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## Research Article

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# Effects of Plaque Morphology and Vascular Curvature on Rupture Risk in Coronary Atherosclerosis: A Parametric Computational Fluid Dynamics Study

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## Abstract

Plaque rupture is a leading mechanism underlying acute myocardial infarction and has been associated with elevated wall shear stress (WSS) and wall shear stress gradient (WSSG). This study uses a parametric computational fluid dynamics (CFD) approach to investigate the effects of plaque morphology and vascular curvature on coronary hemodynamics. Idealized coronary artery geometries containing atherosclerotic plaques were modeled using OpenFOAM. The parameters included percent area stenosis, plaque longitudinal skewness, eccentricity, and vascular curvature. The incompressible Navier–Stokes equations were solved using a non-Newtonian blood viscosity model and pulsatile flow boundary conditions. WSS and WSSG were then evaluated from the resulting flow fields. For concentric plaques, increasing vascular curvature produced approximately linear increases in both WSS and WSSG, with symmetric responses at positive and negative curvature angles. In contrast, eccentric plaques exhibited asymmetric and nonmonotonic responses to vascular curvature. At an eccentricity of 0.2, the lowest peak WSS and WSSG occurred at a curvature angle of  $-15^\circ$ . Proximally skewed eccentric plaques combined with large positive curvature angles produced the highest WSS and WSSG. These findings demonstrate that plaque morphology and vascular geometry interact to strongly influence local coronary hemodynamics, highlighting the importance of accounting for both factors when assessing hemodynamic conditions associated with plaque vulnerability.

*Keywords:* CFD, atherosclerosis, hemodynamics, vascular curvature, plaque morphology

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## 1. Introduction

Cardiovascular disease is a leading cause of death worldwide (Młynarska et al., 2022), with plaque rupture underlying approximately 60%–75% of acute myocardial infarctions (Kaminsky et al., 2024). The progression and rupture of atherosclerotic plaques are governed by complex interactions among anatomical, biological, and hemodynamic factors (Veneziano et al., 2026; Wang et al., 2025). However, hemodynamic quantities within the coronary vasculature are not typically available to clinicians with sufficient spatial and temporal resolution for direct assessment.

Over the past two decades, increasing efforts have focused on establishing and quantifying the relationships between hemodynamic forces and the progression, remodelling, and rupture of atherosclerotic plaques (Stone et al., 2007; Dhawan et al., 2010; Bajraktari et al., 2021; Ebrahimi et al., 2022). Kinnoshita and coworkers (Kinnoshita et al., 2024) reported notable differences in plaque morphology between acute coronary syndromes (ACS) and chronic coronary syndromes (CCS) lesions using computed tomography angiography (CTA). Russo et al. (2023) conducted a comprehensive investigation of the hemodynamic and biological characteristics of unstable (ACS) and stable (CCS) plaques and suggested that differences in wall shear stress (WSS) between the two groups may be associated with differences in their underlying molecular profiles.

The relationship between disturbed flow and plaque development has also been investigated experimentally and clinically. In-vitro fluid-mechanical studies of the spatial distribution of atherosclerotic lesions in human coronary arteries have shown that atherosclerosis was predominantly observed in regions exposed to disturbed flow, including branch points and curved segments (Asakura et al., 1990). Studies combining in vivo medical imaging techniques—such as angiography and intravascular ultrasound (IVUS)—with numerical models have demonstrated that plaques progress substantially more in regions exposed to low endothelial shear stress (ESS) (Stone et al., 2007; Kok et al., 2015; Wahle, 2006; Chaichana et al., 2011).

Conversely, several studies have reported associations between elevated WSS and fibrous-cap instability. Elevated wall shear stress gradients (WSSG) have also been associated with endothelial cell misalignment and dysfunction, potentially promoting endothelial activation and local inflammation.

Ebrahimi et al. (2022) used finite element analysis (FEA) to investigate the effects of different patterns of coronary artery tortuosity (CAT) on plaque stress concentration and blood-flow patterns within atherosclerotic arteries. They observed that maximum WSS occurred in the stenotic region and suggested that elevated WSS may increase plaque susceptibility to rupture. More recently, Zhao et al. (2024) performed a computational modelling study using in-vivo tomographic data from patients with ruptured and unruptured coronary plaques. They reported that the average WSS in ruptured lesions was 77% higher than that in unruptured lesions.

Direct measurement of coronary blood flow using an intracoronary Doppler guidewire provides an important means of characterizing hemodynamic conditions from which WSS and WSSG can be estimated. However, such measurements are invasive and carry inherent procedural risks. Moreover, they provide limited spatial resolution and are strongly dependent on patient-specific anatomy and physiological conditions. Consequently, CFD simulations have increasingly been employed to investigate coronary hemodynamics and its relationship to cardiovascular disease. Malota et al. (2018) conducted a numerical investigation of the effects of flow rate, heart rate, vessel geometry, and degree of stenosis on coronary hemodynamic indices. Their results demonstrated that vessel geometry strongly influences both the magnitude and spatial distribution of WSS and other hemodynamic parameters. In particular, bends and bifurcations generated flow characteristics substantially different from those observed in straight vessels. Choi et al. (2015) performed a CFD analysis of 114 coronary lesions from 81 patients using coronary CTA data and reported that axial plaque stress (APS), and consequently the associated rupture risk, was higher in lesions dominated by upstream plaque morphology. Thondapu et al. (2021) investigated 37 patients with acute coronary syndromes and found that WSS did not differ significantly between plaque erosion and plaque rupture, whereas WSSG demonstrated significant differences between the two lesion types. High WSSG was therefore independently associated with plaque rupture risk.

Hossain et al. (2024) conducted a parametric CFD study of three-dimensional plaque morphology and its effects on local hemodynamics and plaque stability using a finite-element model. Their investigation considered plaque eccentricity and longitudinal skewness and demonstrated that proximally skewed eccentric plaques exhibited the greatest vulnerability. However, because the vascular geometries in that study were patient-specific, isolating the effects of individual geometric parameters and deriving generalized relationships

remains challenging. Furthermore, two potentially important parameters—percent area stenosis (PAS) and vascular curvature—were not considered.

The primary objective of the present study is to extend previous parametric investigations by systematically incorporating vascular curvature and percent area stenosis together with plaque skewness and eccentricity into a CFD analysis of coronary atherosclerosis. The effects of these geometric parameters on local hemodynamics, particularly WSS and WSSG, are investigated, with particular emphasis on their individual and combined effects. A generic, non-bifurcated vascular geometry is employed to isolate the effects of individual geometric parameters and facilitate the development of more generalizable conclusions.

In the longer term, this work aims to contribute to the development of a comprehensive CFD database spanning a broad range of plaque morphologies and vascular geometries. Such a database could support the development of machine-learning models for rapid and cost-effective estimation of plaque-related hemodynamic risk in patient-specific applications (Malek et al., 2025), potentially providing an additional tool for clinical assessment and treatment planning.

## 2. Methods

### 2.1. Geometry Generation

Figure 1 shows the three-dimensional computational model of a representative coronary artery connected to a segment of the ascending aorta (Updegrave et al., 2016). Proximal coronary segments, such as the left main coronary artery and proximal right coronary artery, typically have diameters of approximately 3.0–4.0 mm. The mid-to-distal branches, including the left anterior descending (LAD) and left circumflex (LCx) arteries, generally taper to diameters of approximately 1.5–2.5 mm. The characteristic lengths of the major coronary arteries and their branches range from approximately 15 to 100 mm. In the present study, a vessel diameter of 3 mm and a total vessel length of 35 mm were selected to represent a typical coronary artery segment containing an atherosclerotic plaque.

The morphology of the coronary plaque and the vascular geometry in which it develops are characterized by the vessel centerline profile, percent area stenosis, plaque eccentricity, plaque longitudinal skewness, and vascular curvature. Figure 2a illustrates the minimum lumen area (MLA) cross section relative to the reference lumen area (RLA). Plaque eccentricity is

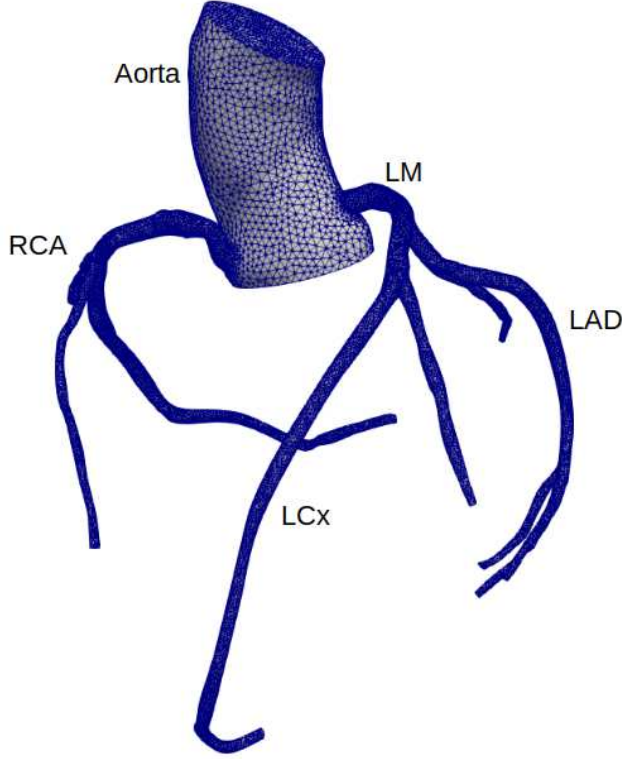


Figure 1: Three-dimensional model of the coronary arteries connected to a segment of the ascending aorta.

defined as the distance between the center of the reference lumen and the center of the minimum lumen, normalized by the reference vessel diameter (RVD) 1. An eccentricity of zero corresponds to a concentric plaque configuration, whereas increasing eccentricity indicates increasing displacement of the minimum lumen relative to the reference lumen.

$$Eccentricity = \frac{\Delta y}{R} \quad (1)$$

Figure 2b shows the variation of lumen area along the axial direction of the artery, while Figure 2c illustrates the corresponding vessel centerline profile. They are both generated by fitting the control points with c-splines. The longitudinal skewness of the plaque is characterized by the dimensionless skewness factor, defined as

$$x = \frac{l_1}{l} - 0.5, \quad (2)$$

where  $l_1$  is the axial distance from the proximal edge of the plaque to the location of the MLA, and  $l$  is the total axial length of the plaque. A value of  $x = 0$  represents a symmetric plaque profile along the axial direction, whereas negative and positive values indicate proximal or distal skewness, respectively. The vascular curvature angle,  $\theta$ , is defined as the acute angle between the vessel centerlines upstream and downstream of the plaque. A positive value of  $\theta$  denotes counterclockwise curvature of the artery viewed from the angle shown in Fig. 2c, whereas a negative value denotes clockwise curvature.

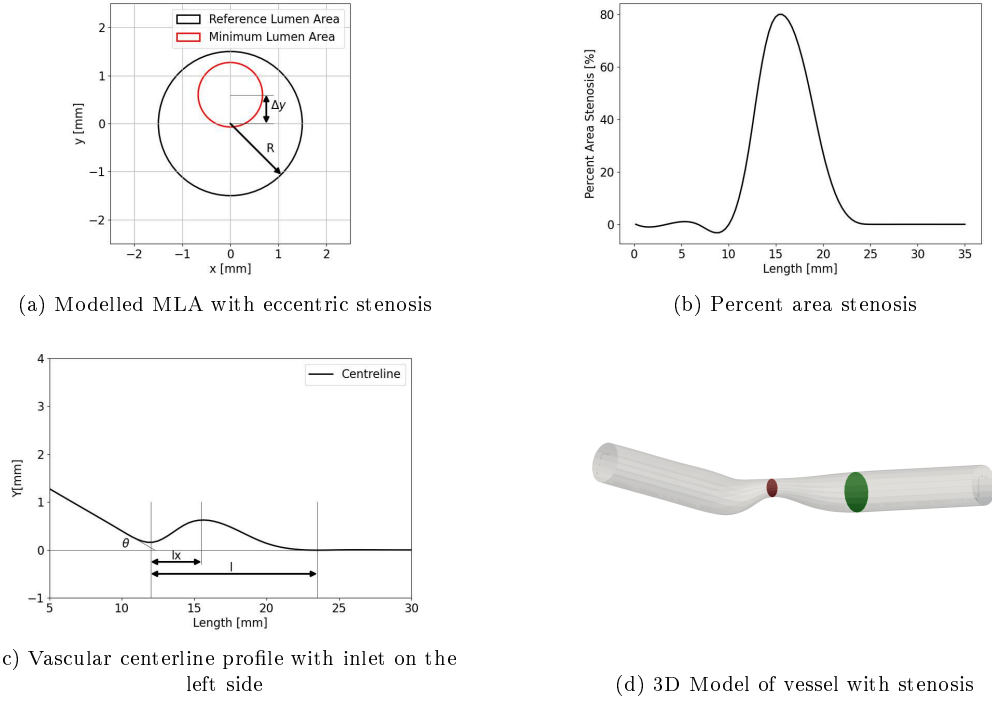


Figure 2: Definition of plaque profile and vascular geometry

A list of CFD cases with the values of their key parameters defining the plaque morphology and vascular curvature is given in Table 1. Case 4, a longitudinally symmetric, concentric plaque with zero curvature and 80 PAS is used as a baseline case.

## 2.2. Governing Equation and Boundary Conditions

The hemodynamic flow is governed by the incompressible Navier-Stoke's equations. The continuity and momentum equations from the Navier-Stoke's equations can be written as:

$$\nabla \cdot \mathbf{u} = 0 \quad (3)$$

$$\frac{\partial \mathbf{u}}{\partial t} + (\mathbf{u} \cdot \nabla) \mathbf{u} = -\frac{1}{\rho} \nabla p + \nu \nabla^2 \mathbf{u} + \mathbf{f} \quad (4)$$

The pulsatile inflow waveform reported for the left anterior descending (LAD) coronary artery by Seligman et al. (2022) was adopted as the velocity boundary condition at the inlet. Figure 3 shows the inlet velocity profile for a single cardiac cycle.

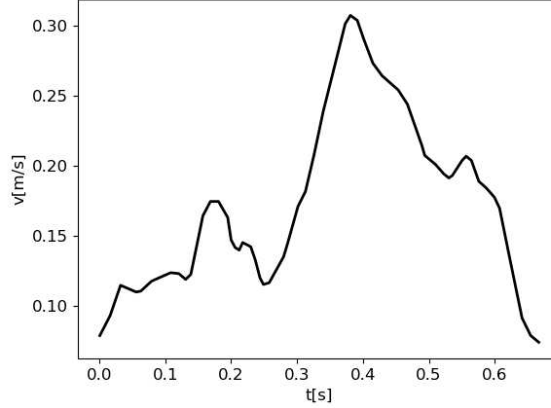


Figure 3: Inflow velocity profile

For the outlet pressure boundary condition, a three-element Windkessel model (Tricarico et al., 2023), also known as the RCR model, was employed. The Windkessel model represents the downstream vascular resistance and compliance using three lumped parameters that are analogous to the components of an electrical RC circuit.

$$dP(t) = (R_p + R_c)Q(t) + R_p R_d C \frac{dQ(t)}{dt} - R_d C \frac{dP}{dt} \quad (5)$$

Here,  $R_p$  is proximal resistance, which represents the resistance to blood flow in the vessels immediately downstream of the modelled coronary segment;  $R_c$

is the distal resistance, which represents the resistance of the smaller downstream vessels, particularly the arterioles and microcirculation;  $C$  is vascular compliance, which represents the ability of the downstream vascular system to store blood volume as pressure changes. It reflects the elastic storage capacity of the arterial and microvascular system.  $Q(t)$  is the volumetric flow rate.

The key parameters used in the Windkessel model and their values are listed in Table 2. These values are close to the range reported by Jiang et al. (2022) for coronary artery simulations.

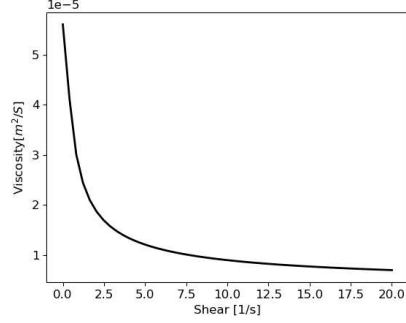
### 2.3. Hemodynamic Viscosity

Blood is a non-Newtonian fluid, whose viscosity changes with the strain rate of the flow field. In the current study, the The Bird-Carreau model (Carreau, 1972) was used to capture the strain-rate-dependant hemodynamic viscosity.

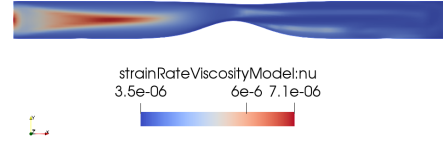
$$\nu(\dot{\gamma}) = \nu_{\infty} + (\nu_0 - \nu_{\infty})[1 + (\lambda\dot{\gamma})^2]^{\frac{n-1}{2}} \quad (6)$$

where  $\dot{\gamma}$  is the shear strain rate in  $1/s$ ;  $\nu_{\infty}$  and  $\nu_0$  are the limiting viscosity at high and zero strain rate respectively;  $\lambda$  is the relaxation time constant and  $n$  is the power-law index. Table 3 lists the values used in the current study. These values align with those reported by Perktold and Rappitsch (1995).

Figure 4a shows the calculated dynamic viscosity as a function of strain rate. The viscosity decreases exponentially with increasing strain rate, consistent with the shear-thinning behaviour of the non-Newtonian blood model employed in the present study. Figure 4b presents a snapshot of the viscosity field for a case with zero-degree vascular curvature. The reduction in viscosity in regions of high strain rate, particularly near the vessel wall, is clearly evident.



(a) Hemodynamic viscosity as a function of strain rate



(b) Hemodynamic viscosity at peak systolic velocity (PSV)

Figure 4: Hemodynamic viscosity from Bird-Carreau model

#### 2.4. Solver Setup

The CFD simulations were performed using OpenFOAM (Weller et al., 1998) v10.1, an open-source CFD framework widely used in academic research and industrial applications. For each case, three consecutive cardiac cycles were simulated to ensure that the flow field approached a periodic state. The results from the third and final cardiac cycle were used for post-processing and subsequent analysis. Figure 5 shows the temporal variations of WSS and WSSG over the three consecutive cardiac cycles, demonstrating the cycle-to-cycle convergence of the hemodynamic quantities.

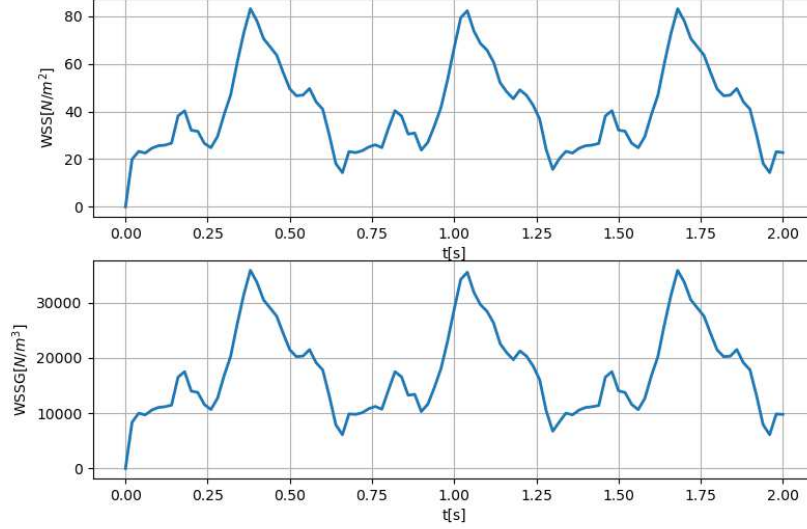


Figure 5: Example of WSS and WSSG over three consecutive cardiac cycles

A hybrid solver Weller et al. (1998), which combines the Pressure-Implicit with Splitting of Operators (PISO) solver (Issa, 1986) and Semi-Implicit Method for Pressure-Linked Equations (SIMPLE) (Patankar and Spalding, 1972) algorithms, was used to solve the governing partial differential equations. A second-order Gauss linear scheme was employed for spatial discretization, while a first-order Euler scheme was used for temporal discretization.

### 2.5. WSS and WSSG Calculation

The wall shear stress (WSS) is defined as

$$\boldsymbol{\tau}_w = \mu \frac{\partial \mathbf{u}}{\partial y} \Big|_{y=0}, \quad (7)$$

where  $\mu$  is the dynamic viscosity and  $y$  denotes the direction normal to the vessel wall. The wall shear stress gradient (WSSG) is defined as the spatial gradient of WSS along the vessel wall:

$$WSSG = \frac{\partial \boldsymbol{\tau}_w}{\partial s}, \quad (8)$$

where  $s$  denotes the tangential direction along the vessel wall. The tangential component of the gradient of the wall shear stress can be obtained by projecting its spatial gradient onto the wall surface:

$$\nabla_s \boldsymbol{\tau}_w = \nabla \boldsymbol{\tau}_w - (\nabla \boldsymbol{\tau}_w \cdot \mathbf{n}) \mathbf{n}, \quad (9)$$

where  $\nabla_s$  denotes the surface (tangential) gradient and  $\mathbf{n}$  is the unit normal vector to the vessel wall. Finally,

$$WSSG_{\text{signed}} = \|\nabla_s \boldsymbol{\tau}_w\| \operatorname{sgn}(\boldsymbol{\tau}_w \cdot \nabla_s \boldsymbol{\tau}_w). \quad (10)$$

Note that the positive sign of WSSG is defined as the direction in which the magnitude of WSS increases. In this study, the maximum WSSG refers to the maximum positive value of WSSG.

### 3. Results and Discussion

#### 3.1. Sensitivity of WSS and WSSG with respect to plaque morphology

Figure 6 shows a snapshot of the flow field at peak systolic velocity (PSV) for a vessel with a positive  $15^\circ$  curvature and 80% area stenosis. The flow inlet is located on the left side of the domain. The highest flow velocity and WSS occur near the minimum lumen area (MLA). The WSSG exhibits a characteristic positive-to-negative variation across the stenotic region, with the maximum positive WSSG occurring slightly upstream of the location of maximum WSS and the maximum negative WSSG occurring slightly downstream. Thus, the maximum positive WSSG is located on the proximal side of the stenotic throat, whereas the maximum negative WSSG occurs on the distal side.

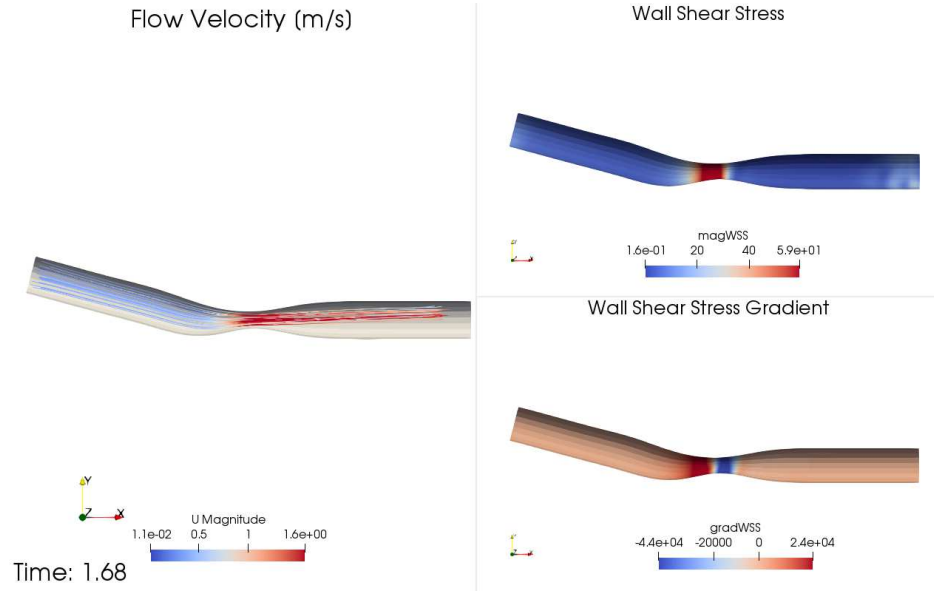
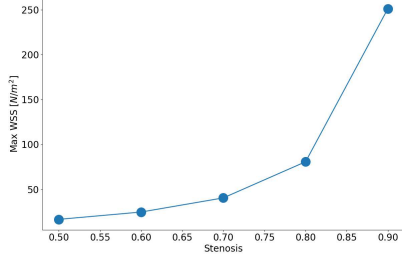
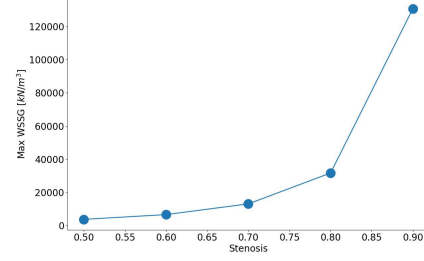


Figure 6: Representative velocity, WSS, and WSSG fields at peak systolic velocity for a vessel with  $15^\circ$  curvature and 80% area stenosis.

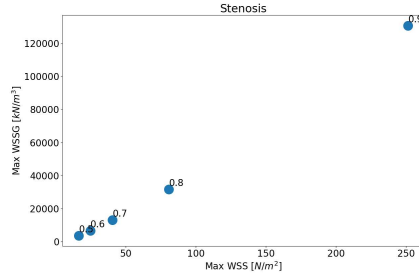
Figure 7 shows the sensitivity of maximum wall shear stress (WSS) and wall shear stress gradient (WSSG) to the degree of stenosis over a cardiac cycle. The results correspond to simulation cases 1–5 in Table 1, which represent concentric and longitudinally symmetric plaques in a vessel with zero curvature. As shown in Figs.7a and 7b, when the inlet velocity profile is held constant, both WSS and WSSG exhibit nonlinear relationships with percent area stenosis (PAS), which results from both increased velocity and reduced lumen dimensions simultaneously. Furthermore, Fig.7c shows that the relationship between maximum WSS and maximum WSSG remains approximately linear across the range of PAS investigated.



(a) PAS Vs. maximum WSS



(b) PAS Vs. maximum WSSG



(c) Maximum WSS Vs. maximum WSSG

Figure 7: Sensitivity of Maximum WSS and WSSG with respect to PAS

Figure 8 shows the sensitivity of the maximum WSS and WSSG to plaque eccentricity for cases 6–9 in Table 1. These cases correspond to longitudinally symmetric plaques with a PAS of 0.8 in a straight vessel. Unlike the non-linear, approximately first- to second-order relationship observed between WSS/WSSG and PAS, the relationships between WSS/WSSG and eccentricity are approximately linear. Both maximum WSS and WSSG increase moderately with increasing eccentricity. Consequently, as shown in Fig. 8c, the relationship between maximum WSS and maximum WSSG remains approximately linear across the range of eccentricities investigated.

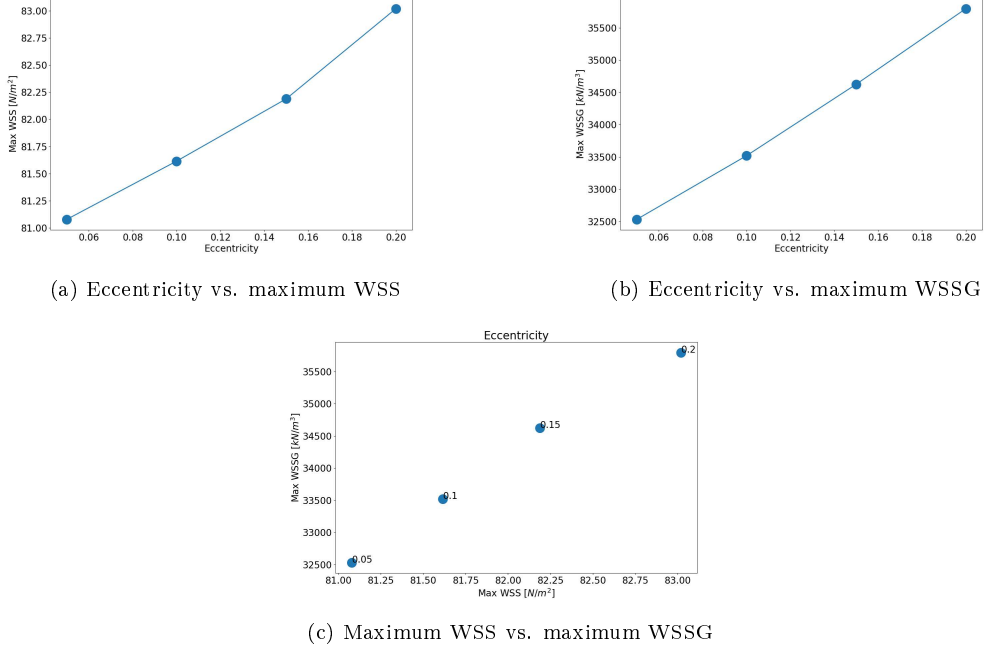
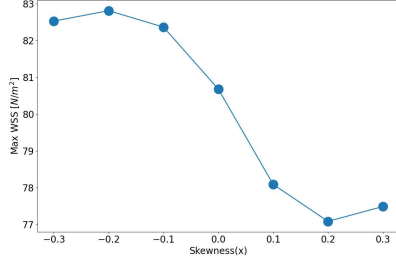


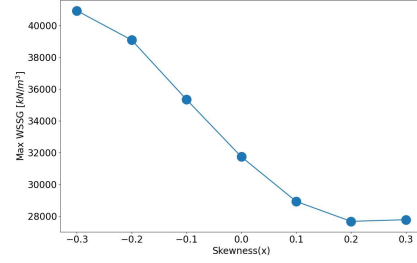
Figure 8: Sensitivity of maximum WSS and WSSG to plaque eccentricity.

Figure 9a shows the sensitivity of maximum WSS to the longitudinal skewness of the plaque profile for cases 10–16 in Table 1. In general, the effect of plaque skewness on WSS and WSSG is more pronounced than that of eccentricity. Maximum WSS decreases with increasing  $x$  in the range  $-0.2 < x < 0.2$ , indicating that proximally skewed plaques tend to produce higher WSS. This finding is consistent with the results reported by Hossain et al. (2024). Interestingly, maximum WSS levels off for  $x < -0.2$  and  $x > 0.2$ , suggesting that the effect of further increasing the degree of proximal or distal skewness becomes relatively limited beyond these ranges.

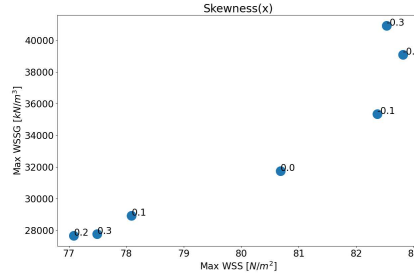
A similar trend is observed for maximum WSSG (Fig.9b), which increases with increasing proximal skewness and exhibits a relatively weaker dependence on  $x$  for  $x < -0.2$  and  $x > 0.2$ . Figure 9c shows that the WSS–WSSG relationship becomes nonlinear in the regions of  $x < -0.2$  and  $x > 0.2$ .



(a) Skewness Vs. maximum WSS



(b) Skewness Vs. maximum WSSG



(c) Maximum WSS Vs. maximum WSSG

Figure 9: Maximum WSS Vs. maximum WSSG with respect to longitudinal skewness (zero skewness at  $x=0$ )

Figure 10 compares the WSS and WSSG profiles at three different levels of plaque skewness. The maximum WSS decreases as the plaque morphology transitions from proximal skewness ( $x = -0.2$ ) to distal skewness ( $x = 0.2$ ). The maximum positive WSSG occurs upstream of the stenotic throat, with proximally skewed plaques producing higher positive WSSG values. In contrast, distal skewness promotes more negative WSSG values, with the minimum WSSG occurring downstream of the MLA following flow separation and the associated development of a re-circulation region.

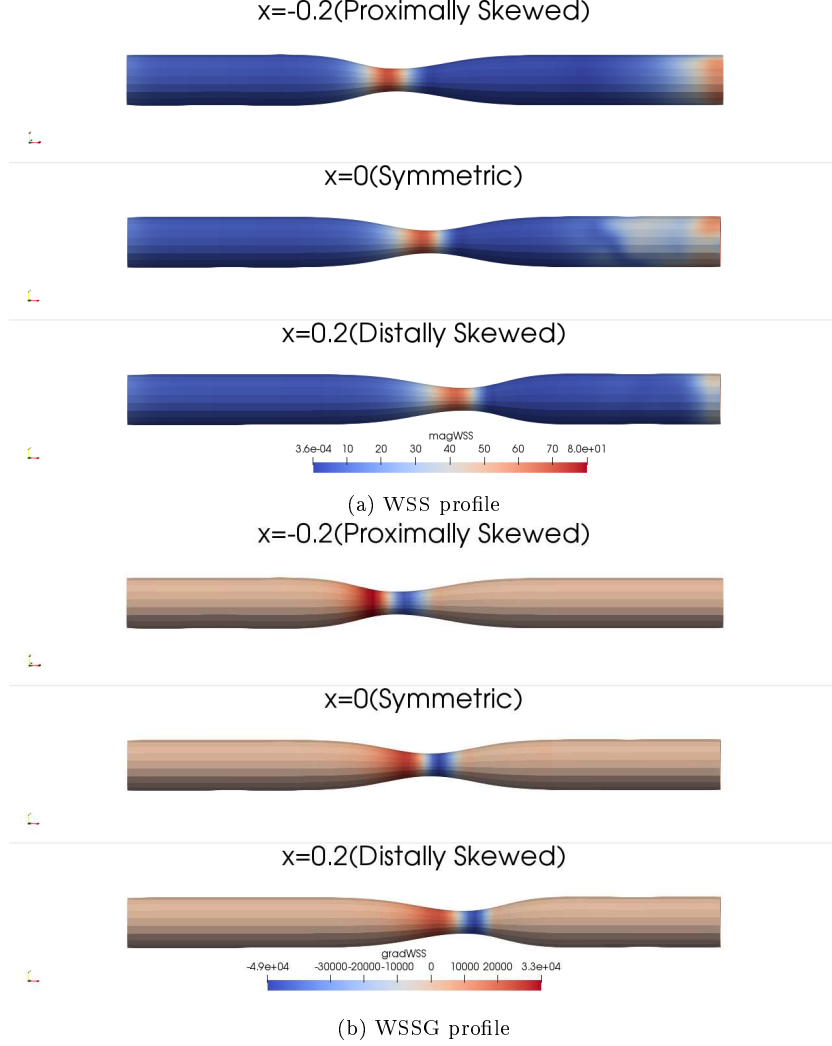
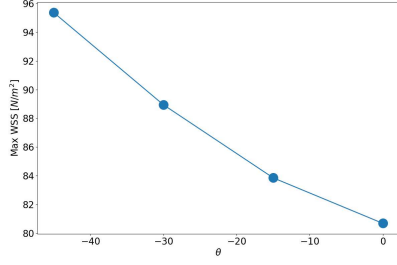


Figure 10: WSS and WSSG profiles at different levels of plaque skewness.

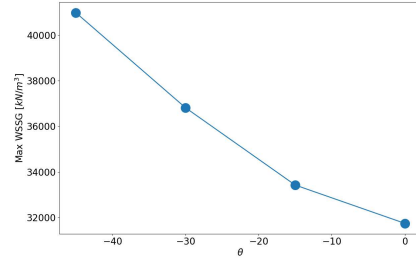
### 3.2. Effect of Vascular Curvature on WSS and WSSG

In the present study, the vascular curvature angle  $\theta$  was varied from  $-45^\circ$  to  $+45^\circ$ . Figure 11 shows the variation of maximum WSS and WSSG with curvature angle for a longitudinally symmetric, concentric plaque. The simulations correspond to cases 17–20 in Table 1. Both maximum WSS and WSSG increase with increasing curvature magnitude. For the concentric plaque configuration, the maximum WSS and WSSG decrease approximately linearly as the magnitude of the curvature angle decreases. Furthermore,

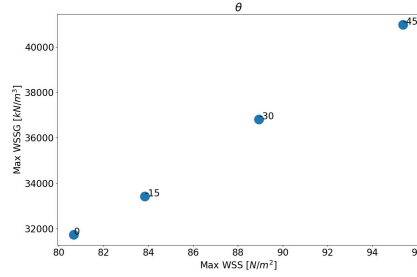
positive and negative curvature angles of equal magnitude produce nearly identical results, owing to the geometric symmetry of the concentric plaque configuration. As shown in Fig. 11c, maximum WSSG also exhibits an approximately linear relationship with maximum WSS across the range of curvature angles investigated.



(a) Vascular curvature vs. maximum WSS



(b) Vascular curvature vs. maximum WSSG



(c) Maximum WSS vs. maximum WSSG

Figure 11: Variation of maximum WSS and WSSG with vascular curvature.

Figure 12 compares the WSS and WSSG fields at PSV for cases 17 and 20, corresponding to curvature angles of  $-45^\circ$  and  $0^\circ$ , respectively. For the case with a large curvature angle of  $\theta = -45^\circ$ , substantial flow separation develops downstream of the MLA, causing the streamlines to be displaced toward the lower portion of the flow domain. Meanwhile, the location of maximum velocity near the MLA is shifted toward the upper wall. This asymmetric velocity distribution results from the secondary flow induced by the strong curvature and the associated centrifugal effects.

As the curvature increases, the flow experiences a stronger radial pressure gradient required to maintain the curved flow path. The resulting redistribution of momentum across the vessel cross section shifts the high-velocity

region toward the outer wall and modifies the local velocity gradient near the vessel wall. In the present geometry, this produces a thinner near-wall boundary layer and a larger velocity gradient near the upper wall. Consequently, the local WSS and WSSG increase with increasing vascular curvature.

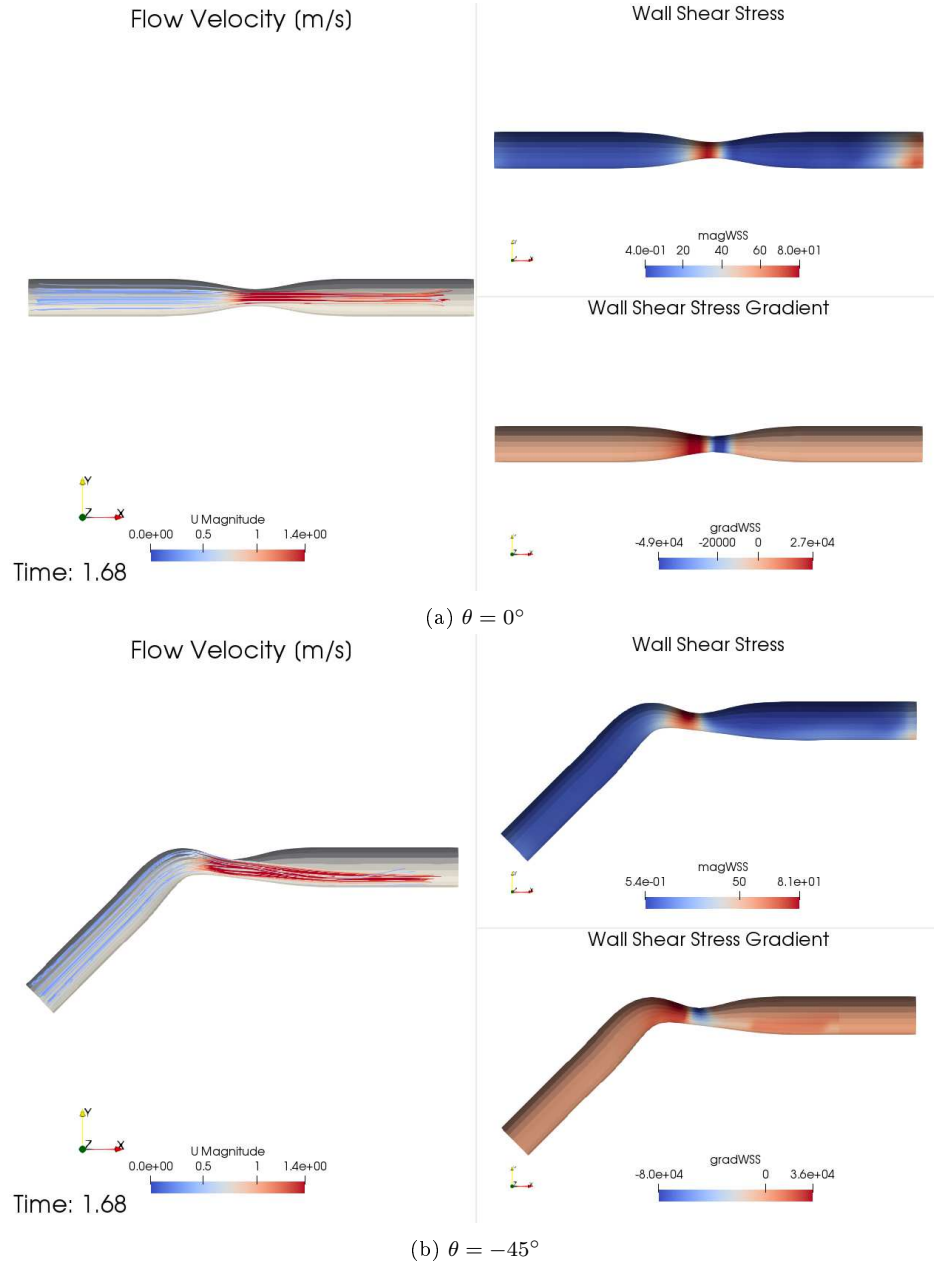


Figure 12: Comparison of streamlines, WSS, and WSSG at peak systolic velocity for  $\theta = 0^\circ$  and  $\theta = -45^\circ$ .

Figure 13 compares the velocity profiles near the minimum lumen area (MLA) for curvature angles of  $-45^\circ$  and  $0^\circ$ . The near-wall boundary layer

along the upper wall is thinner for the  $-45^\circ$  case than for the straight-vessel case, indicating a larger local velocity gradient near the wall.

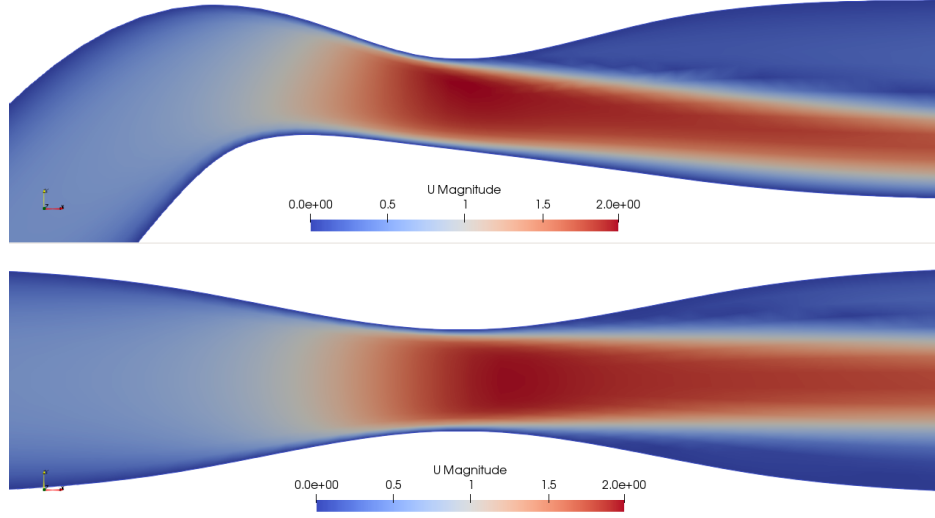


Figure 13: Velocity profiles near the minimum lumen area for curvature angles of  $-45^\circ$  (upper) and  $0^\circ$  (lower).

As the curvature angle increases, the location of maximum velocity shifts toward the opposing wall, resulting in an increased velocity gradient near the wall. Because WSS is directly related to the near-wall velocity gradient, this redistribution of the velocity profile leads to an increase in WSS, as shown in Fig. 14. The resulting increase in WSS is consistent with the observations of Foutrakis et al. (1999), who reported that curvature-induced centrifugal effects can redistribute high-velocity blood toward the outer wall, increasing local impact pressure and maximum WSS. Such elevated hemodynamic loading may contribute to plaque instability and increase the potential risk of rupture when atherosclerotic plaques are located along the outer wall of a curved vessel.

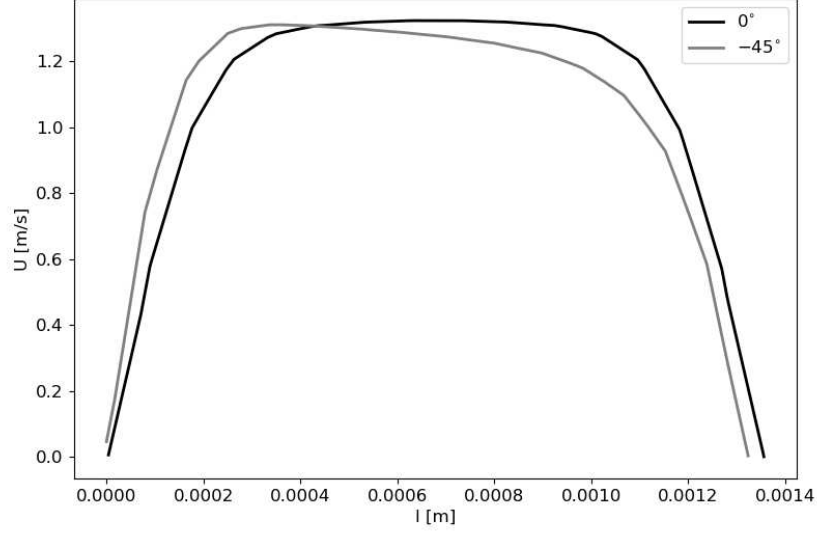
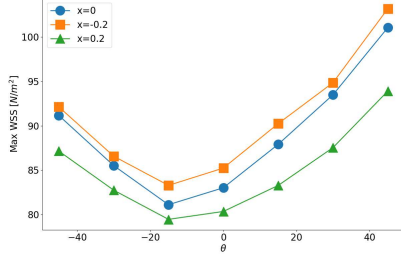
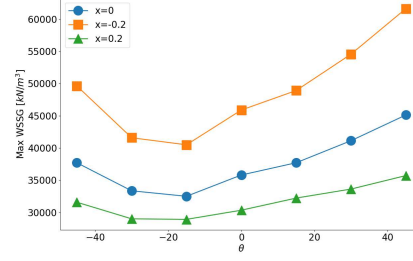


Figure 14: Comparison of cross-sectional velocity profile between 0 and 45 degree vascular curvatures

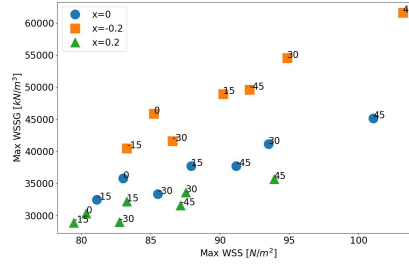
Figure 15 shows the relationship between the vascular curvature angle  $\theta$  and maximum WSS and WSSG for eccentric plaques with different levels of longitudinal skewness (cases 24–37 in Table 1. All plaques have a PAS of 80%. The three cases exhibit similar trends with respect to  $\theta$ . Interestingly, the minimum values of both WSS and WSSG occur at a curvature angle of approximately  $-15^\circ$  rather than at  $\theta = 0^\circ$ . Furthermore, positive curvature angles produce higher WSS and WSSG than negative curvature angles of the same magnitude, indicating an asymmetric response to vascular curvature in the presence of plaque eccentricity. Among the three levels of skewness, the proximally skewed plaque exhibits the highest WSS and WSSG, whereas the distally skewed plaque produces the lowest values.



(a)  $\theta$  vs. maximum WSS



(b)  $\theta$  vs. maximum WSSG



(c) Maximum WSS vs. maximum WSSG

Figure 15: Effect of vascular curvature angle on maximum WSS and WSSG for eccentric plaques with different levels of longitudinal skewness.

The low WSS and WSSG observed at  $\theta = -15^\circ$  for the eccentric plaque demonstrates the interaction between the plaque morphology and vascular curvature. Figure. 16 shows the velocity, WSS, and WSSG distributions within the flow domain at PSV. In this configuration, the plaque is located along the inner bend of the artery. The curvature-induced redistribution of the flow causes the high-velocity region to be directed towards the opposing wall around the bend, reducing the effect of rising topology at the inner bend due to the plaque. Consequently, the flow approaches the stenotic region with a smaller change in flow direction than in the corresponding straight-vessel configuration. This reduced directional change appears to mitigate the local velocity gradients near the stenosis, resulting in lower WSS and WSSG.

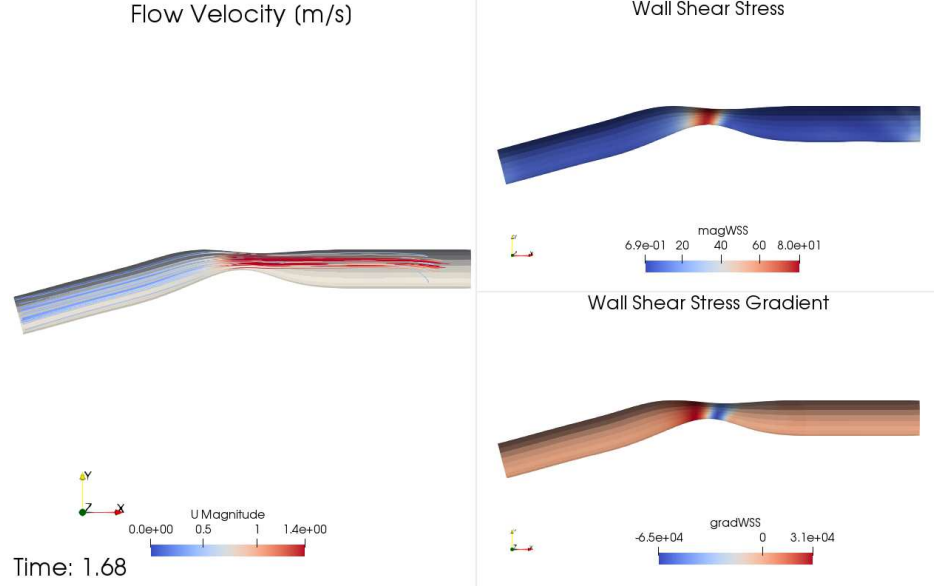


Figure 16: Velocity, WSS, and WSSG fields for an eccentric plaque at PSV with a curvature angle of  $\theta = -15^\circ$ .

The WSS and WSSG fields for an eccentric, proximally skewed plaque under strong positive and negative vascular curvature are shown in Fig. 17. The plaque has a skewness factor of  $x = -0.2$  and an eccentricity of 0.2. For the negative-curvature case, the curvature-induced redistribution of the flow produces a relatively smooth transition toward the MLA. In contrast, the positive-curvature configuration produces a sharper contraction of the flow passage, owing to the nearly perpendicular orientation between the incoming flow direction and the proximal plaque surface. This geometric configuration results in a more abrupt change in the flow direction and a more concentrated local hemodynamic response. These observations highlight the complex interaction between plaque morphology, including eccentricity and longitudinal skewness, and vascular geometry, particularly curvature.

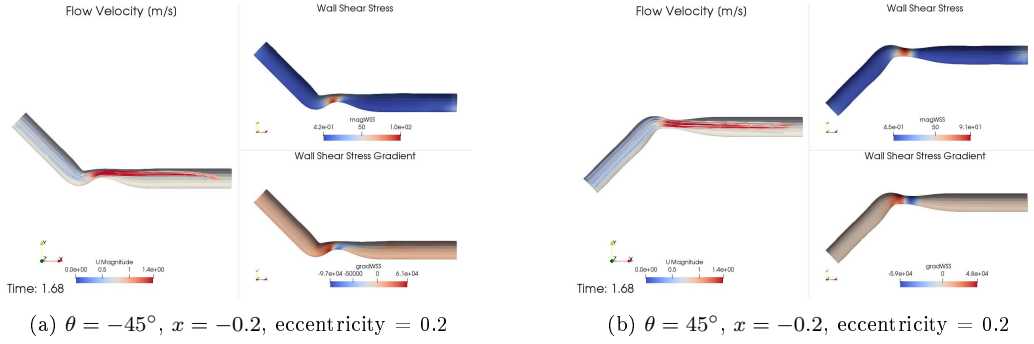


Figure 17: Velocity, WSS and WSSG surface fields for a proximally skewed eccentric plaque at curvature angles of  $\pm 45^\circ$ .

#### 4. Conclusion

A parametric CFD study using OpenFOAM was conducted to systematically investigate the effects of vascular curvature and plaque morphology on wall shear stress (WSS) and wall shear stress gradient (WSSG) in idealized coronary arteries with atherosclerotic plaques. The parameters investigated included percent area stenosis (PAS), plaque longitudinal skewness, eccentricity, and vascular curvature.

The results demonstrate that increasing the degree of stenosis substantially increases both WSS and WSSG. The relationship with PAS exhibits nonlinear behaviour, reflecting the combined effects of increased flow velocity and reduced lumen dimensions within the stenotic region. Plaque skewness also has a pronounced effect on the local hemodynamics. Proximally skewed plaques produce substantially higher WSS and WSSG, whereas distally skewed plaques produce lower values. The effect of skewness levels off for  $x$  above 0.2 or below -0.2. In comparison, plaque eccentricity has a more moderate and approximately linear influence on both WSS and WSSG.

For concentric plaques, vascular curvature exhibits an approximately linear relationship with both WSS and WSSG, with greater curvature magnitude producing higher values. For eccentric plaques, however, the effect of curvature is asymmetric and non-monotonic, depending on the relative orientation of the vascular curvature and plaque eccentricity. For a plaque with an eccentricity of 0.2, the lowest peak WSS and WSSG were observed at a curvature angle of  $-15^\circ$ , whereas positive curvature angles produced substantially higher values. These findings demonstrate that plaque morphology and vascular geometry can interact to produce markedly different

local hemodynamic environments.

Overall, the findings highlight the importance of incorporating detailed plaque morphology and vascular geometry when evaluating hemodynamic conditions associated with coronary plaque vulnerability. The systematic parametric framework developed in this study provides a foundation for further investigation of the interactions among plaque morphology, vascular geometry, and hemodynamic risk factors. In the longer term, this framework may support the development of comprehensive CFD databases and data-driven models for rapid estimation of plaque-related hemodynamic risk in patient-specific coronary arteries, potentially providing an additional tool for clinical risk assessment and treatment planning.

### **Declaration of Competing Interest**

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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## References

- Asakura, T., et al., 1990. Flow patterns and spatial distribution of atherosclerotic lesions in human coronary arteries. *Circulation Research* 66, 1045–1066. doi:10.1161/01.RES.66.4.1045.
- Bajraktari, A., et al., 2021. High coronary wall shear stress worsens plaque vulnerability: A systematic review and meta-analysis. *Angiology* 72, 706–714. doi:10.1177/0003319721991722.
- Carreau, P., 1972. Rheological equations from molecular network theories. *Transactions of the Society of Rheology* 16.
- Chaichana, T., et al., 2011. Computation of hemodynamics in the left coronary artery with variable angulations. *Journal of Biomechanics* 44, 1869–1878. doi:10.1016/j.jbiomech.2011.04.033.
- Choi, G., et al., 2015. Coronary artery axial plaque stress and its relationship with lesion geometry. *JACC Cardiovasc Imaging* 8, 1156–1166. doi:10.4208/cicp.030714.101014a.
- Dhawan, S., et al., 2010. Shear stress and plaque development. *Expert Review of Cardiovascular Therapy* 4, 545–556.
- Ebrahimi, S., et al., 2022. Investigation of coronary artery tortuosity with atherosclerosis: A study on predicting plaque rupture and progression. *International Journal of Mechanical Sciences* 223. doi:10.1016/j.ijmecsci.2022.107295.
- Foutrakis, G., et al., 1999. Saccular aneurysm formation in curved and bifurcating arteries. *American Journal of Neuroradiology* 20, 1309–1317.
- Hossain, S., et al., 2024. A parametric study of the effect of 3d plaque shape on local hemodynamics and implications for plaque instability. *Biomech Model Mechanobiol* 23, 1209–1227. doi:10.1007/s10237-024-01834-6.
- Issa, I., 1986. Solution of the implicitly discretised fluid flow equations by operator-splitting-in-fact. *Journal of Computational Physics* 62, 40–65.
- Jiang, X., et al., 2022. A hemodynamic analysis of the thrombosis within occluded coronary arterial fistulas with terminal aneurysms using a blood stasis mode. *Front. Physiol* 13, 906502. doi:10.3389/fphys.2022.906502.

- Kaminsky, L., et al., 2024. From atherosclerotic plaque to myocardial infarction-the leading cause of coronary artery occlusion. *Int J Mol Sci.* 25, 7295. doi:10.3390/ijms25137295.
- Kinnoshita, D., et al., 2024. Relationship between plaque burden and plaque vulnerability: Acute coronary syndromes versus chronic coronary syndrome. *J Cardiovasc Comput Tomogr* 18, 559–566. doi:10.1016/j.jcct.2024.09.002.
- Kok, A., et al., 2015. The influence of multidirectional shear stress on plaque progression and composition changes in human coronary arteries. *EuroIntervention* 15, 692–699.
- Malek, S., et al., 2025. Machine learning-based prediction of hemodynamic parameters in left coronary artery bifurcation: A cfd approach. *Heliyon* 11. doi:10.1016/j.heliyon.2025.e41973.
- Malota, Z., et al., 2018. Numerical analysis of the impact of flow rate, heart rate, vessel geometry, and degree of stenosis on coronary hemodynamic indices. *BMC Cardiovascular Disorders* 18, 132. doi:10.1186/s12872-018-0865-6.
- Młynarska, E., et al., 2022. The importance of healthy lifestyle behaviors in the prevention of cardiovascular disease. *Prog Cardiovasc Dis* 70, 8–15. doi:10.1016/j.pcad.2021.12.001.
- Patankar, V., Spalding, B., 1972. A calculation procedure for heat, mass and momentum transfer in three-dimensional parabolic flows. *International Journal of Heat and Mass Transfer* 15, 1787–1806.
- Perktold, K., Rappitsch, G., 1995. Computer simulation of local blood flow and vessel mechanics in a compliant carotid artery bifurcation model. *Journal of Biomechanics* 28, 845–856.
- Russo, G., et al., 2023. Coronary artery plaque rupture and erosion: Role of wall shear stress profiling and biological patterns in acute coronary syndromes. *International Journal of Cardiology* 370, 356–365. doi:10.1016/j.ijcard.2022.10.139.

- Seligman, H., et al., 2022. Phasic flow patterns of right versus left coronary arteries in patients undergoing clinical physiological assessment. *Eurointervention* 17, 1260–1270.
- Stone, P., et al., 2007. Regions of low endothelial shear stress are the sites where coronary plaque progresses and vascular remodelling occurs in humans: an in vivo serial study. *European Heart Journal* 28, 705–710. doi:10.1093/eurheartj/ehl575.
- Thondapu, V., et al., 2021. High spatial endothelial shear stress gradient independently predicts site of acute coronary plaque rupture and erosion. *Cardiovascular Research* 117, 1974–1985. doi:10.1093/cvr/cvaa251.
- Tricarico, R., et al., 2023. Non-invasive estimation of the parameters of a three-element windkessel model of aortic arch arteries in patients undergoing thoracic endovascular aortic repair. *Front Bioeng Biotechnol* 11, 1127855. doi:10.3389/fbioe.2023.1127855.
- Updegrove, A., et al., 2016. Simvascular: An open source pipeline for cardiovascular simulation. *annals of biomedical engineering. Annals of Biomedical Engineering* 44, 2485—2493. doi:10.1007/s10439-016-1762-8.
- Veneziano, F., et al., 2026. Plaque progression and rupture in obstructive coronary artery disease: A review of current imaging modalities. *Rev Cardiovasc Med* 27, 48015. doi:10.31083/RCM48015.
- Wahle, A., 2006. Plaque development, vessel curvature, and wall shear stress in coronary arteries assessed by x-ray angiography and intravascular ultrasound. *Med Image Anal.* 10, 615–631. doi:10.1016/j.media.2006.03.002.
- Wang, L., et al., 2025. Role of biomechanical factors in plaque rupture and erosion: insight from intravascular imaging based computational modeling. *npj Cardiovasc Health* 2, 12. doi:10.1038/s44325-025-00048-8.
- Weller, H., et al., 1998. A tensorial approach to computational continuum mechanics using object-oriented techniques. *Computers in Physics* 12, 620–631. doi:10.1063/1.168744.
- Zhao, C., et al., 2024. Plaque ruptures are related to high plaque stress and strain conditions: direct verification by using in vivo OCT rupture data and FSI models. *Arterioscler Thromb Vasc Biol* 44, 1617–1627. doi:10.1161/ATVBAHA.124.320764.

Table 1: CFD Cases and Parameter Values

| Case No. | skewness(x) | Curvature( $\theta$ ) | eccentricity | stenosis | Remarks  |
|----------|-------------|-----------------------|--------------|----------|----------|
| 1        | 0           | 0                     | 0            | 0.5      | Baseline |
| 2        | 0           | 0                     | 0            | 0.6      |          |
| 3        | 0           | 0                     | 0            | 0.7      |          |
| 4        | 0           | 0                     | 0            | 0.8      |          |
| 5        | 0           | 0                     | 0            | 0.9      |          |
| 6        | 0           | 0                     | 0.05         | 0.8      |          |
| 7        | 0           | 0                     | 0.1          | 0.8      |          |
| 8        | 0           | 0                     | 0.15         | 0.8      |          |
| 9        | 0           | 0                     | 0.2          | 0.8      |          |
| 10       | -0.3        | 0                     | 0            | 0.8      |          |
| 11       | -0.2        | 0                     | 0            | 0.8      |          |
| 12       | -0.1        | 0                     | 0            | 0.8      |          |
| 13       | 0           | 0                     | 0            | 0.8      |          |
| 14       | 0.1         | 0                     | 0            | 0.8      |          |
| 15       | 0.2         | 0                     | 0            | 0.8      |          |
| 16       | 0.3         | 0                     | 0            | 0.8      |          |
| 17       | 0           | -45                   | 0            | 0.8      |          |
| 18       | 0           | -30                   | 0            | 0.8      |          |
| 19       | 0           | -15                   | 0            | 0.8      |          |
| 20       | 0           | 0                     | 0            | 0.8      |          |
| 21       | 0           | 15                    | 0            | 0.8      |          |
| 22       | 0           | 30                    | 0            | 0.8      |          |
| 23       | 0           | 45                    | 0            | 0.8      |          |
| 24       | 0           | -45                   | 0.2          | 0.8      |          |
| 25       | 0           | -30                   | 0.2          | 0.8      |          |
| 26       | 0           | -15                   | 0.2          | 0.8      |          |
| 27       | 0           | 0                     | 0.2          | 0.8      |          |
| 28       | 0           | 15                    | 0.2          | 0.8      |          |
| 29       | 0           | 30                    | 0.2          | 0.8      |          |
| 30       | 0           | 45                    | 0.2          | 0.8      |          |
| 31       | -0.2        | -45                   | 0.2          | 0.8      |          |
| 32       | -0.2        | -30                   | 0.2          | 0.8      |          |
| 33       | -0.2        | -15                   | 0.2          | 0.8      |          |
| 34       | -0.2        | 0                     | 0.2          | 0.8      |          |
| 35       | -0.2        | 15                    | 0.2          | 0.8      |          |
| 36       | -0.2        | 30                    | 0.2          | 0.8      |          |
| 37       | -0.2        | 45                    | 0.2          | 0.8      |          |

Table 2: Windkessel model parameters for pressure outlet boundary condition

| Parameter | Value | Unit       |
|-----------|-------|------------|
| $R_p$     | 5e8   | $Pa.s/m^2$ |
| $R_d$     | 5e9   | $Pa.s/m^2$ |
| $C$       | 3e-10 | $m^3/s$    |

Table 3: Coefficients for Bird-Carreau viscosity model

| Parameter    | Value  | Unit    |
|--------------|--------|---------|
| $\nu_0$      | 5.6e-5 | $m^2/s$ |
| $\nu_\infty$ | 3.5e-6 | $m^2/s$ |
| $\lambda$    | 3.313  |         |
| $n$          | 0.357  |         |