A patient-specific computational model of fluid–structure interaction in abdominal aortic aneurysms

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Abstract

It is generally believed that knowledge of the wall stress distribution could help to find better rupture risk predictors of abdominal aortic aneurysms (AAAs). Although AAA wall stress results from combined action between blood, wall and intraluminal thrombus, previously published models for patient-specific assessment of the wall stress predominantly did not include fluid-dynamic effects. In order to facilitate the incorporation of fluid–structure interaction in the assessment of AAA wall stress, in this paper, a method for generating patient-specific hexahedral finite element meshes of the AAA lumen and wall is presented. The applicability of the meshes is illustrated by simulations of the wall stress, blood velocity distribution and wall shear stress in a characteristic AAA. The presented method yields a flexible, semi-automated approach for generating patient-specific hexahedral meshes of the AAA lumen and wall with predefined element distributions. The combined fluid/solid mesh allows for simulations of AAA blood dynamics and AAA wall mechanics and the interaction between the two. The mechanical quantities computed in these simulations need to be validated in a clinical setting, after which they could be included in clinical trials in search of risk factors for AAA rupture.

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Keywords: Abdominal aortic aneurysm; Patient-specific mesh; Computational fluid dynamics; Fluid–structure interaction; Wall stress; Wall shear stress

1. Introduction

An abdominal aortic aneurysm (AAA) is a dilatation of the abdominal aorta which may be prone to rupture. In current clinical practice, surgical treatment of AAA is generally considered necessary when the maximal diameter exceeds 5–6 cm [1]. Although rupture is highly related to increased aneurysm size [2,3], rupture also occurs in smaller aneurysms [1,4]. To find more accurate rupture risk predictors, both geometric properties other than the diameter and non-geometric patient characteristics have been considered [5,6]. Yet, due to recent improvements in imaging and segmentation, the most prominent trend in AAA rupture risk assessment is to study the mechanical behaviour of AAA by means computational models [7–20]. As rupture is characterised by mechanical failure, it is generally believed that the wall stress distribution computed with these models could help to better assess AAA rupture risk.

Several studies of AAA wall stress by means of computational models have been reported in literature. Models based on idealised geometries were used to show that AAA wall stress is significantly reduced in the presence of intraluminal thrombus (ILT), depending on its size and constitutive properties [8–10,18]. Similarly, the presence of atherosclerotic plaques was shown to have an increasing effect on the wall stress [8]. Further, the wall stress distribution was shown to be dependent on the actual shape of the AAA as well as...
its maximum diameter [7,11,13]. As the in vivo shape of AAA is highly complex and far from axisymmetric [21], this means that for patient-specific wall stress assessment, finite element models need to be based on detailed three-dimensional descriptions of the AAA geometry. Indeed, models based on patient-specific geometries show complex wall stress distributions [12,15,16], which again are influenced by the presence of ILT [17]. In some studies, the peak wall stress has been related to arterial wall remodelling [23] and specific patterns of high and low WSS occurring in aneurysms have been related to thrombus formation [24], modelling the WSS could be essential for modelling the pathogenesis of AAA and its development over time.

2. Methods

2.1. Imaging and segmentation

The patient-specific geometry used in this study is derived from a CT angiographic (CTA) image of an AAA that contained virtually no thrombus. The image was acquired at the University Hospital Maastricht using a Toshiba Aquilion spiral CT scanner (Toshiba Medical Systems Corporation, Tochigi, Japan). A typical AAA CTA protocol was applied, in which the scan was performed during one breath-hold of the patient to reduce motion artefacts. The slice thickness was 2 mm and the 2D image size was 400 mm × 400 mm. At a resolution of 512 × 512, the resulting voxel size was 0.78 mm × 0.78 mm × 2 mm. A coronal cross section of the image is depicted in Fig. 1(a)–(c).

From the CTA image, descriptions of the centrelines and the boundary surface of the lumen of the AAA are determined by means of semi-automated centreline tracking and segmentation using a 3D Active Object (3DOM) [25]. The centre-lines are tracked between user-specified end points using a minimum cost path approach [26]. The 3DAO-technique of segmentation is a deformable-model type of technique, a prototype of which is implemented in the EasySCHL software platform, a development version of the EasyVision clinical image processing workstation (Philips Medical Systems, Best, The Netherlands) [27]. The 3DOM is initialised in a tubular shape around the tracked centrelines, after which it is incrementally inflated until it matches the lumen boundary. The image considered in this study was segmented between

<table>
<thead>
<tr>
<th>Nomenclature</th>
<th>Description</th>
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<tbody>
<tr>
<td>( a )</td>
<td>viscosity model parameter</td>
</tr>
<tr>
<td>( A )</td>
<td>inflow boundary area (m²)</td>
</tr>
<tr>
<td>( B )</td>
<td>Finger tensor</td>
</tr>
<tr>
<td>( d )</td>
<td>projection distance (m)</td>
</tr>
<tr>
<td>( D )</td>
<td>rate of deformation tensor (s⁻¹)</td>
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<tr>
<td>( F )</td>
<td>deformation gradient tensor</td>
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<tr>
<td>( G )</td>
<td>shear modulus (Pa)</td>
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<tr>
<td>( h )</td>
<td>transformation increment</td>
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<tr>
<td>( n )</td>
<td>viscosity model parameter</td>
</tr>
<tr>
<td>( m )</td>
<td>surface or boundary normal</td>
</tr>
<tr>
<td>( N )</td>
<td>set of nodal point indices</td>
</tr>
<tr>
<td>( p )</td>
<td>hydrostatic pressure (wall deformation problem) (Pa), wall pressure load (wall deformation problem) (Pa), fluid pressure (blood flow problem) (Pa)</td>
</tr>
<tr>
<td>( q )</td>
<td>flow rate (m³ s⁻¹)</td>
</tr>
<tr>
<td>( t )</td>
<td>time (s)</td>
</tr>
<tr>
<td>( t )</td>
<td>tangential boundary vector</td>
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<tr>
<td>( v )</td>
<td>fluid velocity (m s⁻¹)</td>
</tr>
<tr>
<td>( w )</td>
<td>displacement weight</td>
</tr>
<tr>
<td>( \omega )</td>
<td>fluid domain velocity (m s⁻¹)</td>
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<tr>
<td>( x )</td>
<td>point coordinates (m)</td>
</tr>
</tbody>
</table>

Greek letters:

| \( \gamma \) | shear rate (s⁻¹) |
| \( b \) | displacement (m) |
| \( \varepsilon \) | displacement criterion (m) |
| \( \eta \) | fluid viscosity (Pa s) |
| \( \lambda \) | smoothing factor (mesh generation), viscosity model parameter (fluid problem) (s) |
| \( \rho \) | fluid density (kg m⁻³) |
| \( \sigma \) | Cauchy stress tensor (Pa) |
| \( \tau \) | wall shear stress magnitude (Pa) |
slightly above and below the levels of the renal arteries and the iliac bifurcation, respectively. The tracked centrelines are shown in Fig. 1(a). The tubular and final shapes of the 3DAO are shown in Fig. 1(b) and (c).

2.2. Mesh generation

In order to generate a patient-specific combined mesh of the AAA lumen and wall, a standardised bifurcated mesh is transformed based on the centrelines and the boundary surface of the segmented AAA. The standardised mesh comprises both fluid and solid domains. The geometry of the mesh is based on the shape of three intersecting tubes, as shown in Fig. 2(a). The topology of the fluid domain is chosen such that it contains smaller elements at locations closer to the wall. Both the fluid and wall domains consist of quadratic 27-noded elements. The boundary surface is represented by a surface triangulation (see Fig. 2(b)). Due to the bifurcation, there are two centrelines, each running from the proximal end of the trunk of the bifurcation to either of the left and right distal branch ends (see Fig. 2(c)). The centrelines are represented by sets of discrete points.

First, based on the centrelines, the fluid domain of the standardised mesh is transformed into a patient-specific initial shape that is orientated along the lumen of the AAA. To this end, the original centrelines are reconfigured as depicted in Fig. 2(c), and of each of the centrelines a continuous description is determined (see Appendix A). Next, the fluid domain of the standardised mesh is transformed to an initial patient-specific mesh, that subsequently is orientated along the reconfigured centrelines. The latter transformation is based on local coordinate systems that are defined along the centre-
lines based on their continuous descriptions (see Appendix B). Finally, to correct for distorted elements that may occur at the connection of the trunk and the two branches, Laplacian smoothing is applied to the nodal points of the mesh. For a point $x_i$, a new position $x'_i$ is calculated as

$$x'_i = x_i + \lambda \Delta x_i,$$  

with $\lambda$ a scale factor and $\Delta x_i$ the discrete Laplacian, defined as

$$\Delta x_i = \sum_{j \in N_i} w_{ij} (x_j - x_i),$$  

with $N_i$ the set of indices of the nodal points connected to point $i$ through element edges and $w_{ij}$ the weights of these points, defined as

$$w_{ij} = \frac{||x_i - x_j||^{-1}}{\sum_{k \in N_i} ||x_k - x_i||^{-1}}.$$  

In this procedure, the planarity of the inflow and outflow surfaces is retained by means of hierarchical constraints, as described by Taubin [28]. The procedure is repeated 10 times with $\lambda = 0.25$. These parameters were chosen to obtain an only moderate degree of smoothing, to prevent the collapse of outer layer mesh elements.

Next, based on the triangulated boundary surface, the initial fluid mesh is incrementally deformed to eventually fit the segmented AAA. First, for every point of the fluid/solid boundary of the mesh, the projection distance $d$ to the triangulation is determined as described in Appendix C. The transformation of the mesh into the patient-specific shape is determined by solving a linearly elastic deformation problem, where the inflow and outflow boundaries are constrained to in-plane motion. To each of the fluid/solid boundary points, a displacement is prescribed in a direction $n$ that is determined by Laplacian smoothing of the local surface normal $n$ (see Fig. 10(e)). The smoothing procedure, necessary to prevent mesh warping, is again described by (1)-(3), where in (1) and (2), $x$ is substituted by $n$. To obtain a high degree of smoothing, the procedure is repeated 50 times with $\lambda = 0.5$. After every step, $n$ is normalised. The displacement $\delta$ of a point is based on $d$, where again Laplacian smoothing is applied between neighbouring points. This time, the smoothing procedure is only repeated 10 times. To promote smooth mesh deformation, the mesh is transformed in an incremental fashion, where in every increment, $\delta$ equals $d/2$. In addition, during a selected number of initial steps, $\delta$ is limited to $d$ divided by the number of initial increments left:

$$\delta = \frac{d}{h_i} \quad \text{for} \ h < h_i,$$

$$\delta = \frac{d}{2} \quad \text{for} \ h \geq h_i,$$  

with $\delta$ the increment and $h_i$ the number of initial increments. The transformation process is defined to converge after the increment when the mean variation of $d$ over all boundary points has decreased below a criterion $\varepsilon$:

$$\bar{d}[h_i - d|h_{i-1} \leq \varepsilon, (5)$$

with $\bar{d}[h]$ the mean projection distance at increment $h$ and $h_i$ the increment of convergence.

Next, the wall mesh is generated by transformation of the solid domain of the standardised mesh based on the determined shape of the fluid domain. First, for every node of the mesh, an associated node on the inner wall surface is found, based on the constant topology of the different layers of nodal points throughout the thickness of the wall. Next, various nodal points are orientated in the direction of the normal to the inner wall surface, evaluated at the associated nodes. To prevent mesh warping, again Laplacian smoothing is applied to the surface normals. Further, the wall cross sections at the inflow and outflow boundaries of the lumen mesh are oriented in the planes associated with those boundaries. The relative transversal nodal point positions with respect to the inner wall surface are retained in the transformation. The total wall thickness is set to a characteristic value.

Finally, to correct for small element distortions that may occur in the transformation of the lumen and wall meshes, the positions of the edge and face mid-points with respect to the vertices of all elements are adjusted. Each of the edge mid-points is translated to the centre of a parabolic curve fitted through the edge end-points and the mid-point itself. Next, each of the face midpoints is determined from the points along the face edges using Serendipity shape functions [29]. These functions are also applied to finally determine the new positions of the element centroids.

2.3. Wall deformation and blood flow models

The stress and strain distributions in the AAA wall are computed by solving the equations of motion and continuity on the solid domain $\Omega_s$ of the AAA model. The inertial and gravitational forces acting on the wall are neglected because they are of a lesser order of magnitude than the wall forces associated with the intravascular pressure load. For an incompressible material with spatial coordinates $x$, the equations then read

$$\nabla \cdot \sigma = 0 \ \text{in} \ \Omega_s, \ \det(F) - 1 = 0 \ \text{in} \ \Omega_s,$$  

with $\sigma$ the Cauchy stress tensor and $F = (\gamma(x))^T$ the deformation gradient tensor with respect to the reference state. The AAA wall is considered a linearly elastic neo-Hookean material, the Cauchy stress tensor of which is defined by

$$\sigma = 0 + G(B - I),$$  

with $\rho$ the hydrostatic pressure, $I$ the unit tensor, $G$ the shear modulus and $B = FF^T$ the Finger tensor. As for the boundary conditions, at the cross sectional surfaces at the inflow and the two outflow locations, $\Gamma_{in}$, $\Gamma_{o1}$ and $\Gamma_{o2}$, respectively, all displacements are constrained. At the fluid/solid boundary,
\( \Gamma_{\text{ex}} \), the normal stress is prescribed in terms of a given pressure load \( p \), while the outer wall boundary \( \Gamma_{\text{sw}} \) is considered stress-free in normal direction:

\[
\mathbf{x} = \mathbf{x}_0 \quad \text{in} \quad \Gamma_{\text{ex}} \cup \Gamma_{\text{ex}} \cup \Gamma_{\text{sw}},
\]

\[
(\sigma \cdot \mathbf{n}) \cdot \mathbf{n} = -p \quad \text{in} \quad \Gamma_{\text{ex}}, \quad (\sigma \cdot \mathbf{n}) \cdot \mathbf{t}_j = 0 \quad \text{in} \quad \Gamma_{\text{ex}},
\]

\[
\mathbf{n} \cdot \mathbf{n} = 0 \quad \text{in} \quad \Gamma_{\text{sw}} \tag{8}
\]

with \( \mathbf{n} \) the outward boundary normals and \( \mathbf{t}_{ij} \) the tangential boundary vectors.

The blood velocity and wall shear stress distributions in the AAA are computed by solving the Navier–Stokes equations on the deforming fluid domain \( \Omega_1 \) of the AAA model based on the arbitrary Lagrangian–Eulerian (ALE) method [22]. The gravitational forces acting on the blood are neglected, since the effects of geometrical factors independent of body posture are of primary interest. For an incompressible fluid with velocity \( \mathbf{v} \) and density \( \rho \), the ALE formulation of the Navier–Stokes equations then reads:

\[
\begin{align*}
\frac{\partial \mathbf{v}}{\partial t} + \rho (\mathbf{v} \cdot \nabla) \mathbf{v} - \nabla p + \frac{1}{\rho} \nabla \cdot \tau &= 0 \quad \text{in} \quad \Omega_1, \\
\nabla \cdot \mathbf{v} &= 0 \quad \text{in} \quad \Omega_1, \\
\end{align*}
\tag{9}
\]

where \( t \) denotes time and with \( \mathbf{w} \) the velocity of the fluid domain. Blood is considered an inelastic generalised Newtonian fluid, the Cauchy stress tensor of which is defined by

\[
\tau = \eta \left( \frac{\partial \mathbf{v}}{\partial t} + \frac{1}{2} \nabla (\mathbf{v} \cdot \mathbf{v}) \right) + \lambda \nabla \cdot \mathbf{v} \tau
\]

with \( \eta \) the viscosity and \( \lambda \) the stress-free in normal direction:

\[
\tau = \eta \left( \frac{\partial \mathbf{v}}{\partial t} + \frac{1}{2} \nabla (\mathbf{v} \cdot \mathbf{v}) \right) + \lambda \nabla \cdot \mathbf{v} \tau
\]

The simulations are performed on a single 900 MHz Itanium II processor of a SGI Altix system. In order to obtain

\[
\mathbf{v} = \mathbf{v}_0 \quad \text{in} \quad \Gamma_{\text{in}}, \quad (\mathbf{v} - \mathbf{w}) \cdot \mathbf{n} = 0 \quad \text{in} \quad \Gamma_{\text{ex}},
\]

\[
\mathbf{n} \cdot \mathbf{n} = 0 \quad \text{in} \quad \Gamma_{\text{sw}} \cup \Gamma_{\text{sw}}, \quad \mathbf{v} - \mathbf{w} = 0 \quad \text{in} \quad \Gamma_{\text{sw}}, \tag{13}
\]

with \( q \) a prescribed flow rate and \( A \) the area of the inflow boundary. The wall shear stress magnitude \( |\tau| \) at the fluid/solid boundary is determined by simple multiplication of the local shear rate with the viscosity:

\[
|\tau| = \eta (\gamma) |\gamma| \tag{14}
\]

More detailed information on the applied equations can be found by van de Vosse et al. [22].

2.4. Implementation and simulations

In order to provide a computed fluid domain velocity to the blood flow simulation, the wall deformation and blood flow problems are solved in a successive manner. First, the wall deformation problem is solved, giving the time-varying displacement field of the wall mesh. From the displacement field, the time-varying wall stress distribution is determined using the wall constitutive behaviour. Next, the motion of the fluid mesh is computed by solving a linearly elastic deformation problem defined on the fluid domain, where the motion of the fluid/solid boundary surface is used as boundary condition. Finally, the blood flow problem is solved, giving the blood velocity and pressure fields with respect to the deforming fluid mesh. From the velocity field, the time-varying wall shear stress distribution is computed using the blood viscosity model. All sets of equations are consequently linearised, discretised and solved using the SEPRAN finite element package [31]. For the interested reader, details on the finite element method can be found for instance in Zienkiewicz and Taylor [29] and Cuvelier et al. [32].

The boundary conditions imposed in the simulations are based on pressure and flow rate signals in the healthy aortic bifurcation as respectively simulated and measured by Olufsen et al. [33]. Both signals are scaled to obtain a time period of 1 s. The prescribed pressure load \( p \) in the wall deformation simulation equals the deviation from the minimum or end-diastolic value of the concerned pressure signal (see Fig. 3). The flow rate \( q \) in the blood flow simulation is scaled to obtain a peak-systolic Reynolds number of 1000, based on the average inlet diameter and the viscosity at infinite shear rate (see Fig. 3).

The wall and blood properties applied in the simulations are set in the order of magnitude of properties applied in literature. In the wall deformation simulation, the shear modulus of the wall material is set to 1.0 MPa, based on the elastic modulus of 2.7 MPa applied by Di Martino et al. [14]. The wall thickness is set to 2 mm, based on measurements by Raghaban and Vorp [12] and Thubrikar et al. [34]. In the blood flow simulation, the parameters of the Carreau–Yasuda viscosity model are determined by least-squares approximation of blood viscosity measurements by Thurston [35], yielding \( \eta_0 = 5.19 \text{ mPas} \) and \( \eta_\infty = 4.76 \text{ mPas} \) and \( \alpha = 0.409 \text{ } \eta_0 \text{ } 0.191 \text{ } \lambda = 0.438 \text{ s} \) and \( \eta_\infty \text{ } \). The density is set to 1080 kg m\(^{-3}\) [30].

The simulations are performed on a single 900 MHz Itanium II processor of a SGI Altix system. In order to obtain
a time periodic solution in the blood flow simulation, four consecutive periods are simulated. The results are evaluated by considering the last simulated period.

3. Results

The incremental transformation of the initial lumen mesh results in a patient-specific mesh that closely resembles the triangulated boundary surface of the segmented AAA (see Fig. 4). Based on the desired minimum variation of the mean distance of the lumen mesh boundary points towards the triangulated boundary surface of $5 \times 10^{-3}$ mm and 8 initial steps, the transformation process converges in 16 increments (see Fig. 5). During the transformation, at the level of the bulge, the initial mesh is inflated to a great extent, while in the inflow region, the mesh is constricted. In the iliac arteries, the degree of inflation varies with the axial position. Both at the bifurcation and in other mesh parts, the inflation process results in a regular element distribution. The transformation of the solid part of the standardised mesh yields a wall geometry that closely surrounds the shape of the lumen (see Fig. 4(d)).

The wall stress distribution in the AAA is characterised by a global increase of stress around the bulge combined with more localised areas of increased and decreased stress distributed over the aneurysm (see Fig. 6). The highest stresses occur at these localised areas. Furthermore, high stresses occur at the apex of the bifurcation at the inner wall. Although the wall stress distribution on the inner wall differs from that on the outer wall, the two are closely related. Either aside or opposite to stress concentrations on one side of the wall, on the other side similar or complementary concentrations occur.
The blood velocity distribution in the AAA is characterised by the consecutive formation and diminishing of 3D vortices (see Fig. 7). At end-diastole, there are few remains of the vortices formed in the previous flow cycle. In the iliac arteries, there is reversed flow near the wall. At peak-systole, all remaining vortices have been washed out and except for a small area of flow separation at the inner bend of the inflow region, there is an attached flow pattern. During systolic deceleration, the area of flow separation has grown into a large 3D vortex migrating from the wall. In the bifurcation region and at local dilatations in the iliac arteries there is vortex formation as well. At end-systole, the vortex in the inflow region has promoted a complicated flow pattern that balances the reversed flow at the inflow boundary. In the iliac arteries, the vortices have migrated towards the main stream and there are large areas of reversed flow near the wall. At mid-diastole, the vortices present at end-systole have translated and/or diminished and areas of reversed flow are not present.

The WSS distribution in the AAA comprises a time-varying pattern of both regions of high and low WSS that are determined by both the local diameter and locally occurring vortices (see Fig. 8). At all instants, the WSS in the bulge is very low compared to the WSS in the inflow region and in the iliac arteries. At end-systole, the bulge WSS has increased. The gradients over the bulge have increased as well, both at the proximal and the distal side. In

Fig. 6. Maximal principal wall stress on the deformed geometry at peak-systole: anterior/exterior (a); posterior/exterior (b); posterior/interior (c); anterior/interior (d).

Fig. 7. Blood velocity distribution in the deforming geometry at end-diastole (a), peak-systole (b), late-systole (c), end-systole (d), and mid-diastole (e) in the fourth period.
the iliac arteries, the WSS is still mainly determined by the diameter. During systolic deceleration, both the bulge WSS and the distal bulge gradient have decreased. At the proximal side, a local area of high WSS has migrated in distal direction. At the outer bend of the inflow region, the WSS has decreased. In the iliac arteries, the WSS has become less dependent on the diameter. At end-systole, the WSS has rather evened out than decreased. Yet, the proximal bulge gradient is still present. Further, in the iliac arteries, the WSS is not primarily determined by the diameter. At mid-diastole, the WSS and WSS gradients have diminished to a great extent. The effect on the WSS of the iliac artery diameter has once again become dominant.

The computational time needed for the simulations approximated 6 days per simulated period. It is expected that this time can be reduced by optimisation of the applied computational algorithms, but this was not pursued in the current study.

4. Discussion

In order to facilitate the incorporation of fluid/structure interaction (FSI) in the assessment of AAA wall stress, a method for generating patient-specific hexahedral finite element meshes of the AAA lumen and wall has been presented. In this method, first, the fluid domain of a standardised bifurcated fluid/solid mesh is transformed into an initial shape that is oriented along the centrelines of a segmented AAA. Next, the initial mesh is incrementally transformed to eventually fit a boundary surface triangulation of the segmented AAA. Finally, the wall mesh is generated by transformation of the solid domain of the standardised mesh based on the determined shape of the lumen mesh. The applicability of the meshes has been illustrated by simulations of the wall stress, blood velocity distribution and wall shear stress in a characteristic AAA.

Although the considered method may take more effort than direct meshing by means of Delaunay methods, it is expected to limit the computational cost of rupture risk assessment models incorporating FSI. First, the application of hexahedral elements provides for more accurate descriptions of the computed quantities than the use of tetrahedral elements [22]. Second, the application of standardised meshes allows for pre-defined element distributions, which can implicitly limit the amount of elements needed to describe certain geometries and computed quantities. Since patient-specific blood-wall interaction simulations are associated with considerable amounts of computational time, increased efficiency of such simulations could be a decisive factor in their clinical impact.

The main assumption in the method of mesh generation as it has been presented is that the segmented aneurysm does not contain thrombus. Irrespective of the morphology of the structures surrounding the lumen, the wall mesh is placed directly against the fluid mesh. To incorporate a thrombus model, the applied surface triangulation should represent the inner boundary of the wall rather than the outer boundary of the lumen. A mural thrombus could then possibly be modelled either by incorporating a standardised thrombus model in the initial mesh and deforming its inner boundary to fit the outer boundary of the lumen or by incorporating the thrombus in the wall model and spatially varying their combined thickness and mechanical properties. A more distributed thrombus could possibly only be modelled using the latter approach.

For the AAA considered in this study, the hexahedral mesh obtained with the presented method closely represents the characteristics of the boundary surface triangulation of the segmentation. To what extent the mesh matches the vascular...
a characteristic blood flow velocity, flow-induced pressure mainly determined by the arterial pressure wave. With that in general, local pressures in the arterial system are using a decoupled approach. This is motivated by the fact it is assumed that the combined behaviour can be solved in the order of 1 mm, the accuracy of the surface triangulation is the main determining factor. 

In the simulations of blood/wall interaction in the AAA, it is assumed that the combined behaviour can be solved using a decoupled approach. This is motivated by the fact that in general, local pressures in the arterial system are mainly determined by the arterial pressure wave. With \( U \) a characteristic blood flow velocity, flow-induced pressure variations are in the order of \( \rho U^2 \), which is negligible compared to the pressure wave magnitude. Based on an inflow boundary area of 220 mm\(^2\) and a peak flow rate of 57 ml s\(^{-1}\), in the current study, the peak inflow velocity is 0.27 m s\(^{-1}\). The flow-induced pressure variations are therefore in the order of 0.1 kPa, while the pressure load is in the order of 10 kPa. Therefore, in this case, the decoupled approach is permitted.

Other assumptions that can have an impact on the results are concerned by the choice of boundary conditions. First, the applied flow rate and pressure load signals are based on a young healthy male [33]. It cannot be expected that these signals represent the specific situations in older patients having cardiovascular disease affecting the arterial haemodynamics such as hypertension and atherosclerosis [36]. Second, at the outflow boundaries of the lumen, the fluid is assumed to be stress-free. In reality, the outflow conditions are determined by the impedance of the distal parts of the arterial tree. To overcome these problems, a one-dimensional model of blood flow in arteries could be adopted, describing the flow and pressure waves in the arterial system (see for instance For-maggia et al. [37]). This could provide physiologic flow and pressure boundary conditions that are in tune to one another. Finally, MRA-based 3D velocity measurements in patients could provide a more specific inflow profile. Since the flow pattern close to the inflow boundary is always affected by the profile that is imposed, the adverse effects of non-physiologic profiles may be considerable in AAA geometries with shorter neck lengths. For the simulated case however, a measured profile was not available. A plug flow was applied instead to simulate inertia-dominated flow.

Although the situation modelled in the current paper is similar to the situation modelled by Di Martino et al. [14], the results are difficult to compare. In the latter paper, no vector fields of the velocity distribution were presented. Furthermore, the applied geometry varied considerably from the geometry applied in the current paper. However, it should be noted that in both studies, the wall displacements were small. The extent to which the relatively larger blood flux associated with these displacements has an impact on the flow characteristics in patient-specific AAAs in general should be subject to further investigation. In the current situation however, the pulse wave velocity, which is in the order of \( \sqrt{\frac{hR}{\rho \omega}} \), with \( h \) a characteristic wall thickness and \( R \) a characteristic vessel radius, is about 11 m s\(^{-1}\), so the wave length is about 11 m. Since this is rather high in relation to the axial length of the model, a comparison with rigid models of AAA flow may be appropriate.

The current study confirms the characteristic velocity patterns and accompanying WSS gradients observed in rigid AAA models. In resting conditions, for asymmetric models, the vortices formed at the proximal side of the bulge during systolic deceleration slowly expand and migrate [38,39]. In the current study, although the Reynolds number applied is in the order of the Reynolds number in resting conditions, vortex formation and translation is less prominent than in the above studies. This may be due to the fact that in the current study, an angulation is present at the neck of the aneurysm, while the above rigid models were straight. Because of the angulation, the vortex formed at the distal end of the neck inner bend is driven into the opposite wall, where it is diminished. The typical WSS gradients promoting thrombus formation [24] are still present. Yet, due to the applied Carreau–Yasuda model, it is expected that the absolute WSS values better approximate the in vivo values. Further, it should be noted that the WSS pattern near the inflow boundary is affected by the imposed inflow profile.

The wall stress simulations confirm previous studies in the sense that the AAA wall stress distribution is complex and to a great extent influenced by local wall curvature. Yet, it should be noted that the curvature-induced stress concentrations in the current study mainly occur at locations at which calcifications are present in the CTA image. The calcifications can be noted in Fig. 1 by the white areas. Due to the high intensity of the calcifications compared to that of the lumen, dents are generated in the segmented lumen boundary surface and therefore in the wall model. This could be solved by more accurate segmentation of the lumen for instance by removal of the calcifications using local subtraction [40]. Further, since the wall curvature in general is dependent on the accuracy of the segmented boundary surface, it should be studied to what extent the computed wall stress is affected by the protocol and the modality that were applied to generate the original image.

The wall simulations may provide good measures of geometry-based wall stress variations to relate to AAA rupture risk. For a more accurate correlation, probably a more dedicated material model will be necessary, such as the one proposed by Raghavan and Vorp [41]. Also, the wall thickness should be varied to distinguish between healthy and diseased tissue [12] and between the aorta and the iliac arteries. Further, it should be considered whether the maximum principal stress as obtained in the current study provides a better criterion for correlation than the Von Mises stress obtained in previous studies [12,17,20]. Although Fillinger et al. report that the results of the two criteria are similar [16], the maximum principle stress criterion may become more important when considering the anisotropic behaviour of the aneurysm wall [34].
If the developed model is applied in rupture risk assessment based on the wall stress to strength ratio rather than peak stress only, it should be considered whether the amplitude-based pressure load yields a better prediction than the peak-systolic pressure load applied in previous studies [12,15,16,20]. Although currently the computed wall stress will be lower than physiologic for lack of an initial stress model, when applying a material model suitable for large displacements, the results obtained with a peak-systolic load may be less realistic. An exact simulation of AAA wall rupture is however further complicated by the need for knowledge on the patient-specific morphology, material properties, and boundary conditions. On the outer wall surface, an external stress and/or kinematic constriction may have to be provided to simulate the presence of external tissues like the spinal column. Also the effects of gravitational forces acting on the blood may then become important.

5. Conclusion

In conclusion, the method of mesh generation that has been presented provides a flexible, semi-automated approach for generating patient-specific hexahedral meshes of the AAA lumen and wall with pre-defined element distributions. The combined fluid/solid mesh allows for simulations of AAA blood dynamics and AAA wall mechanics and the interaction between the two. The mechanical quantities computed in these simulations need to be validated in a clinical setting, after which they could be included in clinical trials in search of risk factors for AAA rupture. In addition, the presented method provides a basis for the development of models for studying the role of blood/wall interaction in processes associated with AAA formation such as wall adaptation and thrombus formation.

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Appendix A. Centreline description

Depending on their orientation with respect to the apex of the boundary surface, different line sections of the original centrelines are assigned to either the trunk of the bifurcation or its left and right branches. Further, the proximal end point of the trunk line is chosen distal to the branch points of the renal arteries, and the distal end point is chosen near the split point of the original centrelines. The proximal end points of the branch lines are determined as the centreline points that are closest to the apex of the boundary surface, and the distal end-points are chosen proximal to the iliac bifurcations (see Fig. 2(c)).

Of each of the trunk and branch lines, a continuous description is determined using piecewise cubic Hermite interpolation [42]. Initially, each of the lines is described by a set of discrete points \( \{ x_0, x_1, \ldots, x_n \} \), with \( x_i = (x_{i1}, x_{i2}, x_{i3}) \) the coordinates of point \( i \equiv n \) the number of points. By interpolation of each of the coordinate sequences, for every line an interpolating function \( C_i \) is derived that for every interval \( [x_n, x_{n+1}] \) can be written as

\[
C_i = \left( \begin{array}{c} a_1 i^3 + b_1 i^2 + c_1 i + d_1 \\ a_2 i^3 + b_2 i^2 + c_2 i + d_2 \\ a_3 i^3 + b_3 i^2 + c_3 i + d_3 \end{array} \right),
\]

where \( 0 \leq \theta_i \leq 1, \; C_i(0) = x_i, \; C_i(1) = x_{i+1}, \) with \( \theta_i \) the interpolation parameter on the interval, and with \( a_j, b_j, c_j, d_j \) the function coefficients.

The interpolation parameter and the index \( i \) are related to the relative axial position \( t \) along the centreline through the following expressions:

\[
\begin{align*}
    i &= (n - 1) \frac{t}{\Delta t} - \text{mod}(n - 1, 1), \\
    \theta_i &= \text{mod}(n - 1, 1),
\end{align*}
\]

where \( 0 \leq t \leq 1 \). In order to obtain smooth interpolating functions, prior to the interpolation, mild second-order penalised least squares smoothing according to Eilers [43] is applied to the original coordinate data.

Appendix B. Mesh transformation

By an initial transformation, the fluid domain is reshaped to a mother channel with two parallel daughter channels (see Fig. 9(a)). The lengths of the channels are determined by the lengths of the reconfigured centrelines. In this respect, the mother channel is extended by the distance between the distal trunk line end point and the initial mesh apex, defined in between the proximal branch line end points (see Fig. 2(c)). The distance between the latter points is applied as the distance between the daughter channel mid-lines. The channel diameters are defined by setting the radius of curvature at the bifurcation equal to the daughter channel radius. Along the centrelines, local coordinate systems \( (n_1, n_2, n_3) \) are defined as depicted in Fig. 9(b). At a certain axial position, \( n_3 \) is determined from the local centreline tangent as

\[
n_3(t) = -\frac{\text{grad}(r)}{\|\text{grad}(r)\|_2} = -\frac{\partial C(r)}{\|\partial C(r)\|_2}.
\]
The axis \( n_1 \) is defined as the normalised projection of \( a_1 \) onto the plane perpendicular to \( n_1 \), where \( a_1 \) is defined as the vector from the apex to the proximal end point of the left branch line (see Fig. 9(b)). Further, \( a_2 \) is defined as the outer product of \( n_1 \) and \( n_2 \):

\[
\mathbf{n}_1(t) = \mathbf{n}_2(t) \times \mathbf{n}_1(t).
\]

The vector \( a_2 \) is also treated as an axis of a coordinate system \( (a_1, a_2, a_3) \) defined at the apex (see Fig. 9(b)). The axis \( a_3 \) is defined as the normalised projection onto the plane normal to \( a_1 \) of the averaged tangent at the proximal branch line end points. Further, \( a_2 \) is defined by

\[
\mathbf{a}_2 = \mathbf{a}_3 \times \mathbf{a}_1.
\]

Further, the trunk line and its local coordinate system are extended towards the apex by a third order polynomial curve that fits both the trunk line tangent and \( a_2 \) (see Fig. 2(c)).

For every cross section of the initially transformed mesh, as a function of the axial position \( t \) along the considered mesh part (see Fig. 9(a)), a rotation matrix \( R \) is defined determining its orientation in the fully transformed mesh. For the trunk, \( R \) is directly defined as:

\[
\mathbf{R}(t) = \begin{bmatrix} \mathbf{g}_1(t) & \mathbf{g}_2(t) & \mathbf{g}_3(t) \end{bmatrix},
\]

with \( \mathbf{g}_j(t), j = 1, 2, 3 \) the column representation of \( n_j(t) \). For the branches, to obtain a smooth transition near the apex, \( R \) is defined by interpolation with the apex local coordinate system:

\[
\mathbf{R}(t) = (1 - t)[\mathbf{a}_1 \mathbf{a}_2 \mathbf{a}_3] + t[\mathbf{g}_1(t) \mathbf{g}_2(t) \mathbf{g}_3(t)].
\]

The complete transformation of a cross section can then be written as:

\[
\mathbf{x'} = \mathbf{R}(t)\mathbf{x}_0 + \mathbf{C}(t).
\]

with \( \mathbf{x}_0 = (x_1, x_2, 0) \) the in-plane position of a cross section point with respect to the mid-line of the considered mesh part, and \( \mathbf{x'} \) its position in the transformed mesh (see Fig. 9(c)).

Appendix C. Point-surface projection

The projection distance \( d \) of a point \( x \) to the triangulated boundary surface is determined as follows. First, the closest vertex of the surface \( x_s \) is found. Next, the orthogonal projections of \( x \) onto the triangles connected to \( x_s \) are determined. Depending on the number of projections that exist, a local surface normal \( \mathbf{n} \) and a projection vector \( \mathbf{v} \) are calculated, distinguishing between the following cases (see Fig. 10):

- If no projections exist, for \( \mathbf{n} \), take the mean of the normals to all triangles attached to \( x_s \); for \( \mathbf{v} \), take the vector from \( x \) to \( x_s \) (see Fig. 10(a)).
- If only one projection \( x' \) exists, for \( \mathbf{n} \), take the outward normal to the triangle containing \( x' \); for \( \mathbf{v} \), take the vector from \( x \) to \( x' \) (see Fig. 10(b)).
- If two or more projections exist at equal distance from \( x \), for \( x' \), take the projection closest to \( x_s \); for \( \mathbf{n} \), take the outward normal to the triangle containing \( x' \); for \( \mathbf{v} \), take the vector from \( x \) to \( x' \) (see Fig. 10(c)).
- If two or more projections exist at equal distance from \( x \), for \( x' \), take either projection; for \( \mathbf{n} \), take the outward normal to the triangle containing \( x' \); for \( \mathbf{v} \), take the vector from \( x \) to \( x' \) (see Fig. 10(d)).

Finally, \( d \) is defined as

\[
d = \text{sign}(\mathbf{n} \cdot \mathbf{v})||\mathbf{n}||_2.
\]

Depending on whether a point is located inside or outside the triangulation, \( d \) is positive or negative (see Fig. 10(e)). If \( d \) equals zero, the point is located on the triangulation itself.

