



## **Effects of Stenosis Severity and Reynolds Number on Viscoelastic Oldroyd-B Blood Flow in a Stenosed Artery**

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### **Abstract**

Arterial stenosis significantly alters blood-flow velocity, flow separation, and wall stress distribution by reducing the vessel cross-sectional area. In this study, blood flow was modeled as a viscoelastic fluid using the Oldroyd-B model in a two-dimensional rigid artery with a cosine-shaped stenosis. The analysis extended previous work by considering four stenosis severities (25%, 35%, 45%, and 55%) and Reynolds numbers ranging from 1 to 300 at a constant Weissenberg number of 0.1. Simulations were performed in COMSOL Multiphysics 6.2 using the finite element method, and validation showed a maximum discrepancy below 2%. The results demonstrated that increasing stenosis severity increases the maximum velocity and produces a flatter core velocity profile at higher Reynolds numbers. For 25% stenosis, flow recirculation appeared at approximately  $Re = 50$  and expanded downstream as the Reynolds number increased. Increasing stenosis severity also markedly intensified flow separation. Moreover, viscoelastic extra stress was concentrated near the stenosis throat, with greater stenosis severity producing higher stress peaks. Overall, both stenosis severity and flow inertia are critical parameters for predicting flow separation and stress concentration in stenosed arteries.

**Keywords:** Oldroyd-B model; Viscoelastic blood flow; Arterial stenosis; Reynolds number; Flow separation.



## 1. Introduction

The cardiovascular system supplies tissues with oxygen and nutrients through the circulation of blood, and any substantial geometric alteration in the vascular pathway can disrupt this essential function. One of the most important geometric abnormalities is arterial stenosis, which may arise from pathological processes such as atherosclerosis, thrombus formation, embolism, and vascular inflammation. Atherosclerosis narrows the arterial lumen, alters the flow pathway, and may generate regions characterized by strong acceleration, flow separation, and abnormal wall stresses [J. Brunette et al, 2008, C. A. Holmstedt et al, 2013]. In addition, thrombus formation and growth are strongly influenced by the local flow field and its interaction with the vascular wall [H. Hosseinzadegan, D. K. Tafti, 2017]. Therefore, a quantitative investigation of blood flow in stenotic regions is important not only from a fluid-mechanical perspective but also for understanding associated biological consequences and supporting the design of therapeutic devices.

One of the major challenges in hemodynamic simulations is the selection of an appropriate rheological model for blood. The Newtonian assumption provides a simple and useful approximation in large vessels, particularly under conditions of relatively high shear rates and limited temporal variations. However, blood behavior may deviate substantially from this approximation in small vessels, unsteady flows, and regions with pronounced velocity gradients [S. Chien, 1984]. Blood is a complex suspension composed of plasma and cellular components, and the deformability and spatial organization of red blood cells imply that its mechanical response cannot be adequately characterized by a constant viscosity alone. Classical rheological studies have demonstrated the viscoelastic nature of blood as well as the dependence of its properties on time and deformation rate [M. Amir et al, 2022, G. B. Thurston, 1972]. Accordingly, several constitutive models have been developed to account for the elastic contribution of blood, among which the Oldroyd model provides a widely used framework for simultaneously representing viscous and elastic effects [W. Phillips, S. Deutsch, 1975].

Previous investigations have examined the effects of unsteadiness and blood viscoelasticity in both idealized and stenotic geometries. Pontrelli investigated pulsatile flow of an Oldroyd-B fluid through a rigid tube [G. Pontrelli, 1998], while Anand and Rajagopal proposed a viscoelastic shear-thinning model for describing blood flow [S. A. Wajihah, D. S. Sankar, 2023]. Bodnár et al. examined the combined influence of shear-thinning and viscoelastic effects under different flow rates [T. Bodnár et al, 2011]. For stenotic configurations, Ikbal et al. performed an unsteady analysis of viscoelastic blood flow through arterial stenosis [M. A. Ikbal et al, 2012], whereas Sarifuddin et al. investigated physiological flow of a shear-thinning viscoelastic fluid through an irregular arterial constriction [Sarifuddin et al, 2013]. Viscoelastic blood-flow models have also been applied to more complex vascular geometries, including abdominal aortic aneurysms and two-dimensional stenosed arteries [A. Elhanafy et al, 2019, H. Bakhti et al, 2018]. Collectively,

these studies demonstrate that both the rheological description of blood and the vascular geometry can substantially affect the predicted velocity and stress fields.

Despite previous advances, many studies have been limited to low Reynolds numbers or a fixed stenosis severity, which may prevent a complete assessment of post-stenotic flow separation and recirculation. In this study, viscoelastic blood flow is investigated over a broader range of stenosis severities (25–55%) and Reynolds numbers (1–300) using the Oldroyd-B model. The combined effects of stenosis severity and flow inertia on the velocity distribution, recirculation behavior, and viscoelastic extra stress are evaluated. Particular attention is given to changes in the velocity profile, development of downstream vortices, and stress concentration near the stenosis throat.

This broader parameter range enables a more systematic characterization of post-stenotic hemodynamics than studies restricted to a single stenosis geometry or low Reynolds numbers. By simultaneously varying stenosis severity and Reynolds number, the present work clarifies the respective roles of geometric narrowing and flow inertia in the onset and growth of flow separation, as well as in the amplification of viscoelastic stresses. The resulting analysis provides a more comprehensive description of the hemodynamic changes occurring in stenosed arteries and helps identify critical flow conditions for further investigation.

## 2. Methods

### 2.1. Geometry, Assumptions, and Parameter Range

All submitted papers will undergo peer review by the conference committee. Papers must The computational domain consists of a two-dimensional symmetric channel with rigid walls and a cosine-shaped stenosis. Owing to geometric symmetry, only the upper half of the domain was modeled. The characteristic dimensions were defined based on the inlet radius,  $R$ . The center of the stenosis was located at  $L_1=6.5R$ , while the stenosis length parameter was set to  $L_0=1.5R$ . A fully developed flow profile was assumed at the inlet, and a no-slip boundary condition was imposed on the vessel wall. In the present study, four stenosis severities,  $h$ , namely 25%, 35%, 45%, and 55%, were investigated (**Figure 1**)

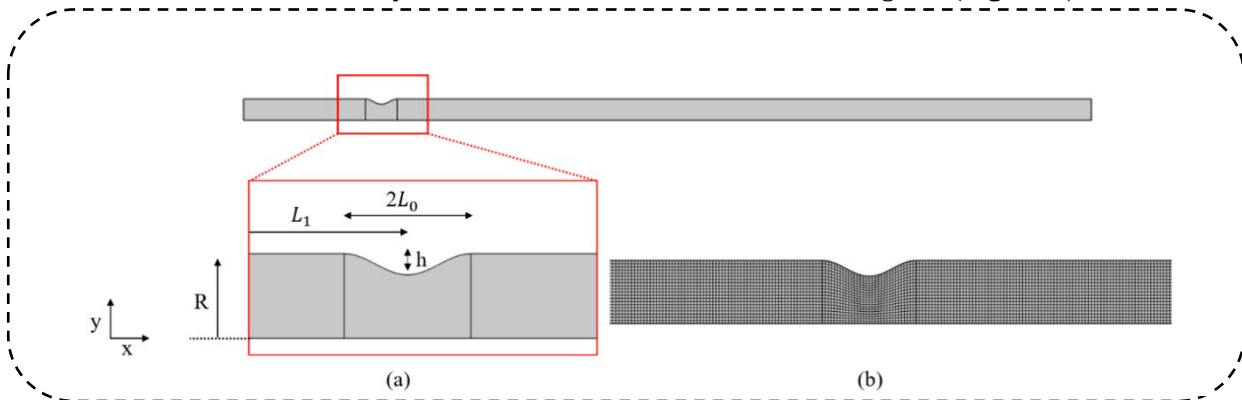


**Figure 1. (a) Two-dimensional geometry of the cosine-shaped arterial stenosis; (b) computational mesh used for the numerical simulations.**

Blood flow through a channel with a cosine-shaped stenosis, as illustrated in Fig. 1, was considered. The flow was assumed to be two-dimensional and symmetric about the channel centerline, with a fully developed velocity profile at the inlet. A two-dimensional Cartesian coordinate system was adopted, and the stenotic geometry was mathematically described as

$$f(x) = 1; x \leq (L_1 - L_0) \quad (1)$$

$$f(x) = 1 - \frac{1}{2}h \left[ 1 + \cos \frac{\pi(x - L_1)}{L_0} \right]; (L_1 - L_0) < x < (L_1 + L_0) \quad (2)$$

$$f(x) = 1; x \geq (L_1 + L_0) \quad (3)$$

$$\nabla \cdot \mathbf{u} = 0 \quad (4)$$

$$\rho \frac{\partial \mathbf{u}}{\partial t} + \rho(\mathbf{u} \cdot \nabla) \mathbf{u} = \nabla \cdot \boldsymbol{\tau} \quad (5)$$

where  $\boldsymbol{\tau}$  denotes the total stress tensor and  $\mathbf{u}$  is the velocity vector. Simultaneous solution of the continuity and momentum equations provides the velocity and pressure fields.

Blood is a complex fluid consisting primarily of red blood cells suspended in plasma, and the presence and deformation of red blood cells introduce viscoelastic characteristics into its rheological behavior. Accordingly, whole blood was modeled as a combination of red blood cells suspended in a Newtonian plasma with viscosity  $\eta_N$ . The total stress tensor is expressed as

$$\boldsymbol{\tau} = -p\boldsymbol{\delta} + 2\eta_N \dot{\boldsymbol{\gamma}} + \mathbf{S} \quad (6)$$

where the rate-of-deformation tensor is given by

$$\dot{\boldsymbol{\gamma}} = \frac{1}{2} [\nabla \mathbf{u} + (\nabla \mathbf{u})^T] \quad (7)$$

Here,  $\boldsymbol{\delta}$  is the identity tensor and  $\mathbf{S}$  represents the additional stress tensor associated with the viscoelastic contribution of red blood cells. According to the Oldroyd-B constitutive model, this tensor is described by

$$\mathbf{S} + \lambda \mathbf{S}^\nabla = 2\eta_{visco} \dot{\boldsymbol{\gamma}} \quad (8)$$

where the upper-convected derivative is defined as

$$\mathbf{S}^\nabla \equiv \frac{\partial \mathbf{S}}{\partial t} + (\mathbf{u} \cdot \nabla) \mathbf{S} - [(\nabla \mathbf{u}) \mathbf{S} + \mathbf{S} (\nabla \mathbf{u})^T] \quad (9)$$

The rheological characteristics of blood are described by three physical parameters: the Newtonian plasma viscosity,  $\eta_N$ , the viscoelastic viscosity associated with red blood cells,  $\eta_{visco}$ , and the relaxation time,  $\lambda$ . Blood was assumed to be incompressible with a constant density  $\rho$ . In this formulation,  $\eta_{visco} = 0.0736 \text{ Pa.s}$  represents the apparent viscosity contribution associated with the viscoelastic component, whereas  $\eta_N = 0.00102 \text{ Pa.s}$  represents the Newtonian plasma viscosity.

## 2.2. Dimensionless Governing Equations

The governing equations were nondimensionalized using the following parameters:

$$\begin{aligned} u &= \frac{u_d}{U}, v = \frac{v_d}{U}, x = \frac{x_d}{R}, y = \frac{y_d}{R}, t = \frac{t_d U}{R} \\ Wi &= \frac{\lambda U}{R}, Re = \frac{\rho U R}{\eta_N}, P = \frac{P_d}{\rho U^2}, S = \frac{S_d}{\rho U^2} \end{aligned} \quad (10)$$

The subscript  $d$  denotes dimensional quantities,  $U$  is the centerline velocity at the inlet, and  $R$  is the half-width of the channel. The Weissenberg number  $Wi$  is a dimensionless parameter representing the relative importance of elastic effects and is defined using the relaxation time, characteristic velocity, and characteristic length scale. For a purely non-elastic fluid, such as the Newtonian plasma component,  $Wi$  approaches zero, whereas increasing  $Wi$  indicates a stronger contribution of fluid elasticity. The total viscosity is defined as

$$\eta = \eta_N + \eta_{visco}$$

and the relative Newtonian and viscoelastic viscosities are expressed as

$$\mu_N = \frac{\eta_N}{\eta} \quad (11)$$

$$\mu_{visco} = \frac{\eta_{visco}}{\eta} = 1 - \mu_N \quad (12)$$

Accordingly, the dimensionless governing equations, including the additional stress tensor described by the Oldroyd-B model, are written as

$$\nabla \cdot \mathbf{u} = 0 \quad (13)$$

$$Re(\mathbf{u} \cdot \nabla) \mathbf{u} = \nabla \cdot (-p \delta + \mu_N [\nabla \mathbf{u} + (\nabla \mathbf{u})^T] + \mathbf{S}) \quad (14)$$

$$\mathbf{S} + Wi((\mathbf{u} \cdot \nabla) \mathbf{S} - [(\nabla \mathbf{u}) \mathbf{S} + \mathbf{S}(\nabla \mathbf{u})^T]) = \mu_{visco} [\nabla \mathbf{u} + (\nabla \mathbf{u})^T] \quad (15)$$

## 2.3. Boundary Conditions

Considering the symmetry condition along the centerline of the computational domain, a fully developed parabolic velocity profile was prescribed at the inlet. The inlet velocity and corresponding stress-tensor components were defined as

$$u = \frac{3}{2} [1 - (0.5y)^2] \quad (16)$$

$$S_{11} = 2\mu_{visco} Wi \left( \frac{\partial u}{\partial y} \right)^2 \quad (17)$$

$$S_{12} = \mu_{visco} \left( \frac{\partial u}{\partial y} \right) \quad (18)$$

$$S_{22} = 0 \quad (19)$$

In the above equations,  $y$  varies within the range  $0 \leq y \leq 2$ . A no-slip boundary condition was imposed on the vessel wall, while constant pressure and a stress-free condition were prescribed at the outlet.

## 2.4. Numerical Solution

The governing equations were solved simultaneously using the finite element method implemented in the commercial software COMSOL Multiphysics 5.4. Figure 1(b) illustrates the structured computational mesh generated for the stenosed arterial geometry. Following a mesh-independence study, a computational grid containing approximately 32,000 elements was selected for the simulations. The nonlinear governing equations were solved using the Newton iterative method, while the PARDISO solver was employed to improve the numerical stability and efficiency of the solution procedure.

## 3. Results and discussion

Prior to extending the parameter range, the numerical method was validated by reproducing the velocity profile at the maximum stenosis section for  $Wi = 0.1$  and two Reynolds numbers,  $Re = 1$  and  $Re = 20$ . As shown in Fig. 2, the results obtained in the present study exhibit good agreement with the reference data. The maximum deviation observed for  $Re = 20$  was less than 2%. Therefore, the mesh configuration, boundary conditions, and numerical solution procedure were considered sufficiently accurate and reliable for the subsequent parametric analyses (**Figure 2**).

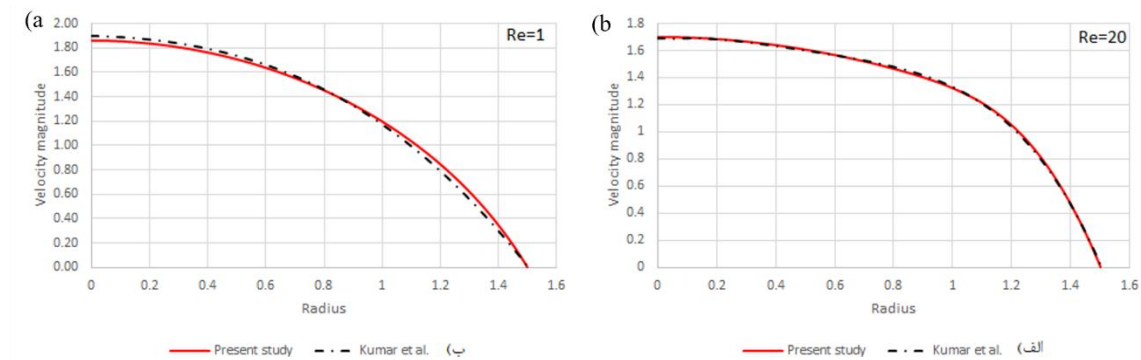


**Figure 2. Validation of the velocity profile at the maximum stenosis section for  $Wi = 0.1$ : (a)  $Re = 20$  and (b)  $Re = 1$ .**

Error! Reference source not found. presents the velocity profiles at the stenosis throat for four stenosis severities and several Reynolds numbers. For each stenosis severity, increasing the Reynolds number modifies the velocity distribution in the central region and causes the profile to become progressively flatter, indicating a stronger influence of inertial effects within the flow core. At the same time, increasing

the stenosis severity reduces the effective radius of the cross-section and consequently increases the dimensionless velocity at the throat. For example, at stenosis severities of  $h = 45\%$  and  $h = 55\%$ , the centerline velocity is noticeably higher than that obtained for  $h = 25\%$ . This change affects not only the velocity magnitude but also intensifies the spatial velocity gradients near the vessel wall and at the downstream end of the stenotic region.

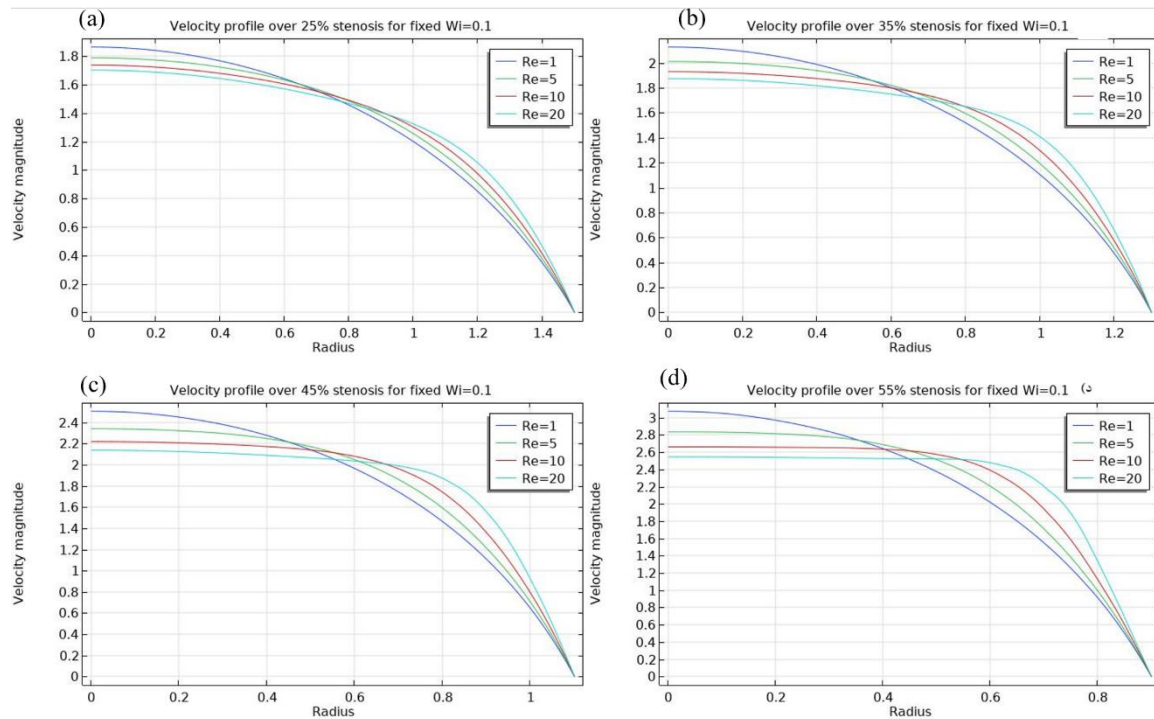


**Figure 3. Velocity profiles at the stenosis throat for  $Wi = 0.1$  and different Reynolds numbers at stenosis severities of (a)  $h = 25\%$ , (b)  $h = 35\%$ , (c)  $h = 45\%$ , and (d)  $h = 55\%$ .**

One of the important differences between the present study and analyses restricted to low Reynolds numbers is the possibility of observing flow separation downstream of the stenosis. Error! Reference source not found. presents the velocity contours and streamlines for a stenosis severity of  $h = 25\%$  over the Reynolds number range from  $Re = 20$  to  $Re = 200$ . At  $Re = 20$ , the flow passes through the stenosis throat and continues downstream without the formation of a distinct recirculation region. As the Reynolds number increases to approximately  $Re = 50$ , a vortical region appears downstream of the stenosis. At higher Reynolds numbers, this recirculation region becomes progressively elongated in the streamwise direction. Therefore, for a stenosis severity of  $h = 25\%$  and  $Wi = 0.1$ ,  $Re \approx 50$  can be regarded as the approximate onset of observable flow separation within the investigated conditions.

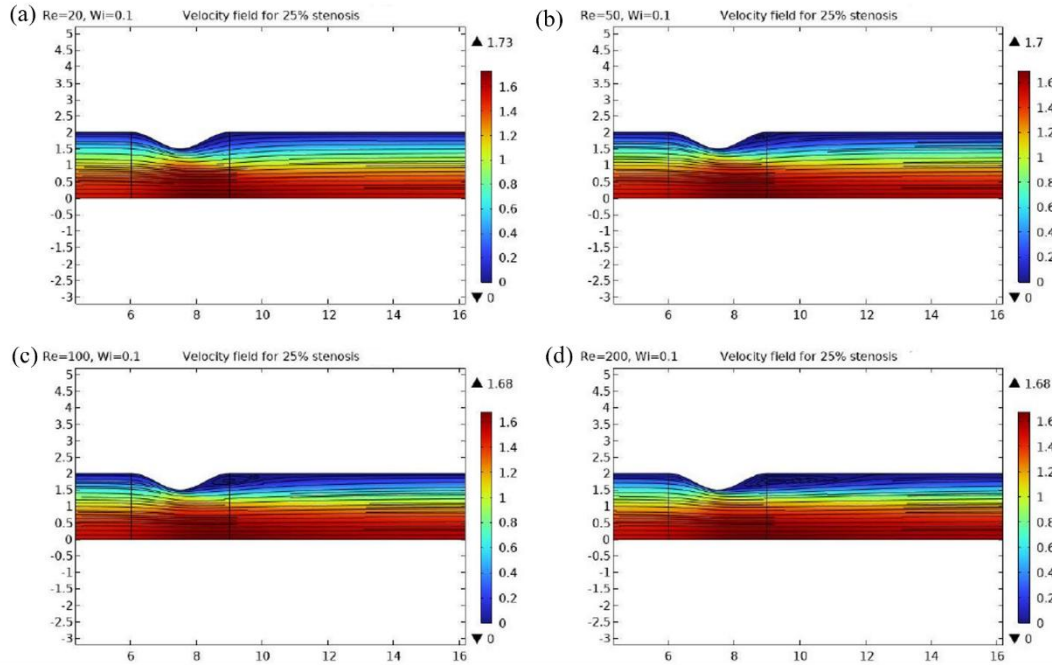


**Figure 4. Velocity contours and streamlines for  $Wi = 0.1$  and  $h = 25\%$  at different Reynolds numbers: (a)  $Re = 20$ , (b)  $Re = 50$ , (c)  $Re = 100$ , and (d).**

The growth of the recirculation region with increasing Reynolds number is directly associated with the increasing contribution of inertial effects relative to viscous diffusion. After accelerating through the stenosis throat, the fluid enters the diverging section and encounters an adverse pressure gradient. As the flow inertia increases, the ability of the fluid to adapt to the expansion of the flow passage decreases, thereby increasing the tendency toward flow separation. These results indicate that restricting the Reynolds number range to  $1 \leq Re \leq 20$  may prevent the observation of one of the most important hydrodynamic phenomena occurring downstream of arterial stenosis.

To distinguish the effect of geometry from that of flow inertia, the Reynolds number was fixed at  $Re = 50$  in Error! Reference source not found., while the stenosis severity was varied from  $h = 25\%$  to  $h = 55\%$ . As the stenosis severity increases, the jet passing through the throat becomes narrower and reaches higher velocities, while the downstream recirculation region expands. In addition to the enlargement of the vortical region, the center of the recirculation structure moves away from the vessel wall and affects a larger portion of the downstream lumen. Comparison with the trend observed in Error! Reference source not found. indicates that, within the investigated range, variations in stenosis severity can exert a stronger influence on the development of flow separation than an increase in Reynolds number alone, although both parameters contribute to the formation and evolution of the vortical structure.

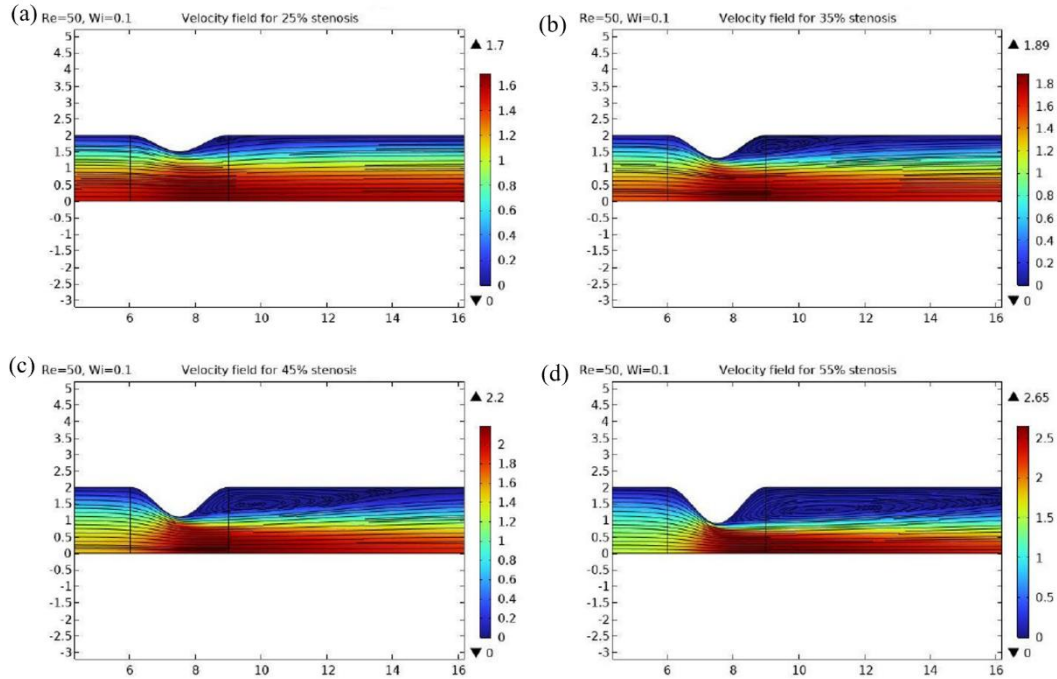


Figure 5. Velocity contours and streamlines at  $Wi = 0.1$  and  $Re = 50$  for different stenosis severities: (a)  $h = 25\%$ , (b)  $h = 35\%$ , (c)  $h = 45\%$ , and (d)  $h = 55\%$ .

**Error! Reference source not found.** shows the variation of viscoelastic extra stress along the upper vessel wall for different stenosis severities. In all cases, the stress decreases slightly upstream of the stenosis throat and then rises sharply near the maximum constriction. Both increasing Reynolds number and stenosis severity enhance the stress peak, indicating a stronger elastic response of the Oldroyd-B model near the throat. Hemodynamically, stenosis creates two distinct mechanical regions: a high-stress zone at the throat, characterized by strong acceleration and velocity gradients, and a downstream recirculation zone associated with separated flow. These effects should be considered together when evaluating vascular wall behavior and therapeutic devices such as stents.

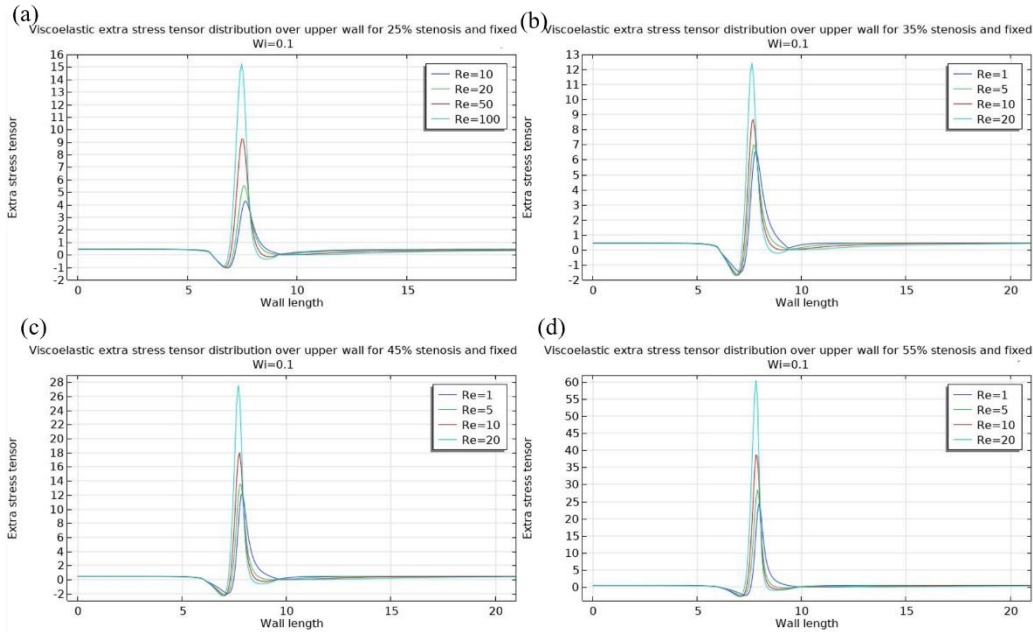


**Figure 6. Viscoelastic extra-stress distribution along the vessel wall for  $Wi = 0.1$  and different Reynolds numbers at stenosis severities of (a)  $h = 25\%$ , (b)  $h = 35\%$ , (c)  $h = 45\%$ , and (d)  $h = 55\%$ .**

Overall, the results demonstrate that extending the computational parameter space in terms of both Reynolds number and stenosis severity reveals flow characteristics that cannot be captured when considering only a fixed geometry and low Reynolds numbers. First, for a stenosis severity of  $h = 25\%$ , the onset of flow separation was identified at approximately  $Re = 50$ . Second, at a fixed Reynolds number, increasing the stenosis severity enhances both the velocity of the jet passing through the throat and the extent of the downstream recirculation region. Qualitative comparison further indicates that the influence of stenosis severity on the development of flow separation is more pronounced than that of increasing the Reynolds number alone. Third, analysis of the viscoelastic extra stress demonstrates that the viscoelastic response in the vicinity of the stenosis throat becomes stronger as the stenosis severity increases. Taken together, these three observations provide a clear framework for interpreting the respective roles of vascular geometry and flow inertia in Oldroyd-B blood flow and may serve as a basis for identifying critical flow conditions in future investigations.

#### 4. Conclusion

In this study, viscoelastic blood flow described by the Oldroyd-B model was numerically simulated in a two-dimensional vessel with a cosine-shaped stenosis using the finite element method. The primary objective was to extend the analysis from a fixed stenosis severity and low Reynolds numbers to a broader parametric investigation covering stenosis severities from  $h = 25\%$  to  $h = 55\%$  and Reynolds numbers ranging from  $Re = 1$  to  $Re = 300$ . Validation of the numerical model at  $Re = 1$  and  $Re = 20$  showed a deviation of less than 2% from the reference results.

The results indicate that increasing stenosis severity leads to higher velocities at the stenosis throat and produces a flatter central velocity profile under higher-inertia conditions. For  $h = 25\%$ , a recirculation



region appears at approximately  $Re = 50$  and extends further downstream as the Reynolds number increases. At  $Re = 50$ , increasing the stenosis severity from  $h = 25\%$  to  $h = 55\%$  significantly enlarges the vortical region, demonstrating the important role of stenosis geometry in flow separation. Furthermore, the peak viscoelastic extra stress near the stenosis throat increases with both Reynolds number and stenosis severity. Therefore, a more realistic analysis of blood flow in stenosed arteries requires the simultaneous consideration of stenosis severity, flow inertia, and viscoelastic effects.

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