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Flow and Deformation in Externally Pressurized Stenosis Model of Arterial Disease

S. Kobayashi[1], J. Ji[1], H. Morikawa[1], D. Tang[2], D. N. Ku[3]

Summary

The increase of the blood velocity in the distal side of the stenosis causes negative transmural pressure, and atheroscrerotic plaque is compressed, furthermore, the stenosis may cause the collpase which leads the rupture of the plaque. The resultant compression may be important in the development of atherosclerotic plaque fracture and subsequent thrombosis or distal embolization. We have developed stenosis models made of polyvinyl alcohol hydrogel, which closely approximate an arterial disease situation, and performed pulsatile flow experiments. Valsalva's maneuver and cough cause a sharp rise in jugular venous pressure to greater than 50 mmHg. Such transient pressure increases within the carotid sheath may augment the external pressure around the carotid artery. We applied external pressure to a stenosis model and discussed the influences of external pressure on steady and pulsatile flow and deformation in the stenosis model.

Introduction

At increase in blood velocity on the distal side of stenosis causes a negative transmural pressure, and atherosclerotic plaque is compressed; furthermore, the stenosis may cause the collapse of artery which leads to the rupture of the plaque [1]. We have developed experimental stenosis models that closely approximate an arterial disease situation, and the characteristics of flow and deformation related to the changing geometry of stenosis and the existence of a lipid core were discussed [2-4].

Valsalva's maneuver and cough cause a sharp rise in jugular venous pressure to greater than 50 mmHg. Such transient pressure increases within the carotid sheath may augment the external pressure around the carotid artery [5]. This study dealt with the influences of external pressure on steady and pulsatile flows and deformation in a stenosis model.

Model and Methods

During the experiment, polyvinyl alcohol hydrogel shaped into the stenosis form was used in a model for a diseased carotid artery. This model is opaque, but its mechanical properties are similar to those of the carotid artery [4]. This model

[1]Department of Functional Machinery and Mechanics, Faculty of Textile Science and Technology, Shinshu University, Tokida, Ueda, Nagano 386–8567, Japan

[2]Mathematical Sciences Department, Worcester Polytechnic Institute, Worcester, MA 01609, USA

[3]School of Mechanical Engineering, Georgia Institute of Technology, Atlanta, GA 30332–0363, USA

can change the degree of stenosis severity and the shape, and the shape of stenosis model is similar to that of a sinusoidal curve. Figure 1 shows the shape of stenosis. The nominal stenosis severity and eccentricity of the stenosis model are given as follows.

Nominal stenosis severity

$$St = (1 - Ds/D) \times 100[\%] \tag{1}$$

Eccentricity

$$Ec = e/((D - Ds)/2) \times 100[\%] \tag{2}$$

We have used a stenosis model of St=70% and Ec=100%. The stenosis model was stretched to 36.5% from its initial length during the experiment.

Figure 2 shows the experimental setup. The hydrogel stenosis model was fixed on rigid pipes and placed in a sealed water tank. The upstream constant head reservoir was pressurized as the direct current component the flow. The computer-controlled gear pump connected to the upstream tube served as the alternative current component for the pulsatile flow. The working fluid used was water. As the input signal for controlling the gear pump, we used the blood flow waveform in the carotid artery [6]. For the measurement of the deformation of the stenosis model, we measured the outer diameter of the stenosis model using a digital video camera.

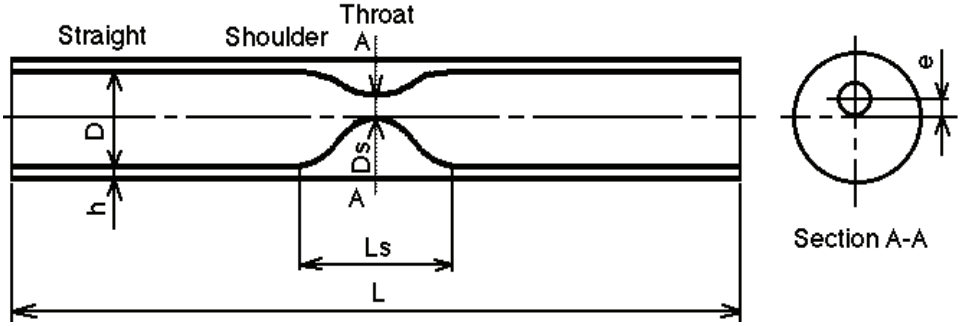


Figure 1: Schematic representation of the stenosis model. (L=110 mm, Ls=4 mm, D=4 mm, h=1 mm)

External pressure was changed by adjusting the height of the head tank and measured using a pressure transducer. Computer-controlled electromagnetic valves were placed between the head tank and the sealed water tank. Another computer-controlled electromagnetic valve was placed between the downstream constant head reservoir and the sealed water tank to release the external pressure. The opening and closing times of the electromagnetic valves were changed using a computer

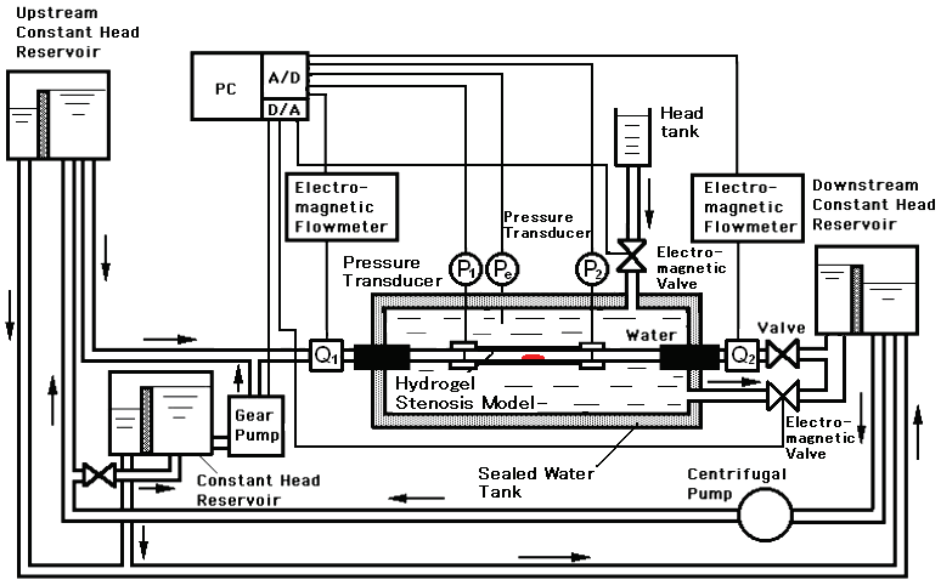


Figure 2: Experimental setup

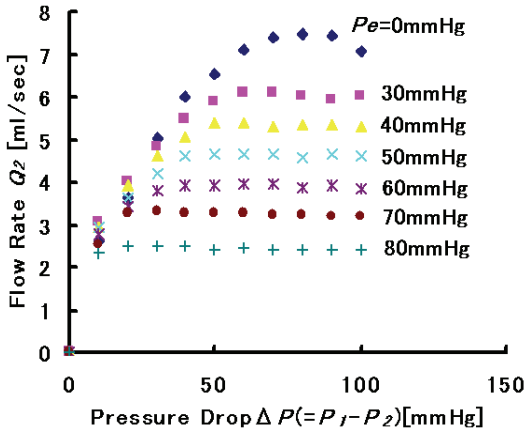
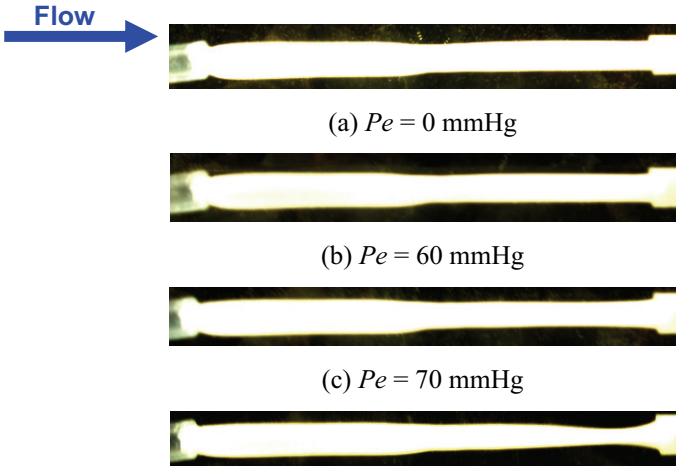


Figure 3: Flow rate versus pressure drop (steady flow). P_1, P_2: upstream and downstream pressures, Q_2: downstream flow rate, P_e: external pressure. P_1=100 mmHg

program. In this experiment, the external pressure can be increased in 0.5 s and released in approximately 1.5 s. The frequency of pulsatile flow was 1.0 Hz.

Results and Discussion

Figures 3 and 4 show the flow versus pressure drop and photographs of stenosis model in steady flow condition. The flow rate decrease with increase in external pressure. Flow choking and the collapse of stenosis occurred when the external



(a) Pe = 0 mmHg

(b) Pe = 60 mmHg

(c) Pe = 70 mmHg

(d) Pe = 80 mmHg

Figure 4: Photographs of stenosis model (steady flow). P_e: external pressure, upstream pressure P_1= 100 mmHg

pressure exceed the downstream pressure.

Figure 5 shows the pressures and flow rates in four cycles for the pulsatile flow condition (average upstream pressure P_{1avg} before pressurization = 95 mmHg, average downstream pressure P_{2avg} before pressurization = 50 mmHg). In this figure, the phase of the maximum external pressure was equal to the phase of the minimum upstream pressure. After pressurization, the downstream pressures and flow rates decreased, and self-excitation of downstream pressure appeared during external pressure was greater than or equal to downstream pressure. As the maximum external pressure increases, the downstream pressure and flow rate decrease.

The outer diameters of the stenosis model at shoulder A, the throat, and shoulder B were measured in the pulsatile flow condition, and the results are shown in Fig 5 (P_{1avg} before pressurization = 95 mmHg, P_{2avg} before pressurization = 50 mmHg). By external pressurization, the outer diameters at the throat and shoulder B were more reduced than that at shoulder A. This indicates that the throat and shoulder B of the stenosis model were more compressed and collapsed. An obvious vibration of the stenosis model induced by the self-excitation of the downstream pressure was not observed. However, there may be a small vibration of the lumenal wall of downstream stenosis. This may affect the stability of atherosclerotic plaque.

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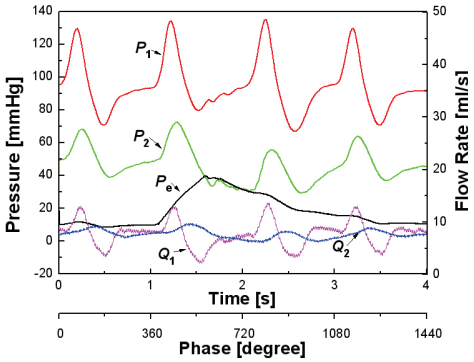
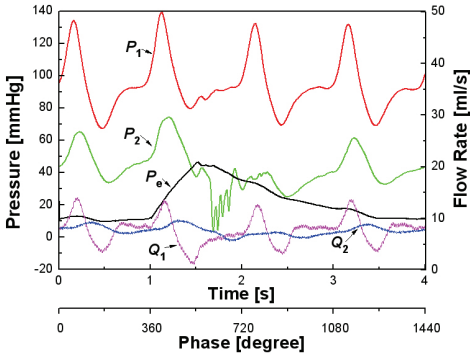
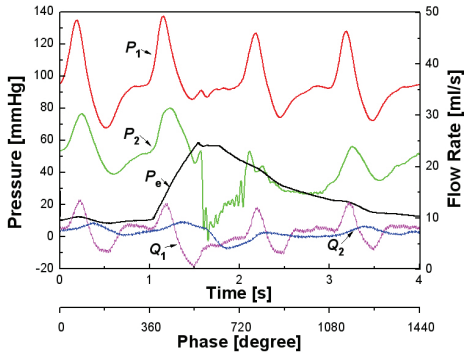


(a) $P_{ema\,x}$= 40mmHg

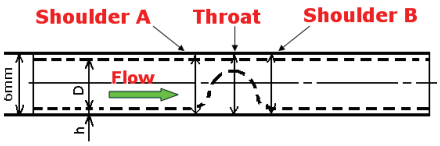


(b) P_{emax}= 50mmHg



(c) P_{emax}= 60mmHg

Figure 5: Time-varying pressures and flow rates (pulsatile flow). P_1, P_2: upstream and downstream pressures, Q_1, Q_2: upstream and downstream flow rates, *Pe*: external pressure



Measured position

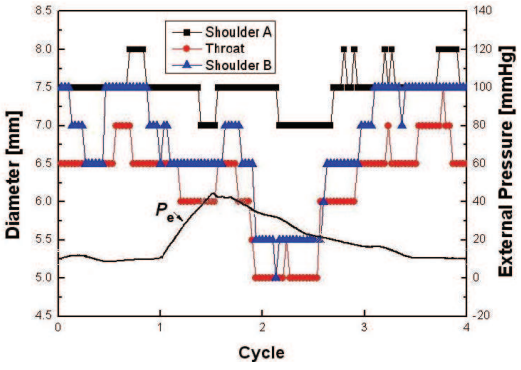


Figure 6: Change in outer diameter (pulsatile flow)

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