A decoupled fluid structure approach for estimating wall stress in abdominal aortic aneurysms

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Abstract

Abdominal aortic aneurysm (AAA) is a localized dilatation of the aortic wall. 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, detailed information on patient-specific wall stress distribution and aortic wall tissue yield stress is required. A complete fluid structure interaction (FSI) study is currently impractical and thus of limited clinical value. On the other hand, isolated static structural stress analysis based on a uniform wall loading is a widely used approach for AAA rupture risk estimation that, however, neglects the flow-induced wall stress variation. The aim of this study was to assess the merit of a decoupled fluid structure analysis of AAA wall stress. Anatomically correct, patient specific AAA wall models were created by 3D reconstruction of computed tomography images. Flow simulations were carried out with inflow and outflow boundary conditions obtained from patient extracted data. Static structural stress analysis was performed applying both a uniform pressure wall loading and a flow induced non-uniform pressure distribution obtained during early systolic deceleration. For the structural analysis, a hyperelastic arterial wall model and an elastic intraluminal thrombus model were assumed. The results of this study demonstrate that although the isolated static structural stress analysis approach captures the gross features of the stress distribution it underestimates the magnitude of the peak wall stress by as much as 12.5% compared to the proposed decoupled fluid structure approach. Furthermore, the decoupled approach provides potentially useful information on the nature of the aneurysmal sac flow.

Keywords: Biofluid mechanics; Aneurysm rupture; Computational hemodynamics; Structural stress analysis

1. Introduction

Abdominal aortic aneurysm (AAA) is a localized dilatation of the aortic wall. The physiological processes associated with AAA development and progression are not as yet fully understood. This pathologic condition has been found to affect 8.8% of the population over the age of 65 (Newman et al., 2001) and if left untreated it may lead to rupture. The size of an aneurysm and its rate of expansion are parameters associated with the risk of rupture. The risk of rupture per year for aneurysms with a maximum transverse diameter below 4 cm is very low but not absent. For transverse aneurysmal diameter between 4 and 5 cm the risk of rupture is 1%. However,
for diameters between 5 and 6 cm this risk becomes 11% rising exponentially with diameter increase (Reed et al., 1997). The decision for surgical intervention for patients with AAA's is complicated by the lack of a reliable and sufficiently accurate rupture risk index. A widely used index, based on the results from a number of clinical studies (Galland et al., 1998; Scott et al., 1999; The UK Small Aneurysm Trial Participants, 1998), is the maximum transverse diameter. In cases where this diameter exceeds 5–6 cm, surgical or endovascular treatment is advised. However, 'small' (< 5 cm) diameter aneurysms, where ‘watchful waiting’ requiring frequent observation is preferred to surgery, are known to rupture (Cronenwett et al., 1985; Darling et al., 1977; Nicholls et al., 1998), while ‘large’ (> 8 cm) diameter aneurysms have been found intact. Therefore, the decision for surgical intervention, associated with a mortality rate of 4–5% (Katz et al., 1992), should not be based exclusively on the maximum transverse diameter and a new more reliable rupture risk index should be introduced.

Recent attempts to establish a reliable AAA rupture risk index were based on the evaluation of the arterial wall stress distribution. Finite element analysis (FEA) has been used to compute the stress distribution in both simplified (Elger et al., 1996; Mower et al., 1993) and anatomically correct (Raghavan et al., 2000; Vorp et al., 1998) AAA models. These studies, however, did not consider flow-induced wall pressure variations. The hemodynamics of the AAA have been extensively investigated experimentally (Egelhoff et al., 1999; Yu, 2000) and computationally in both idealized and anatomically correct models in steady and time varying flow (Budwig et al., 1993; Taylor and Yamaguchi, 1994). The coupling of fluid and structure has also been studied both in realistic (Di Martino et al., 2001) and in idealized (Finol et al., 2003) AAA models. However, both of these investigations considered isolated aneurysmal expansions of the aorta excluding the abdominal bifurcation from their computational model. This model simplification, primarily in AAA’s, is expected to influence the intraluminal flow field and the wall stress distribution.

The role of intraluminal thrombus (ILT) on AAA wall stress distribution still remains uncertain. Some studies support the hypothesis that the ILT introduces a cushioning effect in the transmission of the flow-induced stresses to the wall that reduces the peak wall stress (Mower et al., 1997; Vorp et al., 1996; Wang et al., 2002), while others suggest that thrombus has no effect on the progression of an AAA (Dobrin, 1989; Schurink et al., 2000). Various simplifications have been introduced to the models used in these studies with respect to the shape of the aneurysm, the inclusion and elastic properties of ILT, the thickness and elastic properties of the wall, the role of surrounding structures and, the presence of residual stresses on the AAA wall.

The aneurysmal wall loading distribution is determined by the complex intra-aneurysmal hemodynamics resulting from the irregular geometric configuration of the flow conduit, which results from the pattern of ILT accumulation, and the effects of surrounding structures. To date, the maximum transverse dimension of the AAA is being used routinely in clinical practice as an estimate of rupture risk. However, the use of this parameter alone has led in many cases to the underestimation of rupture risk in ‘small’ (< 5 cm) diameter aneurysms and overestimation of the risk of rupture in ‘large’ (>6 cm) diameter aneurysms 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. Accurate estimation of the patient-specific AAA rupture risk requires detailed information on the distribution of wall loading, the aortic wall and ILT material properties and wall yield stress. However, the AAA wall properties and the stress distribution cannot be measured or even derived with sufficient accuracy from non-invasive measurements in vivo. As an alternative, the wall stress distribution can be numerically approximated. Towards this end, wall constitutive models based on population mean elastic properties obtained by in vitro mechanical testing of excised specimens of the aneurysmal wall are used. Raghavan and Vorp (2000) proposed a two parameter, hyperelastic, isotropic, incompressible material model for the AAA wall utilizing uniaxial loading stress strain measurements on excised AAA specimens. The ILT has been modeled as either an elastic (Di Martino et al., 1998) or hyperelastic (Wang et al., 2001), isotropic, incompressible material.

The wall stress computation should ideally result from a complete fluid structure interaction (FSI) simulation of the wall forces. However, this approach still suffers from modeling assumptions. Ideally, it requires a reliable prediction of the stress free or zero pressure geometry based on the mean arterial pressure geometry obtained in vivo by 3D imaging (Rodríguez et al., 2003). It is also very intensive computationally and thus currently impractical. Furthermore, a study in idealized AAA models (Finol et al., 2003) has shown that the FSI approach yields peak wall stress estimates similar to those obtained by an isolated structural stress analysis. The computational approach most widely used to estimate peak AAA wall stress is the isolated static structural analysis with a uniform peak systolic pressure wall loading. However, this approach neglects the flow-induced pressure distribution on the AAA wall. The aim of the present study was to assess the merit of a decoupled fluid structure approach for AAA wall stress estimation as compared to the isolated static structural stress analysis approach. Towards this end, the stress distribution computed for a uniform wall loading in an
anatomically correct AAA model is compared to the stress computed for the same model, but for the flow-induced pressure wall loading.

2. Methods

A 86 year old male with an intact 10 cm peak transverse diameter AAA was the subject selected for this study. This case was clinically rather unusual as aneurysms commonly rupture before reaching this size. The selected AAA geometry exhibits significant tortuosity of the inflow conduit and the proximal segments of the iliac arteries. These geometric features are expected to strongly influence the intraneurysmal flow field. This geometric configuration is typical of large AAA’s and can be attributed to the asymmetric expansion of the aneurysm sac as a result of the expansion constrains introduced by the proximity to the spinal column. Information on the 3D AAA geometric configuration was extracted in vivo by contrast-enhanced high-resolution spiral computed tomography (CT) angiography. The CT acquisition parameters were prescribed as follows: 160 mAs, 120 kVp, 10.4 s scan time, 22.1 mm feed/rotation ratio, 1.5 mm slice thickness, 1.5 mm reconstruction spacing/increment, 0.5 mm slice overlap and a $512 \times 512$ image matrix size resulting in a 0.742 mm in plane resolution. Angiography was triggered at 120 Houndsfield units. It should be noted that the ILT proliferation in the aneurysmal sac was relatively limited in this case. This was a somewhat unexpected finding considering the size of the aneurysmal sac.

Segmentation and 3D surface reconstruction of the CT images was implemented using purpose-developed software (Giordana et al., 2005). From the segmented CT images two 3D surfaces were reconstructed: the true vessel lumen surface and the external aortic wall surface (Fig. 1). A third surface, the internal aortic wall was generated as an iso-surface 2 mm inwards from the external aortic wall. The third surface, the interface between the AAA wall endothelium and the ILT, could not be extracted from the CT images as the imaging method still lacks the level of spatial resolution and contrast required. The space between this surface and the true vessel lumen surface is primarily occupied with ILT with some regions including patches of calcified wall tissue. The average ILT thickness computed as the distance between the 3D reconstructed surfaces was 3 mm with a maximum of 5.2 mm located at the proximal part of the left common iliac artery. The location of this third surface relative to the external aortic wall surface effectively determines the thickness of the AAA wall. In this study a uniform 2 mm wall thickness was prescribed based on population mean values obtained from an experimental study of excised AAA wall tissue specimens (Raghavan et al., 2000). Abnormal, small scale surface irregularities introduced during the imaging and reconstruction processes applied were excluded from the computational model by applying pixel width constrained smoothing of the reconstructed surfaces prior to mesh generation.

2.1. Flow computation

The computational grid was generated with Gambit v. 2.1.6 (Fluent Inc., NH, USA). It contains 333,150 tetrahedral/hybrid elements and non-uniform grid node spacing to produce higher grid density at the proximal and distal aneurysm neck regions as compared to the bulge region. The numerical grid was locally refined near the wall by imposing a 0.08 $D$ (where $D$ the inlet diameter) viscous layer adjacent to the walls in order to capture the steep gradients in the oscillating boundary layer. Straight tube extensions were added to the common iliac arteries to prevent upwind contamination of the flow domain from flow disturbances caused by forced outflow boundary conditions. A short straight tube extension, which was smoothly blended with the native vessel, was also added to the proximal inflow to create a circular cross section inlet required for the correct application of the exact Womersley solution as the time-dependent inflow boundary condition.

The Navier–Stokes and continuity equations for incompressible flow in the absence of body forces are expressed in vector form as:

$$\nabla \cdot \mathbf{u} = 0,$$

$$\frac{D\mathbf{u}}{Dt} = - \frac{1}{\rho} \nabla p + \nu \nabla^2 \mathbf{u},$$  \hspace{1cm} (1)
where \( D/Dt = \partial/\partial t + \mathbf{u} \cdot \nabla \) is the substantial derivative, \( \rho \) the fluid density, and \( \nu \) the fluid kinematic viscosity.

Fluent v. 6.1.22 (Fluent Inc, NH, USA) was used to solve the flow equations. For the flow field computations, the arterial wall was assumed rigid and blood was modeled as an incompressible Newtonian fluid with a density of 1.05 gr/cm\(^3\) and a viscosity of 4.5 cP. Blood is a suspension of red and white cells, platelets, proteins and other elements in plasma and exhibits an anomalous non-Newtonian viscous behavior when exposed to low shear rates or flows in tubes of less than 1 mm in diameter. However, the Newtonian fluid assumption does not affect the major flow features and is considered an acceptable approximation for modeling blood flow in the macrocirculation (Perktold et al., 1991).

The AAA inflow waveform and the aortic flow split ratio in the iliac arteries were measured in vivo by Doppler US 2 h after CT scanning of the patient. On average, the left iliac artery received 40\% of the AAA inflow and the right iliac artery received 60\%. The discrete Fourier series of the measured AAA inflow waveform can be expressed as:

\[
Q(t) = Q_0 + \sum_{n=1}^{N} Q_n e^{i n \omega t},
\]

where \( Q_0 \) is the steady flow component, \( N = 16 \) represents the number of Fourier modes used, and \( \omega \) the fundamental frequency of the measured flow waveform. From the discrete Fourier series of the volume flow rate in Eq. (2) the fully developed time-varying velocity profile was computed using an expression obtained following Womersley’s derivation (Womersley, 1955):

\[
u(r,t) = \frac{2Q_0}{A} \left( 1 - \frac{r^2}{R^2} \right) + \sum_{n=1}^{N} \frac{Q_n}{A} \left\{ \left( 1 - \frac{J_0(z_n r^{3/2}/R)}{J_0(z_n R^{3/2})} \right) \left( 1 - \frac{2J_1(z_n r^{3/2})}{z_n R^{1/2}J_0(z_n R^{3/2})} \right) \right\} e^{i n \omega t},
\]

where \( J_0 \) and \( J_1 \) are the Bessel functions of the first kind of order zero and one, respectively, \( A \) is the cross sectional area and \( R \) the radius of the straight tube inlet extension and, \( z_n = R \sqrt{4 \omega / \nu} \) is the Womersley parameter of the \( n \)th Fourier mode. The spatiotemporally averaged Reynolds number of the prescribed waveform was \( Re_m = 355 \) and the Womersley parameter for the fundamental frequency of the measured flow waveform was \( z_1 = 16.7 \). The velocity profile given by Eq. (3) with time variation of the mean velocity \( (Q_i/A, i = 0, \ldots, N) \), shown in Fig. 2, was applied as the time-dependent inflow boundary condition. A second-order upwind discretization scheme was applied for the momentum equations and the SIMPLE scheme was used for pressure velocity coupling. A time periodic solution was achieved after 5 flow cycles.

As an indicator of numerical accuracy, the single quantitative value provided by the total force acting on the walls of the AAA was used. The total force coefficient is defined as

\[
C = \frac{|F|}{(1/2) \rho \bar{u}^2 A},
\]

where \( |F| \) is the magnitude of the integrated shear \( \tau \) and pressure \( p \) force over the AAA walls with vector components \( F_i \) along Cartesian direction \( i \) given by

\[
F_i = \int_{walls} (p n_i + \tau_j n_j) \, ds.
\]

\( \rho \) is the fluid density, \( \bar{u} = 0.5 \) m/s is the mean inlet velocity at peak systole and \( A \) the surface area of the AAA walls (0.0336 m\(^2\)).

A systematic time-step and grid size independence study was carried out to assess the accuracy of the numerical computations. The results of this study, summarized in Table 1, show that the change in the total force coefficient \( C \) between grids 1 and 2 is \( \sim 19\% \) of the value obtained with the refined grid 3. However, this difference between grids 2 and 3 drops to less than 3.1\% for all time step sizes selected indicating a good degree of mesh convergence. Less significant differences in \( C \) occur with changing time step size. Halving the time step once, results in a less than 3.9\% change in \( C \) and halving it a second time results in a less than 2\% further change in \( C \) indicating a good degree of time step convergence. Having established mesh and time step independence of the computed results a time step of \( \Delta t = 0.001 \) normalized by the mean inlet velocity per inlet diameter and a grid with \( \sim 3.3 \times 10^5 \) elements were considered sufficient. Finally, pressure and vorticity volume stream ribbons were calculated at a post

![Fig. 2. Mean velocity waveform and instantaneous Reynolds number (Re) prescribed at the AAA inlet.](image-url)
processing stage using a commercial flow visualization package (Tecplot v.10, Amtec Engineering Inc.).

2.2. Finite element stress analysis

MSC.Nastran 2005 (MSC. Software Corp., CA, USA) was used to solve the momentum equations, the wall constitutive equations and the conditions of equilibrium for the static structural stress analysis. The aortic wall was modeled as an incompressible homogeneous isotropic hyperelastic material with a uniform thickness of 2 mm. The finite strain constitutive model proposed by Raghavan and Vorp (2000) was adopted for the arterial wall with a strain energy density function given by

\[
W = \alpha (I_B - 3) + \beta (I_B - 3)^2, \tag{4}
\]

where, \( I_B \) is the first invariant of the left Cauchy–Green tensor \( B \) (\( I_B = \text{tr} \, B \)). The model parameters were set to \( \alpha = 17.4 \, \text{N/cm}^2 \) and \( \beta = 188.1 \, \text{N/cm}^2 \) that correspond to population mean values obtained from uniaxial loading tests on excised AAA wall specimens.

The ILT was modeled as an incompressible, isotropic, homogeneous, linear elastic material with a Young modulus \( E = 0.11 \, \text{MPa} \) and a Poisson ratio \( \nu = 0.45 \). These values of \( E \) and \( \nu \) represent population mean values obtained from uniaxial loading tests performed on ILT specimens harvested during AAA surgery by Di Martino et al. (1998). The AAA model assembly included the ILT solid part with 41,291 linear tetrahedral/hybrid elements and the arterial wall shell part with 9690 linear triangular elements. Mesh convergence was achieved by gradually increasing the grid density of the model until the computed peak stress did not change by more than 1%. Stress analysis results were obtained both for a uniform wall loading using the peak systolic arterial pressure (16 kPa or 120 mmHg) and for the peak non-uniform flow-induced wall pressure loading computed during early systolic deceleration. Mapping of the pressure field from the finer numerical grid used in the flow computations to the coarser grid used in the stress analysis was achieved by inverse-distance interpolation.

A non-slip condition was applied at the AAA wall–ILT interface. The proximal and distal ends of the model were constrained from longitudinal deformation to simulate the tethering forces acting on the AAA model.

3. Results

The results of the time-dependent flow field computation showed that most of the AAA lumen wall surface was exposed to low wall shear stress (WSS) throughout the cardiac cycle. The computed spatiotemporal mean wall shear stress integrated over the AAA bulge walls was 7.7 dynes/cm². This is almost half the \( \sim 15 \, \text{dynes/cm}^2 \) level that normal arteries typically try to maintain by adapting their diameter (Glagov et al., 1988). Regions of locally elevated WSS were located near the proximal and distal neck of the AAA bulge (Fig. 3a). WSS magnitude was normalized by the straight pipe inlet Poiseuille WSS. The computed wall pressure distribution during early systolic deceleration (Fig. 3b) showed a significant deviation from the peak arterial systolic pressure, which has been widely used as a uniform wall loading condition in static structural stress analyses. During early systolic acceleration, regions in the vicinity of the distal neck of the aneurysmal wall were exposed to a pressure loading 15% higher than the peak systolic pressure. Furthermore, most of the aneurysm bulge wall was exposed to pressures 11% higher than the peak systolic pressure.

The highly complex flow field that develops in the aneurysmal sac is depicted in Fig. 4 where stream ribbons color mapped with static pressure (a) and vorticity \( \omega = \nabla \times \mathbf{u} \) magnitude (b), a measure of angular velocity or rotation rate, are shown. The out-of-plane curvature (tortuosity) of the aortic flow conduit that injects blood into the aneurysmal expansion strongly influences the velocity distribution at the aneurysmal bulge inlet. This results in a skewed velocity profile being injected in the aneurysmal expansion thus introducing an asymmetry in the intraneurysmal flow pattern. The vorticity color-mapped stream ribbon graph (Fig. 4b) clearly depicts the pattern of creation, transport and diffusion of vorticity within the tortuous AAA inflow conduit and its convection downstream in the aneurysmal sac. Vortical flow structures in arterial flows have been discussed in detail by Dooryl et al. (2002). Their computational results obtained in arterial bypass graft models also indicate a strong influence of the non-planar graft inflow on the pattern of transport and diffusion of vorticity in the host artery downstream.

The bulk rotation of the flow resulting from the out-of-plane configuration of the AAA inflow conduit and the associated helical flow pattern that develops, can be identified by plotting the helicity density \( H = \mathbf{\omega} \cdot \mathbf{u} \).
which is a scalar and provides a measure of flow stream aligned fluid particle rotation. Fig. 5 shows the computed absolute helicity density cross sectional distribution extracted from the AAA flow domain at various locations along the craniocaudal direction during the early and late deceleration phases. Helicity density increases with distance from the inlet and remains confined near the posterior aneurysmal wall. It also appears that helicity density rapidly dissipates within the proximal half of the aneurysmal expansion and vanishes within the distal half.

Arterial wall stress distributions for uniform wall loading and flow-induced non-uniform pressure wall loading are presented using the Von-Mises stress, a scalar measure of the stress tensor that is proportional to the strain energy density at each point, which is expressed as

$$\sigma_{VM} = \sqrt{1/2[(\sigma_1 - \sigma_2)^2 + (\sigma_1 - \sigma_3)^2 + (\sigma_2 - \sigma_3)^2]}.$$  \hspace{1cm} (5)

where $\sigma_1, \sigma_2, \sigma_3$ are the principal stresses. Three regions of high wall stress were found, one located anteriorly.
and in the distal half of the AAA bulge with a local peak of 52 N/cm² (indicated with an arrow in Fig. 6), a second located at the proximal neck anteriorly and to the left with a local peak of 54 N/cm² (indicated with an arrow in Fig. 7) and a third located posteriorly and approximately halfway between the proximal and distal neck with a local peak of 56 N/cm². However, from inspection of the CT images used in the model reconstruction it was found that a large section of the posterior wall is in contact with the spinal column, a constraint not included in our structural analysis model. This model simplification is expected to influence the computed wall stress distribution at the posterior wall. However, as the degree of influence is unknown the posterior wall local peak was not considered any further.

At the first two sites of local peaks the stress computed applying the isolated structural stress analysis approach was 47 and 48 N/cm², respectively. Thus, application of the non-uniform flow-induced wall loading to the AAA model produced a 12.5% increase in the computed peak wall stress as compared to the uniform wall loading result. It should be noted however, that only the magnitude and not the location of the peak stress regions was altered with the application of the non-uniform flow-induced pressure wall loading.
4. Discussion

The AAA selected for this study had a peak transverse dimension of approximately 10 cm and did not rupture prior to surgery although being almost twice the size above which surgical intervention is commonly advised. Such cases further support the argument that the peak transverse dimension is not an absolutely reliable AAA rupture risk indicator. Using the finite-element method to compute the stress distribution in simplified AAA model geometries Stringfellow et al. (1987) and Mower et al. (1993) showed that the complexity of the geometry influences the stress distribution and that direct application of the law of Laplace, which relates internal diameter and wall stress, yields inaccurate predictions of the stress field even in simplified AAA geometries. Furthermore, Elger et al. (1996), using an analytical model, found that the wall stress distribution is primarily influenced by the shape of the aneurysm with peak stress correlated to wall curvature. This finding is in agreement with the results of the present study where regions of high surface curvature located near the proximal neck of the aneurysm also exhibit high wall stress (see Figs. 6 and 7). However, it is a common observation that the site of rupture is highly variable. This indicates that although surface curvature most strongly influences wall stress distribution among geometric parameters, other parameters not related to geometry such as wall composition, ILT accumulation and wall thickness affect the elastic behavior of the aneurysmal wall and play an important role in the pathogenesis of rupture. These parameters are influenced by the wall remodeling process that is triggered by the non-physiological hemodynamic conditions and wall forces that develop at aneurysmal expansions. Arterial wall remodeling can be triggered by prolonged non-physiological levels of wall shear and hoop stress and is expressed by changes in intima media layer thicknesses and changes in the organization of collagen and elastin in the wall. These changes may lead to a reduction in the structural strength of the AAA wall thus increasing the risk of rupture.

In the patient studied the extent of ILT accumulation was not significant given the size of the AAA expansion. Although the relationship between thrombosis and hemodynamics is highly complex some insight into the extent of ILT deposition in the case studied can be gained from our results. The out-of-plane curvature of the AAA inflow conduit creates a helical bulk flow pattern that is known to suppress flow separation, improve mixing and reduce the wall shear range (Papaharilaou et al., 2002; Zabielski and Mestel, 2000). This in turn reduces the propensity of platelet shear-induced activation (Strony et al., 1993) and may therefore produce a less favorable hemodynamic environment for thrombus formation.

Our results show that although the isolated static structural stress analysis approach captures the gross features of the stress distribution it underestimates the magnitude of the peak wall stress by as much as 12.5% compared to the proposed decoupled fluid structure approach. This value may be different when other AAA cases are considered depending on the aneurysm shape and inflow conditions. However, the intra-aneurysmal flow-induced wall pressure distribution is primarily influenced by the temporal acceleration and deceleration of the flow and to a lesser extent by the size of the aneurysmal sac. Consequently, as our stress computations were based on a typical physiological AAA inflow waveform, the difference in the computed peak wall stress found should be considered representative. In addition to the improved wall stress production, the
decoupled fluid structure approach also yields the local AAA hemodynamic conditions thus allowing for the identification of wall regions exposed to low and oscillatory wall shear stress and high shear gradients, conditions that have been linked to the development of wall lesions. Moreover, this approach allows for the identification of hemodynamically important large scale flow features in AAA’s such as vortical flow structures which can be used to study the transport and mixing of particles such as red blood cells and platelets in arteries (Doorly et al., 2002). This information may then be used to further support prognosis of AAA rupture risk.

The AAA model used in the present study includes a number of simplifications and underlying assumptions. A uniform wall thickness was specified due to the inherent limitations in the imaging technique. This affects the computed stress distribution thus increasing the uncertainty of the results as compared to the exact in vivo conditions. However, as this will have a similar effect on the stress distribution in both computational approaches considered, it does not reduce the value of the comparative results presented. To support this argument, the stress field in both AAA model cases considered was computed prescribing a 1.8 mm aortic wall thickness, 10% thinner then that used originally. The results of this numerical experiment show that although there is an increase in the peak stress computed in both cases, which is expected, the relative difference in the peak stresses between the two AAA models is ~11.5% very close to the original results (Fig. 8). The hemodynamic pressure field was computed assuming a rigid wall model. However, it has been shown both experimentally (Duncan et al., 1990) and computationally (Perktold et al., 1994) that the introduction of wall compliance to arterial models has a minor quantitative effect on the computed wall shear stresses whereas the main flow features are preserved.

The computational mesh used for the structural stress analysis was based on the geometry reconstructed from the CT images obtained throughout the cardiac cycle and over multiple cycles as the acquisition was not gated to the cardiac rhythm of the subject. As a result, the mean geometric representation of the pressure pulse modulated AAA configuration is reconstructed although a zero-stress state is assumed in the computation. As the zero-stress state of the AAA cannot be measured in vivo one could assume that as the diastolic phase occupies most of the abdominal aortic flow cycle the reconstructed AAA geometry is an approximate representation of the diastolic pressure modulated AAA stress state. The aspects of initial stress and strain have not been incorporated in our models. However, their effects, which may be important and will be assessed in a future study, are not expected to invalidate the comparative results of this study.

The material properties used in this study where based on mean values and therefore the computed stress distribution is not expected to represent the exact in vivo wall loading conditions. It should be noted, however, that the aforementioned difficulties in constructing a mathematical model to simulate in vivo AAA wall loading conditions also apply to a further extent to the FSI approach, which is even further complicated by the dynamic effects of wall motion. It is therefore very important to reduce the solution uncertainties identified in the proposed decoupled fluid structure model before introducing wall motion dynamics in a coupled fluid structure model.

The effect of errors associated with the AAA flow and structure modeling in the computed wall stress distribution is worthwhile discussing. Errors in the reconstruction of the geometry can be due to lack of sufficient spatial resolution and contrast of the imaging method. From high-resolution spiral CT images most
hemodynamically significant AAA geometry features can be resolved. Typically, noise in CT images causes geometry reconstruction errors that are constrained to the dimensions of a pixel which, given the resolution of the technique and the AAA size, are expected to have a minor effect on the accuracy of the computed wall stress. However, CT lacks sufficient image contrast and spatial resolution to accurately resolve the thickness of the arterial wall, a limitation that introduces proportionate and potentially significant errors in the computed wall stress. Thus, if clinically useful wall stress estimates are required it is critical that AAA wall thickness is accurately extracted. The uncertainty in the computed wall stress associated with the use of mean population values for the aortic wall properties model has been assessed by Raghavan et al. (2000). They found a disproportionately lower variation in computed peak stress (<4%) when material model parameters were varied within their 95% confidence interval. This result indicates that mean population values can be used as a reasonable modeling approximation.

In order to establish a more 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. The results of this study demonstrate that a degree of under prediction in the computed AAA wall stress occurs when a uniform pressure is applied. Furthermore, the decoupled approach provides potentially useful information on the nature of the aneurysmal sac flow. However, further full FSI studies are necessary to establish the degree of error associated with the modeling simplifications in the decoupled approach.

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