A DE-COUPLED FLUID STRUCTURE APPROACH IN ESTIMATING WALL STRESS IN ABDOMINAL AORTIC ANEURYSMS

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INTRODUCTION

Abdominal aortic aneurysm (AAA) is a localized dilatation of the aortic wall. The physiological processes associated with AAA development and progression are not yet fully understood. The distribution of mechanical stress on the aneurysmal wall is determined by the complex intra-aneurysmal hemodynamics generated by the three dimensional geometric configuration of the intraluminal thrombus modulated flow conduit. The maximum normal cross sectional AAA diameter is being used routinely in clinical practice as an estimate of rupture risk. However, this index has proven inaccurate leading to either under or over estimation of rupture risk thus compromising the quality of patient management.

The lack of an accurate AAA rupture risk index remains an important problem in the clinical management of the disease. To accurately estimate AAA rupture risk patient specific wall stress distribution and aortic wall tissue yield stress are required. A complete fluid structure interaction (FSI) simulation of the wall forces is very intensive computationally and currently impractical. Furthermore it has been shown in idealized AAA models to produce peak wall stress estimates close to those produced by a structures only approach [1]. A static structural analysis on the other hand that assumes a uniform wall loading is a widely applied approach. However this approach neglects the flow modulated wall stress distribution. Aim of this study was to assess the value of a de-coupled fluid structure approach in AAA wall stress estimation.

METHODS

Information on the 3D AAA geometric configuration was extracted *in vivo* by CT angiography. Segmentation and 3D surface reconstruction of the CT images was conducted using in house developed software [2]. From the segmented CT images two 3D surfaces were reconstructed: the true vessel lumen surface and the external aortic wall surface. A third surface, the internal aortic wall was generated as an iso-surface 2 mm inwards from the external aortic wall. To exclude from the computational model abnormal small scale surface irregularities introduced during imaging and reconstruction, pixel width constrained smoothing of surfaces was applied.

The computational mesh was generated using Gambit and the time varying flow field was computed using Fluent 6.1.22. For the flow field computations, the arterial wall was assumed rigid and blood was assumed a Newtonian and incompressible fluid with a density of 1.05 gr/cm^3 and a kinematic viscosity of 4.5 10^{-3} Pa·s. The AAA inflow waveform and the aortic flow split in the iliac arteries were obtained *in vivo* by Doppler US two hours after CT scanning of the patient. Based on the discrete Fourier series of the measured waveform, the fully developed Womersley solution was prescribed at the model inlet.

ABAQUS 6.4.1 was used for the static structural analysis. The aortic wall was modeled as an incompressible, homogenous, isotropic hyperelastic, material with a constant thickness of 2 mm using the model parameters proposed by Rhagavan et al. [3]. The intraluminal thrombus (ILT) was modeled as an incompressible, isotropic, homogenous, linear elastic material with parameters proposed by Di Martino et al. [4]. Stress analyses results were obtained both for a uniform wall loading using the peak systolic arterial pressure (16 kPa or 120 mmHg) and a non-uniform wall loading using the computed hemodynamic peak wall stress. A non-slip condition was applied at the AAA wall - ILT interface. The proximal and distal ends of the model were constrained longitudinally.

RESULTS & DISCUSSION

The results of the time dependent flow field computation showed that most of the AAA lumen wall surface was exposed to very low wall shear stress (WSS) throughout the cardiac cycle. Regions of locally elevated WSS were located near the proximal and distal neck of the AAA bulge (Figure 1).

The computed wall pressure distribution during early systolic deceleration (Figure 2) showed a significant deviation from the peak arterial systolic pressure which has been widely used as a wall loading condition in static structural analyses. Application of the non-uniform hemodynamic wall loading to the AAA model produced a 12 % in-

crease in the computed peak wall stress as compared to the uniform wall loading result (Figures 3 and 4). Two regions of high stress were found, one located anteriorly in the distal half of the AAA bulge with a local peak of 52 N/cm^2 (Figure 3 arrow) and the other located at the proximal neck anteriorly and to the left with a local peak of 54 N/cm^2 (Figure 4 arrow).

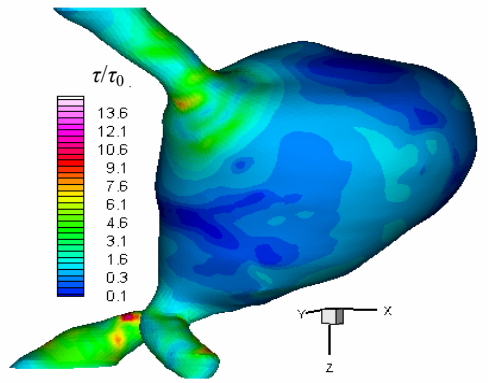

Figure 1. Computed WSS magnitude during early systolic deceleration normalized by the inlet equidiameter straight pipe WSS.

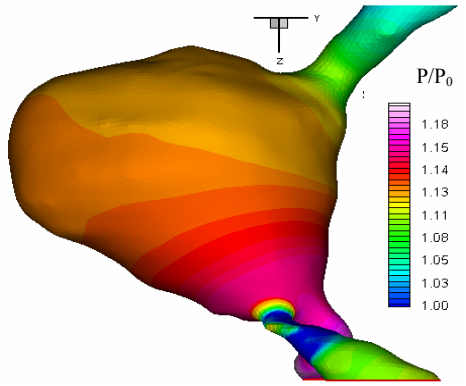

Figure 2. Computed static pressure on the wall of the true lumen during early systolic deceleration normalized by peak systolic arterial pressure.

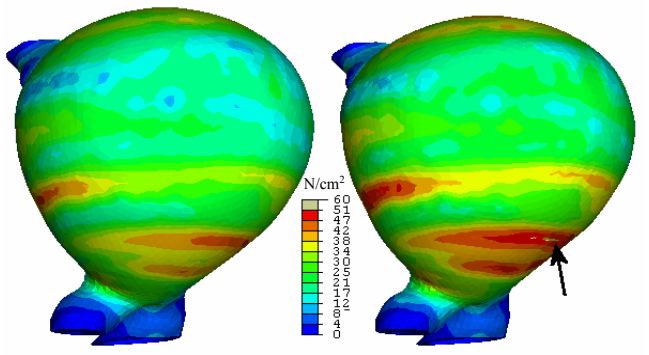

Figure 3. Computed Von Mises stress for uniform peak systolic pressure loading (left) and flow computed non-uniform wall loading (right)

Use of a uniform wall thickness does not reduce the value of the results presented as it will have a similar effect in the stress distribution on both computational approaches considered. Our results show that although the structure only analysis captures the gross features of the stress distribution it underestimates the magnitude of the peak wall stress as compared to the proposed de-coupled fluid structure approach. Furthermore, the simultaneous computation of the flow field allows for the localization of disturbed flow patterns such as low wall shear stress and high shear gradients associated with the development of wall lesions. Information derived from the local AAA hemodynamic conditions may be used to support AAA prognosis.

In order to establish a reliable patient specific index of AAA rupture risk it is necessary to further improve the accuracy of the computational models used. This requires imposing realistic boundary conditions extracted from the patient *in vivo* to a computational model that couples fluid and solid dynamics. This study shows that a de-coupled fluid structure approach is a practical alternative to the more complete but computationally expensive FSI study.

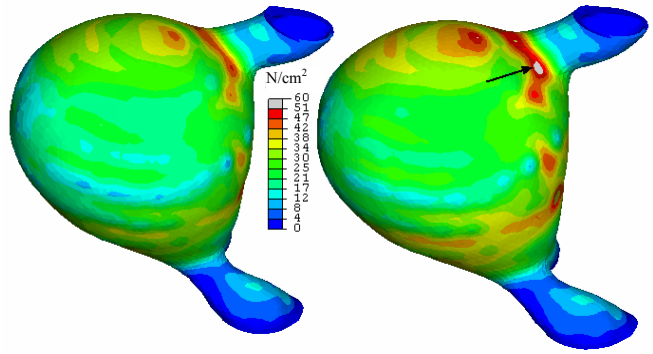

Figure 4. Computed Von Mises stress for uniform peak systolic pressure loading (left) and flow computed non-uniform wall loading (right).

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