# FLUID-STRUCTURE INTERACTION AND STRUCTURAL ANALYSES OF AN ANEURYSM MODEL

**Ender A. Finol (1), Elena S. Di Martino (2), David A. Vorp (2,3), Cristina H. Amon (1,4)**

(1) Institute for Complex Engineered Systems, Biomedical Engineering Carnegie Mellon University

(2) Surgery University of Pittsburgh Pittsburgh, PA

(3) Bioengineering University of Pittsburgh Pittsburgh, PA

(4) Mechanical Engineering Carnegie Mellon University Pittsburgh, PA

## INTRODUCTION

Abdominal aortic aneurysm (AAA) is a degenerative process whose ultimate event is the rupture of the aorta. In the lack of a reliable prognosis for aneurysms, the advantages of an early surgical procedure must be weighted against the risks of the procedure itself. Accurate prediction of AAA rupture is critical for patient management. We believe that geometric parameters alone are not sufficient to discriminate between "safe" and "at risk" aneurysms. In the last two decades computational models have been applied to study AAA biomechanics. The abdominal aorta in the presence of an aneurysm is a multi-physics system. The pressure due to the blood flowing in the vessel lumen produces a deformation of the complex structure composed of the wall and the intraluminal thrombus (ILT), which in turn alters the flow dynamics in the lumen. The elements required to build a biomechanical model of the aneurysm are the geometry, the constitutive equations for the solid and fluid domains and the boundary conditions, including constraints to eliminate rigid body motion in the segment of the vessel under study and the time-varying pressure and velocity conditions of the fluid. Many simplifications have been adopted to solve this complex problem. The internal mechanical forces that cause wall stress are initiated and maintained by the dynamic action of blood flow within the aneurysm. If we neglect the fluid domain, the stresses in the aneurysm wall can be studied applying a uniform pressure to the internal lumen of the vessel.

We have recently reported pulsatile blood flow patterns and shear stresses in a patient-specific AAA model [1] and performed a comparison between its hemodynamic pressure and wall stress distribution using a de-coupled fluid and structural analysis approach [2]. Additionally, we predicted for two patient-specific AAA models that hemodynamic pressure does not vary significantly within the aneurysm sac at any particular time stage of the flow [3]. The objective of this work is the comparison between time-dependent fluid-structure interaction (FSI) simulation and structural static simulation utilizing the same aneurysm model, to establish a practical method for the accurate calculation of AAA wall stresses, which are responsible for the rupture of the aneurismal sac.

## METHODS

A three-dimensional (3D) asymmetric aneurysm model was constructed based on a previously used geometry [4], but with geometric dimensions (maximum transverse diameter, proximal neck diameter and length) that are commonly found in a human abdominal aorta in the presence of an aneurysm. A layer of laminated thrombus was also modeled, partially occluding the lumen of the artery. The dimensions of the model aneurysm, illustrated in Fig. 1(a) are: inlet diameter, d = 2.0 cm; maximum aneurysm diameter, D = 6.0 cm; total length of the aortic segment, L = 18.0 cm; wall thickness, t = 2.0 cm.

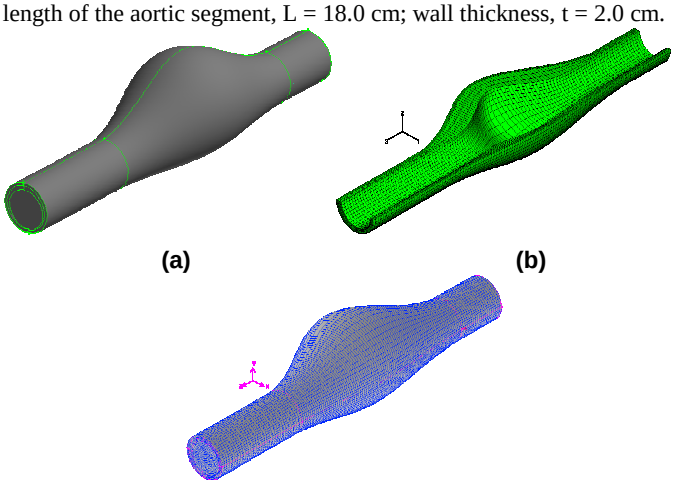


**Figure 1. Aneurysm model: (a) solid form representation; (b) 3D axisymmetric computational grid utilized for the structural analyses; (c) 3D computational grid utilized for fluid-structure interaction (FSI) simulation.**

The computational domain generated for the structural static analyses consists of 25,323 hexahedral elements for a total of 20,800 nodes. For the FSI analysis, the computational domain is composed of

122,470 hexahedral elements that provide 100,956 nodes. The finite-element method (FEM) was utilized for all the computational studies reported in this work. Extensive mesh-independency analyses were performed as part of the structural simulations, which are supported by previous work [5].

For FSI, incompressible, homogeneous, Newtonian flow is simulated for average resting conditions (heart rate = 60 bpm). Average blood properties are considered: molecular viscosity $\mu$ = 0.00319 Pa·s and density $\rho$ = 1,050 kg/m$^3$. The boundary conditions are imposed as follows: (i) parabolic velocity profile at the inlet, and (ii) uniform systemic pressure at the outlet. For pulsatile flow, the velocity and pressure are time-dependent and the volume flow rate is oscillatory, as described in [1]. The pulsatile waveforms are represented by discrete Fourier series based on the in-vivo measurements reported first by Mills et al. (referenced in [2]), which are triphasic pulses appropriate for average resting hemodynamic conditions in the abdominal segment of the human aorta. The wall and thrombus material are modeled as linearly elastic and nearly incompressible (Poisson's ratio, $\nu$ = 0.45) with Young's moduli of elasticity, E, equal to 5.0 MPa and 2.5 MPa, respectively [6].

For static stress simulations, the upper and lower surfaces of the wall and thrombus are constrained on the x-, y- and z- directions. A homogeneous pressure of 15.6 kPa (equivalent to 116.8 mmHg) is applied to the luminal surface. This value represents the peak systolic pressure of the outlet pressure waveform imposed for the FSI analysis.

## RESULTS AND DISCUSSION

Figures 2(a) and (b) depict the wall stresses obtained from static simulations of the peak systolic pressure. Non-linear material properties are used for the wall in Fig. 2(a) [4] and linear properties in Fig. 2(b). The maximum wall stress is dependent on the material characteristics and the geometry of the aneurysm. The pressure acting on the AAA inner wall determines the stresses on the wall itself. The simplification of assuming linearly elastic material properties for the wall results in an underestimation of the maximum wall stress.

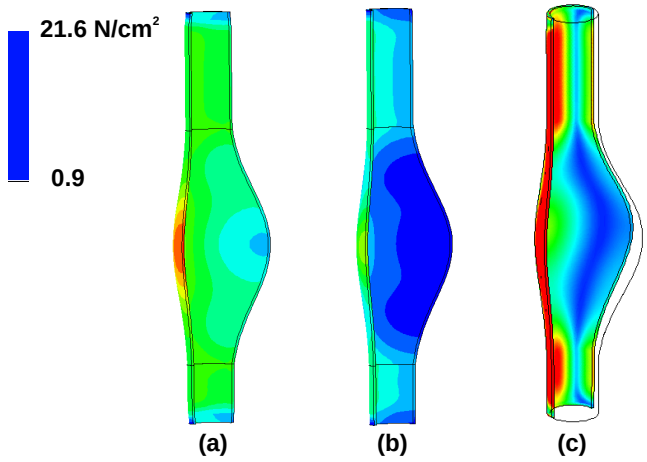


**Figure 2. Von Mises stress distributions at peak systolic pressure: (a) static structural simulation, hyperelastic constitutive model; (b) static structural simulation, linearly elastic constitutive model; (c) FSI simulation. In each frame, the half geometry is viewed from the luminal perspective, as only the wall is shown.**

The wall stresses obtained from the FSI simulation at the instant of peak systolic pressure are illustrated in Figure 2(c). During the cardiac cycle, the instantaneous fluid forces acting on the thrombus lining deform the wall. In turn, the wall motion alters the velocity field until equilibrium is reached. This is true for each instant of the cardiac cycle. If one ignores the entrance effects caused by the flow adjusting itself within the inlet and outlet extensions, the location of the maximum wall stress (along the lateral-anterior surface) obtained by FSI corresponds well with the structural analyses.

Based on a comparison of computational times required to perform structure-only and FSI simulations, a structural analysis of AAA wall biomechanics appears to be the most practical approach for the prediction of wall stresses, if there is interest in the peak systolic stresses alone. However, a true FSI simulation allows computation of the flow and pressure fields in the aneurysm, simultaneously with the wall stresses. This provides a mean to validate the computational simulation with clinical diagnostic data, such as Echo Doppler flow visualization. Moreover, a de-coupled approach of fluid and solid dynamics is not representative of the blood and wall interaction, which only a FSI analysis can illustrate. FSI of a patient-specific AAA model has been previously reported by Di Martino et al. [7]. Conversely, to the authors' knowledge, the present work is the first reported study where FSI of an aneurysm model is compared with its structural static analysis.

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