Modeling Of Blood Flow In Stenosed Lad Coronary Artery

Atherosclerosis is the most common causes of vascular diseases. Thus, looking for the physical factors and correlations that explain the phenomena of existence the atherosclerosis disease in LAD artery in some people rather than the others is achieved in this study by analysis data from coronary angiography as well as estimating the blood velocity from coronary angiography scans without having a required data on velocity by using some mathematical equations and physical laws.

It found some information that may explain the ability of small arteries to develop the lesions with time mainly in proximal site of LAD artery. For investigation the fluid-structural response to pulsatile Newtonian and non-Newtonian blood flow through an axisymmetric stenosed coronary artery; the simulation is built up by using ANSYS11 to evaluate the biomechanical parameters in the atherosclerotic process. Rigid and Newtonian cases were investigated to provide an understanding on the effects of incorporating Fluid-Structural Interaction FSI into model.

For validation of the models and methods used, the computation results are compared with previous studies. Read more Read less. Kindle Cloud Reader Read instantly in your browser. Register a free business account. Tell the Publisher! I'd like to read this book on Kindle Don't have a Kindle?

Beyond your wildest dreams. Listen free with trial. Customer reviews. How are ratings calculated? Instead, our system considers things like how recent a review is and if the reviewer bought the item on Amazon. It also analyzes reviews to verify trustworthiness. No customer reviews. The results are tabulated in Table 2. Additionally, a sensitivity test of the inlet turbulence intensity $Tu$ was carried out by using the SST transition model. The experimental results of Ahmed and Giddens [23] were used for the initial validation of the sensitivity test. The test was carried out by using the idealized geometry and steady flow conditions depicted in Deshpande and Giddens [24]. The tube was extended upstream and downstream of the throat by and , respectively.

The inlet and outlet lengths are those found in Ryval et al. Figure 4 shows different levels of $Tu$ between 1. In the turbulence intensity profiles, the radial position $r$, was normalized by the local radius where corresponds to the upper wall of the model, while corresponds to the center of the tube. It is shown that values between 1. Hence, for the simulations performed in this study, a $Tu$ value of 1. The results obtained for this sensitivity test are in complete agreement with those performed by Tan et al. Furthermore, a validation test was conducted in order to confirm that the SST transition model is capable of predicting turbulence under pulsatile flow conditions.

The pulsatile flow conditions were based on the experiments conducted by Ahmed and Giddens [26]. The fluid properties used were those based on the experiments by Ahmed and Giddens [23] where they used a solution of water and glycerin. The amplitude of the transient flow ranged
from of to All velocities were normalized by , one-half of the sum of the maximum and minimum upstream centerline velocities.

It can be seen from the figure that the model represents the experimental data fairly well. The results obtained for the validation test employed are also in good agreement with those obtained by Tan et al. The simulations have been performed for protrusions of Type I through Type IV see Figure 1 using the physiological waveform depicted in Figure 2. In this section, a comparison of the similarities and differences in the flow characteristics amongst these types is performed.

The flow characteristics of interest are the three components of the mean velocity profiles, the wall shear stresses WSS due to the viscous and turbulent terms, the turbulent kinetic energy TKE, and the turbulence intensity Tu, where the last two characteristics are an important representation of the turbulent state of the flow.

First, the differences and similarities in the axial component of the mean velocity are analyzed during the peak systolic phase of the cardiac cycle. Figure 6 shows axial component of the mean velocity. Seven different axial locations along the length of the artery have been selected for the analysis.

In the mean velocity profiles, the wall normal distance has been scaled by the lumen height where and correspond to the lower wall and upper wall, respectively. Such similarity is expected since the blood flow is natural and has not encountered the stenotic region. For Type I, the profile is no longer symmetric where the maximum centerline mean velocity is shifted towards the lower wall.

In comparison, for Type II and Type III, the profile is no longer symmetric where the maximum centerline mean velocity is shifted towards the upper wall. For Type IV, the profile remains almost unchanged. Type IV exhibits minimal flow reversal at the upper wall. Similarly, the differences and similarities in the radial component of the mean velocity are analyzed during the peak systolic phase.

Figure 7 shows the radial component of the mean velocity for the four types. At the inlet region, the flow is unidirectional with no radial component of velocity for the four types. As the flow approaches the proximal region of the plaque, the flow becomes three-dimensional with a nontrivial radial component of velocity. At the proximal region, for Type I, Type II, and Type IV, the flow is directed towards the wall in the upper-half region of the lumen, while the flow is directed away from the wall in the lower-half region of the lumen.

In contrast, for Type III, the flow is directed away from the wall near the upper and lower wall regions. At the peak region, for Type I and Type IV, the flow is now directed away from the wall near the upper and lower wall regions, whereas for Type II, the flow is directed away from the wall near the upper wall region while remaining unidirectional on the lower wall.

In comparison, the flow for Type III is directed towards the wall in the upper-half region of the lumen and directed away from the wall in the lower-half region of the lumen.

Furthermore, the three mean velocity components have been averaged in the plane and plotted against the wall normal distance in order to study the overall effect of the stenosis in the natural blood flow.

Figure 8 shows the mean velocity profiles averaged in plane plotted against wall-normal distance during peak systole for the axial, radial, and circumferential components of the mean flow for Type I through Type IV. For the axial mean velocity component, Type I and Type IV exhibit minor disturbances in the natural blood flow. Type I and Type IV are almost symmetric with a peak velocity of around 0.

This profile is similar to those observed in the inlet region in Figure 6. For both types, the profile is no longer symmetric with peak velocities concentrated above the centerline. For the radial mean velocity component, Type IV exhibits minor disturbances in the overall radial flow, behaving unidirectionally.

In comparison, Type I and Type II exhibit major disturbances in the radial mean flow where the flow is overall directed away from the wall in the lower-half region of the lumen and directed towards the wall in the upper-half region, whereas for Type III, the flow is overall directed away from the wall in the lower-half region of the lumen while remaining unidirectional in the upper-half region. For the circumferential mean velocity component, the circumferential flow overall remains undisturbed and unidirectional with low magnitudes for all types, whereas for Type II, the circumferential flow attains a maximum of 0.

Next, an analysis of flow parameters such as turbulence intensity Tu, turbulence kinetic energy TKE, and wall shear stresses WSS is presented. Such analysis is of importance because these parameters are true representations of the turbulent nature of the flow.

First, the differences and similarities in the turbulence kinetic energy profiles for Type I through Type IV are analyzed during the peak systolic phase of the cardiac cycle. Similar to the mean velocity profiles, seven different axial locations along the length of the artery have been selected, whereas the wall normal distance has also been scaled by the lumen height.

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Additionally, the differences and similarities in the turbulence intensity Tu profiles for Type I through Type IV are also analyzed during the peak systolic phase of the cardiac cycle. Similar to the other profiles, seven axial locations were also selected. No significant differences in the Tu profiles are observed in the inlet region of the stenosis. The conclusions are similar to those observed in the TKE profiles.

Additionally, the viscous and turbulent wall shear stresses WSS at the wall along the length of the stenosis have been computed during the peak
systolic phase. The viscous and turbulent components in the mean flow direction normal to the wall have been included.

Figure 11 shows the viscous shear stresses at the wall along the length of the artery for the four types. Furthermore, the trend followed by the viscous WSS stress values is in complete agreement with the numerical simulations performed by Bhaganagar et al. Patient-specific simulations with realistic physiological flow conditions are conducted to understand the effect of plaque morphology in altering the flow characteristics in diseased coronary artery.

In our previous study Bhaganagar et al. This classification based on morphology is of great significance for fluid dynamics analysis. Bhaganagar et al. The previous study was restrictive as it assumed 1 laminar flow conditions and 2 steady forcing flow.

We use transition model to predict the location of flow transition to turbulent state. For the purpose of analysis, we select several axial locations along the length of the artery. The mean velocity time and phase averaged profiles exhibit significant differences between the types. All the types except Type IV exhibit flow reversal at distal portion of the stenosis. However, the location of the peak mean velocity shifts to the upper half of the artery for Types II and III, whereas this shift is towards the lower half of the artery for Type I.

Next, we analyze the flow disturbances. In particular, we are interested in understanding the differences in the location of the transition to turbulence, total stresses, and intensities of turbulent fluctuations between the types.

The transition for Type III occurs at much proximal location compared to other types suggesting that flow alters at earlier location for this type. The flow continues to be in a disturbed state close to the exit region for Type II and Type III, whereas the flow relaminarizes to laminar state beyond the distal location of the plaque for Type I.

This study clearly shows that, for the same degree of stenosis, a the presence of turbulence, b location of transition to turbulence, c turbulence intensity, and d region of turbulence are type-dependent. This study is of great significance to determine the risk of rupture of the plaques and thus identify the vulnerable plaques prone to rupture.

It should be noted that transition to turbulence is just one of the several biomechanical factors that contribute to risk of rupture plaque see Fukumoto et al.

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We will be providing unlimited waivers of publication charges for accepted articles related to COVID-19. Sign up here as a reviewer to help fast-track new submissions. Journal overview.

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Transient calculations of a timestep size of 0. Such timestep sizes resulted in an approximation of , and number of timesteps per cycle, respectively.

Five cardiac cycles were simulated for each transient calculation in order to ensure statistical convergence or periodicity. The results are tabulated in Table 2. Additionally, a sensitivity test of the inlet turbulence intensity Tu was carried out by using the SST transition model. The experimental results of Ahmed and Giddens [23] were used for the initial validation of the sensitivity test.

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