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Stress Analysis in Layered Aortic Arch model: Influence of Arch Aneurysm and Wall Stiffness

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Summary

Patients with aortic aneurysm, especially aortic arch aneurysm, are prone to aortic dissection. For investigation of the effects of aneurysm and wall stiffness on wall stress distribution, a nonaneurysm arch model as well as an aneurysm arch model was constructed. The fluid structure interaction was implemented in the arch model of aorta. The results show that the stresses are much higher at inflection points in the aneurysm model than in nonaneurysm model, and the stresses at media in stiffened wall are higher than in unstiffened wall. The high composite stress is located at inflection points and is much higher in the aneurysm model. The arch aneurysm and wall stiffening are important determinants of peak wall stress in aortic wall.

Introduction

Cardiovascular disease is the No. 1 killer in developed countries, and is responsible for millions of deaths and disabilities every year. Aortic aneurysm is a degenerative process whose ultimate event is the rupture of the aorta. Aortic dissection is the commonest catastrophe affecting the aorta, occurring more frequently than rupture [1]. A classic aortic dissection begins with a laceration of the aortic intima and inner layer of the aortic media, forming an entrance tear, which allows entering blood to split the aortic media. Many aortic dissection cases with aortic aneurysm were found throughout the literature [2, 3, 4]. In Mehta's [3] paper, of 383 patients with acute type B dissection, 18.2% had prior aortic aneurysm. Among the 83 patients of aortic arch aneurysm in Kitamura's [4] paper, 51 cases had aortic dissection. Patients with aortic aneurysm, especially aortic arch aneurysm [4], are prone to aortic dissection.

From the mechanical point of view, aortic wall ruptures if the stresses acting on the wall rise above the ultimate value for the wall tissue. Aortic wall stress is the outcome of several factors, such as characterization of wall material, the shape of the aorta, and the dynamic interaction of the wall with blood flow. Since the internal mechanical forces are maintained by the dynamic action of blood flowing in the aorta, the quantification of the hemodynamics is essential for the characterization

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of the biomechanical environment. In our prior studies, the simulation has been done on layered aortic arch model [5]. In the present study, our objective was to examine the role of aortic arch aneurysm, as well as wall stiffening, on wall stress distribution. The fully-coupled FSI methodology is utilized to analyze the distribution of stress on the aortic wall with arch aneurysm. The effects of aneurysm and wall stiffness on the mechanical stresses were determined.

Method

For investigation of the effects of aneurysm on wall stress distribution, the nonaneurysm arch model [5] and the aneurysm arch model were constructed.

The virtual aortic arch aneurysm models were generated with the geometry and mesh generation software Gambit. The models are comprised of a fluid domain representing the aortic lumen, and a solid domain representing the aortic wall. The fluid domain is characterized by circular cross sections with an undilated diameter, d=2cm, and a maximum diameter, D=1.5d, at the midsection of the arch aneurysm sac. Aortic aneurysm was defined as a diameter greater than or equal to 1.5 times the average aortic diameter. Important locations on the wall of arch aneurysm are the inflection points on the wall where the surface of aneurysm changes from concave outward to concave inward. The aortic wall is assumed to be an isotropic, linear, elastic solid with a density p=1050kg/m3 and Poisson's ratio v=0.45. The mean Young's modulus is set E=840kPa.

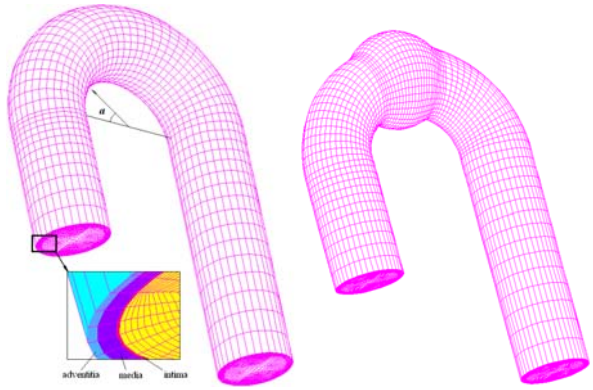

Figure 1: Meshes for aortic arch nonaneurysm model and arch aneurysm model.

For investigation of the effect of wall stiffness on wall stress distribution in aortic aneurysm, Young's modulus of the aortic wall was changed from a healthy value to a stiffer value. The Young's modulus at inflection points was nearly two times higher than that in un-dilated points, and the Young's modulus at mid-sac was a little higher than that at inflection points [6]. The medial stiffness accounts for arterial stiffening. In this study, the medial Young's modulus at aneurysm wall was changed to 2.7MPa [7], at inflection point it was changed to 2.6MPa, and at

un-dilated portion it was changed to 1.3MPa.

Results

Figure 2 shows a comparison of the stresses in nonaneurysm model and aneurysm model, for both circumferential and longitudinal stress. For arch aneurysm model, the high stresses were located proximally and distally to the aneurysm sac (inflection points). Figure 3 shows composite stress (composition of the circumferential stress vector and the longitudinal stress vector) in aneurysm model, compared with nonaneurysm model. High composite stress is located at inflection points, and is much higher in the aneurysm model.

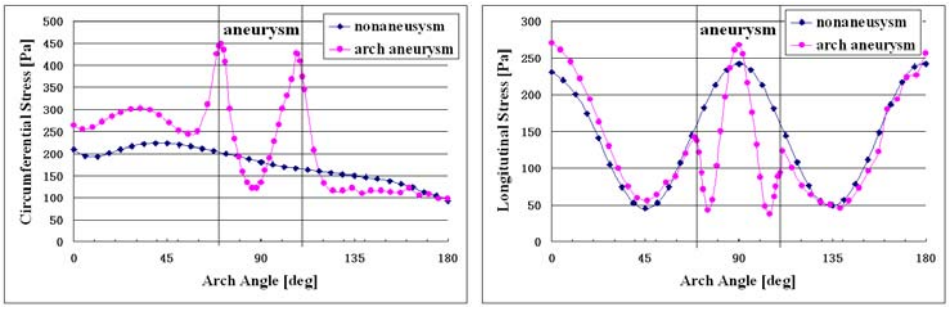

Figure 2: Comparisons of wall stress distribution in nonaneurysm model and aneurysm model, for circumferential stress and longitudinal stress.

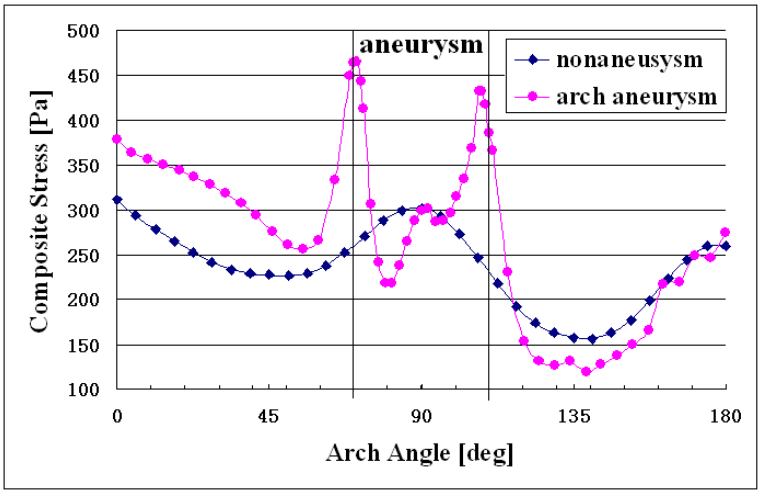

Figure 3: Comparison of wall stress distribution in nonaneurysm model and aneurysm model for composite stress.

Figure 4 shows comparison of the stresses in unstiffened model and stiffened model for circumferential stress and longitudinal stress. At the inflection points, the circumferential and longitudinal stress are much higher in the stiffened model

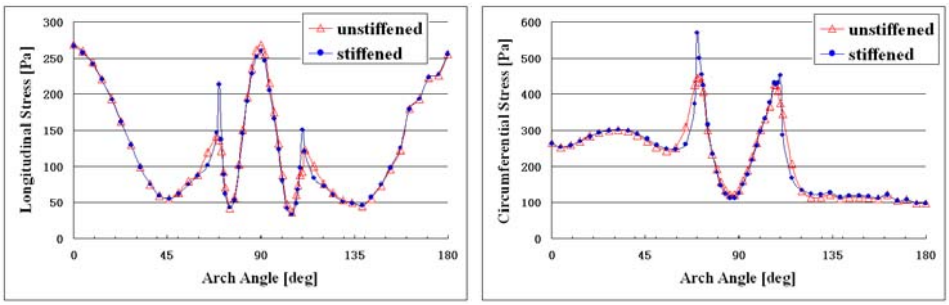

Figure 4: Comparisons of wall stress distribution in unstiffened aneurysm model and stiffened aneurysm model.

than in the unstiffened model.

Four positions were selected to describe the effects of aneurysm and wall stiffness on wall stress across wall thickness. Figure 5 shows the variation of stresses across wall at section A (ascending portion), section B (proximal position to the aneurysm sac), section C (mid aneurysm sac), and section D (distal position to the aneurysm sac). Section A is undilated portion for aneurysm model. Sections B, C, and D in nonaneurysm model are positions with an arch angle equal to that in the aneurysm model. At ascending portion, proximal position to the aneurysm sac, and distal position to the aneurysm sac (A, B, and D), the stresses are higher in aneurysm model than in nonaneurysm model, and the stresses at media in stiffened wall are a little higher than in unstiffened wall. At inflection points (B and D), the stresses reach a peak value at media near adventitia and wall stiffness increases the peak value. At Section C, the variations of stresses are similar in nonaneurysm model, unstiffened aneurysm model, and stiffened aneurysm model.

Discussion and Conclusion

This study shows the stress analysis of aortic arch aneurysm models to investigate the effects of aneurysm and wall stiffness on wall mechanical stresses. The wall stresses on the aneurysm model were indicated to be distributed in a complex ways with large regional variations at inflection points. The wall stresses on the nonaneurysm model were relatively low and uniformly distributed. The previous studies found that the failure strength of the aneurysm wall was lower than that of the nonaneurysm wall [8], and the peak wall stress on aneurysm was from 45% to 69% of its failure strength, whereas the peak wall stress of nonaneurysm aorta was less than 10% of its failure strength [9]. The maximum stresses occur at the proximal and distal ends of an aneurysm. This result suggests that maximum stresses do not occur at the location of the maximum diameter, but at the regions of high curvature associated with the proximal and distal ends. This agrees with previous studies [7, 8, 9]. Most aneurismal ruptures occur at the posterolateral wall, which

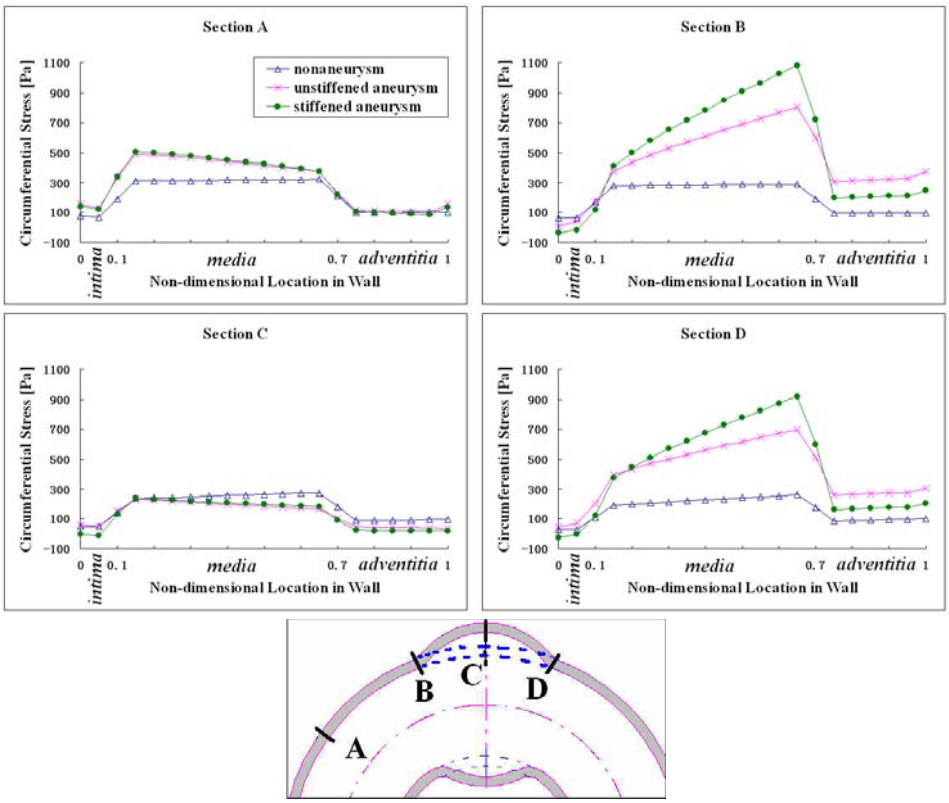

Figure 5: Comparisons of wall stress distribution across wall at sections A, B, C, and D for nonaneurysm wall, unstiffened aneurysm wall, and stiffened aneurysm wall.

correlates to the area of high stress concentration [8, 9]. Also, the actual propensity for rupture for other surfaces of an aneurysm depends on the comparative local value of wall strength, since it has been found that the strength of tissue near the neck or undilated ends of an aneurysm is greater than that in the midsection, where diameter is maximum.

As observed clinically, many aortic dissection cases with aortic aneurysm were found in the literature [2, 3, 4], typically patients with aortic arch aneurysm. Our results indicate the aortic arch aneurysm increases stresses on aortic wall, and the high stress concentrations are correlated with aortic tear and dissection. Thus, treatment of aortic aneurysm may have an important role in decreasing the risk of aortic dissection.

The arterial stiffness was significantly higher in aneurysm wall compared with healthy aortic wall [7, 10]. It has been suggested that aneurysms develop as a result of an alteration in the connective tissue metabolism, and that this might change

arterial wall stiffness [10]. Our results indicated that wall stiffness in aneurysms increases maximum stresses at inflection points. It has been reported that aortic dissection originated in a distal aortic arch aneurysm [11]. Combining the effects of aneurysm and wall stiffness, it is possible that the patients with aortic aneurysm, especially aortic arch aneurysm [4], are prone to aortic dissection. In this study, stresses are found to be higher in the media, and reach a peak value in the media near the adventitia at inflection points, and the medial stiffening increased the stress in media and the peak value of the stress.

The aortic arch aneurysm and wall stiffening have a direct impact on the mechanical stresses acting on the aortic wall. The wall stiffening, in addition to aneurysm, is an important determinant of peak wall stress in aortic wall. This information may be important in understanding the natural history of aneurysm and aortic dissection, and in clinical management of these diseases.

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