	100–2000	Pa
τ_w	Wall shear stress	1–15	Pa
Re	Reynolds number	200–1200	—
α	Womersley number	2–8	—
K	Phase interaction coefficient	10^5	kg m ³ s ⁻¹

Figure 7.2: Velocity profile for different stenosis severities

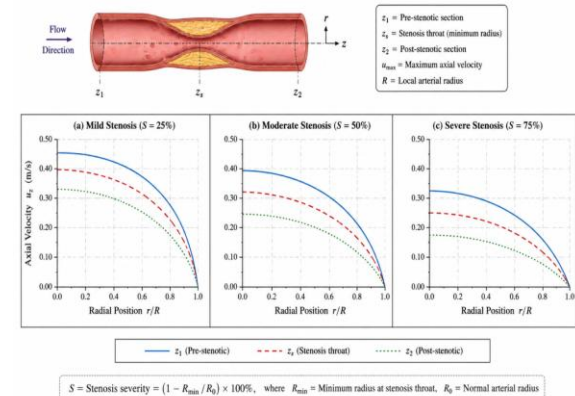
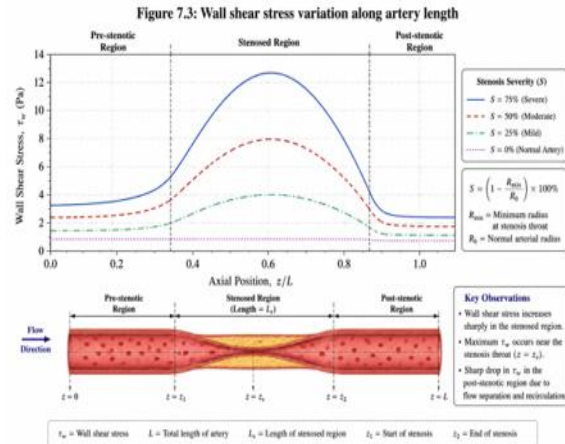


Figure 7.2 presents the axial velocity profiles of blood flow for three different stenosis severities, namely mild (25%), moderate (50%), and severe (75%), illustrating how arterial narrowing influences the velocity distribution across the vessel radius.

The upper schematic identifies the pre-stenotic region (z_1), the stenosis throat (z_s), and the post-stenotic region (z_2), indicating the direction of blood flow and the radial and axial coordinate system. In each velocity profile, the highest velocity occurs at the centerline of the artery ($\frac{r}{R} = 0$) and gradually decreases toward zero at the arterial wall ($\frac{r}{R} = 1$) due to the no-slip boundary condition. The blue solid curve represents the velocity in the pre-stenotic region, the red dashed curve corresponds to the stenosis throat, and the green dotted curve represents the post-stenotic region. As the stenosis severity increases from 25% to 75%, the overall velocity magnitude decreases in the plotted sections, reflecting the increasing resistance to blood flow and enhanced energy losses caused by the narrowing of the artery. The decrease of velocity is at its greatest extent for the severe stenosis case and this can be taken to indicate that a large amount of arterial constriction will have adverse effects on blood flow through the diseased artery and alter hemodynamics throughout the diseased artery.



The figure (7.3) shows how wall shear stress varies at different distances from a narrowed artery segment during four levels of narrowing (normal, mild, moderate, and severe). The x-axis measures distance (z) relative to the total length (L) of the narrowed area, and the y-axis measures wall shear stress (τ_w) in units of pressure (pascals).

The artery is depicted as being broken down into three distinct areas: pre-stenotic area; stenosed area; and post-stenotic area. Wall shear stress remained relatively consistent throughout the pre-stenotic area for each level of narrowing. Once the blood enters the stenosed area, where there is an increase in velocity due to the constriction of the artery's diameter, wall shear stress increases dramatically and reaches its highest point at or just past the narrowest part of the artery ($z = z_s$). This wall shear stress continues to increase with the severity of the narrowing. The increased friction produced by this high-velocity blood flow will continue to have effects on the arterial walls. Once past the narrow portion of the artery, it begins to widen again in the post-stenotic area and undergoes flow reversal, recirculation, and ultimately returns to typical values. Thus, severe narrowing creates significantly greater wall shear stress than do both mild and moderate narrowing.

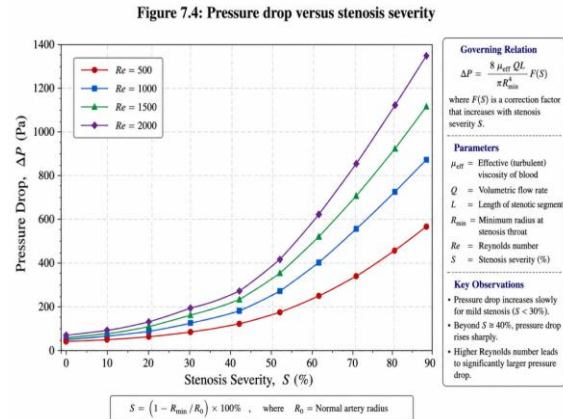


Figure 7.4 shows how pressure loss varies with the degree of atherosclerotic stenosis in arteries for three levels of flow velocity ($Re=500, 1000$, and 2000).

Pressure loss is represented by the vertical axis and the stenosis severity by the horizontal axis. The pressure loss will be low for minor degrees of stenosis; however, once the stenosis exceeds about 40%, there is a sharp rise in pressure loss. This is due to the considerable decrease in diameter of the artery and hence in its cross-sectional area. The increased resistance of the narrowed section results from both the wall shear stress and the turbulent flow generated. At each level of stenosis, higher values of Reynolds number give a greater pressure loss since the inertial forces and the associated turbulence result in additional dissipation of kinetic energy.

As expected, the greatest pressure losses were found at $Re = 2000$ and the least at $Re = 500$. The equation provided in the diagram illustrates that the pressure loss is dependent upon blood viscosity (the effective viscosity of blood), volumetric flow rate (the volume of fluid per unit time passing through the artery), length of the stenosis, and inversely upon the fourth power of the minimum diameter of the artery. These factors emphasize that it is not just the presence of narrowing of the artery which generates a high hemodynamic resistance but also its extent. Overall, this plot demonstrates that increasing either stenosis or flow velocity can cause a large increase in the pressure needed to circulate blood through an obstructed artery. Therefore, pressure drop may be used as an index of the degree of occlusion of an artery.

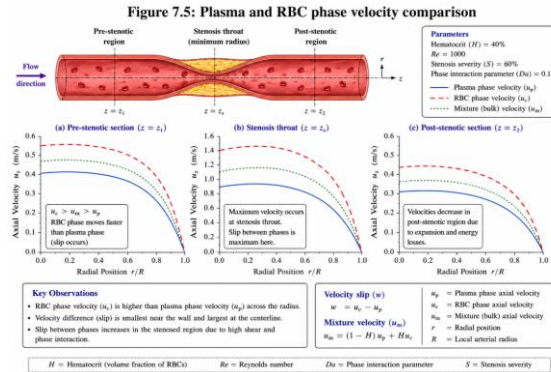


Figure 7.5 illustrates how the axial velocity profile for the plasma phase, red blood cell (RBC) phase and bulk-mixture phase varies at three distinct locations within a stenosed artery; namely the pre-stenotic region (z_1), the stenosis throat ($z = z_s$), and the post-stenotic region ($z = z_2$).

An illustration of the geometry of the artery and the direction of flow through the stenosis is presented schematically on top. Three separate plots show how the axial velocity changes from $r = 0$ (the centre-line of the artery) to $r = R$ (the artery wall) for each section. For all sections of the plot, the velocity will be at its peak along the centre line of the artery and reduce to zero at the artery wall due to the presence of a no-slip boundary.

The velocity of the RBC phase (u_c) was greater than the velocity of the plasma phase (u_p), while the velocity of the mixture (u_m) falls somewhere between u_c and u_p , since it represents the combined motions of both phases. Due to the constriction of the artery at the stenosis throat, there will be an acceleration of the flow causing the highest velocities and therefore a larger velocity difference or slip between the plasma and RBC phases. In addition, after passing over the stenosis throat, there will be a reduction in velocities as the artery begins to expand resulting in a reduction in flow rates and an increase in energy loss.

As shown by Figure 7.5, the velocity slip ($u_c - u_p$) will be greatest closest to the stenosis throat where the effect of shear and interactions between phases will be most pronounced, while it will be lower in the pre- and post-stenotic regions. Therefore, overall, this figure has demonstrated how arterial stenoses have an impact on the individual phase velocities and their interaction, which can play a major role in defining the

hemodynamics of blood flow through diseased arteries.

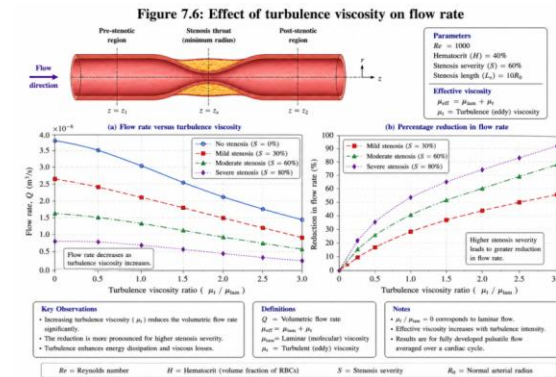


Figure 7.6 demonstrates how turbulence viscosity impacts volumetric flow rates within an artery narrowed by stenosis under varying degrees of stenosis.

The top diagram illustrates the anatomy of the artery. In this diagram, it is shown that there are three regions where the blood is flowing; the pre-stenotic region, the stenotic region and the post-stenotic region. The graphs show volumetric flow rates versus the turbulence viscosity ratio ($\mu_t / \mu_{\text{turb}}$) for arteries that have undergone normal, mild, moderate or severe stenosis. As can be seen from these plots, the volumetric flow rate continues to decrease as the turbulence viscosity ratio is increased since the total blood viscosity is increased thereby reducing flow rates due to additional resistance.

This reduction of flow rate is very minimal when an artery has been subjected to normal flow conditions however will become more profound as the degree of stenosis becomes greater. The most extreme flow condition exists for a severely narrowed artery with the greatest degree of reduction existing at higher levels of turbulence viscosity ratios. The right-hand graph displays the percent reduction in flow rates over the same level of turbulence viscosity ratios as was depicted in the left-hand graph.

These plots illustrate that both the increase in turbulence viscosity and the increase in stenosis reduce flow rates more profoundly than do either variable alone. Under extreme stenosis conditions, the percent reduction in flow rate exceeds ninety percent. However, for less severe forms of stenosis such as mild stenosis, there is a relatively small amount of reduction in flow rate. The bottom portion of the plot provides boxes that list some physiological parameters

used in the study along with their respective values. They include Reynolds numbers, hematocrits, stenosis severities and lengths. Also listed in this section are definitions regarding how to calculate the effective viscosity as being equal to the sum of both laminar and turbulent viscosities. Therefore, this plot clearly illustrates how the effects of turbulence viscosity can enhance viscous losses in arteries that have disease processes occurring within them. More specifically, the data illustrated in this figure demonstrate how turbulence and arterial disease can interact to produce altered hemodynamic conditions that may contribute to vascular diseases and thrombosis formation.

Table 7.4. Effect of Hematocrit on Effective Viscosity

Hematocrit, (H) (%)	Effective Viscosity, μ (eff)(mPa·s)	Flow Characteristic
30	2.8	Low viscosity, high flow
35	3.2	Moderate flow
40	3.8	Normal physiological flow
45	4.5	Increased flow resistance
50	5.3	High viscosity, reduced flow
55	6.2	Very high resistance

V. CONCLUDING REMARKS

The analytical model presented here has been used to study the effects of turbulence and plasma-RBC phase interactions on blood flow in stenotic arterial vessels. The analysis indicates that significant alterations in the physical properties of blood flow occur due to stenosis which include increased wall shear stress, increased pressure loss, increased flow resistance, reduced volumetric flow rate. Turbulent viscosity has been found to be an important addition to the model since it has improved predictions of distorted flow in the region of the stenosis. A two-phase model was also shown to predict the effect of plasma and RBC velocity slip on each other. Analytical expressions have been developed from these models that indicate the location of the maximum velocity and wall shear stress occurs at or very near the throat of the stenosis. In this area both turbulence and phase interaction is most intense. Increased hematocrit (the ratio of packed

red cells to total volume) increases the effective viscosity of blood resulting in a greater amount of resistance to flow and larger pressure drops. Therefore, the three major factors controlling arterial hemodynamics are stenosis severity, turbulence intensity, Reynolds numbers and hematocrit.

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