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Biomechanics of Abdominal Aortic Aneurysms: Flow-Induced Wall Stress Distribution

Christine M. Scotti[1], Sergio L. Cornejo[2] and Ender A. Finol[3]

Summary

Abdominal aortic aneurysm (AAA) rupture is believed to represent the culmination of a complex vascular mechanism partially driven by the forces exerted on the arterial wall. In the present investigation, we present fully coupled fluid-structure interaction (FSI) and finite element analysis (FEA) computations of a patient-specific AAA model. This work advances previous FSI AAA modeling by including localized intraluminal thrombus and the comparison of FSI- and FEA-predicted wall stress distributions. The FSI transient fluid and wall dynamics resulted in a maximum wall stress 21% higher than that obtained with FEA, demonstrating the importance of modeling blood flow for the assessment of AAA wall mechanics.

Introduction

Diagnosed AAAs are characterized by their suitability for (surgical or endovascular) repair based on maximum diameter or aneurysm growth rate measured over time. Patients with aneurysms smaller than 4 cm are placed under clinical surveillance while those greater than 5.5 cm (on average) or growing at a rate $\geq$ 0.5 cm/year are recommended for intervention. It is a known fact, however, that basing the clinical management of this disease on growth rate or maximum transverse dimension alone is not an adequate criterion for estimating individual rupture risk. This is evident by the number of small aneurysms that rupture prior to reaching the critical diameter of 5.5 cm and the many more that are diagnosed at an advanced stage of growth having exceeded the cutoff size for intervention and yet did not rupture. This inability to fully assess the individual at-risk status of an AAA has led to extensive research into other potential indicators of rupture or evaluative criteria for assessing the need for repair. Primary among the biomechanical factors linked with AAA rupture is wall stress, frequently quantified either using the Von Mises criterion or the maximum principal component of the stress tensor "mapped" on the diseased arterial wall. Peak intraluminal pressure has been determined as the most important patient-based variable predicting this stress, and thus the great majority of numerical modeling studies of AAA mechanics have focused on quasi-static computational solid stress (CSS) predictions. In such studies the effect of blood flow has been completely ignored on the basis that fluid pressure on the wall of the

[1]Biomedical Engineering Department, Carnegie Mellon University, Pittsburgh, PA 15213, USA

[2]Department of Mechanical Engineering, Universidad de Santiago de Chile, Santiago, Chile

[3]Institute for Complex Engineered Systems and Biomedical Engineering Department, Carnegie Mellon University, Pittsburgh, PA 15213, USA

aneurysm does not change significantly from the proximal neck of the AAA to the iliac bifurcation and, thus, has minimal effect on the wall stress distribution.

Idealized fusiform and saccular models have shown that wall stress increased with bulge diameter and asymmetry [1]. Real AAAs can grow into unique shapes with equally unique lumens. Hence, using maximum diameter can over or underestimate the wall stress. Fillinger *et al.* [2] demonstrated that maximum wall stress correlated more closely with the risk of rupture than maximum diameter. Wall stress was calculated in this study by using FEA applied to patient-specific anatomy obtained from CTs and peak systolic pressure. A similar study was undertaken by Venkatasubramaniam *et al.* [3] with 27 patients, from which 15 AAAs ruptured. They found that ruptured AAAs had significantly higher peak wall stress than non-ruptured AAAs (77 N/cm^2 vs. 55 N/cm^2). Both studies found a strong correlation between areas of high stress and the rupture site, based on quasi-static computational solid stress calculations applying a uniform intraluminal pressure directly on the wall. AAA wall stress is the outcome of several factors, such as characterization of the wall material, the shape and size of the aneurysm sac (geometry), the presence of intraluminal thrombus (ILT), and the dynamic interaction of the wall with blood flow. Di Martino and colleagues [4] first provided the notion of interaction between solid and fluid domains as it contributes to aneurysm rupture potential. More recently, the use of FSI techniques has been applied to patient-specific geometries modeling the wall with linearly elastic properties in the absence of intraluminal thrombus [5], or with a decoupled fluid/solid technique that does not account for the deformation of the fluid domain imposed by the wall motion [6].

In the present investigation, fully coupled FSI computations of a patient-specific AAA model are presented and compared with static and transient computational solid stress (CSS) analyses to identify the effects of fluid flow and assess the significance of patient-specific features on the wall stress and the biomechanical environment of abdominal aortic aneurysms. To the authors' knowledge this work represents the first fully coupled nonlinear FSI model of a patient-specific AAA with localized ILT.

Methods

The computed tomography (CT) scan images of an eighty year old male patient chosen for elective AAA repair were reconstructed based on an automated thresholding technique applied using Mimics (v9.0, Materialise, Ann Arbor, MI). The areas of interest in the abdominal aorta were digitally segmented into two specific sets: the boundary of the arterial wall and the region of blood flow through the arterial segment, known as the lumen. The difference between these two sets was assumed to be ILT, which is attached to the inner surface of the wall. Due to

limitations of CT scan imaging and the segmentation algorithms, the actual thickness of the arterial wall could not be distinguished. Consequently, a uniform wall thickness of 1.5 mm was used in this study, as reported in [1]. Figure 1 illustrates the stages of the CT image reconstruction.

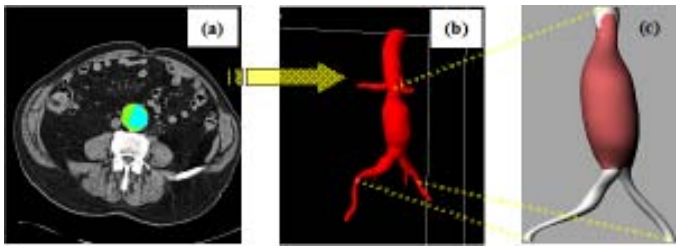

Figure 1: Stages of the geometry reconstruction process: (a) abdominal CT image with colored masks for lumen and ILT; (b) 3D reconstructed and smoothed aortic lumen; (c) computational model of AAA lumen and ILT (in red).

The software Adina (v8.2, ADINA R&D, Inc., Watertown, MA) was utilized for the numerical simulation of FSI between the wall and the lumen, as well as the alternative CSS analyses involving only the aneurysmal wall. The governing equations for blood flow are the Navier-Stokes formulations with the assumptions of laminar, homogenous, incompressible, and Newtonian flow. With a diameter that is greater than 0.5 mm, an assumption of Newtonian flow through the aorta is reasonable due to the fact that blood viscosity is relatively constant at the high rates of shear (100/sec) typically found in the aorta. Blood is modeled to have a density of ρ_f=1.05g/cm^3 and a dynamic viscosity of μ_f=3.5cP. The applied boundary conditions on the fluid domain are (i) a time dependent inlet uniform velocity profile and (ii) a time dependent normal traction (due to luminal pressure) at the distal iliac outlets, as shown in Figure 2. A no-slip boundary condition was applied on the lumen-wall interface for all FSI analyses.

For the CSS analyses, blood flow is disregarded in an attempt to obtain comparatively accurate results by applying a spatially-uniform pressure on the inner wall surface. The CSS$_T$ method (transient CSS) applies the pressure waveform from Figure 2(b) as a transient pressure function (but spatially-uniform) to simulate the effect of luminal pressure acting on the inner wall. Similarly, the CSS$_S$ method (static CSS) applies p(t=0.5) in a quasi-static formulation to model wall mechanics at peak systolic pressure. In these two approaches we utilize only the solid domain with prescribed zero translation at the proximal and distal ends, and with a pressure boundary condition.

The AAA wall is assumed to be a non-linear, isotropic, hyperelastic material with a density ρ_s=1.2g/cm^3, which is represented as a simplified, general Mooney-Rivlin material model for the strain energy density functions $({}^t_0W)$ for the AAA

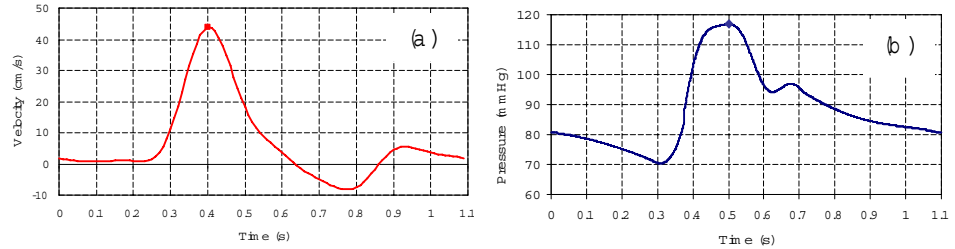

Figure 2: Inlet fluid velocity (a) and outlet pressure (b) waveforms indicating the peak systolic conditions of the cardiac cycle.

wall in Equation (1) and the ILT in Equation (2):

$$ {}^{t}_{0}W = C_1\left({}^{t}_{0}I_1 - 3\right) + C_3\left({}^{t}_{0}I_1 - 3\right)^2 \tag{1} $$

$$ {}^{t}_{0}W = C_2\left({}^{t}_{0}I_2 - 3\right) + C_5\left({}^{t}_{0}I_2 - 3\right)^2 \tag{2} $$

where I_i represents the ith variant of the left Cauchy-Green tensor and C_i are based on the means of the best-fit material parameters of the data examined in [7]. For the FSI analyses, the numerial approach implements an arbitrary Lagrangian-Eulerian (ALE) formulation to account for the movement of the fluid domain and a typical Lagrangian formulation of the solid domain. To accommodate the moving reference velocity, the fluid governing equations are updated as follows:

$$ \rho\frac{\partial\vec{v}}{\partial t} + \rho\left(\left(\vec{v} - \vec{w}\right)\cdot\vec{\nabla}\right)\vec{v} - \vec{\nabla}\cdot\overline{\overline{\tau}} = f^B \tag{3} $$

where the term $\vec{w}$ is used to reflect the reference velocity which in this case is the moving mesh velocity vector. The governing equation for the solid domain is the momentum conservation given by Equation (4):

$$ \nabla\cdot\tau_s + f_s^B = \rho_s\ddot{d}_s \tag{4} $$

where τ_s is the solid stress tensor, $\mathbf{f}_s^B$ are the body forces per unit volume, and $\ddot{d}_s$ is the local acceleration of the solid. The solid and fluid domains are coupled through traction equilibrium and displacement compatibility. A more detailed description of the FSI methodology employed in the present study is described in [8,9]. The mesh size used for the FSI analyses is 221,779 linear tetrahedral elements, of which 145,449 elements correspond to the solid domain. The simulations were run on a Tru64 Unix platform with up to eight 1.15 GHz processors and 8 GB of RAM.

Results and Discussion

Pulsatile blood flow interacting continuously with the compliant AAA wall and ILT appears to influence the wall stress of an AAA. Figure 3 compares the three computational techniques utilized in this study and shows that the maximum wall stress is significantly larger for the FSI technique than the CSS analyses. To quantify wall stress, the Von Mises criterion is considered in this analysis because it represents a material failure criterion, taking into consideration all of the principal stresses within the system. As evident by Figure 3, the flow dynamics within the aneurysm affects the magnitude of the maximum stress rather than its location, with the FSI-predicted maximum wall stress being 35.4 N/cm^2 while the CSS$_S$ and CSS$_T$ counterparts are 28.0 (-20.9%) and 28.1 (-20.6%) N/cm^2, respectively.

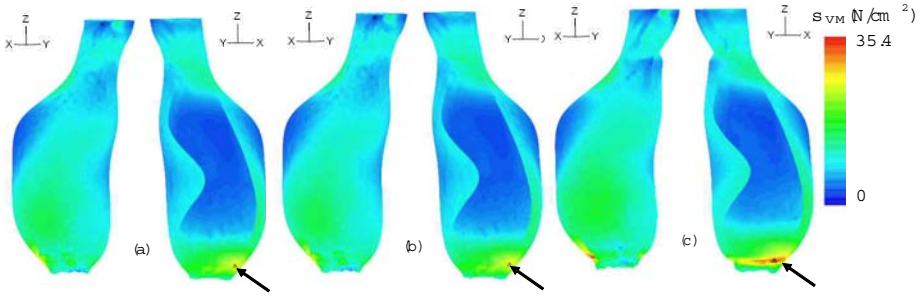

Figure 3: Wall stress distribution along the inner wall surface (with thrombus) using three computational techniques: (a) CSS$_S$, (b) CSS$_T$, and (c) FSI. All models are scaled to the maximum stress obtained by the FSI technique, with the location of the maximum for each method indicated by an arrow.

The comparison between the static and transient CSS analyses yield markedly similar results, with a less than 1% difference in wall stress, while the combination of fluid flow and the induced non-uniform intraluminal pressure on the arterial wall yields a maximum wall stress 21% higher. The intraluminal thrombus effectively shields the arterial wall from the flow-induced pressure gradients, with the location of the maximum wall stress residing where the localized thrombus is thinnest or absent. The primary argument for utilizing CSS methods for predicting AAA wall stress distribution is the small fluid pressure drop across the AAA, estimated at 0.1 kPa (0.075 mmHg) by Wolters *et al.* [10]. However, as shown in [8,9], the pressure within the AAA sac exceeds the imposed outlet normal traction, while the pressure gradient ranges from 1.8 – 4.6 mmHg. Consequently, the assumption that the maximum stress to be considered for aneurysm rupture prediction occurs at intraluminal peak systolic pressure needs to be re-examined for application to patient morphologies, as the traditional FEA methods underestimate the maximum wall stress when compared to the FSI technique.

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