Coupled fluid–structure interaction hemodynamics in a zero-pressure state corrected arterial geometry

V. Vavourakis, Y. Papaharilaou, J.A. Ekaterinaris

Abstract

Hemodynamic conditions in large arteries are significantly affected by the interaction of the pulsatile blood flow with the distensible arterial wall. A numerical procedure for solving the fluid–structure interaction problem encountered in cardiovascular flows is presented. We consider a patient-specific carotid bifurcation geometry, obtained from 3D reconstruction of in vivo acquired tomography images, which yields a geometrical representation of the artery corresponding to its pressurized state. To recover the geometry of the artery in its zero-pressure state which is required for a fluid–structure interaction simulation we utilize inverse finite elastostatics. Time-dependent flow simulations with in vivo measured inflow volume flow rate in the 3D undeformed artery are performed through the finite element method. The coupled-momentum method for fluid–structure interaction is adopted to incorporate the influence of wall compliance in the numerical computation of the time varying flow domain. To demonstrate the importance in recovering the zero-pressure state of the artery in hemodynamic simulations we compute the time varying flow field with compliant walls for the original and the zero-pressure state corrected geometric configurations of the carotid bifurcation. The most important resulting effects in the hemodynamic environment are evaluated. Our results show a significant change in the wall shear stress distribution and the spatiotemporal extent of the recirculation regions.

1. Introduction

Three-dimensional numerical modeling of fluid–structure interaction (FSI) of blood flow/distensible arterial vessel is a challenging task and has received increasing attention in recent years. Various computational techniques have been proposed in order to simulate blood flow in the vasculature. These techniques broadly fall into two main categories, where the wall is assumed rigid (Taylor et al., 1998; Oshima et al., 2001; Cebral et al., 2003; Shojima et al., 2004; Salmon et al., 2003) or compliant (Quarteroni et al., 2000; di Martino et al., 2001; Formaggia et al., 2001; de Hart et al., 2003; Wolters et al., 2003; Torii et al., 2006; Scotti and Finol, 2007; Khanfer et al., 2009). The rigid wall approximation was primarily adopted due to the difficulty in solving the coupled FSI blood flow/vessel problem. In large healthy arteries, the vessel diameter variation during the cardiac cycle is relatively small (approximately 5–10%) and is further reduced in less compliant diseased vessels (Nichols and O’Rourke, 2005); thus, the rigid wall simplification could be acceptable. However, recent studies including full FSI analysis in vascular conduits indicate that the rigid wall assumption precludes wave propagation phenomena (Formaggia et al., 2001; Vignon-Clementel et al., 2006; Figueroa et al., 2006), which influence the character of the numerical solutions.

FSI modeling of pulsatile blood flow in a compliant artery requires the solution of both the equations describing fluid motion and the elastic wall motion. This can be achieved either by solving the governing equations separately in a staggered iterative manner (Papaharilaou et al., 2007; Järvinen et al., 2008) or simultaneously in a fully coupled manner (di Martino et al., 2001; de Tallec and Mouro, 2001; Hron and Mádlík, 2007). The numerical method most widely used for fully coupled FSI is the arbitrary Lagrangian–Eulerian (ALE) formulation in the finite element method (FEM) framework (Hughes et al., 1981; Donea et al., 1982; Nomura, 1994; Bathe et al., 1995). However, ALE formulations are computationally expensive as they require mesh updating, due to the continuous geometry update of both fluid and solid domains, and introduce errors, when projecting the solution from the old mesh to the updated one. Furthermore, a suitable sub-iteration process must be established to converge to the correct wall velocity that appears in the fluid momentum equations. Alternative FSI formulations are the immersed boundary method (Peskin and McQueen, 1995), transpiration techniques based on linearization principles (Fernandez and de Tallec, 2003),...
the sequentially coupled arterial FSI technique (Tezduyar et al., 2009), the coupled-momentum method (CMM) (Figuerola et al., 2006), and the fictitious domain method schemes proposed by Baaijens (2001) and van Loon et al. (2006).

Most of the aforementioned works considered patient-specific models with a geometry reconstructed from diagnostic images (CT or MRI). The image-based geometry definition, however, corresponds to a deformed pressurized state of the arterial structure. It was common for researchers so far to take the in vivo geometry as the initial configuration and proceed to simulate the arterial wall deformation for stress distribution evaluation or to solve the fluid–structure interaction problem. This approach can introduce errors (Lu et al., 2007b; de Putter et al., 2007; Gee et al., 2009; Torii et al., 2009), which are related to the magnitude of the deviation between the stress-free and image-based geometric configurations of the vascular structure.

Govindjee and Mihallic (1996) first proposed a method for solving the inverse elastostatic problem via the FEM. This procedure was later extended by other researchers to include anisotropic elastic solids via a total (Lu et al., 2007a; Fachinotti et al., 2008) and an updated (de Putter et al., 2007; Gee et al., 2009) Lagrange formulation. Lu et al. (2007b), de Putter et al. (2007) and Gee et al. (2009) solved the inverse elastostatic problem for image-based models of abdominal aortic aneurysms and computed the wall stress through finite element analysis (FEA). Recent works by Tezduyar et al. (2008) and Bazilevs et al. (2010) have proposed methods to account for the prestress state of image-based arterial structures in their FSI simulations. In Tezduyar et al. (2008), a rudimentary technique is used to estimate the zero-pressure geometry and the wall thickness distribution, whereas in Bazilevs et al. (2010) a different approach is followed whereby the stress state of the reference image-based geometry is computed and then used as an initial condition for the FSI analysis. However, these studies do not quantify the effects of correcting for the prestress state of image-based arterial structures on FSI analysis based hemodynamics.

The objective of this work is to offer a robust methodology in recovering the zero-pressure state configuration of image-based arterial structures based on inverse finite elastostatics and assess its significance in FSI analysis of compliant arterial hemodynamics. Towards this an image-based geometric representation of the carotid bifurcation (pressurized state) is obtained and the corresponding zero-pressure state is recovered applying an inverse finite element elastostatics formulation. FSI analysis is then performed on the zero-pressure configuration by adopting the CMM for blood flow analysis in compliant arteries. For this purpose, a patient-specific carotid bifurcation extracted in vivo from a healthy 35-year-old volunteer is examined. The CMM technique is chosen for our FSI simulations as it offers a computationally efficient way to strongly couple the degrees of freedom of the vessel wall with the fluid boundary. This is achieved by representing the solid and fluid domain kinematic equations using the same Eulerian frame of reference, thus, circumventing the numerical drawbacks of the ALE formulation.

2. Methods

2.1. Coupled-momentum FSI technique

The coupled-momentum FSI method offers a solution of the fluid/solid kinematic equations and the fluid continuity equation in a coupled manner. The Galerkin weak form for the coupled-momentum FSI (obtained by combining Eqs. (A.1) and (A.2) as discussed in the Appendix) reads

\[ \int_U \left[ (\rho_f \mathbf{w} - \left( \frac{\partial \mathbf{w}}{\partial t} + \mathbf{v} \cdot \nabla \mathbf{w} \right) + \mathbf{w} \cdot (\mathbf{v} \cdot \nabla) - \nabla \cdot (\rho_f \mathbf{w} \mathbf{a} - \mathbf{F}_s) \right] \cdot \mathbf{d}V - \int_{\Gamma_f} \mathbf{w} \cdot \mathbf{dS} + \int_{\Gamma_t} \mathbf{q}_w \cdot \mathbf{dS} = 0, \]

where \( \mathbf{v} \), \( \rho_f \), are the fluid velocity and normal to \( \Gamma_f \), \( \mathbf{t} \), \( \rho_t \), are the viscous stress tensor, pressure, fluid density, respectively; and \( \mathbf{a} \), \( \mathbf{t} \), \( \rho_t \) are the Cauchy stress tensor, the prescribed traction vector on \( \Gamma_t \), and the solid wall density, respectively. The quantities \( \mathbf{w} \) and \( \mathbf{q}_w \) are weighting functions for the velocity and pressure fields, respectively, while \( \mathbf{I} \) is the identity matrix and \( h \) the thickness of the solid wall. The last three integrals in Eq. (1) are the terms added to the rigid wall theory fluid/solver to account for FSI. Further details on the numerical technique, the boundary conditions and the space–time-discretization procedure are provided in the Appendix and in Figuerola et al. (2006).

The main steps of the coupled-momentum FSI numerical procedure are the following: (a) The fluid solver is utilized to obtain a steady flow solution for rigid wall; (b) the arterial wall is loaded with an average pressure—evaluated in step (a)—and the vessel wall initial state is obtained; (c) the steady flow coupled FSI analysis starts with fluid and solid domain initial conditions obtained from steps (a) and (b), respectively; (d) once a convergent solution is reached in step (c), then unsteady flow is applied and the FSI analysis initiates until time-periodicity is reached. The CMM FSI numerical simulations were carried out through the open-source project simvascular.
Newton–Raphson technique performing up to 12 nonlinear iterations for each increment to achieve convergence. The volume reduction of the MRI carotid bifurcation (see Fig. 2(a)) following the inverse FEA is depicted in Fig. 2(b), where it can be clearly seen that radial rather than axial deformations dominate. This is the result of fixing the ends of the carotid bifurcation for the inverse analysis in order to avoid rigid body motion. CMM FSI solutions—starting with the MRI-based or the zero-pressure configurations—were carried out until time-periodic response was reached. Time-periodicity was achieved after three cycles and all results presented next correspond to time-periodic FSI numerical solutions.

In Fig. 3, contours of the differential pressure \( (p - p_0) \) corresponding to the deformed MRI geometry and zero-pressure initial configurations of the carotid bifurcation at peak systole and early diastole are compared. The pressure distribution at late diastole (not shown here) was found similar to that shown for the early diastole stage. The pressure difference is calculated at post-processing and taken as the difference between the numerically evaluated pressure and the inlet pressure \( p_{in} \) at the corresponding cardiac phase. From the original pressure contours, the pressure range is 99.6–103.2 mm Hg higher in the zero-pressure case during peak systole, due to the reduction in the vascular lumen size as compared to 97.9–100.7 mm Hg of the deformed MRI case. This effect is less pronounced during the diastolic phases, where the pressure range is 83.7–84.9 mm Hg for the zero-pressure case and 84.5–85.5 mm Hg for the MRI case.

Comparisons of the wall shear stress (WSS) magnitude distribution and the \( Z \)-component of fluid velocity of the MRI and zero-pressure geometries are shown in Figs. 4 and 5, respectively. The WSS distribution and \( Z \)-velocity component variation depicted in these figures are normalized, respectively, by the corresponding cycle averaged WSS \( (\tau = 4\mu r/\tau) \) and mean velocity \( (V = \bar{V}/\tau^2 = 24.8 \text{ cm/s}) \) at the inlet of the domain. As expected, the wall shear stress attains its maximum value at the apex of the bifurcation in both cases, while high WSS was observed on the ECA branch (see Fig. 4(a)) at the diastolic phase. Fig. 4 shows that although a region of low WSS on the ICA wall is present in both configurations it is larger in the MRI-based geometry. To obtain a quantitative assessment of the differences in WSS distribution between MRI and zero-pressure geometries we selected three representative points on the fluid domain lateral surface (see figure in Table 1) and present the extracted WSS values in Table 1. These results show a physiological range of WSS variation in both cases but a consistent underestimation of the WSS in the MRI geometry. The \( Z \)-velocity variation of Fig. 5 indicates that flow develops faster in the ECA for the zero-pressure geometry as compared to the MRI-reconstructed geometry. In the ICA branch of the MRI geometry, flow recirculation regions and vortices develop slightly upstream of the bifurcation. In the zero-pressure geometry the backflow effect is less pronounced, resulting in a reduced area of low WSS exposure as compared to the MRI-extracted geometry case.

In Fig. 6, a comparison of the vorticity magnitude contours in the carotid MRI and zero-pressure geometries is shown. The non-dimensional vorticity vector field is calculated at the post-processing stage through the relation: \( \nabla \times (v/\tau) \). As expected due to the reduced lumen volume, the vorticity magnitude at peak systole is higher in the zero-pressure configuration as compared to the MRI-extracted geometry. However, in both cases peak vorticity appears near the inner walls of the bifurcation where it is also generated, although interestingly it is not spatially related to the presence of regions of flow reversal in both the ICA and ECA branches, as it can be seen in Fig. 5. During early diastole, flow entering the bifurcation from the common carotid artery attains a lower vorticity magnitude in the case of the MRI-extracted

Fig. 1. (a) Geometry of the MRI-acquired carotid bifurcation and (b) the flow rate history on the inlet of the carotid artery.

Fig. 2. Comparison between the MRI and the zero-pressure finite element mesh geometries. (a) MRI and (b) Z-P.
geometry, affecting primarily the vorticity distribution in the ICA. The vorticity is different in the two geometric configurations considered. Such changes in the vorticity distribution entering the carotid bulb are expected to increase flow disturbances; thus, influencing the propensity for flow detachment and the generation of regions of flow recirculation exposing the wall to unfavorable hemodynamics. This effect is further accentuated during late diastole.

The total volume of the carotid bifurcation depicted in Fig. 7 obtained through MRI measurements is 1.324 cm³, while the corresponding volume of the zero-pressure geometry is 1.081 cm³. When the latter geometry is loaded by end diastolic pressure 82 mm Hg on the initial stage of the FSI analysis, in order to evaluate the initial state of the vessel wall as described in Section 2.1, the volume becomes 1.372 cm³. Raising the applied inner pressure to 90 mm Hg the volume becomes 1.399 cm³. Thus the initial MRI

![Fig. 3. Fluid domain surface and internal distribution of pressure on the MRI and zero-pressure carotid, respectively. (a) MRI: peak systole, (b) MRI: early diastole, (c) Z-P: peak systole and (d) Z-P: early diastole.](image)

![Fig. 4. Non-dimensional fluid domain log₁₀[WSS] distribution on the surface of the MRI and zero-pressure carotid, respectively. (a) MRI: peak systole, (b) MRI: early diastole, (c) MRI: late diastole, (d) Z-P: peak systole, (e) Z-P: early diastole and (f) Z-P: late diastole.](image)
geometry is recovered with a total volume deviation of 9.4%. A detailed comparison of the MRI measured geometry and the ‘recovered’ zero-pressure configuration under 90 mm Hg inner pressure can be found in the Appendix.

Significant differences of the computed displacements, at peak systole and early diastole, obtained from FSI with the MRI and zero-pressure geometries are highlighted in Fig. 7. A detailed quantitative analysis of the computed deformations is carried out next. Fig. 7 shows various sections and sub-volumes considered, in order to quantify surface and volume changes during the cardiac cycle of the MRI and zero-pressure geometries. The corresponding measurements are given in Tables 2 and 3, respectively. The relative area deviation of each section is taken as 

$$\frac{|A_{REF,MRI} - A_{MRI(t)}|}{A_{REF,MRI}}$$

for the MRI geometry and 

$$\frac{|A_{REF,MRI} - A_{ZP(t)}|}{A_{REF,MRI}}$$

for the zero-pressure geometry, where $A_{REF,MRI}$ is the reference area of the corresponding MRI-acquired geometry section and $A_{MRI(t)}$ and $A_{ZP(t)}$ the area of the deformed geometry section at the respective cardiac phase obtained through FSI analysis on the MRI and the zero-pressure geometry respectively.

The information presented in Tables 2 and 3 indicates that, as expected, the maximum deformation occurs during peak systole. The maximum area variation occurs in sections C, D and G of the corresponding sub-volumes I, II and III, which can be attributed to their close proximity to the apex. However, during the diastolic phases the sectional variation of the MRI and zero-pressure geometries is similar, except for sections D and G which are proximal to the bifurcation apex. This finding is also supported by the pressure distribution depicted in Fig. 3, where the pressure range between the MRI and zero-pressure geometries is similar.

The following important observations can be made from the results presented in Tables 2 and 3: (i) when the MRI geometry is taken as the initial state of the arterial wall for the FSI computation, a sectional area increase of up to 37% and 31% and a volumetric increase of up to 32% and 27% approximately occurs during peak systole and diastole, respectively; (ii) when the zero-pressure arterial wall geometry is used for the FSI analysis, a sectional area increase of up to 18% and 11% and a volumetric increase of up to 11% and 6% approximately is observed during peak systole and diastole, respectively. We should note that the differences between MRI and zero-pressure corrected geometry reported in Tables 2 and 3 are influenced by the selection of the wall material properties. It is expected that these differences would be reduced for a subject with a less compliant wall (i.e. a healthy but older subject or a patient with atherosclerotic disease). A similar effect is expected if a hyperelastic material model was used in the forward FE and FSI analyses. The inconsistency of the solid domain representation in the FSI simulation, where linearly elastic material was used, and the inverse approach, where a hyperelastic material assumption was adopted in order to obtain a more realistic zero-pressure geometry is a limitation of our analysis approach. One way to address this issue would be to modify the elastic modulus used in the FSI analysis so as to minimize the difference between the MRI measured geometry.

Table 1

<table>
<thead>
<tr>
<th>WSS (dyn/cm²) at point on</th>
<th>Peak systole</th>
<th>Late diastole</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MRI</td>
<td>Z-P</td>
</tr>
<tr>
<td>CCA</td>
<td>11.0</td>
<td>20.8</td>
</tr>
<tr>
<td>ICA</td>
<td>8.8</td>
<td>17.3</td>
</tr>
<tr>
<td>ECA</td>
<td>36.7</td>
<td>43.5</td>
</tr>
</tbody>
</table>

Fig. 5. Non-dimensional velocity z-component contours of the MRI and zero-pressure carotid geometry, respectively. (a) MRI: peak systole, (b) MRI: early diastole, (c) MRI: late diastole, (d) Z-P: peak systole, (e) Z-P: early diastole and (f) Z-P: late diastole.
geometry and the zero-pressure configuration obtained under 90 mm Hg inner pressure (see Appendix for a measure of this difference). Work is in progress to alleviate this inconsistency by implementing a nonlinear hyperelastic material model for the description of the distensible arterial wall for the full FSI/FEM combined with an anisotropic, variable thickness description of the wall. However, even using the current set of parameters, which are within the physiological range for a young healthy individual, and relying on simplifying assumptions related to the mathematical

Fig. 6. Non-dimensional vorticity magnitude and iso-surface contours of the MRI and zero-pressure carotid geometry, respectively. (a) MRI: peak systole, (b) MRI: early diastole, (c) MRI: late diastole, (d) Z-P: peak systole, (e) Z-P: early diastole and (f) Z-P: late diastole.

Fig. 7. (a) Sections and (b) sub-volumes of the carotid bifurcation geometry used to compute area and volume changes.

<table>
<thead>
<tr>
<th>Section</th>
<th>Peak systole (%)</th>
<th>Early diastole (%)</th>
<th>Late diastole (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>29.9</td>
<td>7.6</td>
<td>25.5</td>
</tr>
<tr>
<td>B</td>
<td>37.1</td>
<td>9.1</td>
<td>31.5</td>
</tr>
<tr>
<td>C</td>
<td>28.3</td>
<td>11.0</td>
<td>24.0</td>
</tr>
<tr>
<td>D</td>
<td>35.7</td>
<td>10.1</td>
<td>30.1</td>
</tr>
<tr>
<td>E</td>
<td>30.9</td>
<td>10.7</td>
<td>26.0</td>
</tr>
<tr>
<td>F</td>
<td>27.1</td>
<td>8.9</td>
<td>22.9</td>
</tr>
<tr>
<td>G</td>
<td>29.9</td>
<td>18.4</td>
<td>25.0</td>
</tr>
<tr>
<td>H</td>
<td>24.5</td>
<td>17.1</td>
<td>20.3</td>
</tr>
<tr>
<td>I</td>
<td>26.9</td>
<td>18.8</td>
<td>22.1</td>
</tr>
</tbody>
</table>

Table 2
Area relative deviation on each section considered in Fig. 7(a) of the MRI/zero-pressure geometry at three cardiac phases.

<table>
<thead>
<tr>
<th>Sub-vol.</th>
<th>Peak systole (%)</th>
<th>Early diastole (%)</th>
<th>Late diastole (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>30.5</td>
<td>10.1</td>
<td>25.8</td>
</tr>
<tr>
<td>II</td>
<td>24.4</td>
<td>10.0</td>
<td>20.6</td>
</tr>
<tr>
<td>III</td>
<td>32.1</td>
<td>11.2</td>
<td>27.0</td>
</tr>
</tbody>
</table>

Table 3
Volume relative deviation on each part considered in Fig. 7(b) of the MRI/zero-pressure geometry at three cardiac phases.

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implementing a nonlinear hyperelastic material model for the description of the distensible arterial wall for the full FSI/FEM combined with an anisotropic, variable thickness description of the wall. However, even using the current set of parameters, which are within the physiological range for a young healthy individual, and relying on simplifying assumptions related to the mathematical
description of the wall kinematics (i.e. linearly elastic, isotropic material of uniform thickness), our approach yields a substantial quantitative assessment of the importance of considering the unloaded state of image-based arterial structures, when these are used in FSI vascular modeling and provides a robust methodology to achieve this. We should also note that the residual stresses of the unloaded artery were not taken into account in our analysis. The effects of including residual strains in stress analysis in arteries are discussed in Delfino et al. (1997), where an idealized carotid bifurcation is considered and in the review paper of Rachev and Greenwald (2003).

The total wall displacement field is presented in Fig. 8 for the MRI and zero-pressure geometries at peak systole and early diastole. The relative radial wall deformation obtained from the zero-pressure corrected configuration is approximately 5%, whereas for the MRI geometry the arterial wall deformation is significantly larger yielding an unrealistic FSI evaluation of the hemodynamic conditions. It should be noted that apart from the radial deformation, there is also a bulk wall motion that contributes to the displacement field depicted in Fig. 8. This bulk wall motion can be primarily attributed to the lack of 'fictitious' constraints to simulate wall tethering to surrounding tissue.

In the recent paper of Merkx et al. (2009), the wall displacements on the aneurysmatic abdominal aorta obtained via dynamic MRI and those computed via the FEM were compared. The authors derived the initial unloaded geometry of the artery through a backward incremental method (de Putter et al., 2007). Applying a mean diastolic pressure for the forward FEA they observed that the median of the relative cross-sectional area difference between MRI measured and FEA computed values was 12.4% when the MR measured geometry is used in FEA and 1.7% when the unloaded configuration of the geometry is used. These differences are of the same order of magnitude with those presented in Table 2 herein. However, we should note that in contrast to our work the wall displacement evaluation in Merkx et al. (2009) is based on analyzing solely the structural part, thus neglecting FSI.

4. Conclusion

Significant differences in the computed WSS and pressure distribution, as well as in the velocity field, and vorticity patterns are observed between the zero-pressure and the MRI-reconstructed configurations of the healthy human carotid bifurcation examined. Our results highlight the importance of recovering the zero-pressure configuration of vascular structures used in FSI simulation as direct utilization of the image-based configuration was shown to cause unrealistic wall deformation and underestimation of wall shear stress. Computation of the wall stress in

Fig. 8. Arterial wall displacement magnitude contours of the MRI and zero-pressure carotid geometry, respectively. (a) MRI: peak systole, (b) MRI: early diastole, (c) Z-P: peak systole and (d) Z-P: early diastole.
conjunction with the hemodynamic parameters examined in the present work could provide a better understanding of the coupled dynamic system of blood flow and arterial wall deformation.

Conflict of interest statement

The authors declare that there exist no conflicts of interest.

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