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VORTEX DYNAMICS IN THORACIC AORTIC ANEURYSMS

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Abstract. As described in this paper, a numerical simulation of blood flow in the thoracic aorta is presented. Patient-specific aorta shapes are used in a centerline-fitted generalized coordinate system in which the Navier–Stokes equation is discretized using finite-difference approximation. The main target of this study is long-term adverse events that occur after endovascular stent-graft treatment. Thoracic endovascular aortic repair (TEVAR), or stent-graft treatment, has become widely accepted as an important option for treatment of thoracic aortic diseases. Many studies have proven the safety and efficacy of TEVAR with satisfactory short-term to mid-term outcomes. Nevertheless, even if the initial TEVAR treatment technically succeeds, some patients show recurrence and progression of diseases many years after treatment. Based on long-term follow-up examinations, such long-term morphological change and effects of hemodynamic flow apparently interact synergically. This study is intended to investigate the constant effect of vascular hemodynamics on the long-term adverse events using computational fluid dynamics based on finite-difference approximation with an immersed boundary/fictitious domain approach. Some cases with aneurysms or strongly tortuous vessels show swirling flows that remain even at the late diastole. Streamwise vortex cores, which are initially generated at the systole of the cardiac cycle, merge to form a single vortex core. This phenomenon apparently affects the long-term morphological change of aorta and long-term adverse events.

1 INTRODUCTION

Thoracic endovascular aortic repair (TEVAR), or stent–graft treatment, has become widely accepted as an important option for treatment of thoracic aortic diseases. Many studies have proven the safety and efficacy of TEVAR with satisfactory short-term to mid-term outcomes. Even if the initial TEVAR treatment technically succeeds, some patients demonstrate recurrence and progression of diseases many years after treatment.

Based on long-term follow-up examinations, such long-term morphological change and effects of hemodynamic flow seem to interact synergically. Constant pulsatile hemodynamic effects from blood flow seem to induce degeneration of the underlying aorta to induce its morphological change and induce minor morphological change to alter the hemodynamic state. These changes eventually engender long-term adverse events. This study investigates the constant effect of vascular hemodynamics on longterm adverse events using computational fluid dynamics.

2 NUMERICAL METHODS

Navier–Stokes equations for an incompressible viscous fluid with a continuity equation are used, where blood is assumed to be a non-Newtonian fluid. In this study, flow equations are discretized using finite difference approximations. Finite difference using meshes are generated centerline data detected from CT scans using the median axis transform technique [3]. Then the shapes of aortic walls are reconstructed in the centerlinefitted generalized coordinates (ξ, η, ζ) using radius data at respective points on the centerline. Figure shows 1 centerlines and the finitemesh difference generated around them, one of which coordinate axes are nearly centerlines. parallel The to shapes of aorta are represented by characteristic functions λ



Fig. 1: Centerlines and finite-difference meshes



Fig. 2: Contour surfaces of the characteristic functions

 (ξ, η, ζ) , which are used in the fictitious domain method. Figure 2 depicts finite difference meshes and contour surfaces of λ . The combination of centerline-fitted mesh and fictitious domain method is expected to realize higher accuracy with reasonable computational cost. A collocate arrangement of flow variables on the generalized coordinate has been adopted. For approximation of the advection term, a third-order upwind scheme is used. A Poisson equation for the pressure is solved using GP-BiCG method with incomplete LU factorization as a preconditioner.

3 NUMERICAL RESULTS

Flow fields over five cardiac cycles are computed and the time-averaged stress on the aortic wall. Then the following two quantities are computed to assess hemodynamic effects on the long-term deformation of the aorta.

$$E(s) = \int_{\Gamma(s)} \sigma_n d\Gamma$$
$$B(s) = \int_{\Gamma(s)} \sigma_n n d\Gamma$$

Therein, σ_n signifies the time-averaged outward normal component of the stress exerted on the aortic wall. Parameter *s* stands for the length along the centerline, and $\Gamma(s)$ represents the cross-section perpendicular to the centerline at *s*. Outward normal vector *n* can be computed as a gradient of the characteristic function $n = \text{grad } \lambda$. Figure 3 shows a 1D representation of these forces, in which the colors of the centerline represent E(s), whereas the arrows on them indicate B(s) for different cases.



Fig. 3: 1D representations of force components

Large differences in force distribution are apparent. One reason for the differences in Fig. 3 might be the swirling flow in the diastole period shown in Fig. 4.



Fig. 4: Swirling flow in an aneurysm

To elucidate why such a swirling flow is generated, we compute the streamwise component of the vorticity. Figure 5 portrays contour surfaces of streamwise vorticity at the four instances of cardiac cycle. In this figure, streamwise vortex cores, which are generated initially at the systole of the cardiac cycle, merge to form a single vortex core.

Lee et al. [5] extensively investigated steady flows in nonplanar double bends. Our results for unsteady flows in realistic aorta morphology reveal similar characteristics to those reported from that study [5]. This phenomenon apparently affects the long-term morphological change of the aorta and long-term adverse events.



Fig. 5: Contour surfaces of streamwise vorticity

4 CONCLUSIONS

Our numerical results demonstrate that a swirling flow persists even in the diastole period, especially in an aorta with large torsion. These swirling flows are explainable as a flow in a nonplanar bend. This remaining vortex apparently accounts for the increase of the total wall stress, which is responsible for aortal deformation in long-term adverse events.

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