Original Article

The effect of angulation in abdominal aortic aneurysms: fluid–structure interaction simulations of idealized geometries

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Abstract Abdominal aortic aneurysm (AAA) represents a degenerative disease process of the abdominal aorta that results in dilation and permanent remodeling of the arterial wall. A fluid structure interaction (FSI) parametric study was conducted to evaluate the progression of aneurysmal disease and its possible implications on risk of rupture. Two parametric studies were conducted using (i) the iliac bifurcation angle and (ii) the AAA neck angulation. Idealized streamlined AAA geometries were employed. The simulations were carried out using both isotropic and anisotropic wall material models. The parameters were based on CT scans measurements obtained from a population of patients. The results indicate that the peak wall stresses increased with increasing iliac and neck inlet angles. Wall shear stress (WSS) and fluid pressure were analyzed and correlated with the wall stresses for both sets of studies. An adaptation response of a temporary reduction of the peak wall stresses seem to correlate to a certain extent with increasing iliac angles. For the neck angulation studies it appears that a breakdown from symmetric vortices at the AAA inlet into a single larger vortex significantly increases the wall stress. Our parametric FSI study demonstrates the adaptation response during aneurysmal disease progression and its possible effects on the AAA risk of rupture. This dependence on geometric parameters of the AAA can be used as an additional diagnostic tool to help clinicians reach informed decisions in establishing whether a risky surgical intervention is warranted.

Keywords Aneurysm · FSI · Risk of rupture · Iliac angle · Neck aneurysmal angle

1 Introduction

Abdominal aortic aneurysm (AAA) rupture is currently the 13th leading cause of death in the United States claiming the lives of approximately 15,000 patients each year [23]. Aneurysmal dilation of the aorta occurs in 2–4% of males over the age of 65 in the western world. The disease risk is increased in patients who have evidence of coronary, carotid, or peripheral vascular disease, hypertension, a history of smoking, or a family history of aneurysmal disease [20, 36, 52, 56]. Its physiological and biomechanical manifestation is increased wall stiffness due to loss of elastin, increased wall stress, drastic differences in wall shear stress (WSS) in different regions of the aneurysm, all of which weakens the aneurysmal region of the aorta, resulting in rupture.

For patients at high risk of rupture, surgical intervention in the form of open surgery or endovascular repair is recommended if the aneurysmal diameter is greater than or equal to 5 cm and has a growth rate of 0.8 cm/year [23, 26], based on patient follow-ups every 6 months or every year. However, this diameter based criterion fails to identify small aneurysms that may rupture [31]. Attempting to refine predictions of AAA rupture in order to help surgeons determine the need for a risky surgical procedure,
several researchers developed models for predicting AAA rupture, based on relevant biomechanical and medical parameters. Kleinstreuer et al. has developed a model which takes into account eight different indices [23, 26]. This model outputs an aneurysmal severity parameter between 0 and 1 which can be used by surgeons in their decision whether to operate the AAA. Watton et al. [53] developed an advanced mathematical model combining mechanical and biological properties that takes into account the vessel wall properties during the development of the aneurysm. Antiga et al. [2] have developed a modeling framework (Vascular Modeling Toolkit) for patient-specific computational hemodynamics. Breeuwer et al. investigated the feasibility of a patient-based hemodynamic modeling of AAA. This methodology is an efficient (improved therapeutic decision) and a robust (insensitive to variation in the quality of input data) approach to risk assessment of AAA [8].

Accurate estimation of the rupture potential of individual AAA requires that the wall strength distribution be taken into account. A statistical model of noninvasive parameters for in vivo calculation of the wall strength distribution was recently developed by the Vorp group [46]. It lump together significant predictors of local AAA strength, both clinical and geometric, to yield a local value of the wall strength. This model can be used to generate 3D mapping of the wall strength that can be compared to the local stress distribution, to generate a rupture potential index, RPI, which is defined as the locally acting wall stress divided by the local wall strength.

Currently, the state of the art approach for modeling the biomechanical behavior of the AAA wall is the fluid structure interaction (FSI) approach, in which the coupling of the aortic wall with the blood flow depicts a dynamic behavior of the AAA during the cardiac cycle. Previously, researches have focused their attention on static structural (SS) simulations as a less computationally demanding approach for modeling the biomechanical behavior of AAA, justified on the basis of small differences between SS and FSI results for particular cases [25]. Substantial work has been done in AAA on SS simulations and mechanical tissue behavior by Vorp and collaborators [32, 33, 45, 49]. Helderman et al. [21] showed, with the use of finite element method (FEM), exponential expansion of aneurysm and revealed that the location of peak wall stress drifts over time. The roles of intraluminal thrombus (ILT) and wall calcifications in patient-specific wall stress of AAA were studied showing the importance of these structures in the evaluation of risk of rupture [10, 42, 51]. Anisotropy of the aortic wall, owing to the collagen fibers orientation within the vessel wall may have a significant impact on wall stress distribution, which needs to be accounted for on the assessment of rupture risk [47]. Geometric features of the aneurysm also play a role in the stresses developing within the AAA wall, with AAA asymmetry playing an important role on the increase of the wall stresses [13]. Excessive bulging of one surface of the aneurysmal wall results in elevation of wall stresses on the opposite surface.

Recently, several researchers have performed FSI simulations in realistic geometries of the abdominal aorta. Scotti and Finol [40] showed that the FSI transient fluid and wall dynamics resulted in a maximum wall stress 21% higher than the SS simulations. Additionally, they have demonstrated that the asymmetry and non-uniform wall thickness of the pathological aorta plays an important role in the assessment of the rupture risk [41]. Di Martino et al. [11] demonstrated that the ILT can significantly reduce the stress on the wall. Papaharilaou et al. [29] presented a decoupled approach for estimating the wall stresses on AAA. Our group studied the role of ILT by applying FSI methodology with patient-specific reconstructed geometries [5]. Recently our group also studied the effect of ILT and calcifications, combined with anisotropic fiber orientation characterization of the wall with patient-based AAA geometries [36, 37].

Individual patient risk factors that were mostly overlooked in the past, may contribute significantly to AAA risk of rupture. Those include anatomical geometric characteristics such as the AAA neck angle and the iliac arteries bifurcation angulation that affect the AAA hemodynamics and undoubtedly contribute to the wall stresses. There were several studies that took into account the contribution of such factors to aneurysmal disease. Some of these studies focus on considerations for endovascular repair with aortic grafts [3, 35, 50]. Others analyze anatomical characteristics of AAA patients like the diameter, length, and volume of the aneurysmal area and iliacs, for determining the potential of elevated risk of rupture [14, 22, 44]. Few studies report the iliac bifurcation and aortic neck angles [3, 35]. The paucity of research in this direction highlights the need for a parametric study of the effect that iliac and neck angulation may have in AAA disease progression and AAA risk of rupture. This will facilitate providing clinicians with better predictive capabilities as to a specific patient AAA progression and risk of rupture.

In this study a three-dimensional FSI methodology is introduced. The methodology takes into consideration the wall tissue anisotropy and the interaction of blood with the aneurysmal wall. The effects of iliac bifurcation and the AAA neck inlet angulations were studied parametrically, based on a range of geometric parameters that were measured in patients.
2 Methods

2.1 Parametric studies in patients’ AAA

To establish the range of angles that were applied to the numerical models, the geometric parameters were measured in a cohort of AAA patients. CT scans were acquired in 26 patients with AAAs and reconstructed in three dimensions using MMS software (MMS, Medical Metrx Solutions Inc., West Lebanon, NH). Several measurements were performed in the reconstructed geometries. These included the iliac bifurcation angles, aneurysmal bending angle, tortuosity, diameters, and AAA lengths. Informed consent was obtained retrospectively. The protocol was approved by Stony Brook University Institutional Review Boards (IRB) Committees on Research Involving Human Subjects.

2.1.1 Iliac bifurcation and aortic bending angles

Two types of iliac bifurcation angles were measured: the lateral bifurcation angle and the anterior–posterior bifurcation angle with respect to the sagittal plane of view. The lateral bifurcation angle was measured using the centerline to mark two points on each of the iliacs and a third point immediately before the bifurcation, thus forming a triangle, as depicted in Fig. 1a (used by permission from MMS).

For the anterior–posterior bifurcation angle, an axial inferior view was obtained and two points were marked on each of the iliac centerlines and another point on the aortic centerline. Two angles were measured for the left and right iliacs, respectively, using the sagittal plane passing through the aortic centerline mark as the reference line. The axial inferior view, iliac angles above the reference line were considered positive and those below the reference line were considered negative, to account for the aortic orientation. To obtain the final anterior–posterior bifurcation orientation, the absolute values of the right and left angles were added.

The bending angle of the aorta was also calculated using the MMS software. Three points were marked on the centerline. One point was chosen at the maximum aortic bending and the other two are located approximately equidistant above and below on the centerline where the aortic orientation returns to normal, as shown in Fig. 1b. The final aortic bending angle was calculated by subtracting the obtained angle from 180°.

2.1.2 Tortuosity measurements

For the evaluation of tortuosity of the iliacs and the aorta a script (MATLAB, The MathWorks Inc., Natick, MA) was developed based on Eq. 1. This formula sums the Euclidian distances between the centerline points defined by the MMS software and divides it by the distance of the straight line formed by the maximum and minimum points on the centerline.

\[
T = \frac{\sum_{i=1}^{N} S_i}{L} \quad \text{where} \quad S_i = \sqrt{(x_i - x_{i-1})^2 + (y_i - y_{i-1})^2 + (z_i - z_{i-1})^2} \\
\text{and} \quad L = \sqrt{(x_{\text{max}} - x_{\text{min}})^2 + (y_{\text{max}} - y_{\text{min}})^2 + (z_{\text{max}} - z_{\text{min}})^2}
\]

(1)

2.1.3 Aneurysm length and diameter

Maximum aneurysm diameter was measured in the region of maximal extent of orthogonal dilation for 25 patients.

Fig. 1 Angulation measurements: a Lateral bifurcation angle as measured from MMS software. b Bending angle of the aorta was calculated using the MMS software.
The aneurysm length was measured axially from the beginning to the end of the aortic dilation (the region where the aneurysm is located). The smaller aneurysmal length identifies a saccular aneurysm where a large aneurysmal length identifies a fusiform one.

2.2.2 Parametric studies in numerical models

Based on the measurements in the patients’ reconstructed AAA geometries described above, a range of the pertaining geometric parameters was established for the streamlined AAA geometries used in the numerical simulations, and a series of parametric studies was conducted. Streamlined ideal AAA geometries with 5 cm (4.8 inner, 5.2 outer) maximum diameter were constructed. The idealized geometries included iliac bifurcation angles that range from $\phi = 30^\circ$ to $\phi = 150^\circ$. All the geometries have a constant length, $l$, of 12.5 cm from the neck to the base of the bifurcation. To reduce the number of variables under consideration, the vessel wall was considered as a material with uniform thickness of 2 mm. We chose to concentrate on the lateral plane iliac angulation effects, because such angulation is more common in human anatomy during aneurysmal disease progression, and because it demonstrates better the effect it has on the wall stress distribution. In addition, angle variations of the AAA neck were considered based on our experimental measurements. These angles range from the vertical, $\theta = 0^\circ$, to $\theta = 40^\circ$. The neck diameter remained constant and similar to the inlet aortic diameter (approx. 2.0 cm) for all neck cases ($0^\circ$–$40^\circ$) and the iliac diameter was 1.0 cm for all cases. The ideal AAA geometry and the various angles for the iliac bifurcation, $\phi$, and the neck of the aorta, $\theta$, are depicted in Fig. 2a.

2.2.1 Material models for the AAA wall

In this study we employ higher order terms in the isotropic Mooney–Rivlin (M–R) strain energy function to account for the departure from neo-Hookean/Gaussian behavior at large stretches [9], combined with the Holzapfel orthotropic material formulations [39]. The Holzapfel strain energy function models the vessel wall as fiber-reinforced composite material with the fibers corresponding to the collagenous component of the material. Similar models that combine higher order isotropic terms with Fung type exponential terms have been used recently to describe the properties of atherosclerotic coronary arteries [55]. The isochoric elastic response for our isotropic material model formulation, $\Psi_{\text{iso}}$, is given in Eq. 2.

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

$$I_1 = \text{tr}(C_{ij}).$$

where $I_1$, is the first invariant of the Cauchy–Green tensor. The isochoric elastic response for the orthotropic material model formulation, $\Psi_{\text{aniso}}$, is given in Eq. 3.

$$\Psi_{\text{aniso}} = \Psi_{\text{iso}} + \frac{k_1}{2k_2} \sum_{i=4,6} [e^{2(k_{ij})(I_1 - 1)^2} - 1],$$

$$J_4 = J^{-1/3} I_4, \quad I_4 = C_{ij}(n_x)(n_x), \quad J_6 = J^{-1/3} I_6,$$

$$I_6 = C_{ij}(n_y)(n_y), \quad J = \det(C_{ij}).$$

where $J$, is the third invariant of the Cauchy–Green tensor $C_{ij}$, $n_x$ and $n_y$ are the directions of the fibers defined by two angles, $a_x$ and $a_y$, which are offset from the material axes. In this phenomenological higher order model formulation it is important to guarantee that the constants chosen must result in realistic and stable constitutive responses in all deformation states [7]. Stability (Drucker stability) was achieved by calculating the tangential stiffness matrix (Hessian) and establishing that it is positive. A nonlinear least-squares estimate was used by our group to fit the strain energy curves to previously published AAA patient specimens experimental data [37].

2.2.2 Governing equations

The fluid domain is governed by the Navier–Stokes and the continuity equations. The Arbitrary Lagrangian–Eulerian (ALE) moving mesh approach was utilized for re-meshing the fluid domain at each time step. The momentum and continuity equations for the fluid domain are shown in Eqs. 4 and 5 for a moving reference frame.

$$\nabla \cdot \ddot{\mathbf{q}} = 0, \text{continuity},$$

$$\rho \left( \frac{\partial \ddot{\mathbf{q}}}{\partial t} + (\dddot{\mathbf{q}} - \dddot{\mathbf{q}}_g) \cdot \nabla \mathbf{q} \right) = -\nabla p + \mu \nabla^2 \dddot{\mathbf{q}}, \text{fluid momentum.}$$

where $\dddot{\mathbf{q}}$ is the fluid velocity vector and $\dddot{\mathbf{q}}_g$ is the local coordinate velocity vector, $p$ is the static pressure, $\rho$ the blood density, and $\mu$ is the dynamic viscosity. The numerical simulations utilized direct coupling between the fluid (blood) and solid (vessel wall) domains. Large strains and large deformations were considered. The dynamics of the flexible wall were calculated using the linear dynamics response of the system shown in Eq. 6 [38].
$M \ddot{\mathbf{U}} + C \dot{\mathbf{U}} + K \mathbf{U} = \mathbf{R}$, solid momentum, \hfill (6)

$M$, $C$, and $K$, represent mass, damping, and stiffness matrices, respectively. $\mathbf{R}$ is the vector of externally applied loads. $\ddot{\mathbf{U}}$, $\dot{\mathbf{U}}$, and $\mathbf{U}$ are the vectors of acceleration, velocity, and displacement of the structural domain. A first order finite-element scheme was used to solve the set of motion and fluid equations using the commercial software Adina (ADINA, ADINA R&D Inc, Watertown, MA).

2.2.3 Boundary conditions

For the fluid domain, the pressure and velocity waveforms were prescribed at the outlet and inlet of the AAA geometry, respectively. At the inlet, velocity waveform spans the entire cardiac cycle with velocity range from 0.3 to $-0.05$ m/s, and outlet pressure boundary condition with range between 123 and 83 mmHg, as shown in Fig. 2b. Blood was modeled as a Newtonian fluid, with a density of 1,050 kg/m$^3$ and a viscosity of 3.5 cP [37]. The flow and pressure waveforms applied were extracted from Olufsen et al. [28]. A no-slip condition was applied at the wall boundaries. Due to the complexity of the geometries, residual stress field in the unloaded configurations was not considered. Instead, all models were assumed to be initially at zero stress state, followed by initial stress loading that was achieved by pressurizing the AAA from 0 to 90 mmHg with zero flow for 1 s, before the FSI waveforms were applied at the inlet and outlet. While this may lead to a minor degree of geometric exaggeration, it is an effective
way to impose physiological fluid pressure boundary conditions. There are several groups who apply specific techniques to prevent geometric inflation [18]. In our future studies we will consider such approaches. For the solid domain, all degrees of freedom were fixed at the inlet and outlets.

2.2.4 Grid independence study

Three different mesh densities were used in order to ensure grid independency of the results. The computational grids are composed of tetrahedral four node finite elements for both fluid and solid domain. Table 1 summarizes the three different computational grids composed of fluid and solid structures. Figure 2c shows the von Mises stress averaged over a cross-section of the solid wall for the three different computational meshes and for the case where the iliac angle is $100^\circ$. A 19.0% difference was observed at the peak systole between the coarse and the fine grids, and only a 5.9% difference at the peak systole between the fine and finer computational meshes. During the diastolic phase the differences between the three grids were very small. Since the difference between fine and finer meshes was small enough, we have performed the large number of parametric simulations using the fine mesh. This mesh provides accurate replicable results, without the need for further refinement that would become computationally prohibitive.

The time step size used in the simulations was $\Delta t = 0.005$ s. We used an automatic time stepping (ATS) method that controls the time step size in order to obtain a converged solution. If there is no convergence with the user-specified time step, the program automatically subdivides the time step until it reaches convergence [1].

2.2.5 Parallel computing

All FSI simulations were performed on a high performance computing cluster composed of four quad core Xeon CPUs with a shared memory of 64 GB RAM. The CPU time for each simulation was approximately 1.6 days for the fine computational grid, increased to 4.1 days for the finer mesh. The coarse grid required only 4.0 h of CPU time. A direct numerical approach was used for the integration of the highly non-linear coupled system of equations. This approach increases the computational time but is considered more appropriate for FSI simulations since it fully couples the solid and fluid matrices. However, there are differing views in the literature for using coupled versus uncoupled numerical solvers [25, 29, 37, 43].

3 Results

3.1 Measurements in patients’ AAA

Several measurements were performed in the reconstructed geometries. These included the iliac bifurcation angles, aneurysmal bending angle, tortuosity, diameters, and AAA lengths. These results are summarized below.

3.1.1 Iliac bifurcation and aortic bending angles

Two types of iliac bifurcation angles were measured: the lateral bifurcation angle (mean: $41.7^\circ \pm 9.7^\circ$) and the anterior–posterior bifurcation angle (mean: $42.5^\circ \pm 21.8^\circ$) with respect to the sagittal plane of view. Additionally, the aortic bending angle was measured (mean: $40.1^\circ \pm 16.3^\circ$). Table 2 represents the mean value and standard deviation for the three angles as well as the maximum and minimum values measured from the studied AAA ($n = 26$). In several patients it was difficult to estimate the length, diameter, and tortuosity of the aneurysmal structure and these patients were not selected for the specific measurements.

3.1.2 Tortuosity measurements

Tortuosity calculations were divided into three categories. In 20 patients, the tortuosity for the right iliac, TRIL, had a mean of 1.137 with a standard deviation of $\pm 0.051$. Likewise, tortuosity for the left iliac, TLIL, had a mean of 1.144 with a standard deviation of $\pm 0.0518$. The aortic tortuosity, TAO, had a mean of 1.030 with a standard deviation of $\pm 0.027$. These results indicate a more tortuous iliac nature with respect to the aortic tortuosity and the left iliac has a larger mean tortuosity than the right iliac by 0.6%, Table 2.

3.1.3 Aneurysm length and diameter

Maximum aneurysm diameter was measured in the region of maximal extent of orthogonal dilation for 25 patients. The mean luminal diameter of the reconstructed aneurysmal geometries was $38.37 \pm 7.99$ mm, and $53.97 \pm 12.09$ mm with the wall and ILT included. The aneurysm length was measured axially from the beginning to the end of the aneurysmal dilation. The mean length was $80.74$ mm with a standard deviation of $\pm 24.43$ mm. The smaller aneurysmal length identifies a saccular aneurysm where a

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Number of tetrahedral elements for grid sizes tested to ensure grid independence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of elements</td>
<td>Coarse mesh</td>
</tr>
<tr>
<td>Fluid domain</td>
<td>17,827</td>
</tr>
<tr>
<td>Solid wall</td>
<td>52,641</td>
</tr>
<tr>
<td>Total</td>
<td>70,468</td>
</tr>
</tbody>
</table>
large aneurysmal length identifies a fusiform one. Our measurements indicate a maximum measured lateral bifurcation angle of 65.2° and the maximum measured anterior–posterior bifurcation angle of 84.8°, in accordance with previously published results [27]. However, additional studies report larger iliac bifurcation angles. Resch et al. [35] report right iliac angles (RIA): 74° ± 39° and left iliac angles (LIA): 81° ± 36°, with a mean value for the total iliac angle at 155°. Beebe et al. [3] report a maximum bifurcation angle of 127°. We have extended the range of the iliac bifurcation angles in our parametric study accordingly to 30°–150°, corresponding to the pathophysiological range reported in the literature.

Based on the above measurements two series of parametric studies were conducted for examining possible critical indicators for AAA vulnerability. In the first group of parametric studies the effects of the iliac angle on the AAA lumen hemodynamics and the resulting wall stresses were investigated. In the second series the role of the AAA inlet neck angle and the effect of the aneurysm angulation were studied.

3.2 Iliac bifurcation angle

In this parametric study, the contribution of AAA iliac bifurcation angle on the wall stresses was explored. The velocity field and the WSS during the cardiac cycle were analyzed; the pressure distribution within the aneurysm was examined during the cardiac cycle. This is a parameter that was not accounted for in many previous studies.

3.2.1 Velocity field

The velocity field is presented in four different time frames of the cardiac cycle for two iliac angles (60° and 120°, Fig. 3). The four time frames are: (1) after the peak systole, 0.33 s, (2) early diastole, 0.55 s, (3) mid diastole, 0.78 s, and (4) late diastole, 0.9 s. The velocity fields were similar for both angles at peak systole. However, the maximum velocity magnitude for the 120° angle geometry was 3% lower than the maximum velocity magnitude for the angulation of 60°. As the cardiac cycle progressed, the flow field developed during diastole into several recirculation zones. At early diastole two counter-rotating vortices were observed near the inlet influencing the inlet jet of the blood entering the aneurysm. These vortices were retained throughout the diastolic period but lost energy towards the early systole, with the forward flow restored. As the cardiac cycle was progressing, this flow reversal was reduced and became almost zero during late diastole. This phenomenon was more pronounced (by 19.5% for the maximum velocity magnitude) for smaller iliac angles (e.g., 60°), as compared to the larger ones (e.g., 120°).

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Mean and SD</th>
<th>Max. value</th>
<th>Min. value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lateral iliac angle (°)</td>
<td>41.7 ± 9.7</td>
<td>65.2</td>
<td>21.1</td>
</tr>
<tr>
<td>Anterior iliac angle (°)</td>
<td>42.5 ± 21.8</td>
<td>84.8</td>
<td>5.0</td>
</tr>
<tr>
<td>Aortic bending angle (°)</td>
<td>40.1 ± 6.3</td>
<td>66.5</td>
<td>6.7</td>
</tr>
<tr>
<td>TRIL</td>
<td>1.14 ± 0.05</td>
<td>1.36</td>
<td>1.03</td>
</tr>
<tr>
<td>TLIL</td>
<td>1.14 ± 0.051</td>
<td>1.36</td>
<td>1.03</td>
</tr>
<tr>
<td>TAO</td>
<td>1.14 ± 0.051</td>
<td>1.36</td>
<td>1.03</td>
</tr>
<tr>
<td>AAA diam. (mm)</td>
<td>38.37 ± 7.99</td>
<td>80.74 ± 24.13</td>
<td>35.4</td>
</tr>
<tr>
<td>Lumen diam. (mm)</td>
<td>38.57 ± 12.09</td>
<td>85.2</td>
<td>40.5</td>
</tr>
<tr>
<td>AAA length (mm)</td>
<td>38.57 ± 12.09</td>
<td>85.2</td>
<td>40.5</td>
</tr>
</tbody>
</table>

Table 2: Measurements extracted from the three-dimensional reconstructions with MMS.
3.2.2 WSS

Abnormally high or low WSS, especially patterns of high followed by low WSS, is a known causative factor of vasoconstriction, coagulation, and platelet aggregation and deposition, and is likely to promote ILT formation [4–6, 30, 34]. The peak WSS was analyzed for several iliac angulations and the WSS distribution near the iliac branches is presented for several angles of 40°, 60°, 100°, 120°, and 150°, correspondingly (Fig. 4, blue dots). The peak WSS of the aneurysm—excluding the iliacs and the stagnation area in the iliac bifurcation where rupture is unlikely to occur, was also plotted for these five angles and compared to the peak WSS of the whole structure. The maximum peak WSS occurred at 60° angle (blue - dark dots, $R^2 = 1$). Locally at the aneurysmal area the trend is opposite with lowest local WSS at 60° angle (red - light dots, $R^2 = 1$).
iliacs and the stagnation area was reduced at 60° iliac angle compared with the other four angles (Fig. 4, red dots). As discussed earlier, the flow reversal due to recirculation zones during early diastole increased the WSS as well.

3.2.3 Pressure distribution

The pressure distribution generated by the dynamic blood flow patterns during the cardiac cycle within the AAA lumen is yet another significant mechanism for increasing the wall stresses and exacerbating the risk of rupture. The results indicate highest pressure, $p$, and highest pressure drop, $\Delta p$, during peak systole. As the iliac angle increased, the pressure inside the lumen changed significantly. The pressure reached its peak value during peak systole at two distinct bifurcation angles of 40° and 100°, respectively (it was approx. 149 mmHg, 1 cm above the iliacs). It was slightly lower for the 60° and 80° angles. As the angle increased the peak pressure decreased (e.g., for 120°, 140°, and 150° peak pressure was approx. 133 mmHg). The pressure drop $\Delta p$, defined as the pressure difference between the inlet pressure and the iliac bifurcation pressure at peak systole, decreased as the iliac angle increased. It remained around 1.0 mmHg at peak systole for small iliac angles through 100° ($\Delta p = \text{approx. 1.0 mmHg}$), and dropped to 0.5 mmHg for 120° and above. Pressure distribution remained almost constant throughout the aneurysmal area and dropped drastically in the iliacs at peak systole and for a 60° iliac angle.

3.2.4 von Mises stress within the solid wall

Analysis was performed for the peak and mean von Mises stress within the aneurysmal wall for the isotropic material model formulation—shown for four representative iliac angles (Fig. 5, ranging from 40° to 140°). The orthotropic model formulation for three iliac angles (40°, 60°, and 100°) was also analyzed and the principal axis stresses were reported. The anisotropic material model formulation predicted higher principal axis stresses than the isotropic formulation. The von Mises stress distribution for these four representative iliac angles indicate that as the angle increased there was an overall reduction of the stress in the aneurysmal wall.

The peak value of the von Mises stress increased as the iliac angle increased (Fig. 5, red dots). At low iliac angulation the peak stress remained less than 1.0 MPa with the lowest value observed at 60° angulation (0.57 MPa). As the angle increased the peak von Mises stress increased, reaching maxima of 1.38 MPa at 100° and 150°,
correspondingly. However, exclusion of the high stress values characterizing the iliac arteries bifurcation stagnation points (where rupture is unlikely to occur) revealed a different pattern of peak stress distribution as a function of the iliac angulation (Fig. 5, green dots), with two minima at 60° and 120°. The peak stresses at 50°, 100°, and 120° angle decreased, but the trend of increasing peak stress with increasing angulation remained. The mean wall stress in the whole domain demonstrated a different pattern, with an overall decrease in the mean stress with increasing iliac angle as depicted in Fig. 5 (blue dots). This decrease in the mean stress can be attributed to the changing hemodynamics within the AAA lumen as the iliac angle increases. This change leads to a formation of an extensive stagnation area and different WSSs distribution in the iliac branches.

3.2.5 Anisotropic material model formulation for iliac angulation

Additional orthotropic simulations were performed for three different iliac angles, 40°, 60°, and 100° and compared with the isotropic simulations. Due to tissue anisotropy principal axis stresses were used in lieu of von Mises stresses (valid only for isotropic material formulation). In all anisotropic simulations the peak stress was higher compared to the isotropic simulations, as reported in our previous studies [37]. In 40° iliac angle the principal axis peak stress, $p_1$, increased for the orthotropic material (32.5% increase). For 60° iliac angle the principal axis peak stress, $p_1$, slightly decreased for the orthotropic material (1.3% decrease) but all other principal axis stresses, $p_2$ and $p_3$, increased by 1.4 and 2.3%, respectively. For 100° iliac angle the principal axis peak stress, $p_1$, increased for the orthotropic material model (25.5% increase). Hemodynamic differences between the two material model formulations were marginal (less than 5%).

3.3 Aneurysmal neck angle

The contribution of AAA inlet angle on the stresses developing within the AAA wall was studied. The FSI simulations predict a strong correlation between inlet angle and peak stress on the aneurysmal wall indicating an increase in wall stresses as the neck angle increased. Velocity field, WSS, and fluid pressure during the cardiac cycle were also analyzed.

3.3.1 Velocity field

The velocity field is presented at four different time frames of the cardiac cycle for inlet angles of 20° and 40°, with an iliac bifurcation angle of 60° (corresponding to the 0° inlet angle in Fig. 3, top). The major effect of the inlet neck angle is that while for the 0° inlet angle a pair of counter-rotating vortices is formed (Fig. 3, top), the neck angulation breaks this symmetric structure and the vortices merge into a stronger single vortex. The flow field for 40° inlet angle was accordingly substantially different from that of 20° and 0°. During systole a stronger jet was observed entering the structure for both neck angles (Fig. 6). This jet was stronger and its maximum velocity was 7% higher than that of the 0°. During diastole the inlet recirculation zone became more pronounced and dominant. The vortex was retained throughout diastole, but lost its energy towards early systole (Fig. 6). The recirculation zone was smaller for the 20° neck angle. During early and mid diastole an additional smaller recirculation zone was observed distal to the inlet for the 20° neck angle.

3.3.2 WSS

The WSS distribution of four different inlet angle configurations, 0°, 20°, 30°, and 40° at peak systole of the cardiac cycle is depicted in Fig. 7. The iliac angle was kept constant for all inlet angle simulations—at 60°. A peak WSS level was observed at 0° inlet angle (2.66 Pa during peak systole). The peak WSS decreased for 20° inlet angle (1.74 Pa), and increased again for larger inlet angles, (1.82 Pa for 30° and 1.95 Pa for 40° during peak systole).

3.3.3 Pressure distribution

As the inlet angle increased, the pressure at peak systole inside the lumen (1 cm above the iliac bifurcation) decreased. It was highest for the zero neck angle (it was approx. 147 mmHg) and decreased to approx. 133 mmHg for 20° and 30° neck angulation, reaching a minimum for 40° (approx. 132.7 mmHg). The pressure drop, $\Delta p$ (the pressure difference between the inlet pressure and the iliac bifurcation pressure at peak systole), also decreased as the inlet angle increased. It was the highest at peak systole for zero inlet angle (approx. 1.0 mmHg) and decreased for 20° and 30° inlet angles (to 0.84 mmHg), reaching a minimum for 40° inlet angle (0.76 mmHg). The pressure distribution remained almost constant throughout the aneurysmal area for all inlet angles and dropped drastically in the iliacs, as also reported in the iliac angulation parametric study.

3.3.4 von Mises stress on the aneurysmal wall

Analysis was performed for the peak and the mean von Mises stresses of the whole wall domain for three inlet angles, ranging from 20° until 40°, Fig. 8. The peak von Mises stress increased as the inlet angle increased. At zero inlet angle the peak stress was 0.57 MPa, increasing to a maximum of 0.93 MPa at 20°. After that a slight decrease
Fig. 6  Velocity field in the aneurysmal area for 20° and 40° inlet angles in comparison to the flow field of Fig. 3 at four different time frames of the cardiac cycle (units: m/s)

Fig. 7  A cross-sectional plane of WSS distribution near the inlet wall for four different inlet angles: 0°, 20°, 30°, and 40° during peak systole (units: Pa)

Fig. 8  Peak von Mises stress versus inlet angle of the AAA for the isotropic material model formulation ($R^2 = 0.97$). It was observed a substantial increase of the peak stress for 20° and after that a decrease to slightly smaller peak stresses
of peak stress was observed (0.81 MPa at 40°). At the same time, a substantial decrease of the mean stress was observed as the inlet angle increased. This decrease was of the order of 9.5% for the case of 40° inlet angle in comparison with 0° inlet angle.

4 Discussion

The FSI methodology presented in this study accounts for fluid pressure dynamics that directly influences the stresses developing within the aneurysmal wall, as well as the WSSs that act on the wall as a result of the dynamically evolving flow patterns within the AAA lumen. Those also strongly depend on the specific geometric features characterizing aneurysmal disease progression and adaptation response. Our study focused on specific geometric parameters that characterize the progression and adaptation response during aneurysmal disease progression, such as marked changes in the iliac bifurcation angulation and changes in the aortic neck angle at the AAA inlet. Idealized streamlined AAA geometries were preferred for this parametric study, to minimize the effects of patient-specific geometric variability. Our parametric studies in these streamlined geometries indicate that in patient-specific geometries, physiological features related to these parameters are likely to amplify the combined contribution of flow and pressure, synergistically acting to elevate wall stresses resulting from these geometry dependent stress concentrations.

4.1 Iliac angle

The blood flow is strongly influenced by the iliac angulation. As the angle increased the flow patterns within the AAA lumen drastically changed, affecting fluid pressure and WSS. Stronger reverse flow and increased velocity gradient near the wall resulted in elevated WSS with highest fluid stresses occurring at 60° angle. The impingement area was increased as the iliac angle increased (Fig. 5). This area increase leads to a decrease of the local WSS at the iliac bifurcation, as depicted in Fig. 4 (blue). Maximum peak WSS was observed at 60° where the flow field was more intense. This maximum peak WSS was located at the iliacs. The blood was exposed to lower peak WSSs as the angle increased and the velocity reduced by 3% from 30° to 60° during systole and almost 20% during diastole-indicating a reduced WSS and fluid pressure in larger angles. The radial displacement was enhanced as the iliac angle increased leading to elevated wall stresses.

Focusing on the aneurysm only, excluding the iliacs and the stagnation area at their bifurcation, a local reduction of the WSS was observed for the 60° angle, as compared to the four other iliac angles studied. Our analysis indicates an increase in the peak wall stresses as the iliac angle increased. The correlation between angle and peak stresses can be attributed to the fact that larger iliac bifurcation angles result in larger flow stagnation areas. Flow impingement on this region combined with increased surface area contributed to the formation of higher stresses within the vessel wall.

Similar behavior was observed in the anisotropic material model formulation. The anisotropic model predicted increased stresses within the wall as compared to the isotropic material mode, while retaining the same correlation between iliac angle and peak stresses as with the isotropic material model formulation. It is clearly more realistic to model the aortic wall as a reinforced material composed of two sets of collagen fibers [39] than an isotropic material model formulation. The anisotropic model predicted increased stresses within the wall (excluding the stagnation area) and was in the range of the measured angles from these 26 patients. The anisotropic material model formulation used in this study showed a similar stress reduction as with the isotropic model for 60° iliac angle.

These findings lead us to hypothesize that there may be a remodeling adaptation mechanism that increases the iliac angle in order to reduce the mean stresses within the wall, as well as reduce the peak wall stresses in the interim. As the disease progresses increase of iliac angle may lead to a temporary reduction of hemodynamic factors like the WSS. We postulate that this is an attempt of the vasculature involved (the abdominal artery and its branching iliacs) to reduce the wall stresses, thus the risk of rupture of the aneurysm itself, by transferring part of the load to the iliacs. In turn this may lead to an iliac aneurysm that is sometimes observed in patients with increased iliac angulation. The theoretical foundation for such hypothesis of remodeling adaptation mechanism that may increase the iliac angle in order to reduce the mean stresses within the wall, as well as reduce the peak wall stresses in the interim, is motivated by classic papers by Y. C. Fung who correlated the stresses to the remodeling of blood vessel wall and introduced a stress-growth law [16]. In his book he further introduced the notion that the remodeling of a vessel is a three-dimensional phenomenon since the stress
changes are usually non-homogeneous [17]. The remodelling of the vessel wall due to stress variation is further analyzed in later works [12, 19]. Specifically, while conducting our extensive parametric study of the effects of iliac angulation, we have discovered the existence of two minima in the peak stresses at specific iliac angles. This led us to hypothesize that it may represent an adaptation response where the vasculature attempts to reduce the stresses in the AAA. However, in the lack of information regarding the rate of change of the iliac angulation in patient-based geometries further studies are required in order to confirm this hypothesis.

Consistent with the above finding was the observed decrease of the pressure drop, $\Delta p$, in the aneurysmal area during peak systole as the iliac angle increased. The larger angle made it more difficult for blood to flow from the aneurysm into the iliacs. A substantial increase of the wall displacement normal to the direction of flow (radial displacement) was also observed. It was more evident for larger iliac angles, and was the largest for 140°. Pressure distribution remained almost constant throughout the aneurysmal area and drastically dropped in the iliacs for all angles. The regions approaching the iliac bifurcation experienced the highest pressure drops, which may be related to regions more susceptible to rupture.

4.2 Inlet aneurysmal angle

The neck aortic angle parametric studies revealed an increase of the peak stress in the aneurysmal wall as the inlet angle increased. However, there was a substantial decrease in the mean stress as the inlet angle increased. Recirculation zone was present during systole and was more pronounced during diastole. The recirculation zone decreased the cross-sectional area available for the incoming flow through the inlet, thus producing a stronger inlet jet. The pressure drop, $\Delta p$, of the blood inside the lumen decreased as the inlet angle increased, similar to what was observed in the previous iliac angulation parametric studies. At peak systole, non-zero inlet angles produced lower peak WSS values. The above results may indicate a remodeling mechanism of the aorta, likely intended to restore closer to normal physiological flow conditions. Changes in the inlet angle reduced the peak fluid pressure, the pressure drop and altered the flow field. This led to a reduced WSS which was related to lower mean von Mises stresses. The increased neck angle lowered the mean stresses while increased the peak stresses. The overall reduction of the mean stress was probably not as significant as compared to the elevated peak wall stress that led to an increased risk of rupture. In previous work in patient-based geometries using the FSI methodology by our group we observed that regions of high stress concentrations are found at the neck of the aneurysm, supporting our findings in this parametric study for the effect of the neck angle [54]. For the iliac angle, in our previous studies we have found that severity of the condition correlates with larger iliac angles. These results are in accordance with our current FSI calculations in idealized geometries. However, our previous studies in a small number of patient

<table>
<thead>
<tr>
<th>Subject</th>
<th>Subject representation</th>
<th>Age</th>
<th>Max diam. (cm)</th>
<th>Inlet diam. (cm)</th>
<th>AAA length (cm)</th>
<th>Iliac angle (degrees)</th>
<th>Isotropic peak stress (MPa)</th>
<th>$p_1$ peak stress, ILT no Ca (MPa)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td></td>
<td>66</td>
<td>1.80</td>
<td>1.80</td>
<td>--</td>
<td>38.8</td>
<td>0.44</td>
<td>0.54</td>
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<tr>
<td>non-rAAA</td>
<td></td>
<td>--</td>
<td>5.73</td>
<td>2.15</td>
<td>6.98</td>
<td>35.8</td>
<td>1.36</td>
<td>--</td>
</tr>
<tr>
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<td>2.35</td>
<td>10.0</td>
<td>50.9</td>
<td>1.09</td>
<td>1.37</td>
</tr>
<tr>
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<td>2.58</td>
<td>11.3</td>
<td>84.0</td>
<td>2.10</td>
<td>2.17</td>
</tr>
</tbody>
</table>

non-rAAA nor-ruptured AAA, rAAA ruptured AAA, ILT intraluminal thrombus, Ca calcification
reconstructed AAA geometries [54] (2 normal aortas, 1 non-ruptured, and 2 ruptured AAA) cannot provide statistically significant results about the effects of the iliac angle. In Table 3 we present several cases from this Xenos et al. study [54] that illustrates the significance of the iliac angle in realistic patient-based geometries. It is interesting to note that the ruptured AAA with the largest iliac angle was not the largest AAA diameter-wise, however, it exhibited the highest wall peak stress.

4.3 Limitations of the current study

There are very few studies that attempt to examine geometric and biomechanical parameters that characterize aneurysmal disease and its progression. A systematic parametric study of AAA entails a large number of computationally demanding numerical simulations that dictate applying several simplifying assumptions. The flow was considered laminar in all regimes (systole and diastole) of the cardiac cycle. This assumption may underestimate the magnitudes of the stresses generated by the fluid–structure interaction. While this can be remedied by incorporating advanced turbulence models suitable for transient flows, it may handicap such parametric studies. In all the simulations a uniform wall thickness of 2 mm was assumed. This may lead to underestimation of the maximum wall stress if a certain decrease in local wall thickness is present. The anisotropic model assumes a uniform fiber orientation and does not take into consideration possible effects of the wall degradation, e.g., gradual and local weakening of the fibers [24]. The use of an idealized AAA geometry made it possible to conduct this parametric study in an efficient way. However, this study establishes important geometric parameters that characterize aneurysmal disease and may have cardinal effects on its progression. A natural next step will be to apply the same approach to patient-based geometries, although the control over the parametric changes will be limited. The maximum difference of 5.9% between fine and finer meshes used in this study may appear higher than the commonly regarded threshold. However, this difference occurs only at peak systole and not throughout the cardiac cycle (during diastole the difference is almost zero). Since strongly coupled FSI simulations are very demanding we had to choose a trade-off between accuracy and efficiency.

The outflow boundary condition used in this study can easily lead to the creation of reflecting pressure waves but we would expect these reflections to provide fluctuations in the pressure field rather than increasing the pressure because of the formation of complex flow patterns. More details about pressure waves can be found elsewhere [54]. In many situations the outflow pressure is not known or needs to be measured. An alternative approach is to couple the solution at the outflow with lumped parameters or one-dimensional models of the downstream domain [15, 48]. The maximum aneurysmal diameter and the ratio between iliac legs to AAA diameter remained constant in all simulations. In future studies we will consider the parametric investigation of the relationship between AAA diameter and iliac angle or iliac length.

In conclusion, a large parametric study was conducted in order to delineate disease progression and adaptation responses during aneurysmal disease, and the possible effects that such geometric and biomechanical parameters have on AAA risk of rupture. The FSI methodology employed represents a dynamic picture of the AAA during the cardiac cycle and is more accurate than static pressurization or fluid flow studies alone. Peak von Mises stress within the AAA indicates a general trend of increasing stress as the iliac angle increased. A closer examination, by excluding stresses at the iliac bifurcation stagnation point region where rupture is unlikely to occur, indicates that there are two local minima at 60° and 120° iliac bifurcation angles. Anisotropic material model formulation predicted higher principal axis stresses than the isotropic formulation. The strong correlation between angle and stress is also observed in the anisotropic formulation. Total WSS peaked at 60° iliacs angle and decreased for larger iliac angulation. Locally on the aneurysmal area the WSS was the smallest at 60° iliacs angle compared to the other four. Highest pressure and highest pressure drop occurred during peak systole, with luminal pressure drop decreasing as the iliac angle increased. Peak wall stress increased as the inlet angle increased. However, there was a decrease of the mean stress as the inlet angle increased. The peak WSS decreased for 20° inlet angulation and increased again for larger inlet neck angles, 30° and 40°. The luminal pressure drop decreased as the inlet angle increased.

Our simulations indicate a possibility that the adaptation response of the body during aneurysmal disease progression may lead to particular iliac bifurcations and inlet neck angles that may offer a certain reduction of the peak wall stresses in the interim. However, additional studies are needed to confirm whether the formation of these “optimal” angles is indeed an adaptation response during aneurysmal disease, and for how long these angles can be maintained during the AAA progression. It will require further monitoring of the AAA progression in patients, to indicate whether such angles are maintained for a while, or whether those are arbitrary chance manifestations during the disease progression.

References


