Fluid–structure interaction analysis of a patient-specific right coronary artery with physiological velocity and pressure waveforms

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SUMMARY

Coupled fluid–structure interaction (FSI) analysis of the human right coronary artery (RCA) has been carried out to investigate the effects of wall compliance on coronary hemodynamics. A 3-D model of a stenosed RCA was reconstructed based on multislice computerized tomography images. A velocity waveform in the proximal RCA and a pressure waveform in the distal RCA of a patient with a severe stenosis were acquired with a catheter delivered wire probe and applied as boundary conditions. The arterial wall was modeled as a Mooney–Rivlin hyperelastic material. The predicted maximum wall displacement (3.85 mm) was comparable with the vessel diameter (~4 mm), but the diameter variation was much smaller, 0.134 mm at the stenosis and 0.486 mm in the distal region. Comparison of the computational results between the FSI and rigid-wall models showed that the instantaneous wall shear stress (WSS) distributions were affected by diameter variation in the arterial wall; increasing systolic blood pressure dilated the vessel and consequently lowered WSS, whereas the opposite occurred when pressure started to decrease. However, the effects of wall compliance on time-averaged WSS (TAWSS) and oscillatory shear index (OSI) were insignificant (4.5 and 2.7% difference in maximum TAWSS and OSI, respectively).

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1. INTRODUCTION

Atherosclerotic coronary artery disease is a major cause of mortality and morbidity in the developed and developing world, of which one of the outcomes is heart attack. Atherosclerosis shows a focal distribution, suggesting that mechanical factors related to blood flow may contribute to its development. There is considerable evidence implicating mechanical forces resulting from intravascular pressure and flow (e.g. circumferential tensile stress and wall shear stress (WSS)) in the regulation of blood vessel structure under physiological or pathological circumstances \[1–4\]. However, there is a complex question regarding the precise relationship between hemodynamics, mechanical factors and atherosclerotic changes in the arterial wall. Addressing these questions is made difficult as it is not possible to measure local flow patterns and mechanical forces \textit{in vivo} with sufficient accuracy using imaging techniques alone, although evidence has been building for the responses to WSS of arterial wall (endothelial and smooth muscle) cells and their internal molecules, including atherogenic processes \[5\].

We have been investigating coronary hemodynamics using computational fluid dynamics (CFD) combined with medical imaging to determine detailed patterns of blood flow and WSS in anatomically realistic coronary arteries. Coronary hemodynamics has unique characteristics in the human cardiovascular system owing to diastolic-dominant flow \[6\] and cardiac-induced dynamic vessel motion. Myers \textit{et al.} \[7\] investigated the effects of arterial geometry, pulsatile flow condition and dynamic vessel motion on flow patterns in a realistic model of the right coronary artery (RCA). They concluded that arterial geometry was the dominant factor and flow pulsatility came second. Dynamic vessel motion was found to be less important than the other two factors. However, their model did not include the effect of arterial wall compliance. Recent advances in computing resources and numerical algorithms have made it possible for more complicated physics such as fluid–structure interactions (FSI) to be included in computational simulations. FSI has been applied to a large number of cardiovascular problems since it is essential in some of those, e.g. motion of the natural/artificial heart valves \[8\]. FSI is also considered important in parts of the arterial system such as abdominal/thoracic aortas \[9\] including aortic aneurysms \[10\], carotid artery bifurcations \[11, 12\], cerebral aneurysms \[13, 14\] and coronary arteries \[15–17\]. Conversely, there is also a study indicating that the effect of arterial wall deformation on arterial flow can be insignificant \[18\]. Experimental data showed that wall motion of non-stenosed coronary arteries was approximately less than 8.4% of the vessel diameter \((5.4\pm2\% \ [19] \text{ and } 2\% \ [20] \text{ in terms of strain, } 8.4\% \text{ in diameter } [21])\). However, studies on stenosis and plaque structure \[22–25\] have demonstrated the importance of FSI in stenosed arteries. FSI analyses of coronary arteries are still sparse, largely due to lack of \textit{in vivo} information such as subject-specific material properties, flow and pressure waveforms. The aim of the present study was to determine the effect of wall compliance on blood flow patterns and WSS in a human RCA using computational FSI analysis with \textit{in vivo} anatomical, flow and pressure information.

2. COMPUTATIONAL METHOD

2.1. Vascular geometry reconstruction

A 3-D model of the RCA of a patient with a mild stenosis was reconstructed based on multislice computerized tomography (CT) images acquired at mid-diastole, 75% into the duration of the
cardiac cycle, using a Philips Mx8000 IDT 16-detector multi-slice CT scanner (Philips Electronics U.K. Ltd, Guildford, U.K.). The in-plane image resolution was approximately 0.43 mm/pixel and the slice interval was 0.5 mm. The RCA was first delineated with CMRtools (CVIS, London, U.K.). The planes perpendicular to the centerline were computed at 1 mm intervals and used to re-slice the data set with cubic B-spline resampling [26]. The RCA was then semi-automatically segmented from the resulting stack of cross-sectional images [27]. The lumenal surface was constructed by interpolation of the extracted cross-sectional outlines using cubic B-splines. Because it is difficult to obtain the outer boundary of the RCA wall from the CT images, the wall was artificially constructed based on the lumenal surface. First, a lumenal surface without stenosis was constructed by eliminating the stenosis and interpolating the lumenal surface assuming smooth variation in diameter along the centerline. Then the surface without the stenosis was extruded outward by 0.5 mm to obtain the outer wall surface based on wall thickness measurement made with an intravascular ultrasound probe [28]. The RCA model, which has a 62% stenosis (area reduction), is shown in Figure 1. The wall thickness at the stenosis throat is approximately 1.2 mm. The inlet and outlet were extended in length by five diameters in the direction normal to the in/outlet cross sections to prevent interactions between boundary conditions and region of interest. The reconstructed geometries were meshed using ICEM CFD (ANSYS, Inc.) with hexahedral elements for both the fluid and wall domains. The number of nodes and elements were 113 715 and 106 760 for the fluid domain, 63 000 and 50 240 for the wall domain, respectively.

2.2. Computational fluid–structure interactions

The dynamic coupling of blood flow and wall motion was achieved using a commercial computational mechanics suite ANSYS and ANSYS CFX (ANSYS Inc.). ANSYS is finite-element-based software for structural mechanics analysis and ANSYS CFX is finite-volume-based software for fluid mechanics computations. These are coupled and solved iteratively within each time step by applying appropriate kinematic and dynamic conditions at the fluid–structure interface until the residual of the system is below a specified tolerance. For fluid mechanics computations, the 3-D Navier–Stokes equations for incompressible flow with treatment of moving domain,

\[
\frac{d}{dt} \int_{V(t)} \rho \, dV + \int_{s} \rho (U_j - W_j) \, dn_j = 0
\]

\[
\frac{d}{dt} \int_{V(t)} \rho U_i \, dV + \int_{s} \rho (U_j - W_j) U_i \, dn_j = - \int_{s} P \, dn_j + \int_{s} \mu \left( \frac{\partial U_i}{\partial x_j} + \frac{\partial U_j}{\partial x_i} \right) \, dn_j
\]
Table I. Computational schemes used for fluid and structural mechanics computations, in ANSYS CFX and ANSYS mechanics.

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*Numerical integration in FVM is implemented with finite element interpolation function and hence interpolation function is needed.
†Velocity prediction method in SIMPLE scheme is modified to avoid ‘checkerboard’ numerical oscillation due to collocated grid [29].
‡Second-order central difference and first-order upwind are blended; blending parameters are determined locally to avoid numerical oscillation and to minimize the effects of upwinding [30].

were solved with time-dependent physiological velocity and pressure boundary conditions. Here $U_j$ and $W_j$ are the flow velocity and the velocity of the control volume boundary, and $P$ is pressure. $V$ and $s$ respectively denote volume and surface regions of integration, and $dn_j$ are the differential Cartesian components of the outward normal surface vector. Density and dynamic viscosity of the fluid are denoted as $\rho$ and $\mu$. For structural mechanics, momentum balance equations were solved with stress boundary conditions at the fluid–structure interface and constraint conditions. The computational mesh for fluid mechanics was updated with respect to boundary displacement by solving the Laplace equation so that the wall boundary displacement could be propagated through all nodes within the domain. The numerical schemes used in fluid and structural mechanics computations are summarized in Table I.

2.3. Material properties

The arterial wall was modeled as a nine parameter Mooney–Rivlin hyperelastic material in this study. Although arterial wall is known to have heterogeneous and anisotropic structure owing to multiple compositions including collagen fibers [31], the homogeneous and isotropic material model was adopted due to lack of in vivo data on heterogeneity and fiber direction, which governs anisotropic behavior of the tissue. The strain energy function is

$$W = c_{10}(\tilde{I}_1 - 3) + c_{01}(\tilde{I}_2 - 3) + c_{20}(\tilde{I}_1 - 3)^2 + c_{11}(\tilde{I}_1 - 3)(\tilde{I}_2 - 3) + c_{02}(\tilde{I}_2 - 3)^2$$

$$+ c_{30}(\tilde{I}_1 - 3)^3 + c_{21}(\tilde{I}_1 - 3)^2(\tilde{I}_2 - 3) + c_{12}(\tilde{I}_1 - 3) (\tilde{I}_2 - 3)^2 + c_{03}(\tilde{I}_2 - 3)^3 + \frac{1}{d} (J - 1)^2$$

(3)

where $\tilde{I}_1$ and $\tilde{I}_2$ are the first and second deviatoric strain invariants and $J$ is the determinant of the elastic deformation gradient tensor. The model parameters were set to $c_{10} = 0.070$, $c_{20} = 3.2$, $c_{21} = 0.07160$ MPa and $c_{ij} = 0.0$ MPa for the others, following the FSI analysis of the human RCA.
by Koshiba et al. [16]. The material incompressibility parameter \( d (=2/K, K \text{ is bulk modulus}) \) was set to \( 1.0 \times 10^{-5} \text{Pa}^{-1} \). Plaque components and residual stress were not considered in this study for the sake of simplicity. The blood was assumed to be Newtonian because the flow is not stagnant and shear rate is consequently large enough to maintain a flow regime with nearly constant viscosity (shear rate \( \dot{\gamma} > 100 \text{s}^{-1} \)). The density and viscosity of the blood were set to 1050 kg/m\(^3\) and \( 4.0 \times 10^{-3} \text{Pas} \) [6].

2.4. Physiological boundary conditions

Time-dependent velocity profiles were prescribed at the inlet of the RCA as the inflow boundary conditions. The conditions at the outlet were time-dependent pressure, which was assumed to be uniform over the cross section. Both the inflow and outflow boundary conditions were based on \textit{in vivo} measurements. The velocity and pressure waveforms were simultaneously acquired with an electrophysiology (ECG)-gated intravascular ultrasound Doppler and pressure probe (ComboWire, Volcano\textsuperscript{TM} Corporation) [32, 33] in the proximal and distal RCA of a patient with a severe stenosis during percutaneous coronary intervention (PCI), i.e. balloon angioplasty followed by stent insertion. The acquisition was made in addition to standard clinical protocol for PCI, but the additional risk is minimal because a catheter insertion is necessary in PCI procedure in order to deliver a balloon and stent, and waveform acquisition requires an additional 0.356 mm diameter probe. The patient was well consented by a clinician, was fully aware of the risk and agreed with the waveform acquisition. Although the procedure is invasive, this is the only way to obtain coronary pressure waveform that is one of the key pieces of information in this study. The acquired data were aligned with reference to the ECG R-wave. The velocity and pressure waveforms were first decomposed using Fourier transform. The Fourier coefficients obtained were used in functions prescribing pulsatile velocities at the inlet [34] and pressure at the outlet:

\[
w(r, t) = B_0 \left[ 1 - \left( \frac{r}{R} \right)^2 \right] + \sum_{n=1}^{N} \frac{B_0}{2} \left[ \frac{1 - J_0 \left( \frac{a_n r}{R} \right)^{3/2} / J_0 (a_n i^{3/2})}{1 - 2 J_1 (a_n i^{3/2}) / (a_n i^{3/2} J_0 (a_n i^{3/2}))} \right] e^{-2\pi n i w t}
\]

\[
p(t) = \sum_{n=0}^{N} C_n e^{-2\pi n i w t}
\]

where \( B_n \) and \( C_n \) are Fourier coefficients, \( r \) is the cylindrical coordinate and \( t \) is the time. \( R \) denotes the radius of the inlet cross section. Here \( J_0 \) and \( J_1 \) are the Bessel functions of the first kind of order 0 and 1 and \( a_n = R \sqrt{n/m/v} \), where \( m = 2\pi/T \), is based on one cardiac cycle (= 1.0 s). The Womersley parameter \( \alpha = R \sqrt{\sigma/v} \) is 2.60 and the Reynolds number is 531 at peak flow and 276 based on the cycle-averaged velocity. The velocity and pressure waveforms applied in the simulations are shown in Figure 2. In the actual computation, pulse pressure, \( p(t) - p_{\text{ref}} \) (\( p_{\text{ref}} = 70 \text{mmHg} = 9310 \text{Pa} \)) was specified at the outlet since the reconstructed RCA geometry corresponded to a diastolic state (i.e. subject to diastolic pressure). The reference pressure (\( p_{\text{ref}} \)) was determined as the minimum pressure during the cardiac cycle. Wall boundary condition was no-slip, i.e. velocities at the lumen-wall interface in the wall domain were the same as those in the fluid domain. The proximal and distal ends of the artery were fixed with no axial or transaxial motion. Cardiac-induced dynamic vessel motion was not considered in this study in order to isolate the effects of wall compliance on coronary hemodynamics.
3. RESULTS AND DISCUSSION

The wall deformation patterns were examined first. Figure 3 shows the displacement contours, caused by the internal pressure, on the lumen-wall interface at three time points during the cardiac cycle. At peak flow in systole, the maximum displacement, 3.50 mm, occurs in the middle of the RCA distal to the stenosis. The maximum displacement increases to 3.85 mm when peak pressure is reached in mid-cycle, but occurs at a location more distal than that at peak flow. In diastole, the displacement pattern remains similar to that at peak pressure but the maximum displacement, 1.48 mm, is much smaller. Ding et al. [19] reported a maximum displacement of 60 mm for the RCA using biplane cineangiograms, while Zeng et al. [17, 21] showed transaxial RCA motion larger than several vessel diameters (RCA diameter ~ 4 mm), also based on biplane cineangiograms. The RCA displacement observed in vivo is much larger than that found in this study, but it is unclear how much of the measured displacement is owing to coronary compliance and how much to cardiac wall motion. Our results show that the contribution of arterial compliance to the total RCA wall motion is small. With more realistic conditions such as tethering effects of the surrounding myocardium and pericardium, the wall displacement would be restricted even further. One of the other parameters of interest in RCA physiology is the temporal variation of vessel diameter. Figure 4 shows changes in RCA diameter at three different locations: at the throat of the stenosis (L1), in the post-stenotic region (L2) and in the distal region (L3). The results show that the temporal variation of RCA diameter is nearly in phase with pressure, which is consistent with previous experimental and computational studies [11, 35]. Diameter variations during a cardiac cycle are 0.134, 0.266 and 0.486 mm at L1-3, respectively. By normalizing the changes with their corresponding initial diameters, the diameter variation ratios were calculated as 4.71, 5.94 and 13.6% at L1-3, respectively. These values can be compared with previous data on tissue strain and temporal variation of cross-sectional area. For instance, Zeng et al. [21] presented 8.4% of temporal radius variation (15% variation in cross-sectional area averaged along the RCA centerline) using multi-slice CT. Ding et al. [19] reported the total wall strain of 5.4% using biplanar angiograms and Schaar et al. [20] reported a less than 2% circumferential strain using...
ultrasound-based intravascular palpography. Because the circumferential strain in a pressurized vessel is approximately \( \varepsilon_\theta = \frac{u}{r} \), where \( u \) is radial displacement, the corresponding diameter variation is estimated to be 1–2%. Our FSI simulation results show comparable diameter variations to experimental findings in the proximal region, but larger in the distal region particularly when compared with the palpography measurement. One reason could be that the pressure waveform used in the model was acquired from a patient who was also hypertensive; the pressure variation during

Figure 3. Predicted displacement patterns on the lumen-wall interface at peak flow (top left), peak pressure (top right) and in mid-diastole (bottom). The displacement is superimposed onto the original geometry, which is also displayed (transparent). The corresponding time point in each panel is indicated by the vertical line in the velocity and pressure curves.
the cardiac cycle is 84.0 mmHg, which is twice as large as normotensive pulse pressure. Another reason is the lack of surrounding tissue such as myocardium and pericardium in our model, which would restrict the radial motion. However, when compared with the total displacement (maximum 3.85 mm), the diameter variation, 0.486 mm at a maximum, is one order of magnitude smaller, implying that the effect of wall displacement on RCA hemodynamics is likely to be small.

Since low and oscillatory WSS plays an important role in atherogenesis, WSS-related hemodynamic parameters such as time-averaged WSS (TAWSS) and oscillatory shear index (OSI) need to be examined [2, 36, 37]. These parameters were compared between the FSI and rigid-wall models to assess the importance of FSI in determining coronary hemodynamics. As shown in Figure 5, the TAWSS is focally high at the throat of the stenosis for both cases, due to flow acceleration at the throat causing high velocity gradient near the wall. Dilatation of the throat due to pulsating blood pressure (cf. Figure 4) causes reduction in blood velocity at the throat and consequently slightly lower WSS (8.62 Pa for rigid wall and 8.23 Pa for FSI). This can be seen in the velocity contours and vectors around the stenosis shown in Figure 6. For the rigid-wall case at peak flow, the area of high velocity persists far downstream, and the post-stenotic flow is slightly curved and skewed toward the upper wall. Conversely, the high velocity jet attenuates more rapidly in the post-stenotic region with the FSI model, and the downstream flow remains straighter because of the dilated
throat. The same trend is observed at peak pressure and mid-diastole. It is also notable that the flow recirculation in the post-stenotic region, which causes low and oscillatory WSS, is not affected by wall distensibility. As a result, the overall TAWSS distributions for the FSI and rigid-wall cases are almost identical. The effect of wall compliance on instantaneous WSS distributions is also examined. Figures 7 and 8 show comparison of instantaneous WSS contours between the FSI and rigid-wall models at five different time points. It is presented in Figure 7 that WSS patterns for the two models are similar but quantitatively different at all time points except mid systole.
Figure 7. Comparison of instantaneous WSS contours at five time points during the cardiac cycle. Each contour is unwrapped from the RCA surface and mapped onto a rectangle. L4 is on the pericardium-facing side (along outer wall) and L5 is on the epicardium-facing side (along inner wall): (a) reference lines; (b) at peak flow ($t=0.14s$); (c) mid systole ($t=0.20s$); (d) at peak pressure ($t=0.40s$); (e) early diastole ($t=0.50s$); and (f) mid diastole ($t=0.70s$).
Figure 8. Comparison of instantaneous WSS profiles along L5 (inner wall, in the left panels) and L4 (outer wall, in the right panels). The locations of L4 and L5 are shown in Figure 7. The distance from the proximal end is normalized by the total length of the line.
Figure 9. Temporal WSS profiles at the throat of the stenosis (P1, top right), distal to the stenosis (P2, bottom left) and in the distal region (P3, bottom right). P1 and P3 are the intersections of L1, L3 and L5 (cf. Figure 4).

(t = 0.20s). The difference at mid systole is more pronounced in the distal region. Figure 8 enables detailed comparison along the inner and outer walls (L4 and L5). Before peak pressure (t = 0.4s), WSS for the FSI model is lower than that in the rigid-wall case over the entire RCA, whereas WSS for the FSI model is higher than the rigid-wall model after peak pressure. This is because the increasing pressure dilates the vessel and WSS is consequently lowered before t = 0.4s, and the opposite occurs when pressure starts to decrease. The difference between FSI and rigid-wall models is more pronounced in the region distal to the stenosis (normalized distance = 0.25) and larger in the distal region in diastole (t = 0.50, 0.70s). These observations are consistent with Zeng et al. [21] who reported more significant influence of RCA compliance on WSS. In the present study, the reason for larger WSS difference between the FSI and rigid-wall models in the distal region can be explained by the larger diameter variation (13.6%). Temporal variations of WSS at three different locations were also examined (Figure 9). At the throat of the stenosis, the peak WSS values are almost the same for the two models, although WSS magnitude is generally lower for the FSI case before peak pressure (t = 0.40s) and the relationship is inverted after peak pressure. This opposing effect of wall compliance on WSS results in almost identical TAWSS contours between the two models. However, difference in WSS temporal variations is more significant in the distal
region of the RCA, especially at P3. The results show differences in oscillatory nature of WSS due to wall compliance.

The OSI profiles are shown in Figure 10. OSI is defined as

\[
\text{OSI} = 0.5 \left(1 - \frac{\int_0^T |\tau dt|}{\int_0^T |\tau| dt}\right)
\]

where \( T \) is the period of a cardiac cycle and \( \tau \) is WSS as a vector. It tends to be high where the direction of WSS varies in time. It can be seen that OSI is high in the post-stenotic region and at the inside of the relatively sharp bend in the distal RCA (vicinity of L3 in Figure 4), approaching 0.5 for both the FSI and rigid-wall cases. In these regions the flow is likely to be highly disturbed and the results show that the flow disturbance is present regardless of how the wall is modeled. Although the profiles for the two cases look similar, quantitative differences can be observed. OSI values for the FSI case are slightly higher than for the rigid wall, particularly in the distal region. The local maxima at several locations is extracted for quantitative comparison. At P4, P5, P2 and P3 (from proximal to distal, cf. Figure 10), OSI values are 0.129, 0.473, 0.234 and 0.439 for the FSI model, and 0.0968, 0.486, 0.156 and 0.381 for the rigid-wall model, respectively. The comparison shows that the maximum difference occurs at P6, middle of the distal section. This is consistent with data shown in Figures 7, 8 and 9 where larger WSS temporal variation in the distal region was found.

The main focus of this study was to determine the influence of FSI on coronary artery hemodynamics. Hence the arterial wall was modeled as a simple homogeneous isotropic material and the results were analyzed mostly in terms of hemodynamics rather than structural mechanics. Implementation of more sophisticated material models and more realistic boundary conditions to account for the effects of surrounding tissue would be desirable. Indeed, anisotropic arterial stiffness was
observed in a number of studies, e.g. in human abdominal aortas and abdominal aortic aneurysms [38], although similar information on the coronary artery is not available. Anisotropic material models have been proposed to represent such anisotropic behavior [38] including incorporation of collagenous fiber [39]. Studies on the sensitivity of wall stress to the material model for abdominal aortic aneurysms [40] and coronary artery with atherosclerotic lesion [41] showed that anisotropic material models predicted higher wall stress (up to 30%) than that assuming an isotropic material, but no qualitative difference was noted in the stress distribution. This suggests that incorporating the anisotropic material model is likely to make the artery less deformable and would emphasize the insignificance of FSI on coronary hemodynamics. Moreover, it is extremely difficult to obtain fiber orientation and detailed distribution of material compositions in vivo using clinical imaging techniques. More elaborate wall models are important for wall stress analysis, which is another interesting topic related to rupture of vulnerable plaques [15] and will be investigated in the next stage of this study.

4. CONCLUSIONS

Coupled FSI analysis of the human RCA has been carried out in conjunction with physiological velocity and pressure waveforms to investigate the effects of wall compliance on coronary hemodynamics. Comparison of the computational results between the FSI and rigid-wall models showed insignificant difference in TAWSS and OSI, although the predicted wall displacement was comparable with the vessel diameter. However, differences in instantaneous WSS profiles were noticeable, especially in the distal region of the artery.

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