COMPUTER SIMULATION OF WALL THICKENING IN NON-PLANAR MULTIPLE BEND ARTERY INDUCED BY LOW WALL SHEAR STRESS

Shigeo Wada, Ken-ichi Tsubota, Takami Yamaguchi

Department of Mechatronics and Precision Engineering
Graduate School of Engineering
Tohoku University
Sendai, Miyagi
Japan

INTRODUCTION

It is suspected that hemodynamic factors are involved in the development of vascular diseases such as intimal hyperplasia and atherosclerosis, leading to their localization in arterial system[1]. Although wall shear stress must be an important factor among them, its involvement has been investigated by comparing the theoretical and experimental flow analyses with the observation of localized atherosclerosis which has already occurred in the artery. Since changes in the geometry of blood vessel by atherosclerotic thickening of the wall affect the blood flow through the vessel, it is necessary to follow up the progression of the disease induced by hemodynamic factors in order to determine the driving factor. Computer aided analysis allows us to predict the progression of wall thickening leading to atherosclerosis based on various hypothesis. However, few studies [2] have been carried out to account for the change in geometry of blood vessel with the progression of wall thickening. In this study, we conducted a computer simulation of an adaptive change induced by wall shear stress in the thickness of the wall of a human coronary artery with non-planar multiple bend.

METHOD

Initial Geometry of the Blood Vessel

The outline of a blood vessel with a multiple bend was obtained from a transparent human right coronary artery [3], neglecting the branches stemming from it. It was assumed that the thickness of the vessel wall was uniform everywhere in the initial state, and then the boundary of the inside wall was determined from that of the outside wall. The diameter of the vessel was 4.33 mm at the entrance of the arterial segment, and the length along the central axis was 28 mm. As the first step, an artery model was constructed by assuming that the vessel was symmetrical with respect to its common median plane of multiple bend, and the cross-sections were circular at any location. Next step, a non-planer model was obtained by converting the common median plane to a curved surface along the heart wall with a curvature radius of 40 mm (Figure 1). A hexahedral element with 8 nodes was used for CFD analysis. The vessel was divided into 150 and 80 equal-sized elements in the longitudinal and circumferential directions, respectively, and 41 elements of gradually diminishing size in the radial direction.

Blood Flow Analysis

Under the assumptions that arterial wall is rigid and blood is an incompressible Newtonian fluid with a density of 1.05×10^{3} kg/m^{3} and a viscosity of 3.5×10^{-3} Pa·s, the continuity and Navier-Stokes equations for steady flow were solved by the use of a flow simulation software (Star LT 2001 distributed by CD-adapco JAPAN Co., LTD). Boundary conditions applied were a parabolic velocity profile at the inlet, a uniform pressure at the outlet, and non-slip condition at the vessel wall.

Thickening of the Vessel Wall

Based on the fact that atherosclerosis likely occurs at a low wall shear stress region in the artery [1], it was assumed that the vessel wall

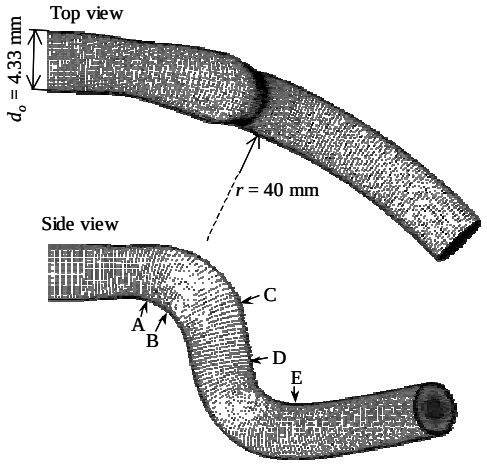

Figure 1. CFD model of a human coronary artery with non-planar multiple bend

thickens inward at the location where wall shear stress is less than a threshold value, τ_{th}. In this study, the amount of thickening during a certain period which corresponds to a computational step is given by

$$\delta = \begin{cases} C\left(\tau_{th} - \tau\right)/\tau_{th} & \text{for } \tau \le \tau_{th} \\ 0 & \text{for } \tau > \tau_{th} \end{cases} \quad (1)$$

where τ is the wall shear stress calculated from the flow velocity in the artery, and C is a coefficient.

Procedure of Computer Simulation

Computer simulations were conducted by repeating calculation of blood flow, evaluation of wall shear stress, and change in the geometry of the vessel by thickening of the wall. A new geometry of the vessel at each step was obtained by moving the nodal points on the wall by δ in Eq. (1) in the direction normal to the lumen. The solution domain was re-meshed for the new geometry. This process was repeated until a stable geometry of the vessel was obtained. The values of τ_{th} and C chosen were 1.2 Pa [4] and 100 μm, respectively.

RESULTS AND DISCUSSION

Calculations were carried out for blood flowing at a physiological flow rate of 2.83×10^3 mm^3/s in the human coronary artery. The Reynolds number defined at the entrance was 250. A stable geometry of the vessel was obtained in 40 steps. Figure 2 shows the contour plot of axial velocity (upper panel) and vector plot of secondary flow (lower panel) in the initial geometry. The location of the cross-section was shown in Fig. 1. The outward lines drawn outside of the vessel in the upper panel indicate the magnitude of wall shear stress at that location. The combination of multiple bends and non-planar geometry of the blood vessel causes complex secondary flow, creating the sites of low wall shear stress which is less than the threshold value at the outer and inner walls of the first and last half of each bend, respectively. It is noted that the low wall shear stress sites are not confined at the inner curved wall along the heart wall.

Figure 3 shows the progress in thickening of the wall from the initial state to the final stable state. It is found that wall thickness increases nonlinearly with computational steps, indicating that change in geometry affects the new development of the wall thickening. Owing to the non-planar geometry of the vessel, the sites of wall thickening are shifted to inner curved wall (lower wall of B) and outer curved wall (upper wall of B and lower wall of D). However, the wall is most thickened around the common median plane at the inner wall of the last half of the first bend (C), where the wall is hogged under the influence of a strong secondary flow.

Figure 4 shows the relationship between the wall shear stress in the initial state and the thickness of the wall in the final state at various locations in the artery. It is found that thickening occurs even at the sites where wall shear stress is larger than the threshold value in the initial state because change in the geometry of blood vessel creates a new site of low wall shear stress. There is a tendency that the wall thickens at the site of low wall shear stress in the initial state. However, lower wall shear stress in the initial state does not always generate thicker wall. This indicates that the final thickness of the wall is determined by not only the value of wall shear stress in the initial state but also by the flow patterns affected by the change in geometry of the blood vessel.

ACKNOWLEDGEMENTS

We would like to thank Prof. T. Karino of RIES, Hokkaido Univ. for his encouragement and support. This work was supported by Research and Development for Applying Advanced Computational Science and Technology from Japan Science and Technology.

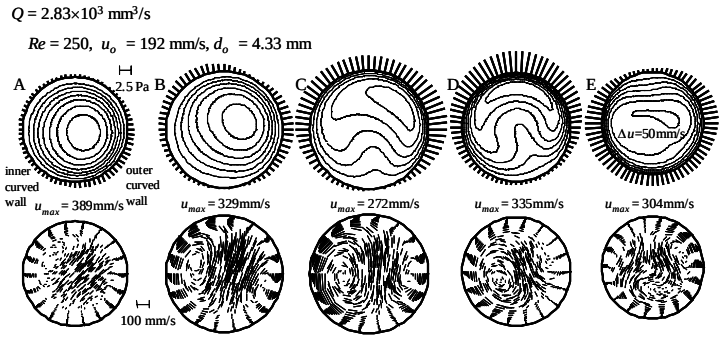

Figure 2. Contour plot of axial velocity (upper) and vector plot of secondary flow (lower) in the initial state

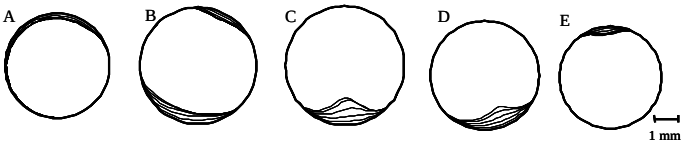

Figure 3. Outline of the inside wall at the initial, 2nd, 5th, 10th, 20th and final step

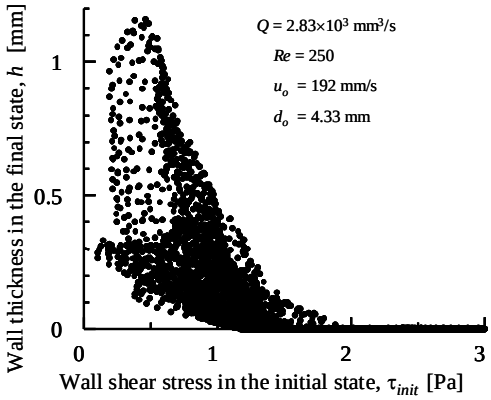

Figure 4. Relationship between wall shear stress in the initial state and the wall thickness in the final sate

REFERENCES

1. Zarins, C. K. et al, 1983, "Carotid Bifurcation Atherosclerosis. Quantitative Correlation of Plaque Localization with Flow Velocity Profiles and Wall Shear Stress," Circ. Res. 53, pp. 502-514.
2. Lee, D. and Chiu, J. J., 1992, "A Numerical Simulation of Intimal Thickening under Shear in Arteries," Biorheology 29, pp. 337-351.
3. Asakura, T. and Karino, T., 1990, "Flow Patterns and Spatial Distribution of Atherosclerotic Lesions in Human Coronary Arteries," Circ. Res. 66, pp. 1045-1066.
4. Kamiya, A. and Togawa, T., 1980, "Adaptive Regulation of Wall Shear Stress to Flow Change in the Canine Carotid Artery," Am. J. Physiol. 239, pp. H14-H21.