

Article

Mathematical Modelling of Sickle-Cell-Induced Variable Viscosity Effects on Magnetohydrodynamic Blood Flow Through a Stenosed Artery with Heat and Mass Transfer

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Abstract

Sickle cell disease is associated with abnormal erythrocyte deformation and altered blood rheology, which may increase resistance to blood flow and contribute to impaired tissue perfusion. The presence of arterial stenosis can further modify the haemodynamic environment and intensify flow resistance. This study develops a mathematical model to investigate the effects of sickle-cell-induced variable viscosity on magnetohydrodynamic blood flow through a stenosed artery in the presence of heat and mass transfer. Blood is modelled as an incompressible, electrically conducting fluid flowing through a symmetric stenosed arterial segment represented within a porous-medium framework. The formulation incorporates concentration-dependent variable viscosity, magnetic resistance, Darcy resistance, thermal and concentration buoyancy, heat transfer, mass diffusion and a prescribed pressure gradient. Appropriate dimensionless variables are introduced to transform the governing momentum, energy and concentration equations into a dimensionless form. A perturbation approach is employed to obtain an analytical expression for the velocity distribution, while the temperature and concentration equations are solved subject to symmetry and wall boundary conditions. Numerical evaluations of the analytical solution are presented using velocity profiles and two-dimensional contour plots for different values of the pertinent parameters. The results show that increasing the favourable pressure gradient, permeability, thermal buoyancy, and concentration buoyancy generally enhances blood velocity. In contrast, increasing magnetic intensity and the sickle-cell-related variable-viscosity contribution suppress the velocity field. Increasing stenosis severity increases flow resistance and generally reduces the overall velocity under the prescribed pressure-gradient condition. The mass-transfer parameter produces comparatively moderate changes in the velocity distribution, while the thermal parameter exhibits stronger sensitivity within particular parameter ranges. The study provides a theoretical framework for understanding the coupled effects of abnormal blood rheology, arterial constriction, magnetic interaction, heat transfer and species transport in sickle-cell-related haemodynamics.



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1. Introduction

Blood flow in the human circulatory system is a highly complex physiological process governed by the interaction of fluid mechanics, vessel geometry, and biochemical properties of blood constituents. Under normal conditions, blood behaves as a non-Newtonian fluid whose viscosity depends on shear rate, haematocrit level, and red blood cell (RBC) deformability [1,2]. However, in pathological conditions such as sickle cell disease (SCD), these properties are significantly altered, leading to abnormal flow characteristics and impaired microcirculation [3,4].

Sickle cell disease is a hereditary blood disorder caused by a mutation in the β -globin gene, resulting in the production of abnormal haemoglobin known as haemoglobin S (HbS) [5]. Under hypoxic conditions, HbS undergoes polymerization, causing RBCs to assume a rigid, sickle-like shape [6,7]. These deformed cells exhibit reduced flexibility, increased adhesiveness, and a higher tendency to aggregate, which significantly increases blood viscosity and disrupts normal flow patterns [8,9]. Consequently, vaso-occlusion occurs, leading to restricted blood flow, tissue ischaemia, and organ damage [10].

The rheological behaviour of sickle blood is markedly different from that of healthy blood. Studies have shown that sickled erythrocytes exhibit increased internal viscosity and reduced deformability, which impair their ability to pass through narrow capillaries [11,12]. Additionally, interactions between sickled RBCs, leukocytes, and endothelial cells contribute to inflammation and vascular dysfunction [13,14]. These processes collectively result in altered haemodynamics, characterised by non-uniform velocity profiles, increased wall shear stress, and enhanced flow resistance [15].

Recent research has highlighted that SCD is not merely a haematological disorder but also a vascular disease due to its significant impact on endothelial function and blood flow regulation [16]. The presence of abnormal cells in circulation leads to endothelial activation, oxidative stress, and nitric oxide depletion, further exacerbating vascular complications [17]. These factors contribute to the development of atherosclerosis, a condition characterised by plaque formation and the narrowing of blood vessels [18]. In such stenosed vessels, blood flow becomes highly restricted, and the effects of viscosity, pressure gradient and external forces become more pronounced [19].

Mathematical modelling plays a crucial role in understanding the complex dynamics of blood flow under such pathological conditions. Blood flow in arteries and capillaries can be described using the Navier–Stokes equations, modified to account for non-Newtonian behaviour and additional physiological factors [20]. In stenosed channels, the geometry of the vessel significantly affects velocity distribution and pressure drop, making analytical and numerical modelling essential tools for investigation [21].

The influence of external magnetic fields on blood flow has also attracted considerable attention. As blood is an electrically conducting fluid due to the presence of ions, its motion can be influenced by magnetic fields through the principles of magnetohydrodynamics (MHD) [22]. The application of a magnetic field introduces a Lorentz force, which opposes fluid motion and leads to a reduction in velocity [23]. This effect has important implications in biomedical engineering, particularly in magnetic drug targeting and diagnostic imaging techniques [24].

In addition to magnetic effects, heat and mass transfer phenomena play significant roles in blood flow behaviour. Temperature variations affect blood viscosity, with higher temperatures generally leading to reduced viscosity and enhanced flow [25]. Similarly, concentration gradients influence mass diffusion and generate buoyancy forces that can alter velocity distribution. These effects are commonly characterised by dimensionless parameters such as the thermal Grashof number and solutal Grashof number, which represent the ratio of buoyancy forces to viscous forces [26].

The presence of porous structures, such as atherosclerotic plaques, introduces additional resistance to blood flow. This effect is often modelled using Darcy's law, where the Darcy number represents the permeability of the medium [27]. A higher Darcy number indicates greater permeability and reduced resistance, allowing for improved flow through the porous region. Furthermore, slip boundary conditions are sometimes applied to account for partial adherence of blood at the vessel wall, especially in microcirculatory systems [28].

Thermal radiation is another factor that can influence blood flow, particularly in cases involving external heating or radiative therapies. Radiation enhances heat transfer within the fluid, leading to changes in temperature distribution and viscosity [29]. The combined effects of radiation, magnetic field, heat transfer, and mass diffusion create a highly coupled system that requires advanced analytical techniques for proper understanding.

Various analytical methods, including the Adomian decomposition method (ADM), homotopy perturbation method (HPM), and numerical simulations, have been employed to study such complex flow systems [30]. These methods provide approximate or exact solutions to the governing equations and enable the analysis of velocity, temperature, and concentration profiles under different physical conditions.

Despite extensive research, there is still a need for comprehensive models that incorporate the simultaneous effects of temperature, concentration, magnetic field, radiation, and arterial constriction in the context of sickle cell disease. Such integrated approaches are essential for accurately predicting blood flow behaviour and for developing effective therapeutic strategies.

Therefore, this study focuses on the mathematical modelling of temperature and concentration effects on sickle-cell-induced atherosclerotic blood flow through a capillary channel in the presence of an applied magnetic field. The model incorporates key parameters such as slip conditions, Darcy number, Grashof numbers, and radiation effects to provide a detailed analysis of velocity distribution. The findings of this study are expected to contribute to a better understanding of haemodynamic alterations in SCD and support the development of improved clinical interventions.

2. Mathematical Formulation

The mathematical formulation describes blood flow through a stenosed artery while incorporating the principal physical mechanisms considered in the study: sickle-cell-induced variation in blood viscosity, magnetic-field effects, porous resistance, heat transfer, mass transfer, thermal buoyancy and concentration buoyancy. The coordinate x^* is measured along the arterial axis, while y^* is measured transversely from the centreline toward the arterial wall. The problem flow chart used in this study is outlined in Appendix A Figure A1.

2.1. Assumptions

The study assumes incompressible, laminar blood flow through a smooth, symmetric and rigid stenosed artery. Blood is represented as an electrically conducting continuum whose effective viscosity varies with the concentration-related sickle-cell contribution. The arterial wall is impermeable and satisfies the no-slip condition, while symmetry conditions are imposed at the vessel centreline. The stenosed region is represented using a homogeneous porous-medium approximation, and the flow is driven by a prescribed axial pressure gradient. A uniform transverse magnetic field is imposed, while the induced magnetic field and externally applied electric field are neglected. Heat and mass transfer are incorporated, including thermal and concentration buoyancy, internal heat effects, molecular diffusion and a simplified concentration reaction. The Boussinesq approximation is adopted for buoyancy effects, and thermophysical properties are assumed constant except for the explicitly modelled variable viscosity. The variable-viscosity contribution is assumed to be sufficiently small to permit perturbation analysis. Vessel-wall elasticity,

turbulence, individual red-cell dynamics and time-dependent stenosis development are neglected. Parameter values are restricted to ranges for which the analytical solution remains mathematically regular.

2.2. Geometry of Stenosis

Following the abovementioned assumptions, the mathematical geometry of the mild stenosis can be represented as follows:

$$h^*(x^*) = R_0 - \frac{\delta^*}{2} \left(1 - \cos \left(\frac{2\pi(x^* - d)}{\lambda_s} \right) \right) \quad (1)$$

3. Governing Mathematical Models

3.1. Continuity Equation

$$\nabla \cdot \vec{V} = 0 \quad (2)$$

3.2. Momentum Equation (MHD Blood Flow)

Under the stated assumptions, the momentum equation is written as follows:

$$\frac{\partial P^*}{\partial x^*} = \mu(\phi) \frac{d^2 u^*}{dy^2} - \sigma B_0^2 u^* - \frac{\mu(\phi)}{k} u^* + \rho g \beta_T (T^* - T_\infty) + \rho g \beta_C (C^* - C_\infty) \quad (3)$$

where u^* is the dimensional axial velocity, P^* is pressure, σ is electrical conductivity, B_0 is the imposed magnetic-field strength, k is permeability, ρ is blood density, g is gravitational acceleration, and β_T and β_C are the thermal and concentration expansion coefficients, respectively.

3.3. Energy Equation (Temperature)

$$\alpha \frac{d^2 T^*}{dy^2} + Q_0 (T^* - T_\infty) = 0 \quad (4)$$

where α denotes thermal diffusivity and Q_0 characterizes the heat-generation or absorption mechanism.

3.4. Concentration Equation (Mass Concentration)

$$D_m \frac{d^2 C^*}{dy^2} - k_0 (C^* - C_\infty) = 0 \quad (5)$$

where D_m is the mass diffusivity and k_0 is the concentration reaction or removal coefficient.

3.5. Variable Viscosity Model (Sickle Effect)

$$\mu(\phi) = \mu_b (1 + \lambda) \quad (6)$$

where $\lambda = \alpha_s \phi$.

The boundary conditions are as follows:

$$\left. \begin{aligned} u^* = 0, T^* = T_w, C^* = C_w, \quad \text{at } y^* = R \\ \frac{du^*}{dy^*} = 0, \frac{dT^*}{dy^*} = 0, \frac{dC^*}{dy^*} = 0, \quad \text{at } y^* = 0 \end{aligned} \right\} \quad (7)$$

4. Dimensional Variables

The variables in the governing equations are now rescaled into dimensionless form. This step converts the raw physical quantities (velocity, temperature, distance) into ratios that allow direct comparison of competing effects. Each dimensionless parameter can be thought of as a dial controlling one physical factor: M controls magnetic field strength, Da controls vessel wall permeability, Gr and Gc control buoyancy from heat and concentration, respectively, and so on. Section 6 varies these dials one at a time to show their individual effect on blood velocity. We introduce the following dimensionless parameters:

$$\left. \begin{aligned} y &= \frac{y^*}{R_0}, u = \frac{u^*}{U_0}, \theta = \frac{T^* - T_\infty}{T_w - T_\infty}, \phi = \frac{C^* - C_\infty}{C_w - C_\infty}, Rd_1 = \frac{Q_0 R_0^2}{\alpha}, Gr = \frac{g \beta_T (T_w - T_\infty) R_0^2}{U_0 v^2}, \delta^* = R_0 \delta \\ Gc &= \frac{g \beta_C (C_w - C_\infty) R_0^2}{U_0 v^2}, Da = \frac{1}{R_0^2}, M^2 = \frac{\sigma B_0^2 R_0^2}{\mu_b}, Pr = \frac{\mu_b c_b}{k_b}, R_T = \frac{k_0 R_0^2}{D_m}, x = \frac{x^* - d}{\lambda_s}, h = \frac{R}{R_0} \end{aligned} \right\} \quad (8)$$

1. Dimensionless Equation

Using the above dimensionless variables, Equations (1)–(7) become the following:

2. Geometry of Stenosis

Following the abovementioned assumptions, mathematical geometry of the mild stenosis can be represented as follows:

$$h(x) = \begin{cases} 1 \\ 1 - \frac{\delta}{2}(1 - \cos(2\pi x)) \\ 1 \end{cases} \quad (9)$$

3. Momentum Equation

$$(1 + \alpha_s \phi) \frac{d^2 u}{dy^2} - \left(M^2 + \frac{(1 + \alpha_s \phi)}{Da} \right) u + Gr \theta + Gc \phi = -P \quad (10)$$

4. Energy Equation

$$\frac{d^2 \theta}{dy^2} + Rd_1 \theta = 0 \quad (11)$$

5. Concentration Equation

$$\frac{d^2 \phi}{dy^2} - R_T \phi = 0 \quad (12)$$

6. Variable Viscosity

$$\frac{\mu(\phi)}{\mu_b} = 1 + \alpha_s \phi \quad (13)$$

The boundary conditions are as follows:

$$\left. \begin{aligned} u &= 0, \theta = 1, \phi = 1, & \text{at } y = h \\ \frac{du}{dy} &= 0, \frac{d\theta}{dy} = 0, \frac{d\phi}{dy} = 0, & \text{at } y = 0 \end{aligned} \right\} \quad (14)$$

5. Method of Solution

The coupled governing equations are solved analytically by first determining the temperature and concentration fields and then substituting these expressions into the momentum equation. Because the blood viscosity varies with the concentration field, the resulting momentum equation is nonlinear in the perturbation parameter. A regular perturbation technique is therefore employed to obtain the velocity distribution in successive orders.

The solutions to the dimensionless Equations (11) and (12) are as follows:

$$\theta(y) = \frac{\cos(\sqrt{Rd_1}y)}{\cos(\sqrt{Rd_1}h)} \quad (15)$$

$$\phi(y) = \frac{\cosh(\sqrt{R_T}y)}{\cosh(\sqrt{R_T}h)} \quad (16)$$

Because α_s represents the contribution of the concentration-dependent variable viscosity, it is assumed to be sufficiently small for a perturbation expansion. The velocity is written as

$$u = u_0 + \alpha_s u_1 + O(\alpha_s^2) \dots \quad (17)$$

neglecting terms of order α_s^2 and higher.

Simplifying Equation (10), we obtain the following:

$$\frac{d^2 u_0}{dy^2} - \left(M^2 + \frac{1}{Da}\right)u_0 + Gr\theta + Gc\phi + \alpha_s \left(\frac{d^2 u_1}{dy^2} - \left(M^2 + \frac{1}{Da}\right)u_1 - \left(\frac{\phi}{Da}u_0\right) + \phi \frac{d^2 u_0}{dy^2}\right) + \alpha_s^2 \left(\phi \frac{d^2 u_1}{dy^2} - \frac{\phi}{Da}u_1\right) = P \quad (18)$$

The zero-ordered equation is as follows:

$$\frac{d^2 u_0}{dy^2} - \left(M^2 + \frac{1}{Da}\right)u_0 = P - Gr\theta - Gc\phi \quad (19)$$

The first-ordered equation at $\alpha_s \neq 0$

$$\frac{d^2 u_1}{dy^2} - \left(M^2 + \frac{1}{Da}\right)u_1 = -\phi \left(\frac{d^2 u_0}{dy^2} - \frac{u_0}{Da}\right) \quad (20)$$

$$\alpha_s^2 \left(\phi \frac{d^2 u_1}{dy^2} - \frac{\phi}{Da}u_1\right) = 0 \quad (21)$$

Let $\beta^2 = M^2 + \frac{1}{Da}$, then Equations (19) and (20) become the following:

$$\frac{d^2 u_0}{dy^2} = P - Gr\theta - Gc\phi + \beta^2 u_0 \quad (22)$$

The first ordered equation at $\alpha_s \neq 0$

$$\frac{d^2 u_1}{dy^2} - \beta^2 u_1 = \phi \left(Gr\theta + Gc\phi - P - M^2 u_0\right) \quad (23)$$

The solution to Equation (22) is as follows:

$$u_0 = \beta_1 \cosh(\beta y) - \frac{P}{\beta^2} + \beta_2 \cos(\sqrt{Rd_1}y) + \beta_3 \cosh(\sqrt{R_T}y) \quad (24)$$

where

$$\beta_1 = \frac{\left(\frac{P}{\beta^2} - \frac{Gr}{\beta^2 + Rd_1} - \frac{Gc}{\beta^2 - Rd_T}\right)}{\cosh(\beta h)}, \beta_2 = \frac{\left(\frac{Gr}{\beta^2 + Rd_1}\right)}{\cos(\sqrt{Rd_1}h)}, \beta_3 = \frac{\left(\frac{Gc}{\beta^2 - Rd_T}\right)}{\cosh(\sqrt{Rd_T}h)} \quad (25)$$

Solving Equation (23), we obtain the following:

$$u_1(y) = \left\{ \begin{aligned} & \frac{BS_b^2}{2\beta^2} [1 - R(y)] + \frac{BS_b^2}{2(4b^2 - \beta^2)} [\cosh(2by) - \cosh(2bh)R(y)] \\ & + \frac{CS_b}{(b^2 - \beta^2)} [\cosh(by) - \cosh(bh)R(y)] + \frac{DS_b}{2(b^2 + 2b\beta)} [\cosh(b + \beta)y - \cosh((b + \beta)h)R(y)] \\ & + \frac{DS_b}{2(b^2 - 2b\beta)} [\cosh(b - \beta)y - \cosh((b - \beta)h)R(y)] \\ & + \frac{AS_a S_b}{(\Delta^2 + 4a^2 b^2)} \left\{ \begin{aligned} & \Delta \cosh(by) \cos(ay) + 2a \sinh(by) \sin(ay) \\ & - [\Delta \cosh(bh) \cos(ah) + 2a \sinh(bh) \sin(ah)] R(y) \end{aligned} \right\} \end{aligned} \right\} \quad (26)$$

Substituting Equations (24) and (26) into Equation (15), we obtain the following:

$$u(y) = \left[\beta_1 \cosh(\beta y) - \frac{P}{\beta^2} + \beta_2 \cos(\sqrt{Rd_1}y) + \beta_3 \cosh(\sqrt{Rd_T}y) \right] + \alpha_s \left\{ \begin{aligned} & \frac{BS_b^2}{2\beta^2} [1 - R(y)] + \frac{BS_b^2}{2(4b^2 - \beta^2)} [\cosh(2by) - \cosh(2bh)R(y)] \\ & + \frac{CS_b}{(b^2 - \beta^2)} [\cosh(by) - \cosh(bh)R(y)] + \frac{DS_b}{2(b^2 + 2b\beta)} [\cosh(b + \beta)y - \cosh((b + \beta)h)R(y)] \\ & + \frac{DS_b}{2(b^2 - 2b\beta)} [\cosh(b - \beta)y - \cosh((b - \beta)h)R(y)] \\ & + \frac{AS_a S_b}{(\Delta^2 + 4a^2 b^2)} \left\{ \begin{aligned} & \Delta \cosh(by) \cos(ay) + 2a \sinh(by) \sin(ay) \\ & - [\Delta \cosh(bh) \cos(ah) + 2a \sinh(bh) \sin(ah)] R(y) \end{aligned} \right\} \end{aligned} \right\} \quad (27)$$

where

$$A = Gr \left(\frac{Da^{-1} + Rd_1}{\beta^2 + Rd_1} \right), B = Gr \left(\frac{Da^{-1} - Rd_T}{\beta^2 - Rd_T} \right), C = -\frac{P}{Da\beta^2}, D = \left(\frac{P}{\beta^2} - \frac{Gr}{\beta^2 + Rd_1} - \frac{Gc}{\beta^2 - Rd_T} \right),$$

$$\Delta = Rd_T - Rd_1 - M^2 - \frac{1}{Da}, a = \sqrt{Rd_1}, b = \sqrt{Rd_T}, S_a = \frac{1}{\cos(h\sqrt{Rd_1})}, S_b = \frac{1}{\cosh(h\sqrt{Rd_T})}$$

$$h = 1 - \frac{\delta}{2}(1 - \cos 2\pi x), R(y) = \frac{\cosh(\beta y)}{\cosh(\beta h)}$$

$$\beta^2 = M^2 + \frac{1}{Da}, \beta_1 = \frac{\left(\frac{P}{\beta^2} - \frac{Gr}{\beta^2 + Rd_1} - \frac{Gc}{\beta^2 - Rd_T}\right)}{\cosh(\beta h)}, \beta_2 = \frac{\left(\frac{Gr}{\beta^2 + Rd_1}\right)}{\cos(\sqrt{Rd_1}h)}, \beta_3 = \frac{\left(\frac{Gc}{\beta^2 - Rd_T}\right)}{\cosh(\sqrt{Rd_T}h)}$$

6. Results

The numerical results obtained from Equation (27) demonstrate the combined effects of magnetic interaction, porous resistance, thermal and concentration buoyancy, variable viscosity, stenosis severity, pressure forcing, heat transfer and mass transfer on the haemodynamic behaviour of the model. The velocity profiles were evaluated at the stenosis throat, $x = 0.5$, while the normalised transverse coordinate is represented by y ,

with the arterial centreline at $y = 0$ and the wall at $y = 1$. The data used for the simulation were obtained from [31–36], and the results for the flow profiles are shown below.

The temperature profiles in Figure 1 demonstrate the influence of Rd_1 on transverse heat distribution. The profiles satisfy the prescribed wall temperature and centreline symmetry conditions. Increasing the thermal parameter modifies the curvature and magnitude of the temperature field, indicating a change in the balance between thermal diffusion and internal heat-generation or absorption effects.

This behaviour is physically significant because the temperature distribution subsequently enters the momentum equation through thermal buoyancy. Hence, temperature variations can indirectly modify blood velocity. The coupling between stenosis, temperature and viscosity agrees with Roy, Saha, Hossain and Gorla [37], who reported substantial changes in heat-transfer characteristics and flow structures when temperature-dependent viscosity was incorporated into stenosed arterial blood flow. Similarly, Fahim, Yang, Li and Mi [38] found that changes in porous and transport parameters modify temperature and heat-transfer characteristics within stenosed arteries.

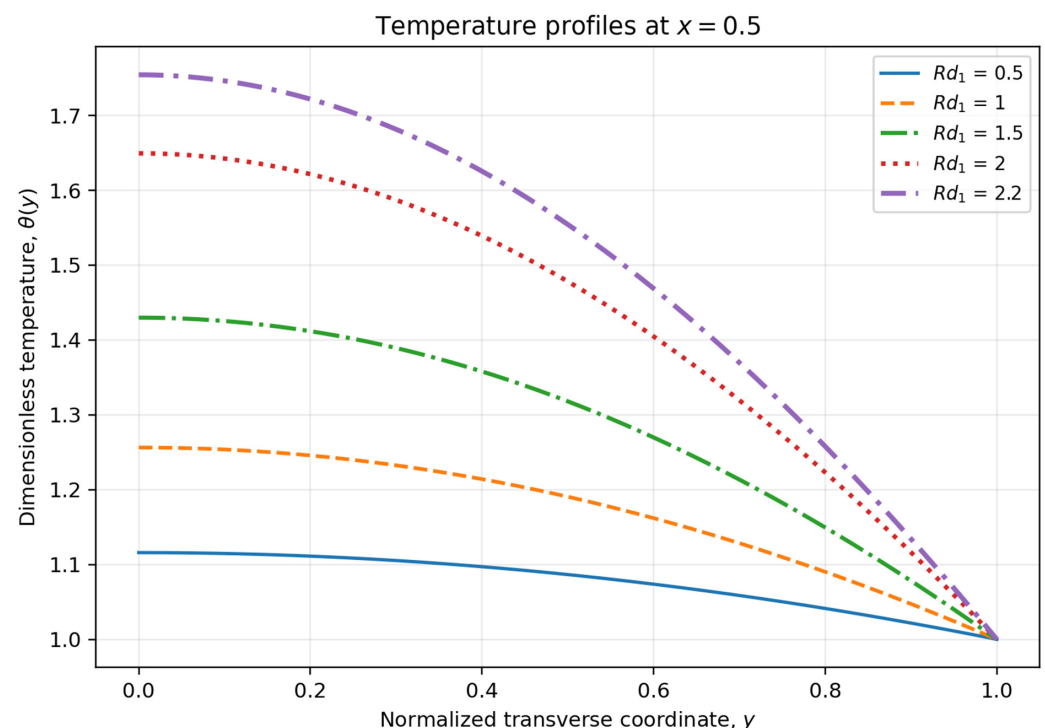


Figure 1. Dimensionless temperature profiles $\theta(y)$ for different values of the heat-transfer parameter Rd_1 at $x = 0.5$.

The concentration profiles demonstrate that increasing Rd_T modifies the transverse concentration gradient. The hyperbolic nature of the analytical concentration solution causes the concentration distribution to become progressively redistributed toward the prescribed wall value as the relative importance of reaction or removal increases, as elucidated in Figure 2. This result is particularly important because concentration is coupled to the velocity through both concentration buoyancy and variable viscosity. Consequently, the concentration field is not merely a passive scalar in the present formulation. The physical importance of concentration and haematocrit in diseased blood flow is supported by Pralhad and Schultz [39], who have demonstrated that suspension concentration substantially affects velocity, resistance and shear stress in stenosed blood flow and who specifically considered Hb SS sickle-cell blood.

The mass-transfer parameter produces a relatively moderate effect on velocity compared with the pressure gradient, magnetic parameter, permeability and variable-viscosity parameter. Within the stable range considered, increasing Rd_T generally causes a modest reduction in velocity, illustrated in Figure 3. This behaviour results from the coupling of the concentration equation with the momentum equation. Changes in mass diffusion or reaction alter the concentration distribution, which subsequently modifies concentration buoyancy and concentration-dependent viscosity.

The result is compatible with the coupled thermo-solutal formulation of Fahim, Yang, Li and Mi [38], who showed that heat and mass diffusion interact with MHD and porous effects in determining velocity, temperature and concentration distributions in stenosed arteries.

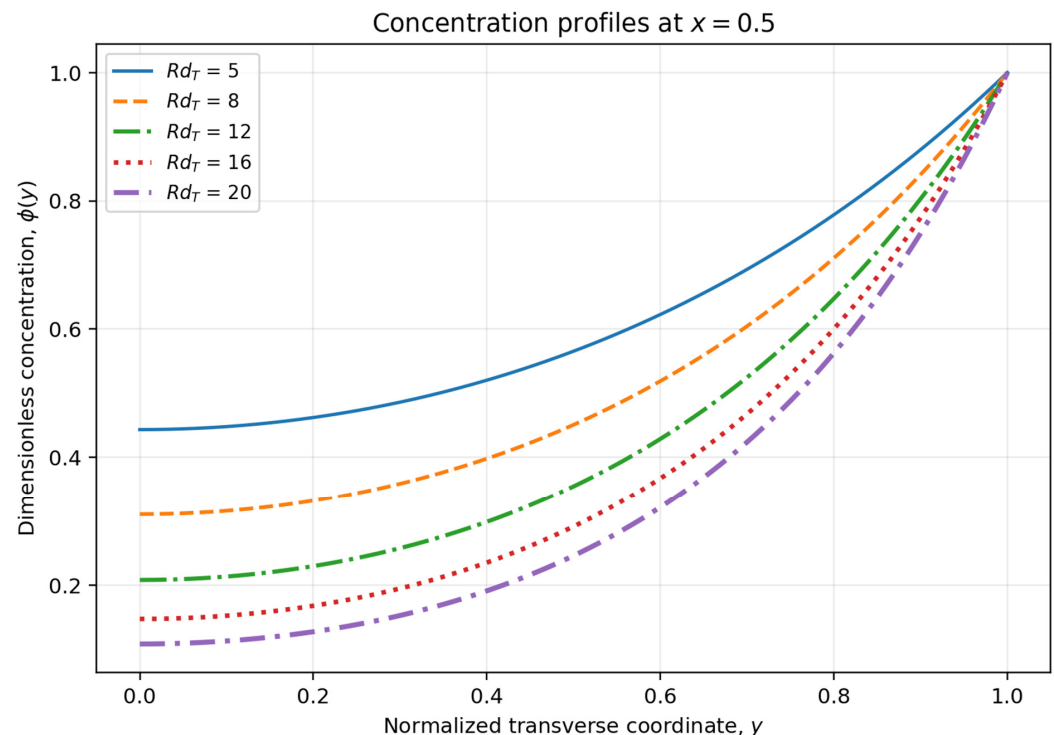


Figure 2. Dimensionless concentration profiles $\phi(y)$ for different values of the mass-transfer parameter Rd_T at $x = 0.5$.

Variation of the heat-transfer parameter produces a comparatively complex response in the velocity field, as shown in Figure 4. Unlike the nearly monotonic effects associated with the magnetic parameter or pressure gradient, the influence of Rd_1 arises through the trigonometric temperature solution and its subsequent coupling with momentum.

The physical interpretation is that modification of the temperature distribution changes thermal buoyancy and consequently modifies the velocity field. The coupling between temperature, viscosity and stenosis is consistent with the findings of Roy, Saha, Hossain and Gorla [37], who demonstrated that temperature-dependent viscosity significantly changes the flow and heat-transfer characteristics of stenosed arterial blood flow. However, particular values of Rd_1 can cause the denominator of the analytical temperature solution to approach zero. Very large velocity responses near such parameter values should therefore be regarded as mathematical sensitivity or near-singular behaviour rather than direct physiological predictions.

Figure 5 shows that velocity increases substantially as the magnitude of the favourable pressure gradient increases. This is physically expected because the pressure difference is the principal driving mechanism for arterial blood movement in the present formulation. A stronger favourable pressure gradient provides additional mechanical energy for over-

coming viscous, porous, magnetic and geometrical resistance. Consequently, the velocity increases throughout the central portion of the vessel while satisfying the zero-velocity condition at the arterial wall.

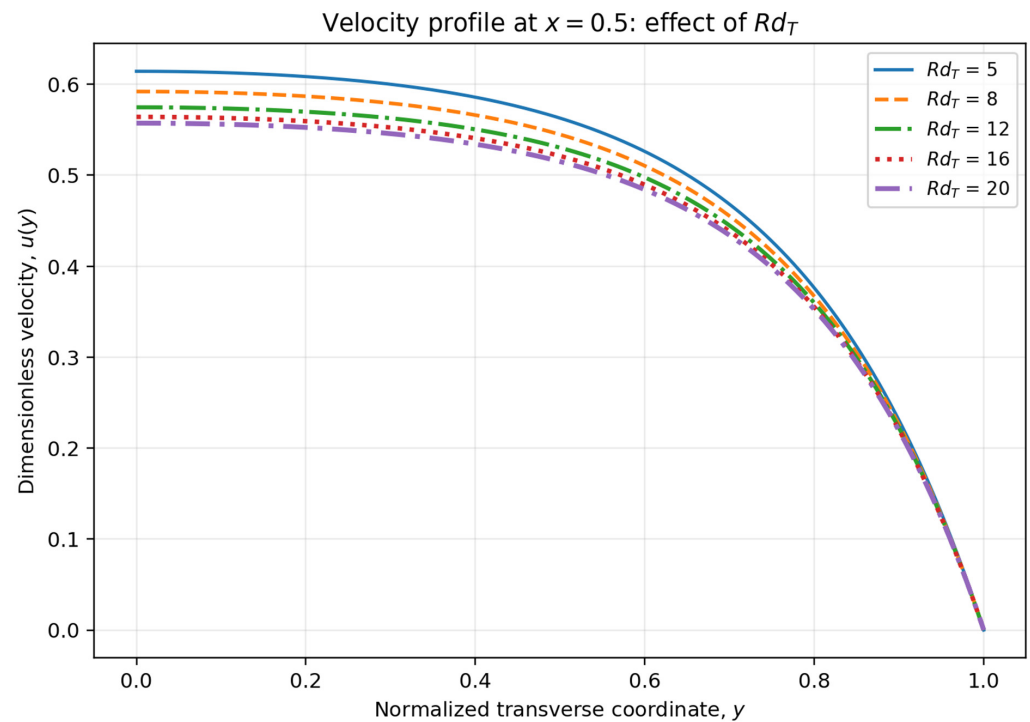


Figure 3. Effect of mass-transfer parameter Rd_T on the dimensionless velocity profile $u(y)$ at the stenosis throat $x = 0.5$.

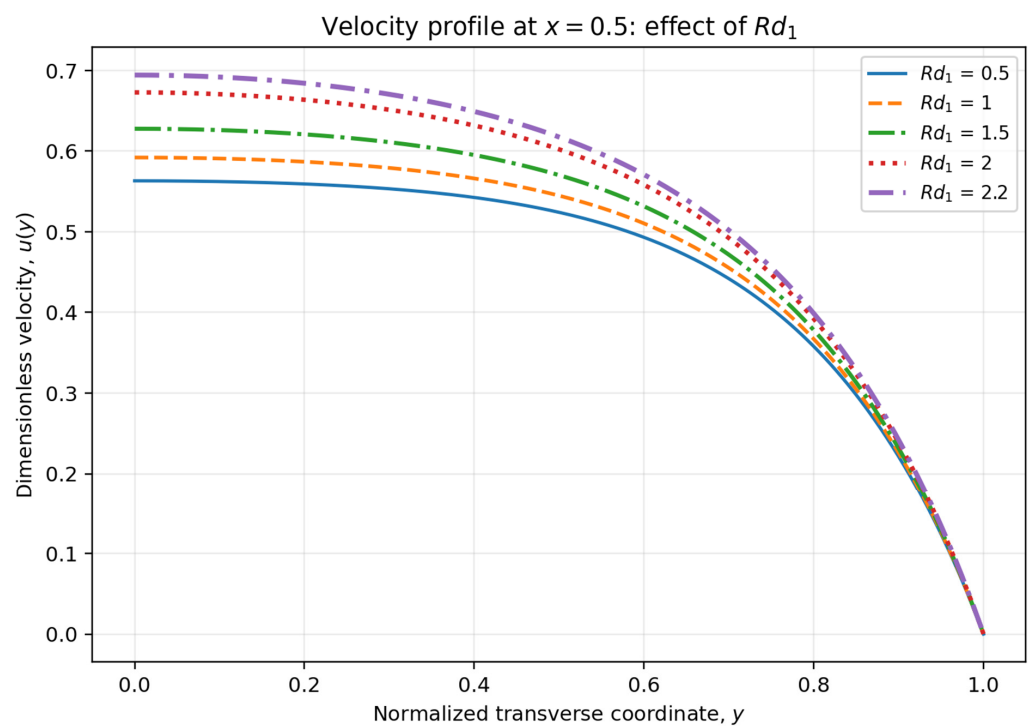


Figure 4. Effect of heat-transfer parameter Rd_1 on the dimensionless velocity profile $u(y)$ at the stenosis throat $x = 0.5$.

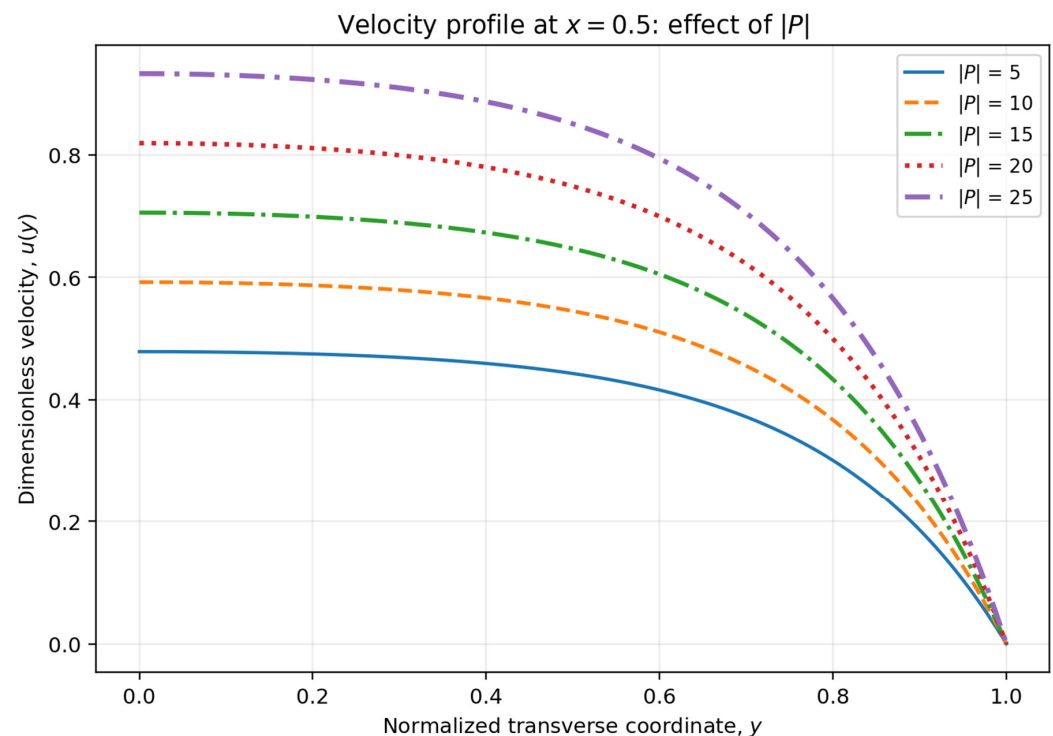


Figure 5. Effect of pressure-gradient magnitude P on the dimensionless velocity profile $u(y)$ at the stenosis throat $x = 0.5$.

This behaviour is consistent with the general stenosed-flow framework employed by Pralhad and Schultz [39], in which pressure-driven blood flow responds to changes in vascular resistance, concentration and stenosis. It is also compatible with the pressure-driven stenotic arterial model considered by Roy, Saha, Hossain and Gorla [37]. Physiologically, this indicates that a greater pressure difference is required to overcome an increase in resistance, although actual cardiovascular compensation involves pulsatility, vascular elasticity and physiological regulation, which are beyond the assumptions of the present model.

The velocity profiles in Figure 6 show that increasing the magnetic parameter reduces the axial blood velocity. The reduction is more pronounced in the central region of the artery, while the velocity approaches zero at the wall in accordance with the no-slip condition. Physically, the magnetic field interacts with the electrically conducting blood and generates a Lorentz force opposing the motion of the fluid. Consequently, increasing magnetic intensity introduces additional resistance and suppresses blood movement.

This result agrees with Farooq, Abbas, Alshammari, Mir and Kolsi [40], who investigated MHD blood flow through a constricted artery containing a porous medium and found that increasing magnetic-field strength suppresses axial velocity, particularly within the stenosed region. Similarly, Fahim, Yang, Li and Mi [38] have reported that magnetic interaction increases flow resistance in a porous multi-stenosed artery. The agreement indicates that the magnetic component of Equation (27) produces the expected electromagnetic braking behaviour.

Figure 7 shows that an increase in the thermal Grashof number enhances the axial velocity in the present computations. This behaviour arises because the thermal buoyancy contribution has been taken as favourable to the direction of blood flow. Increasing thermal buoyancy therefore provides an additional driving force and increases the momentum of the fluid. The importance of thermal effects in stenosed arterial flow has also been demonstrated by Roy, Saha, Hossain and Gorla [37], who studied blood flow through a stenotic artery with temperature-dependent viscosity and showed that temperature, viscosity and stenosis interact to modify the haemodynamic and heat-transfer characteristics.

Furthermore, Fahim, Yang, Li and Mi [38] incorporated simultaneous thermal and solutal transport into a stenosed arterial model and demonstrated significant coupling between the thermal field and blood-flow characteristics.

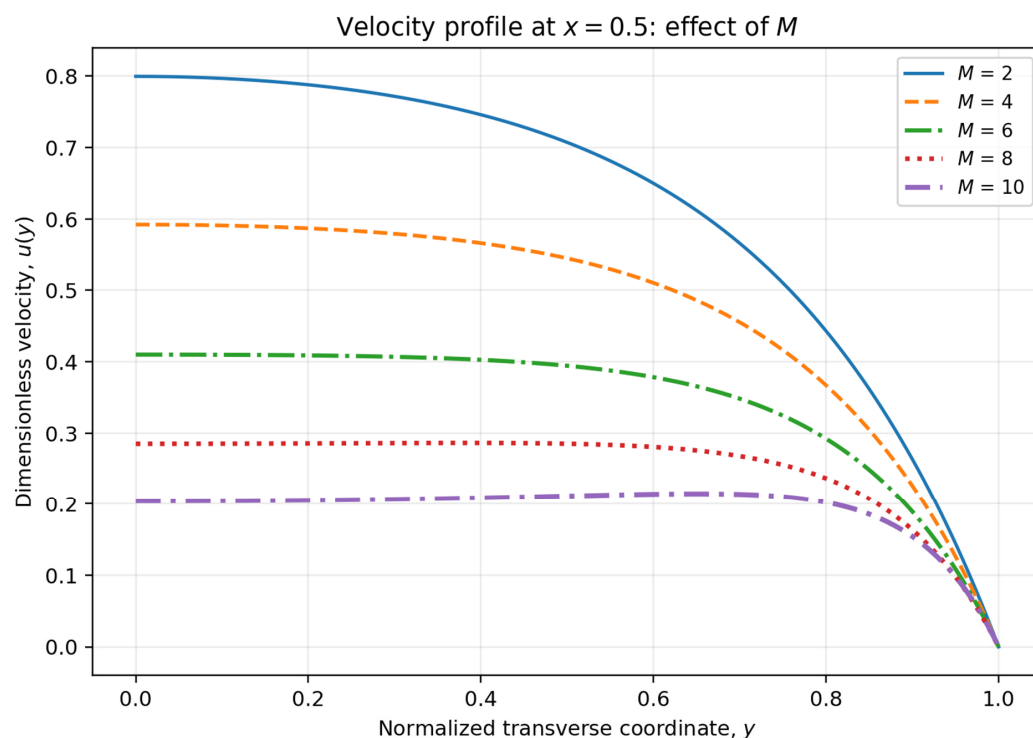


Figure 6. Effect of magnetic parameter M on the dimensionless velocity profile $u(y)$ at the stenosis throat $x = 0.5$.

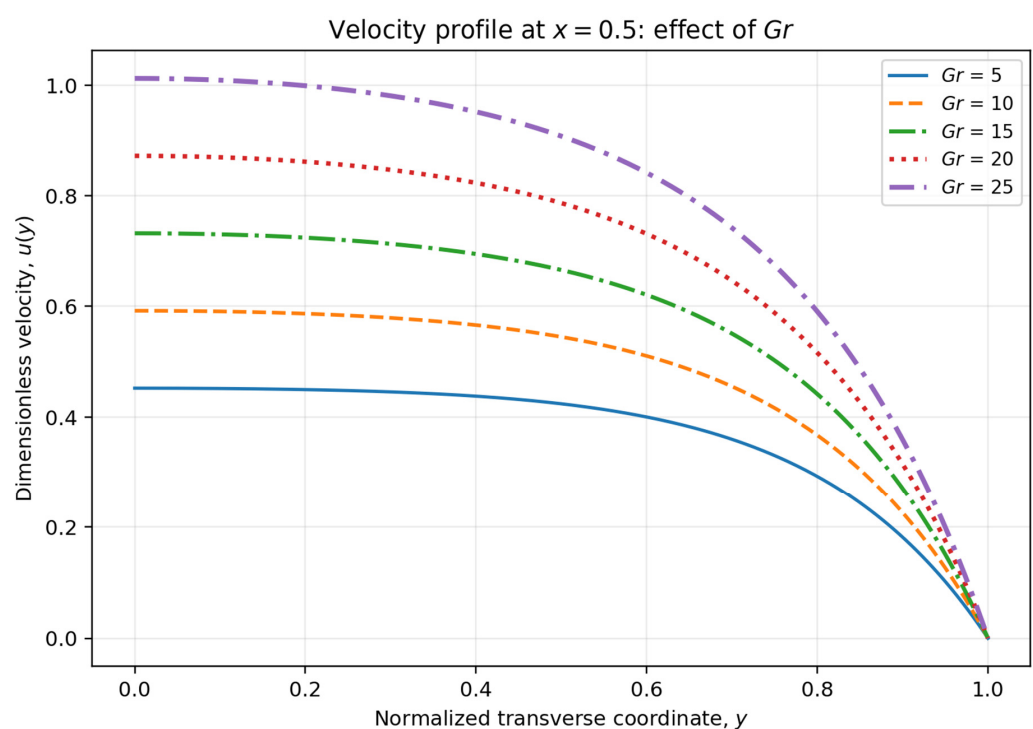


Figure 7. Effect of thermal Grashof number Gr on the dimensionless velocity profile $u(y)$ at the stenosis throat $x = 0.5$.

Increasing the concentration Grashof number increases the predicted velocity, as illustrated in Figure 8. In the present formulation, G_c represents the contribution of concentration differences to the body force. Therefore, when the concentration buoyancy force acts favourably in the direction of the pressure-driven flow, an increase in G_c produces additional momentum.

This finding should not be interpreted as meaning that an increased concentration of sickled erythrocytes necessarily improves blood flow. Concentration has two separate roles in the present model: it contributes to concentration buoyancy and also modifies viscosity. The former may enhance velocity when favourable, whereas the latter can increase resistance and reduce velocity.

This distinction is supported by Pralhad and Schultz [39], who calculated velocity, resistance and shear-stress distributions for different suspension concentrations and explicitly considered Hb SS sickle-cell blood. Their study demonstrated that blood concentration and disease-related rheological properties can significantly alter stenosed arterial flow.

Increasing stenosis severity generally reduces the velocity under the fixed pressure-gradient condition employed in the present model, as seen in Figure 9. A larger stenosis height decreases the effective lumen and increases hydraulic resistance. Consequently, the prescribed pressure force becomes less capable of maintaining the same overall flow. The result is consistent with Pralhad and Schultz [39], whose stenosed-tube model demonstrated important changes in velocity, flow resistance and wall shear stress associated with stenosis and pathological blood conditions. Roy, Saha, Hossain and Gorla [37] have also demonstrated that increasing stenosis substantially alters the haemodynamic and heat-transfer characteristics of arterial blood flow. Their results showed stronger wall skin friction and heat-transfer responses as stenosis increased. More recently, Fahim, Yang, Li and Mi [38] found that increasing stenosis height sharply reduces velocity and flow rate in their porous multi-stenosed artery model.

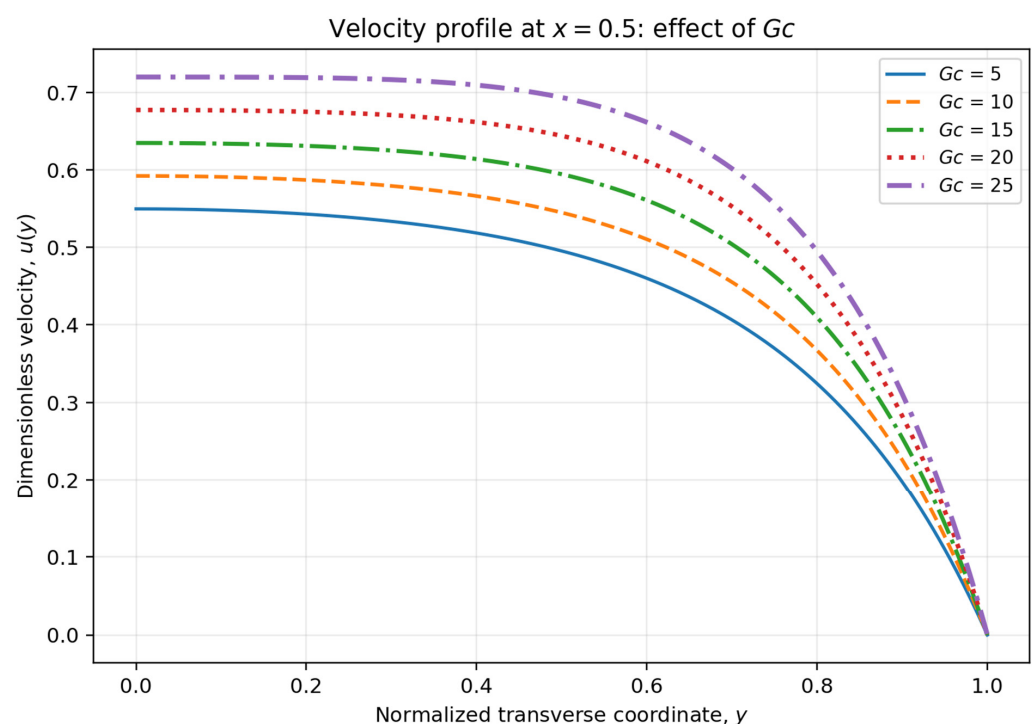


Figure 8. Effect of concentration Grashof number G_c on the dimensionless velocity profile $u(y)$ at the stenosis throat $x = 0.5$.

It is important to emphasise that the present model employs a prescribed pressure-gradient condition. Under a constant-flow-rate formulation, local velocity through a narrowing may increase because the same volume of blood must pass through a smaller cross-sectional area. Thus, the present finding concerns the effect of increasing resistance under the specific pressure-driven assumptions of Equation (27).

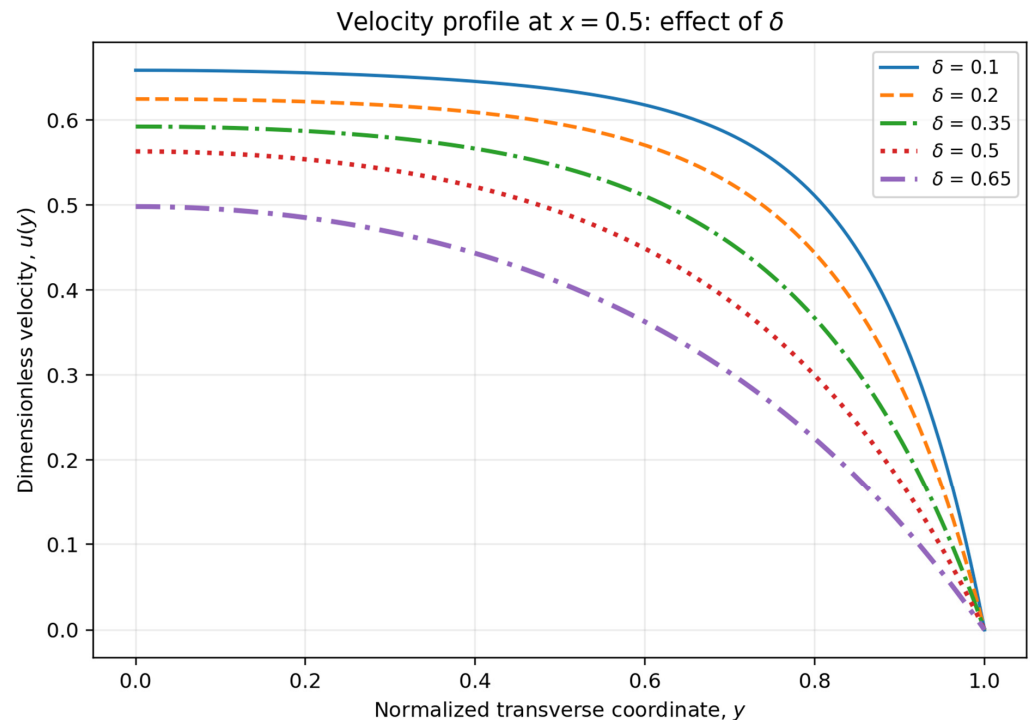


Figure 9. Effect of stenosis height δ on the dimensionless velocity profile $u(y)$ at the stenosis throat $x = 0.5$.

Figure 10 shows that increasing the Darcy number produces an increase in blood velocity. The Darcy number characterizes the permeability of the porous region; therefore, increasing Da represents a reduction in porous resistance. Blood can consequently move more easily through the stenosed region. This observation is consistent with Farooq, Abbas, Alshammari, Mir and Kolsi [40], who found that increased permeability enhances axial velocity in a constricted artery containing a porous medium. It also agrees with Fahim, Yang, Li and Mi [38], whose numerical results show that increasing the Darcy number increases velocity and improves perfusion in a porous stenosed arterial system.

Thus, the Darcy number result demonstrates the competition between permeability and resistance: low permeability restricts blood movement, whereas increased permeability facilitates flow.

Figure 11 shows that increasing the variable-viscosity parameter produces a reduction in blood velocity. This is one of the most important findings of the present study because α_s represents the strength of the concentration-dependent rheological modification incorporated to describe sickle-cell-related changes in blood viscosity. As the contribution of variable viscosity becomes stronger, greater resistance is developed against the deformation and movement of the blood. Under the prescribed pressure-gradient condition, this additional viscous resistance reduces axial velocity.

The result has strong qualitative support from Cima, Discher, Tong and Williams [41]. Their hydrodynamic model of sickle-cell blood flow predicted that moderate increases in plasma viscosity could cause pronounced reductions in blood velocity because of interactions among plasma viscosity, sickle erythrocyte rheology and oxygen transport. Similarly, Pralhad

and Schultz [39] demonstrated that velocity and resistance in a stenosed vessel are influenced by suspension concentration and disease state, including Hb SS sickle-cell blood.

The present result therefore provides an important disease-specific interpretation: increasing sickle-cell-related rheological resistance may impair blood movement, particularly when it occurs simultaneously with vascular narrowing.

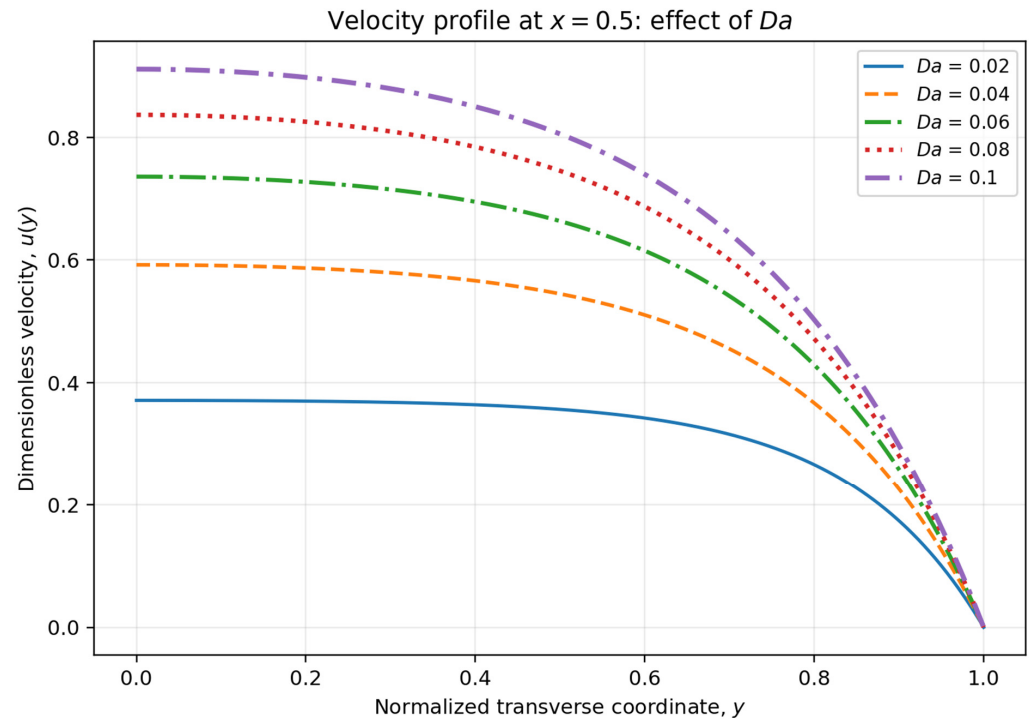


Figure 10. Effect of Darcy number Da on the dimensionless velocity profile $u(y)$ at the stenosis throat $x = 0.5$.

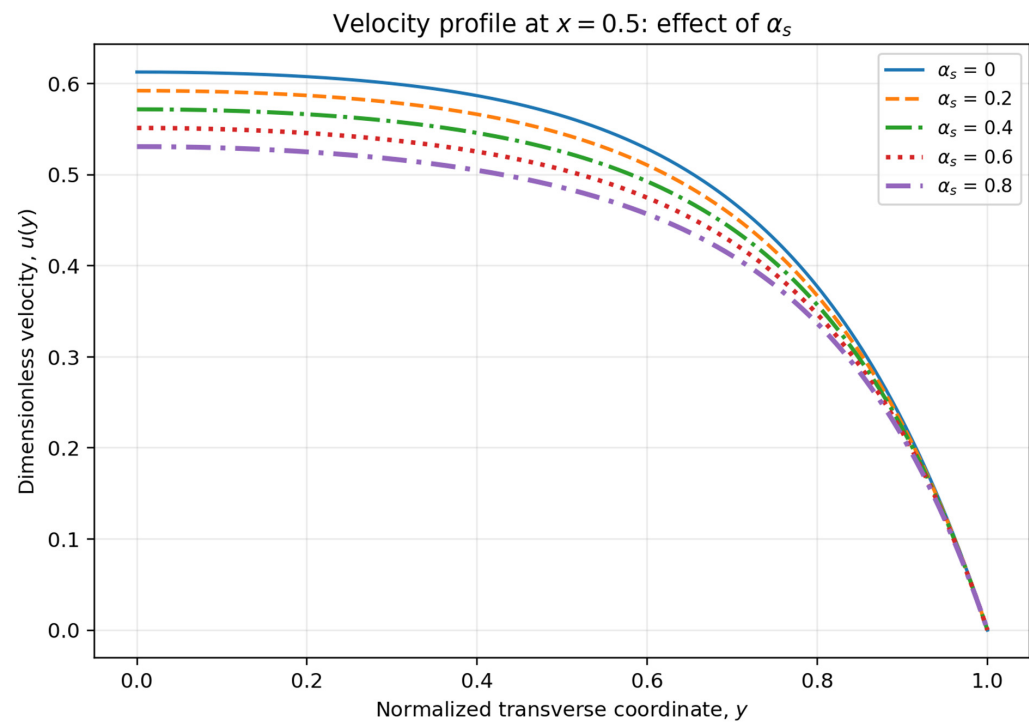


Figure 11. Effect of sickle-cell-related variable-viscosity parameter α_s on the dimensionless velocity profile $u(y)$ at the stenosis throat $x = 0.5$.

7. Discussion

The results presented in the study evaluate temperature, concentration, and velocity responses to the heat-transfer parameter, mass-transfer parameter, pressure gradient, magnetic field, thermal and concentration buoyancy, stenosis height, Darcy number, and the sickle-cell-related variable-viscosity parameter. The velocity plots are evaluated at the stenosis throat, making the results particularly relevant to the part of the artery where haemodynamic disturbance is expected to be strongest.

7.1. Temperature Distribution

Figure 1 shows a systematic increase in dimensionless temperature as the heat-transfer parameter increases from 0.5 to 2.2. The effect is most pronounced near the arterial centreline and progressively diminishes toward the wall, where the temperature curves converge to the prescribed boundary value. This indicates that the heat-transfer parameter controls the amount of thermal energy retained or generated within the flowing blood rather than altering the imposed wall temperature.

The physical implication is that stronger thermal effects enlarge the thermal field within the arterial lumen. Because blood transports thermal energy by both conduction and convection, any parameter that strengthens internal heat transport can raise the fluid temperature away from the arterial wall. The curved profiles further indicate that temperature gradients are greatest close to the arterial boundary, suggesting greater local heat transfer there. This trend is compatible with the numerical investigation of Majee and Shit [42], who studied MHD blood flow and heat transfer in a stenosed arterial segment and showed that thermal conditions can be substantially modified by haemodynamic and magnetic effects. They reported significant changes in temperature distribution and isotherms in stenotic regions and demonstrated the close interaction between velocity, magnetic forcing, and heat transport. Their study is especially relevant because it simultaneously considers stenosis, MHD effects, and blood heat transfer.

The result is also physiologically meaningful because temperature affects blood rheology and can influence sickling kinetics, although the latter process is not explicitly represented by these plots. The classic review of Stuart and Johnson [43] notes that temperature is among the environmental factors capable of changing the delicate rheological state of sickle blood.

7.2. Concentration Distribution

Figure 2 demonstrates that increasing the mass-transfer parameter from 5 to 20 markedly reduces the dimensionless concentration throughout most of the lumen, while all profiles converge at the wall. At the centreline, the reduction is particularly substantial, indicating that the mass-transfer parameter strongly influences solute penetration into the interior of the arterial flow.

The profiles suggest that larger values of the mass-transfer parameter favour more rapid redistribution or depletion of the transported species from the core region relative to the prescribed wall concentration. The increasingly steep concentration gradient near the wall at larger values also suggests enhanced wall-directed mass-transfer activity. Depending on the biological interpretation assigned to concentration in the formulation, this may describe dissolved species, biochemical constituents, oxygen-related species, therapeutic agents, or another transported component.

This aspect is important to sickle-cell haemodynamics because oxygen and other biochemical species strongly influence HbS polymerisation. Keidan et al. [44] demonstrated that polymerisation tendency in sickle-cell anaemia is strongly related to oxygen saturation and erythrocyte deformability. In a similar vein, Li et al. [45] emphasised that deoxygenation promotes intracellular HbS polymerisation and increases sickle-cell

rigidity, directly linking transport conditions to altered rheology. Thus, although the present concentration variable should not automatically be equated with oxygen unless this is explicitly defined in the model, incorporation of mass transport makes the framework more relevant to the physiological processes underlying sickling.

7.3. Effect of the Mass-Transfer Parameter on Velocity

Figure 3 shows that increasing the mass-transfer parameter progressively decreases blood velocity, particularly around the centre and intermediate portions of the artery. All curves approach zero at the wall, which is consistent with the imposed no-slip condition.

The velocity reduction can be interpreted through the coupling between concentration and momentum. Figure 2 already shows that larger mass-transfer parameter values produce lower concentrations in the fluid interior. If concentration contributes positively to the momentum equation through solutal buoyancy, reducing concentration diminishes that driving contribution and consequently lowers velocity. Thus, Figures 2 and 3 are internally consistent.

The reduction is not excessively abrupt, suggesting that mass transport acts as a secondary haemodynamic control compared with parameters such as pressure gradient, magnetic field, thermal buoyancy, or Darcy permeability. This is physically reasonable: mass transfer normally influences blood motion indirectly through concentration-dependent density, buoyancy, viscosity, or reaction terms rather than acting as the principal mechanical driving force.

Studies incorporating simultaneous heat and mass transfer into stenosed blood-flow formulations similarly demonstrate strong coupling among concentration transport and haemodynamics. The broader literature on MHD blood transport through stenosed and porous vessels supports the inclusion of such coupled mechanisms rather than treating momentum, heat, and solute transfer independently.

7.4. Effect of the Heat-Transfer Parameter on Velocity

The opposite trend occurs in Figure 4. As the heat-transfer parameter increases, dimensionless velocity rises appreciably. This agrees directly with Figure 1, where higher values of the same parameter increase the temperature field.

The connection can be explained through thermal buoyancy. Heating produces a stronger temperature difference within the fluid and therefore strengthens the thermal contribution represented by the thermal Grashof term in a coupled momentum equation. This assists the pressure-driven motion and increases velocity. The effect is strongest around the centreline, while the no-slip wall condition forces the curves to meet at zero velocity.

The result agrees qualitatively with MHD heat-transfer investigations in which changes in thermal conditions produce substantial changes in blood velocity. Majee and Shit [42] showed significant coupling between velocity and thermal behaviour in a stenosed arterial segment. More recent thermal studies of Darcy blood flow through stenosed arteries have also reported that thermal parameters can enhance the velocity field under appropriate model definitions.

The clinical interpretation should nevertheless remain cautious. The model indicates that thermal forcing can modify haemodynamics; it does not by itself imply that raising blood temperature is clinically desirable. Temperature changes in biological tissues are tightly regulated.

7.5. Effect of Pressure-Gradient Magnitude

Figure 5 produces one of the strongest responses in the study. Increasing pressure-gradient magnitude from 5 to 25 produces an almost systematic and substantial increase in velocity throughout the arterial lumen. This behaviour is fundamental to pressure-driven arterial flow: a larger axial pressure difference provides a greater force to overcome viscous, magnetic, porous,

and stenotic resistance. The near-proportional vertical separation of the profiles suggests that pressure forcing remains the dominant driving mechanism in the present formulation. Even with sickle-cell-related variable viscosity and arterial stenosis present, adequate pressure forcing can substantially increase local blood velocity.

This observation is consistent with classical stenotic-flow investigations. Chaturani and Samy [46] demonstrated that arterial flow resistance depends strongly on blood rheology and stenosis, so a greater pressure gradient is required to maintain a given flow rate when viscous resistance increases. Misra, Patra and Misra [47] similarly found that stenosis increases resistance to blood flow and wall shear effects. For sickle blood, this relationship is especially important because increased viscosity means that greater pressure forcing may be necessary to achieve comparable perfusion. However, increased pressure does not eliminate the underlying microvascular risks associated with poorly deformable sickled erythrocytes.

7.6. Effect of the Magnetic Parameter

Figure 6 shows a pronounced reduction in velocity as the magnetic parameter increases from 2 to 10. The centreline velocity falls substantially, establishing the magnetic parameter as one of the strongest velocity-suppressing variables in the results. This behaviour has a clear MHD explanation. Blood contains electrically conducting ionic constituents. When an external transverse magnetic field acts on a moving conducting fluid, an electromagnetic resisting force is generated. In conventional MHD formulations this Lorentz force acts opposite to the direction of motion and increases the effective resistance to flow. Consequently, stronger magnetic interaction suppresses axial velocity.

This finding agrees exceptionally well with published studies. Ikbal et al. [48] examined non-Newtonian blood through a stenosed artery under a transverse magnetic field and reported significant Hartmann-number effects on velocity, flux, and wall shear stress. Mustapha et al. [49] found that increasing magnetic-field strength substantially altered velocity and reduced flow-separation regions downstream of arterial stenoses. Majee and Shit [42] likewise explain that a transverse magnetic field tends to decrease blood velocity through Lorentz-force resistance and showed that sufficient magnetic strength modifies recirculation and flow reversal in a stenosed artery. Earlier, Ramamurthy and Shanker [50] investigated MHD blood flow through a porous channel, providing foundational support for combining magnetic and porous resistance in cardiovascular-flow models. The agreement of Figure 6 with several independent MHD investigations provides strong qualitative validation of this component of the present model.

7.7. Effect of Thermal Grashof Number

Figure 7 shows a strong monotonic increase in blood velocity as the thermal Grashof number increases from 5 to 25. The enhancement is particularly large in the central region, with the peak velocity more than doubling over the parameter range shown. The thermal Grashof number measures the relative significance of thermal buoyancy effects. Higher values therefore mean that temperature-induced density differences provide a stronger contribution to fluid motion relative to viscous resistance. In the present formulation, thermal buoyancy acts in the direction of the principal flow and hence accelerates the blood.

Figure 7 also reinforces the conclusions drawn from Figures 1 and 4: temperature is not merely a passive variable in the model. Thermal transport feeds back into the velocity distribution through buoyancy. This thermal-momentum coupling is characteristic of mixed-convection formulations.

Such interaction between heat transfer and haemodynamics is consistent with Majee and Shit [42] and with subsequent porous arterial-flow studies in which thermal parameters substantially modify blood velocity and heat-transfer rates.

7.8. Effect of Concentration Grashof Number

Figure 8 shows that an increase in concentration Grashof number from 5 to 25 increases the velocity distribution throughout the arterial lumen. The effect is clear but somewhat less dramatic than the thermal Grashof-number effect in Figure 7.

The concentration Grashof number represents the momentum contribution arising from concentration-induced density differences. Increasing it strengthens solutal buoyancy and therefore adds an additional driving force to the blood flow. The result confirms that mass transfer and fluid motion are coupled rather than independent processes.

Figures 3 and 8 should be considered together. Increasing the mass-transfer parameter decreases concentration and reduces velocity, whereas increasing the concentration Grashof number strengthens the influence of whatever concentration difference remains and increases velocity. These two apparently different behaviours are therefore physically compatible.

This is particularly useful for biomedical transport models because blood simultaneously transports cells, gases, metabolites, drugs, ions, and other dissolved substances. However, the specific biomedical meaning of the concentration variable should be tied to the definition used in the governing equations rather than assigned retrospectively.

7.9. Effect of Stenosis Height

Figure 9 shows one of the most physiologically important findings. Increasing stenosis height from mild values to the largest value shown progressively decreases the velocity at the stenosis throat. A larger stenosis occupies a greater proportion of the arterial lumen and therefore increases resistance to passage of blood under the fixed forcing conditions represented in the calculation.

This result is consistent with a large body of haemodynamic literature. Misra, Patra and Misra [47] showed that resistance to flow and wall shear increase with stenosis size. Sriyab [51] demonstrated that stenosis geometry and non-Newtonian characteristics substantially influence skin friction and flow resistance. Razavi, Shirani and Sadeghi [52] demonstrated significant changes in pulsatile velocity patterns when carotid stenosis severity was varied between 30 and 60 percent under different rheological models. The finding also agrees with Tandon et al. [53], who analysed blood flow through stenotic tubes and demonstrated substantial alterations in velocity, pressure, wall shear stress, and downstream flow reversal. More recent investigations similarly conclude that stenotic geometry and severity have decisive effects on haemodynamics.

An important nuance is that local velocity can increase inside a narrowing when flow rate is prescribed by continuity, whereas the present plot shows decreasing velocity with increasing stenosis height under the particular pressure-driven, resistive formulation and fixed parameter conditions used in this study. The result should therefore be interpreted within the model's boundary and forcing assumptions rather than as a universal statement that velocity must always decline at every stenosis throat.

7.10. Effect of Darcy Number

Figure 10 shows a large increase in velocity as the Darcy number increases from 0.02 to 0.10. This trend is physically well founded.

Darcy number represents permeability relative to the characteristic geometric scale. A small Darcy number represents a medium with low permeability and therefore strong resistance to fluid motion. As the Darcy number rises, permeability increases, Darcy resistance becomes weaker, and blood can move more freely. The substantial increase in velocity

observed in Figure 10 therefore indicates that porous resistance is a major determinant of flow in the present stenosed arterial model. The result agrees with Ramamurthy and Shanker [50], whose MHD porous-channel model established the importance of permeability in blood flow. It is also consistent with Misra, Sinha and Shit [54], who modelled blood flow in a porous vessel with double stenoses and an external magnetic field. Their work provides particularly close theoretical precedent for combining stenosis, magnetic effects, and porous resistance.

Numerical investigations of porous-saturated stenosed arteries have likewise reported that greater permeability increases velocity and flow rate while reducing flow resistance. Thus, Figure 10 provides another result that is strongly supported by the existing literature.

7.11. Sick-Cell-Related Variable-Viscosity Parameter

Figure 11 represents the central novelty of the study. Increasing the sickle-cell-related variable-viscosity parameter from zero to its largest plotted value progressively reduces blood velocity throughout the artery. Although the difference between successive curves is more moderate than that produced by the magnetic field, pressure gradient, or Darcy number, the direction of the effect is systematic and physiologically important.

An increase in the sickle-cell viscosity parameter effectively represents greater rheological resistance. As sickling becomes more pronounced, erythrocytes become less deformable, intracellular viscosity and membrane stiffness increase, and the effective resistance of blood to deformation and passage through restricted vessels rises. For an unchanged pressure gradient, higher effective viscosity therefore produces lower velocity. This result has very strong experimental and physiological support. Connes et al. [55] describe sickle cell disease as fundamentally a haemo-rheological disorder in which blood viscosity, red-cell deformability, aggregation, and vascular dysfunction contribute to vaso-occlusive complications. Li et al. [45] showed that HbS polymerisation under deoxygenated conditions makes sickle erythrocytes mechanically abnormal and poorly deformable, thereby impairing microvascular flow.

Dufu et al. [56] experimentally demonstrated that deoxygenated sickle blood exhibits hyperviscosity and that improving sickle-red-cell deformability reduces that viscosity. Keidan et al. [44] also established relationships among HbS polymerisation tendency, erythrocyte deformability, and clinical/rheological variables. The recent review by Connes [57] further confirms that altered red-cell deformability and blood viscosity remain central determinants of vascular dysfunction in SCD.

The present Figure 11 therefore translates well-established cellular rheological abnormalities of sickle cell disease into a continuum of haemodynamic consequence: as sickle-cell-induced viscous resistance becomes stronger, axial blood velocity falls. This is one of the most biologically compelling outcomes of the model.

7.12. Implications of Findings

This study investigated the mathematical modelling of sickle-cell-induced variable viscosity effects on magnetohydrodynamic blood flow through a stenosed artery with heat and mass transfer. The model was formulated to examine the combined influence of arterial narrowing, magnetic field, porous-medium permeability, pressure forcing, heat transfer, mass transfer, thermal buoyancy, concentration buoyancy, and sickle-cell-related variable viscosity on the haemodynamic behaviour of blood.

The results showed that the thermal and concentration fields respond significantly to their respective transport parameters. Increasing the heat-transfer parameter increased the dimensionless temperature within the arterial lumen, while increasing the mass-transfer parameter reduced the concentration distribution. These transport processes also influenced the velocity field through their coupling with the momentum equation. Similarly, the velocity

profiles revealed that increasing the pressure-gradient magnitude increased the blood velocity, indicating that pressure forcing remains an important mechanism for overcoming viscous and stenotic resistance. Similarly, increasing the thermal Grashof number and concentration Grashof number enhanced the velocity because stronger thermal and solutal buoyancy forces assisted the flow. Increasing the Darcy number also increased velocity by reducing the resistance associated with the porous medium. On the other hand, increasing the magnetic parameter reduced the velocity profile. This behaviour is attributed to the Lorentz force generated by the interaction between the magnetic field and the electrically conducting blood, which opposes the fluid motion. The results further show that increasing stenosis height reduced the velocity at the stenosis throat, demonstrating that increasing arterial constriction raises flow resistance and impairs blood transport.

A major finding of the study was the effect of the sickle-cell-related variable-viscosity parameter. Increasing this parameter consistently reduced the blood velocity, indicating that greater viscosity associated with sickle-cell conditions increases resistance to blood motion. This behaviour is particularly important because the abnormal rheological properties associated with sickled erythrocytes can further compromise circulation when combined with arterial narrowing. Comparison of the results with existing studies showed broad qualitative agreement with published findings on stenosed arterial flow, magnetohydrodynamic blood flow, porous-medium effects, heat and mass transfer, and sickle-cell haemo-rheology. The results therefore provide reasonable physical support for the mathematical formulation and demonstrate the usefulness of the model for studying the interaction between sickle-cell-related viscosity and stenotic haemodynamics.

8. Conclusions

The study concludes that blood flow through a stenosed artery under sickle-cell-related variable-viscosity conditions is governed by a complex interaction between driving and resisting mechanisms. Pressure forcing, thermal buoyancy, concentration buoyancy, and increased permeability tend to enhance blood velocity, while magnetic resistance, increasing stenosis severity, and increasing sickle-cell-related viscosity tend to suppress the flow.

The magnetic field was found to provide a significant braking effect on the velocity, while increasing Darcy number facilitated blood movement by reducing porous resistance. Increasing stenosis height produced a substantial reduction in velocity, confirming the adverse haemodynamic consequences of progressive arterial constriction. Most importantly, the variable-viscosity parameter associated with sickle-cell conditions produced a systematic reduction in velocity, demonstrating mathematically how increased rheological resistance can contribute to impaired circulation.

The study therefore establishes that the simultaneous occurrence of arterial stenosis and sickle-cell-induced viscosity changes can create a more restrictive haemodynamic environment than either mechanism acting independently. The interaction of increased viscosity with stenotic narrowing may consequently intensify resistance to blood transport, particularly in regions where the arterial lumen is already compromised. Overall, the mathematical model provides a useful theoretical framework for understanding the combined effects of sickle-cell-related viscosity, magnetic forces, porous resistance, heat transfer, mass transfer, and stenosis on arterial blood flow. The study also demonstrates that incorporating variable viscosity into sickle-cell haemodynamic models provides a more realistic representation of the abnormal resistance associated with sickled blood than models based solely on constant viscosity.

9. Limitations of the Study

The study is based on an idealised stenosed arterial geometry, whereas actual arterial stenoses may be irregular, asymmetric, eccentric, or multiple. Sickle-cell blood is represented

mainly through a variable-viscosity parameter, which simplifies the complex rheological behaviour of sickled erythrocytes.

The model does not explicitly incorporate oxygen-dependent haemoglobin S polymerisation and the associated sickling and unsickling processes. Red blood cell adhesion, aggregation, rigidity, and interactions with the vascular endothelium are not explicitly considered. The arterial wall is assumed to have simplified mechanical behaviour, thereby neglecting detailed wall elasticity and fluid–structure interaction. The complex non-Newtonian and shear-dependent rheological properties of sickle-cell blood are not fully represented. The magnetic field is represented in an idealised form without detailed spatial or temporal variations. The porous-medium formulation provides a simplified representation of resistance within the stenosed arterial environment.

Heat and mass transfer are treated using generalised transport parameters without detailed biochemical reactions and metabolic processes. The model does not consider multiple or time-dependent stenoses and progressive changes in arterial geometry. Patient-specific variations in blood properties, stenosis severity, oxygenation, and physiological conditions are not incorporated.

The numerical parameter values are theoretical and are not obtained directly from individual sickle-cell patients. The predicted velocity, temperature, and concentration profiles have not been validated against experimental or clinical haemodynamic measurements. Consequently, the results should be interpreted primarily as theoretical predictions of the interactions among the considered haemodynamic parameters.

10. Further Studies

Future studies should extend the present model by incorporating pulsatile pressure forcing, elastic arterial walls, oxygen-dependent sickling kinetics, red-cell adhesion and aggregation, non-Newtonian rheology, multiple or irregular stenoses, and patient-specific physiological parameters. Experimental and clinical validation should also be pursued to determine realistic ranges for the variable-viscosity parameter and strengthen the biomedical applicability of the model.

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Nomenclature

The nomenclature below is suitable for the mathematical model associated with Equation (27).

Symbol	Definition	SI Unit
x^*	Dimensional axial coordinate along the artery	m
y^*	Dimensional transverse coordinate	m
x	Dimensionless axial coordinate	–
y	Normalised transverse coordinate used in the plots	–
R_0	Reference or normal arterial radius	m
$h^*(x^*)$	Dimensional local arterial radius	m
$h(x)$	Dimensionless local arterial radius	–
L_0	Length of the stenosed arterial segment	m

d	Location parameter for the beginning of stenosis	m
δ^*	Dimensional maximum stenosis height	m
δ	Dimensionless stenosis height/severity	–
u^*	Dimensional axial blood velocity	m s^{-1}
u	Dimensionless axial blood velocity	–
u_0	Zeroth-order dimensionless velocity	–
u_1	First-order variable-viscosity velocity correction	–
U_0	Characteristic/reference blood velocity	m s^{-1}
P^*	Dimensional pressure	Pa
$\partial P^* / \partial x^*$	Dimensional axial pressure gradient	Pa m^{-1}
P	Dimensionless pressure-gradient parameter	–
ρ	Blood density	kg m^{-3}
μ	Dynamic blood viscosity	Pa s
μ_b	Baseline/reference dynamic blood viscosity	Pa s
ν	Kinematic viscosity of blood	$\text{m}^2 \text{s}^{-1}$
α_s	Sickle-cell-related variable-viscosity parameter	–
ϕ	Dimensionless concentration	–
C^*	Dimensional species concentration	kg m^{-3}
C_w	Species concentration at the arterial wall	kg m^{-3}
C_∞	Reference/ambient concentration	kg m^{-3}
T^*	Dimensional blood temperature	K
T_w	Wall temperature	K
T_∞	Reference/ambient blood temperature	K
θ	Dimensionless temperature	–
B_0	Applied magnetic-field strength/magnetic flux density	T
σ	Electrical conductivity of blood	S m^{-1}
M	Magnetic parameter	–
k	Permeability of the porous medium	m^2
Da	Darcy number	–
g	Acceleration due to gravity	m s^{-2}
β_T	Coefficient of thermal expansion	K^{-1}
β_C	Coefficient of concentration expansion	$\text{m}^3 \text{kg}^{-1}$
Gr	Thermal Grashof number	–
Gc	Concentration/solutal Grashof number	–
k_t	Thermal conductivity	$\text{W m}^{-1} \text{K}^{-1}$
c_p	Specific heat capacity at constant pressure	$\text{J kg}^{-1} \text{K}^{-1}$
α	Thermal diffusivity	$\text{m}^2 \text{s}^{-1}$
Q_0	Volumetric heat-generation/absorption coefficient	$\text{W m}^{-3} \text{K}^{-1}$
Rd_1	Dimensionless heat-transfer/source parameter	–
D_m	Molecular mass diffusivity	$\text{m}^2 \text{s}^{-1}$
k_0	First-order species reaction/removal rate	s^{-1}
Rd_T	Dimensionless mass-transfer/reaction parameter	–
a	Thermal auxiliary parameter, $a = \sqrt{Rd_1}$	–
b	Mass-transfer auxiliary parameter, $b = \sqrt{Rd_T}$	–
β	Combined magnetic-porous resistance parameter	–
β^2	Combined resistance parameter, $M^2 + Da^{-1}$	–
β_1	Coefficient appearing in the zeroth-order velocity solution	–
β_2	Thermal-buoyancy coefficient in the velocity solution	–
β_3	Concentration-buoyancy coefficient in the velocity solution	–
A	Auxiliary coefficient in the first-order velocity correction	–
B	Auxiliary concentration coefficient in the first-order correction	–
C	Auxiliary pressure-porous coefficient	–
D	Auxiliary coefficient	–
Δ	Combined auxiliary parameter in the perturbation solution	–

S_a	Thermal auxiliary function, $1/\cos(ah)$	–
S_b	Concentration auxiliary function, $1/\cosh(bh)$	–
$R(y)$	Velocity boundary-correction function	–
Symbol	Meaning	SI Unit
α	Thermal diffusivity	$\text{m}^2 \text{s}^{-1}$
α_s	Variable-viscosity/sickle-cell rheological parameter	–
β_T	Thermal expansion coefficient	K^{-1}
β_C	Concentration expansion coefficient	$\text{m}^3 \text{kg}^{-1}$
δ	Dimensionless stenosis height	–
θ	Dimensionless temperature	–
ϕ	Dimensionless concentration	–
μ	Dynamic viscosity	Pa s
μ_b	Baseline blood viscosity	Pa s
ν	Kinematic viscosity	$\text{m}^2 \text{s}^{-1}$
ρ	Blood density	kg m^{-3}
σ	Electrical conductivity	S m^{-1}
Δ	Perturbation auxiliary parameter	–

Appendix A

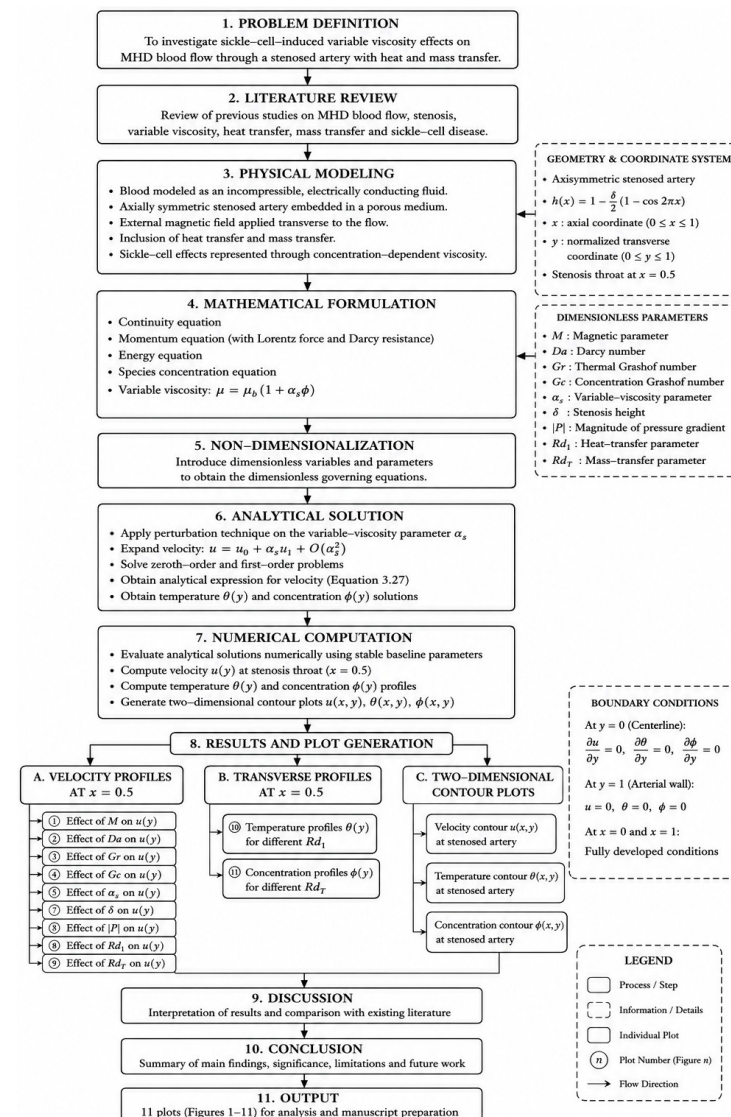


Figure A1. Problem flow chart.

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