

Flow Patterns and Wall Shear Stress Distributions at Atherosclerotic-Prone Sites in a Human Left Coronary Artery - An Exploration Using Combined Methods of CT and Computational Fluid Dynamics

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Abstract – Computed tomography (CT) slices are combined with computational fluid dynamics (CFD) to simulate the flow patterns in a human left coronary artery. The vascular model was reconstructed from CT slices scanned from a healthy volunteer *in vivo*. The spatial resolution of the slices is $0.6 \times 0.6 \times 0.625$ mm so that geometrical details of the local wall surface of the vessel could be considered in the CFD modeling. This level of resolution is needed to investigate the wall shear stress (WSS) distribution, a factor generally recognized as a related to the atherogenesis. The WSS distributions on the main trunk and bifurcation of the left coronary artery of the model in one cardiac cycle are presented, and the results demonstrate that low and oscillating WSS is correlative with clinical observations of the atherosclerotic-prone sites in the left coronary artery.

Keywords—CFD, coronary artery, CT, WSS

I. INTRODUCTION

Numerous investigations of the atherosclerotic plaque distribution in human arterial systems have been done previously, generally revealing that atherosclerotic plaques typically occur at certain regions in the arterial system where the arteries have relative complex geometry that results in “disturbed” blood flow behavior [1, 2]. These observations have led to the general acceptance that local hemodynamic factors, such as blood flow patterns (or velocity distribution) and wall shear stresses (WSS), may play a role in the disease's initiation and, perhaps more importantly, its progression [2]. The entrance regions and segments of the main bifurcation of the left coronary artery are favorite places for atherosclerosis. Here the blood velocity distribution and WSS are not only influenced by the curved structure of the arteries but also influenced by the local geometrical details of the luminal surface of the vessels. To incorporate surface information in a CFD investigation of atherogenesis in coronary arteries has been a challenge because the anatomy of the vessels is too small to be clearly reconstructed using traditional noninvasive medical imaging methods *in vivo*. The CT technique, characterized by higher imaging resolution than MRI and ultrasound, presents a unique imaging opportunity in the investigation of coronary arteries of healthy subjects.

In this work, a CFD model of the left main coronary artery (LM) and the major trunks of the left anterior

descending coronary artery (LAD) and the left circumflex coronary artery (LCX) was reconstructed from CT slices scanned in a healthy volunteer. The blood velocity boundary conditions were obtained from phase contrast MRI scanning. The CFD results are capable of providing local details of WSS that cannot be revealed directly from imaging, and hence enable a better understanding of the role of hemodynamics in plaque development and progression.

II. METHODOLOGY

X-ray CT scanning of the left coronary artery and aortic root of a healthy volunteer was performed. The small size of the coronary arteries requires the CT technique for adequate spatial resolution. The data acquisition was performed using a narrow slice thickness (1.25 mm) with minimum 50% reconstruction overlap, and the final slice resolution was 0.6×0.6 mm with slice thickness 0.625 mm. Using image processing methods, the major lumens of the left coronary arterial tree were segmented [3]. Lacking the flow information in the small branches, only the LM and proximal sections of the LAD and LCX were modeled in the CFD simulation.

NURBS surfaces were selected to construct the arterial model. The lumens of the LM, LAD and LCX after the segmentation appeared as a sequence of independent contours in space. Smoothing methods were used to smooth their shapes and relative positions. Finally, the NURBS surfaces covered these contours to compose a smooth CFD model as shown in Fig. 2.

Because the CT technique can not supply blood flow information, a previous velocity measurement was used as the boundary condition for this CFD simulation [4]. The velocity measurement was performed in the proximal sections of the LAD and LCX of a healthy male using phase contrast MRI scanning. Fig. 1 shows the mean velocity waveforms that were used as the outlet velocity conditions in the computational model, and a traction-free condition was assumed in the inlet section of the LM.

Incompressible, Newtonian fluid and laminar flow were assumed in the calculation of the Navier-Stokes equations, and the solution was performed using the commercial CFD-ACE code.

III. RESULTS

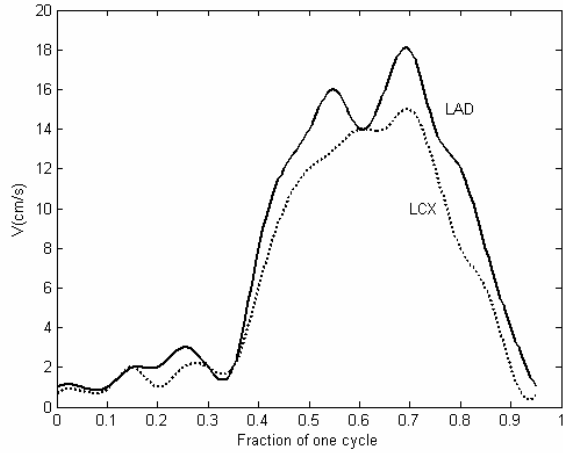


Fig. 1. Flow waveform in LAD and LCX. The horizontal axis expresses the fraction of one cardiac cycle that is normalized to 1.

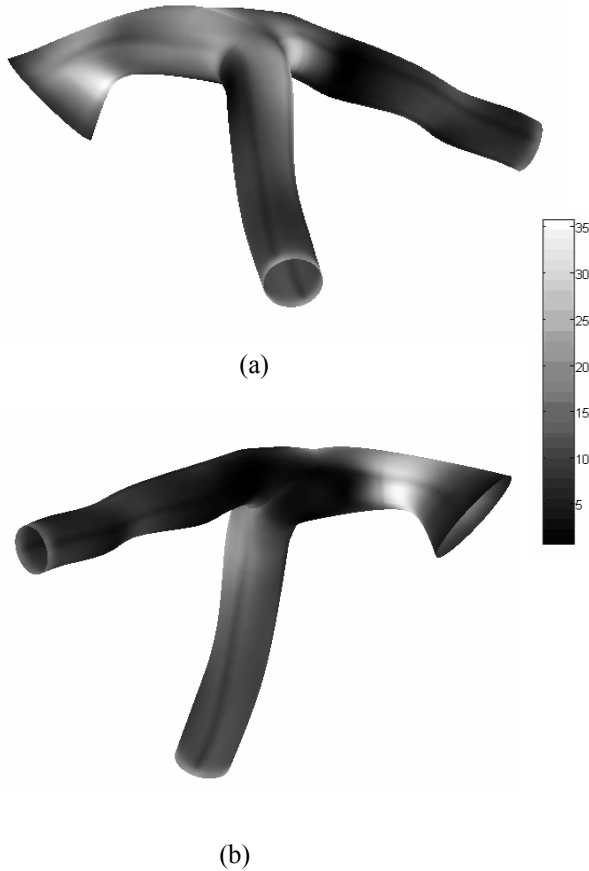


Fig. 2. CFD model and WSS distribution at time 0.8 (see Fig. 1). The front branch in (a) is the LAD and the back is the LCX. Same distribution but different viewing is shown in (b). The WSS gray scale unit is dyne/cm².

The high resolution of the CT slices allows us to explore more details of the flow near wall surfaces of the left coronary artery. The model reconstructed from the slices shows that the LCX surface has an undulating characteristic, while the surfaces of the LAD are smoother (see Fig. 2). Although we do not know the physiological implications of this difference, the pulsatile flow will produce local WSS values with unsteady magnitudes and directions under the influence of surfaces that rise and fall.

The velocity waveforms in Fig. 1 show the velocity boundary conditions assumed on the LAD and LCX outlet sections. The waveforms illustrate typical flow characteristics of the human left coronary artery: flow that is reversed in phase to the flow in aorta (i.e., predominant inflow is during diastole) and moderate mean velocity (≈ 15 cm/s) [5].

In the systolic phase, the net inflow in the LM is very small and the WSS over the entire surface of the artery is small (< 5 dyne/cm²). The mean velocity magnitude gradually rises to its peak value in the early diastolic phase. Simultaneously, the maximum WSS approaches 35 dyne/cm² in the front section of the inner curvature of the LM (Fig. 2). The section of the inner curvature starts from a few millimeters distal to the ostium and extends across the middle section of the LM. Another high WSS region is located at the inner surface of the main bifurcation. The differences in the WSS values over the artery also become the greatest at this time in the cycle, because the WSS always keeps small values (< 5 dyne/cm²) along both the outer surfaces and the myocardial side of the bifurcation. In the late stages of diastole, low and oscillating WSS appears in locations that always keep small WSS values and also where the undulating surfaces are located, the latter reflecting effects of local curvature.

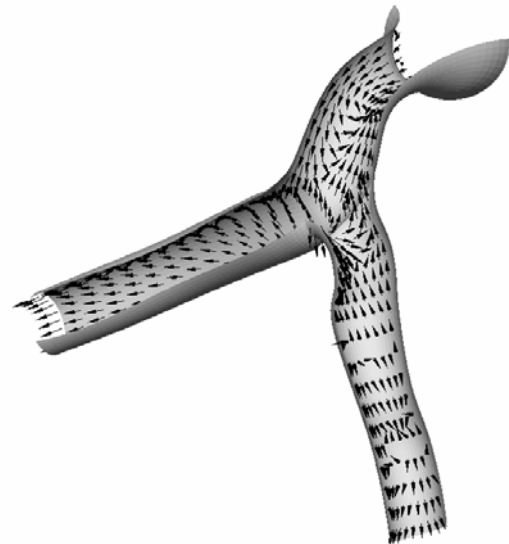


Fig. 3. The vectors of WSS at time 0.9. The length of the arrow is proportional to the WSS magnitude.

Fig. 3 shows the WSS vector distribution of the left coronary artery in late diastole. The WSS vectors that do not point in the downstream direction imply that the WSS is oscillating in direction, as well as in magnitude. Referring to Fig. 2, these locations are the myocardial side of the bifurcation and the downstream regions of the undulating surfaces in the LCX.

IV. CONCLUSION

The middle section of the LM experiences high WSS during much of the cardiac cycle. This implies that there is low probability of atherogenesis midway between the LM ostium and the bifurcation. This suggestion is in agreement with the clinical observations in [5]. The WSS distribution in the main bifurcation of the left coronary artery shows the same distribution characteristics in the results as in the investigations of carotid bifurcation [2], so one would expect the flow divider region of the coronary bifurcation to be spared of early atheroma. The myocardial side of the bifurcation region also exhibits low and oscillating WSS because of the inner curvature of the coronary artery. This region may imply higher probability of atherogenesis, a suggestion that is also in agreement with clinical observations in [6].

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