Abdominal Aortic Aneurysm Risk of Rupture: Patient-Specific FSI Simulations Using Anisotropic Model

Abdominal aortic aneurysm (AAA) rupture represents a major cardiovascular risk, combining complex vascular mechanisms weakening the abdominal artery wall coupled with hemodynamic forces exerted on the arterial wall. At present, a reliable method to predict AAA rupture is not available. Recent studies have introduced fluid structure interaction (FSI) simulations using isotropic wall properties to map regions of stress concentrations developing in the aneurysmal wall as a much better alternative to the current clinical criterion, which is based on the AAA diameter alone. A new anisotropic material model of AAA that closely matches observed biomechanical AAA material properties was applied to FSI simulations of patient-specific AAA geometries in order to develop a more reliable predictor for its risk of rupture. Each patient-specific geometry was studied with and without an intraluminal thrombus (ILT) using two material models—the more commonly used isotropic material model and an anisotropic material model—to delineate the ILT contribution and the dependence of stress distribution developing within the aneurysmal wall on the material model employed. Our results clearly indicate larger stress values for the anisotropic material model and a broader range of stress values as compared to the isotropic material, indicating that the latter may underestimate the risk of rupture. While the locations of high and low stresses are consistent in both material models, the differences between the anisotropic and isotropic models become pronounced at large values of strain—a range that becomes critical when the AAA risk of rupture is imminent. As the anisotropic model more closely matches the biomechanical behavior of the AAA wall and resolves directional strength ambiguities, we conclude that it offers a more reliable predictor of AAA risk of rupture. [DOI: 10.1115/1.3005200]

Introduction

Abdominal aortic aneurysm (AAA) is a pathological expansion of the aorta due to gradual wall weakening. AAA is a common vascular problem with fatal implications. Progressive AAA growth will lead to eventual rupture. Current clinical estimates of AAA patient’s risk of rupture are primarily based on the maximal transverse dimension of the aneurysm (5.5 cm diameter) [1]. Repair is warranted when the risk of rupture exceeds that of the repair. Absolute diameter measurements are poor markers since some smaller diameter aneurysms can rupture while some larger ones can remain intact [2–5]. A better means of predicting AAA rupture—based on patient-specific data—is needed. The mortality rate for an aneurysm rupture is independent of size and exceeds 50% [6]. Some aneurysms can contain an intraluminal thrombus (ILT). Whether or not the ILT offers protection against rupture is subject of ongoing investigation [7–11].

The rupture of an AAA is due to a mechanical failure of the wall tissue because of elevated stresses developing within the AAA wall as a result of the interaction between the pathological AAA hemodynamics and the compromised integrity of the wall. Currently, the clinical modalities available for estimating the aneurysm wall stresses or tissue strength are inadequate. Computational tools, in combination with the available clinical modalities, can be used to better ascertain the risk of rupture. Fillinger et al. [12] showed that even simplified computational models in which a static uniform internal pressure was applied on a AAA wall can be 12% more accurate and 13% more sensitive than using the maximum diameter as a sole predictor for the risk of rupture.

Several studies have used idealized geometries to measure the effects of variations in wall thickness and the effects of including the ILT. Scotti et al. [13] showed that variations in wall thickness result in higher stresses and would increase the risk of rupture as compared to models that use a uniform wall thickness. While Di Martino et al. [7,14] showed that the presence of ILT can significantly reduce the stress on the wall. While these models offer insight into AAA biomechanics and bypass the challenges associated with reconstructing complex patient-specific geometries, they cannot be used to predict a patient-specific AAA risk of rupture. Realistic AAA physiologic geometries are highly complex, and simple geometric characteristics fail to reliably predict abdominal aortic aneurysm wall stresses, as those are strongly coupled to the AAA geometry [15–17]. Advances in clinical imaging modalities had vastly improved the ability of clinicians to extract patient-specific anatomical 3D data that lend themselves to sophisticated numerical modeling approaches. With these advances, patient-specific AAA geometries can be reconstructed from CT angiograms or mesh deformation algorithms [18]. In recent years the more sophisticated fluid structure interaction (FSI) approach is used to calculate the stress against the wall to determine areas of high stress concentration. To date, however, these AAA numerical studies use isotropic material models for the wall behavior, which do not incorporate the characteristic an isotropic behavior of biological tissue [19,20].

In this paper, two patient-specific AAA geometries, denoted as patients A and B, are studied with a fully coupled FSI modeling approach that incorporates an anisotropic material model and are
compared to a commonly used isotropic material model. Additionally, both geometries were simulated with and without the ILT to determine how the ILT influences wall stresses and expansion of the AAA lumen using an anisotropic material model. A total of eight different time dependent FSI simulations was conducted using physiologic flow and pressure waveforms.

Methods

AAA Reconstruction. Images were obtained from abdominal CT scans administered with intravenous contrast from patients diagnosed with AAAs that have been evaluated with abdominal CT angiograms as part of elective evaluation. These patients are routinely referred to the Surgery Department at Stony Brook University Hospital. Informed consent was obtained retrospectively. The protocol was approved by Stony Brook University Institutional Review Boards (IRB) Committees on Research Involving Human Subjects.

The AAA cross-sectional CT scans were reconstructed into 3D geometry using the MEDICAL METRIX software package (MMS, Medical Metrix Solutions Inc., West Labanon, NH). CUSTOM c and MATLAB codes were used to convert the triangular surface mesh, generated by the MMS software, into an input file that was fed to a numerical mesh generating software package—GAMBIT (Fluent Inc., NH). The generated GAMBIT volume was then split into stacked contours so that a smooth surface could be created by interpolating over the contours. Figures 1(a) and 1(c) show the imported MMS triangulated volume of the AAA lumen and ILT for patients A and B, respectively. A uniform thickness of 2 mm was assigned to the AAA wall, as used in previous studies of patient-specific geometries [18,21–23]. To determine the contours of the outer wall surface, the ILT and lumen volumes were fused, the surface of the resulting volume was then meshed, and each mesh node was then translated 2 mm away from the volume (along the average normal direction of each adjacent face) according to Eq. (1).

$$x_i^w = x_i + \sum_{j=1}^{n_i} f_j$$  \(\text{Eq. (1)}\)

where $x_i$ is the current mesh node position, $n_i$ is the total number of element faces connected to node $x_i$, and $f_j$ is the normal of the $j$th face connected to node $x_i$ (the normal is pointing away from the volume). The splitting procedure for triangulated GAMBIT volumes was then repeated to create a smooth exterior volume. The resulting smoothed volume meshes were imported into ADINA 8.4 FSI software package (automatic dynamic incremental nonlinear analysis, Watertown, MA). The lumen was subtracted from the ILT and the exterior wall volume and then the ILT was subtracted from the outer wall volume. The resulting solid volumes of the two patient geometries after the subtractions are depicted in Figs. 1(b) and 1(c), respectively.

Material Models. Uniaxial testing of abdominal aortic tissue specimens performed by Raghavan and Vorp [24] modeled the mechanical behavior of the tissue by using nonlinear hyperelastic wall mechanical properties given by the isotropic Mooney–Rivlin relation [25]

$$\Psi = C_1(I_1 - 3) + C_2((I_1 - 3)^2)$$  \(\text{Eq. (2)}\)

where $\Psi$ is the strain energy, $I_1=1$ is the first invariant of the Cauchy–Green tensor, $C_1$, and $C_2 = 1,881,000$ Pa. FSI simulations using Eq. (2) to model biomechanical behavior of the AAA wall bounding the lumen were done by several authors [12,13,22,26,27]. However, based on the isotropic assumption, Eq. (2) cannot resolve the directional ambiguities associated with abdominal aortic tissue mechanical response to stresses, which may play a major role in the behavior of the tissue under elevated stresses.

To properly model the mechanical response of AAA tissue, Vande Geest et al. [21] used tension controlled biaxial testing to characterize the three-dimensional response of the tissue. A high degree of anisotropy in mechanical response was observed between the circumferential and longitudinal directions. The experimental data were fitted to an exponential strain energy material model proposed by Vito and Hickey [28] of the form

$$\Psi = b_0 e^{(1/2)b_1 e^{b_2/\varepsilon_{LL}^3}} + e^{(1/2)b_4 e^{b_5/\varepsilon_{LL}^3}} - 3$$  \(\text{Eq. (3)}\)

where $b_0$=0.14 kPa, $b_1$=477.0, $b_2$=416.4, $b_3$=408.3, and $\varepsilon_{LL}$ and $\varepsilon_{LL}$ are the strain values in the circumferential and longitudinal directions, respectively. In order to adapt this experimentally observed behavior of AAA wall tissue under mechanical stresses to FSI modeling, we have implemented the orthotropic material model developed by Holza-
pfel et al. [20] for multilayer arterial walls. The Holzapfel strain energy function models the tissue as a fiber-reinforced composite material with the fibers corresponding to the collagenous component of the material. The strain energy potential function used to model the wall of the AAA is given by

$$\Psi = \Psi_{\text{iso}} + \Psi_{\text{aniso}}$$

$$\Psi_{\text{iso}} = C_1 (I_1 - 3) + C_2 (I_1 - 3)^2 + D_1 \left( \exp \left( D_2 (I_1 - 3) \right) - 1 \right)$$

$$\Psi_{\text{aniso}} = \frac{k_1}{2 k_2} \sum_{i=4,6} \exp \left( k_2 (J_i - 1)^2 \right) - 1$$

where

$$J_i = C_{ij} (n_a) (n_b), \quad J_6 = C_{ij} (n_a) (n_b)$$

$C_{ij}$ is the Cauchy–Green deformation tensor and $n_a$ and $n_b$ are the directions of the fibers defined by the angles, $\beta_a$ and $\beta_b$, which are offset from the material axes.

A nonlinear least-squares estimate was accomplished using the R statistics package to fit the strain energy data from Eq. (3) to the strain energy in Eqs. (4)–(6). The parameters that best fit the model were established as follows:

$$C_1 = 8.888 \text{kPa}, \quad k_1 = 1.886 \text{kPa}$$

$$C_2 = 164.9 \text{kPa}, \quad k_2 = 94.75$$

$$D_1 = 0.0487 \text{kPa}, \quad \beta_a = 5 \text{deg}$$

$$D_2 = 53.46, \quad \beta_b = 265 \text{deg}$$

with an $R^2$ value of 0.9987.

Using this orthotropic model, a replication of the bi-axial tensile testing done by Vande Geeste at al. [21] was conducted numerically in order to validate the model parameters. A square 2 cm specimen was recreated in ADINA 8.4, and the stress-strain relationship in the circumferential and longitudinal directions was matched against the experimental results. Loading on each edge started at 0 N/m, then incrementally increased to 120 N/m over 1 s. The stress-strain relationship in both directions was averaged at four elements at the corners of a 5 x 5 mm² square near the center of the plate. A very good agreement was achieved between the stress-strain relationship of the simulated specimen and the experimental measurements (Fig. 2). The Piola–Kirchhoff stress, $S = \partial \Psi / \partial E$, was calculated from Eq. (3) for the circumferential and longitudinal directions. This material model is a more faithful representation of the mechanical behavior of AAA tissue than the more commonly used isotropic hyperelastic Mooney–Rivlin material model of Eq. (2).

The ILT material was modeled as a linear elastic material with Young’s modulus of 0.11 MPa and Poisson’s ratio of 0.45 [14,22].

**Table 1 Number of elements in each simulation**

<table>
<thead>
<tr>
<th></th>
<th>With ILT</th>
<th>No ILT</th>
</tr>
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<tbody>
<tr>
<td>Patient A</td>
<td>55,233</td>
<td>40,314</td>
</tr>
<tr>
<td>Patient B</td>
<td>28,450</td>
<td>18,646</td>
</tr>
<tr>
<td>Fluid</td>
<td>55,233</td>
<td>21,450</td>
</tr>
</tbody>
</table>

**Mesh and Boundary Conditions.** Following the generation and meshing of the various AAA components in GAMBIT, the model geometries were remeshed in ADINA to guarantee smooth...
contact and traction of the AAA components under dynamic FSI conditions. The meshes were then further refined to guarantee mesh density and periodicity independence, as detailed below. For simulations that contained an ILT, faces that were shared between the outer wall and the ILT were linked so that nodes generated on the face are used for both the ILT and the wall. Four node tetrahedral elements were used for the wall and ILT. In the fluid domain, four node tetrahedral flow based control interpolation (FBCI) elements were used. Mesh convergence studies in which flow parameters and stress/displacement analyses with coarser and finer meshes were tested established that the results are independent of mesh density. After establishing mesh size independence, the resulting AAA mesh sizes (with their various components) are summarized in Table 1.

Blood was modeled as a homogeneous, incompressible, and Newtonian fluid, with a density of 1035 kg/m³ and a viscosity of 3.5 cP [29,30]. Time dependent flow and pressure conditions measured by Olufsen et al. [29] (Fig. 3) were applied at the inlet and outlet of the AAA, respectively. Peak systolic pressure occurs at 0.32 s, and peak systolic flow occurs at 0.25 s. A no-slip condition was applied at the fluid-solid interface.

All AAA models were assumed to initially be in a zero stress state. The FSI simulation required pressurization of the flow domain from 0 Pa to 110 kPa with zero flow for 1 s, before the waveforms were applied at the inlet and outlet. After establishing that periodicity was achieved by the second cycle, each computation was continued for four complete flow cycles using the waveform (Fig. 3) and a time step of $4 \times 10^{-3}$ s. The Newton iteration scheme was used for the sparse matrix solver, with 0.001 relative tolerance for the degrees of freedom. The mesh displacement was also analyzed to ascertain if any difference in motion can be seen between the two material models employed. The wall motion was more pronounced for the anisotropic material model (Fig. 4).

Results

The velocity vector fields for patients A and B are depicted in Fig. 5 in the coronal plane—0.2 s after peak velocity, respectively. During this phase of the flow cycle the flow decelerates, leading to the formation of several recirculation zones. This plane was chosen to show the complex flow patterns that develop within the AAA geometries, which play a role in increasing the intraluminal pressure.

The peak stresses and average stresses (over all the elements) for both patients and model configurations (with and without ILT) are summarized in Tables 2 and 3.
The von Mises stress distribution on the outer wall of patient A—simulated with the ILT for each of the material models—is shown in Fig. 6 for the isotropic model with ILT and Fig. 7 for the anisotropic model with ILT, respectively. The stress distribution is presented from several perspectives to indicate regions of stress concentration forming in various regions of the AAA wall. The stress distribution on the outer wall of patient A simulated without the ILT for each material model is depicted in Figs. 8 and 9, respectively. The maximum and averaged von Mises stress over the entire wall as a function of the time in the cardiac cycle is depicted in Fig. 10 for patient A for the case with ILT and without ILT, respectively for both material models. The maximum stress for the isotropic model is 395.2 kPa while the maximum stress for the anisotropic model is 414.3 kPa, and the respective minimum stress values are 235.0 kPa and 217.0 kPa. The maximum of the average stress for the isotropic model is 111.8 kPa, while the maximum of the average stress for the anisotropic model is 116.6 kPa and the respective minima are 69.0 kPa and 62.0 kPa.

Table 2 Minimum and maximum stress values (kilopascals) for patient A. Peak $\sigma$ is the element that contained the maximum stress. Ave $\sigma$ is the average stress over all of the elements in the model.

<table>
<thead>
<tr>
<th></th>
<th>With ILT</th>
<th>Without ILT</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Isotropic</td>
<td>Anisotropic</td>
</tr>
<tr>
<td>Peak $\sigma_{\text{min}}$</td>
<td>235.0</td>
<td>217.0</td>
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<tr>
<td>Peak $\sigma_{\text{max}}$</td>
<td>395.2</td>
<td>414.3</td>
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<tr>
<td>$\sigma_{\text{max}}-\sigma_{\text{min}}$</td>
<td>160.2</td>
<td>197.3</td>
</tr>
<tr>
<td>Ave $\sigma_{\text{min}}$</td>
<td>69.0</td>
<td>62.0</td>
</tr>
<tr>
<td>Ave $\sigma_{\text{max}}$</td>
<td>111.8</td>
<td>116.6</td>
</tr>
<tr>
<td>$\sigma_{\text{max}}-\sigma_{\text{min}}$</td>
<td>42.8</td>
<td>54.6</td>
</tr>
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</table>

Table 3 Minimum and maximum stress values (kilopascals) for patient B. Peak $\sigma$ is the element that contained the maximum stress. Ave $\sigma$ is the average stress over all of the elements in the model.

<table>
<thead>
<tr>
<th></th>
<th>With ILT</th>
<th>Without ILT</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Isotropic</td>
<td>Anisotropic</td>
</tr>
<tr>
<td>Peak $\sigma_{\text{min}}$</td>
<td>151.3</td>
<td>166.4</td>
</tr>
<tr>
<td>Peak $\sigma_{\text{max}}$</td>
<td>249.7</td>
<td>272.1</td>
</tr>
<tr>
<td>$\sigma_{\text{max}}-\sigma_{\text{min}}$</td>
<td>98.4</td>
<td>105.7</td>
</tr>
<tr>
<td>Ave $\sigma_{\text{min}}$</td>
<td>47.3</td>
<td>44.6</td>
</tr>
<tr>
<td>Ave $\sigma_{\text{max}}$</td>
<td>78.9</td>
<td>75.6</td>
</tr>
<tr>
<td>$\sigma_{\text{max}}-\sigma_{\text{min}}$</td>
<td>31.6</td>
<td>31.0</td>
</tr>
</tbody>
</table>

The von Mises stress distribution on the outer wall of patient B—simulated with the ILT for each of the material models—is shown in Fig. 11 for the isotropic model with ILT and Fig. 12 for the anisotropic model with ILT, respectively. The stress distribution is presented from several perspectives to indicate regions of stress concentration forming in various regions of the AAA wall. The stress distribution on the outer wall of patient B simulated without the ILT for each material model is depicted in Figs. 13 and 14, respectively. The maximum and averaged von Mises stress over the entire wall as a function of the time in the cardiac cycle is depicted in Fig. 15 for patient B for the case with ILT and without ILT, respectively (for both material models). The maximum stress for the isotropic model is 879.5 kPa while the maximum stress for the anisotropic model is 1536.5 kPa, and the respective minimum stress values are 533.3 kPa and 951.1 kPa. The maximum of the average stress for the isotropic model is 111.8 kPa, while the maximum of the average stress for the anisotropic model is 116.6 kPa and the respective minima are 69.0 kPa and 62.0 kPa.

The von Mises stress distribution on the outer wall of patient B—simulated with the ILT for each of the material models—is shown in Fig. 11 for the isotropic model with ILT and Fig. 12 for the anisotropic model with ILT, respectively. The stress distribution is presented from several perspectives to indicate regions of stress concentration forming in various regions of the AAA wall. The stress distribution on the outer wall of patient B simulated without the ILT for each material model is depicted in Figs. 13 and 14, respectively. The maximum and averaged von Mises stress over the entire wall as a function of the time in the cardiac cycle is depicted in Fig. 15 for patient B for the case with ILT and without ILT, respectively (for both material models). The maximum stress for the isotropic model is 879.5 kPa while the maximum stress for the anisotropic model is 1536.5 kPa, and the respective minimum stress values are 533.3 kPa and 951.1 kPa. The maximum of the average stress for the isotropic model is 111.8 kPa, while the maximum of the average stress for the anisotropic model is 116.6 kPa and the respective minima are 69.0 kPa and 62.0 kPa.
Discussion

Two patient-specific abdominal aortic aneurysm model geometries have been reconstructed from clinical images and numerically studied with and without ILT, employing two material models used to characterize the AAA wall properties. A major goal of the current study was to analyze and compare the behavior between a simulation that assumes that the wall of the AAA behaves like an isotropic material and one that is based on anisotropic material behavior. The first material model employed was an isotropic hyperelastic Mooney–Rivlin type model that was used in several recent AAA numerical studies. However, recently published experimental results of AAA specimen under biaxial stretching indicate that a more realistic behavior of the AAA wall is anisotropic. Accordingly, an anisotropic material model based on orthotropic formulation was also employed and compared to the isotropic model results. Similar to other recent studies that used routinely acquired CT scans of AAA patients, the results presented are based on the assumption of a uniform 2 mm wall thickness, which may distort the stress values and their distribution. This assumption is necessary due to limitations in the imaging and reconstruction techniques, but should bear only minimal effects on the comparison between the two material models and the effects of the ILT.

The geometry that contained an ILT had a large rotation for the anisotropic material, which was much less pronounced for the isotropic material model (Fig. 4). However, in the geometry without the ILT, the anisotropic simulation did not have rotational movements such as those observed in the simulation with ILT. This effect could be attributed to the intricate interplay between the directionality of strength (inherent to the anisotropic model) and the AAA geometry (with and without the ILT), as contrasted with directional ambiguity inherent to material isotropy. The large variation between the isotropic and anisotropic model rotations may also be attributed to the simulated extraction of the ILT that otherwise dampens this effect. Additionally, the unique geometry of the specific type of saccular AAA may have contributed to its rotational motion. Such behavior was much less pronounced in the fusiform AAA geometry of patient A.

The flow patterns depicted in Fig. 5 for both patients are caused by the narrow neck that is followed by larger diameter region, typical of AAA disease. This configuration generates low pressure zones that induce flow separation and the formation of recirculation zones. In both AAA patient geometries multiple recirculation zones are formed. These recirculation zones represent nonphysiologic flow conditions that distinguish them from flow patterns in healthy abdominal aortas. They offer a long residence time within the AAA and strong flow mixing, with deposition patterns that promote the formation and growth of the ILT observed in both AAAs. The flow patterns may be indicative of the progression of the disease.

The locations of high and low stresses are consistent for both material models for patient A (Figs. 6–9). However, for the case
with the ILT the anisotropic material model exhibits larger variance between the maximum and minimum wall stress values (Fig. 10). The peak stresses for patient A calculated with the isotropic model range over 160.2 kPa, while the range of stresses for the anisotropic material is 197.3 kPa. The differences between the two material models without the ILT were pronounced. When compared to the simulations containing ILT, without a cushioning effect of the ILT the stresses nearly doubled for this patient. Overall, the range of peak stress values and the range of average stress values for the anisotropic material model are larger than that of the isotropic model, indicating that the use of isotropic model may significantly underestimate the peak stresses for a patient-specific AAA geometry, i.e., underestimate the risk of rupture. This is further highlighted by the comparison between the mesh displacement of both simulations (Fig. 4, with and without ILT), in which the displacement for the anisotropic model doubles that of the isotropic model.

Although the geometry of the AAA of patient B is very different from that of patient A, the results obtained for patient B’s AAA simulations follow a similar pattern. The locations of high and low stresses are consistent between the two material models employed (Figs. 11–14). Similar to patient A, the overall stresses are higher with the anisotropic material as compared to that of the isotropic material, with stress values twice as much for the anisotropic material as compared to those computed by the isotropic material for the case without ILT (Figs. 11–13). The range of values of stress for the anisotropic model was again larger than for the isotropic model (Table 3). The displacement was twice as
much for the anisotropic material when simulated without ILT and more than five times as much when simulated with the ILT.

Additionally, the exponential terms in the orthotropic model (Eqs. (5) and (6)) have a faster rate of change than the quadratic
terms of the Mooney–Rivlin isotropic model (Eq. (2)). The difference becomes very significant for larger strain values. As the simulations without the ILT were characterized by higher stress values, they effect greater differences from the isotropic model stress values as compared to the simulations that contained an ILT. Thus, AAA geometries that do not contain a significant amount of ILT to protect the walls from stress are likely to show significantly higher stresses with the anisotropic material model.

It should be emphasized that a patient-specific modeling aimed at AAA diagnostics should incorporate additional patient-specific data beyond geometry. Part of such data could be available by noninvasive methods (e.g., patient-specific hemodynamic parameters measured by echocardiography, although not in our type of retrospective study), while other data are not readily available, if at all (e.g., patient-specific AAA material properties from specimens unless the patient went through a surgical procedure in which such specimen was obtained, etc.). However, our study goes a step beyond the more sophisticated current modeling efforts that incorporate isotropic hyperelastic material models by incorporating an anisotropic material model that is based on material properties measured in specimens obtained from other patients. The pronounced differences strongly indicate that the isotropic models may underpredict the risk of rupture. This is a strong argument in favor of using material models that emulate more accurately AAA wall mechanical deformation under stress.

In addition, it should be noted that certain clinical scenarios are beyond the scope of the methodology presented herein. Intramural leakage, for example, has been shown to double the stress concentrations and is a hallmark of imminent rupture, and decisions whether to operate an AAA are made based on this alone. Our diagnostics methodology is not aimed at this stage at simulating such a complex clinical scenario rather to examine whether the more common case where a risky elective repair of the AAA based on its size alone is warranted (rather than looking at the stress distribution developing within the aneurismal wall as a result of the complex interaction between the geometry, flow field, and the wall mechanical response).

Conclusions

A new anisotropic material model of abdominal aortic aneurysm was applied to FSI numerical models of patient based AAA geometries for calculating the ensuing stresses developing within the aneurismal wall in order to develop a more reliable predictor for its risk of rupture. The anisotropic material model was fitted to previously published results of biaxial testing of AAA tissue specimen. Each geometry was studied with and without an ILT for two material models to delineate the ILT contribution and the dependence of stress distribution developing within the wall on the material model employed. The first material model is the more commonly used isotropic material, and the second is an anisotropic material model that more closely matches observed biomechanical AAA material properties.

Our results clearly indicate larger stress values for the anisotropic material and a broader range of stress values, as compared to the isotropic material. While the locations of high and low stresses are consistent between both material models, the differences between the anisotropic and isotropic models become pronounced at large values of strain–a range that becomes critical when the AAA risk of rupture is imminent. In addition to differences in the stress values, the anisotropic material indicates much larger displacements than those predicted by the isotropic model. Specifically, a large rotational motion is observed when using the anisotropic material model in one patient’s AAA that is not present in the isotropic material model. As the anisotropic model closely matches experimentally observed results of AAA wall material properties while resolving directional ambiguity, we feel confident that the wall stress values it predicts match more closely the physiological behavior of the AAA wall than the predictions offered by the isotropic model. Accordingly, we conclude that the new anisotropic material model allows for more accurate predictions of AAA risk of rupture and that the use of isotropic models may underestimate this risk. To warrant a surgical repair when risk of rupture exceeds that of the intervention a more reliable predictor for patient based AAA risk of rupture is needed. The methodology presented here may offer clinicians a better patient-specific diagnostic tool to determine the need for surgery. Our ongoing and future work aims at establishing the clinical validity of the methodology described herein.

References


