Effects of vessel dynamics and compliance on human right coronary artery hemodynamics with / without stenosis

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Abstract
A computational study of effects of vessel dynamics and compliance on coronary artery hemodynamics with / without stenosis is presented. The coronary artery hemodynamics with stenosis has been a main subject as one of the major cardiovascular diseases induced by atherosclerosis; most computational models assume that the vessel movement and deformation are negligible (Zeng, et al., 2003; Kim, et al., 2010). However, it is still unclear whether the hemodynamic characteristics owing to vessel dynamics and compliance is clinically significant or not particularly under pathological conditions. In this study, we aim at investigating the hemodynamic effects of the vessel dynamics and compliance in right coronary artery under healthy situation without stenosis as well as under diseased conditions with stenosis. We constructed a three-dimensional geometric model of the right coronary artery based on X-ray angiographic images, in which both vessel movement and deformation were taken into account. A specific volumetric flow rate was employed as a boundary condition imposed on inlet. Furthermore, we carried out an extensive study on the inlet waveform dependence and the effects of the vessel compliance on coronary hemodynamics. Our results demonstrate that the conventional assumption on ‘rigid’ artery models holds only in the cases of normal coronary arteries but fails for stenosed coronary arteries where the vessel dynamics and compliance do extend significant influence on distributions of the oscillatory shear indices (OSIs). Moreover, we find that the effects of vessel dynamics and compliance on coronary hemodynamics seem to be independent of both inlet boundary conditions and the vessel compliance.

Key words: Coronary Artery, Compliance, Hemodynamics, Stenosis, Computational Biomechanics

1. Introduction
Cardiovascular disease is one of the leading causes of death especially in developed countries. It is widely known that one of the causes is atherosclerosis, which disturbs blood flow and triggers off the infarction. Coronary arteries that surround heart muscle and play an important role of supplying blood saturated with oxygen to heart; coronary stenosis, which progresses in coronary artery and leads to cardiac infarction in most cases, is one of the major cardiovascular diseases induced by atherosclerosis. Progression of atherosclerosis is presumed to result from permeation of LDL (Low-Density Lipoprotein) cholesterol through endothelial cells and to have close relationship with hemodynamic factors such as the wall shear stress (WSS), the oscillatory shear index (OSI), and the wall shear rate (WSR) (Ku, et al., 1985). Actually, a recent study by Eshtehardi et al., (2012) clarified that both higher WSS and lower WSS in coronary arteries play an important role in rupture and progression of plaque, which points to its clinical importance as well as...
the uniqueness of its hemodynamic characteristics.

Blood flow in coronary arteries is in general characterized by the opposite phase compared with other main arteries, which becomes larger in diastolic phase (Sabiston and Gregg, 1957). On the other hand, coronary arteries also show dynamic movements and deformations, which involve two periodical changes of a global vessel dynamics induced by the dilation or contraction of heart and a local vessel wall deformation owing to vessel compliance.

The global vessel dynamics usually comes out in a form of torsion or translation of coronary arteries; the local vessel deformation results in variation of vessel diameters due to the pressure wave of blood flow. Additionally, coronary arteries are affected by cardiac contraction, which is called “mechanical crosstalk” (Lee and Smith, 1957); a 3~5 mm diameter of major coronary arteries further enhances the difficulty to quantify the three-dimensional flow structures in velocity and pressure. Because of such complexity and difficulty, coronary arteries have often been studied with neglecting both vessel dynamics and compliance (Zeng, et al., 2003; Kim, et al., 2010) in computational fluid dynamic approaches. In the few studies with consideration of the vessel dynamics and compliance, Zeng, et al., (2003) reported that the vessel dynamics had less effect on hemodynamics of human right coronary artery compared with the flow rate waveform imposed on inlet as boundary conditions. On the contrary, Ramaswamy, et al., (2004) pointed out that the dynamics and compliance did influence on the hemodynamics of human right coronary artery. This controversy probably comes from the different conditions employed such as stenosis or non-stenosis, three-dimensional anatomic geometry of blood vessels and vessel dynamics, and boundary conditions imposed on inlet and/or outlets. Actually, the right coronary arterial model by Ramaswamy et al. was stenosed, however, Zeng et al. used a non-stenosed model. Therefore, it is important to perform a systematic study on the effects of dynamics and compliance on hemodynamics in coronary artery under various conditions.

In the present study, we aim at giving a comprehensive answer to this controversy through an extensive study on whether and how the vessel dynamics and compliance in a right coronary artery model affect its hemodynamic characteristics. We focus on investigating the effects of stenosis in the coronary artery model and flow-rate–based boundary conditions at inlet. In section 2 we describe a morphological modeling method for reconstructing a three-dimensional geometric model of the right coronary artery based on X-ray angiographic images, which includes both vessel movement (dynamics) and deformation (compliance), and an in-house computational fluid dynamic method for blood flow simulation. Then computational results are presented and discussed extensively in section 3, which is followed by concluding remarks in section 4.

2. Methods

2.1 Morphological modeling of coronary arteries: geometry, movement and deformation

To reconstruct a three-dimensional morphological model of the right coronary artery with consideration of vessel movement and deformation, we utilized bi-plane X-ray angiographic images, which consisted of 14 frames for a heart beat and provided by the Ninth People’s Affiliated Hospital, School of Medicine, Shanghai Jiao Tong University (Fig. 1). The object was an 82-year-old female who was observed to have a left coronary stenosis and its heart rate was approximately 60 bpm. We first extracted the outline of the right coronary artery based on the two mutually perpendicular X-ray angiographic images, and calculated the coordinates of its centerline and cross-sectional diameters for each frame of 14 frames, which was done using a commercial image-processing software, Rhinoceros (Robert McNeel & Associates, USA). With this geometric information, we then reconstructed three-dimensional geometry of the coronary artery by approximating each cross-section as an elliptic to form a skeleton model and generated computational grids with an in-house grid generator. Because we only had 14 frames in each cardiac cycle, a cubic spline interpolating technique was further introduced to define grids at intervals between frames. Given the computational grids at each time step that provide the information of spatial coordinates of the vessel wall, the vessel dynamics and compliance can be described in detail in terms of vessel movement and deformation. Vessel movement and spatial-averaged deformation during a cardiac cycle derived from constructed model are shown in Fig. 2; the maximum deformation was calculated to be approximately 12%, which was within the physiological range (Fig. 2C).

In this study, we aim at evaluating the hemodynamic effects of vessel dynamics and compliance in non-stenosed and stenosed arteries. Based on the previous prototype model of the right coronary artery shown in Figs. 2A and 2B, we further built up a symmetrically stenosed model with a stenosis rate of 75%, which was artificially added more than 2.5
Fig. 1 Image-based models of non-stenosed (A) and 75% stenosed (B) human right coronary arteries at mid-diastolic: \( t / T = 0 \). The reconstructed computational model is superimposed onto X-ray angiographic images, with a grid of 65100: 100 in axial direction, 31 in circumferential direction and 21 in radial direction.

Fig. 2 Reconstructed coronary artery models in a cardiac cycle: coronal plane (A) and sagittal plane (B). Black lines show the centerline of coronary model. a, b and c indicate the location of coronary model at \( t/T = 0, 0.30 \) and 0.60, respectively. (C): Spatial-averaged rate of diameter change of the reconstructed coronary model. The diameter at each time instant was normalized by that at \( t/T = 0 \). ED and ES are abbreviation for end-diastole and end-systole, respectively.
diameters downstream the inlet (Fig. 1B). Note that the case with a 75% stenosis is clinical criteria because a positive action, such as percutaneous coronary intervention or coronary bypass graft surgery, is usually taken when the stenosis rate exceeds 75%, which is defined as:

\[
\text{Stenosis rate} \ [\%] = \frac{A_s}{A_n} \times 100,
\]

where \(A_s\) and \(A_n\) are the area of stenosed and normal cross-section, respectively.

In order to estimate the effects of dynamics and compliance of stenosed / non-stenosed coronary artery, we further developed physiological, compliance, and dynamic models for each arterial model; Physiological model has both dynamics and compliance, compliance model only compliance, and dynamic model only dynamics. Compliance model and dynamic model were developed using time-averaged datasets of centerline and cross-sectional diameter, respectively.

### 2.2 Computational fluid dynamic modeling of blood flows in coronary arteries

For the unsteady flow in a moving/deforming vessel model we used an in-house computational fluid dynamic (CFD) solver based on a finite volume method (Liu, et al., 1999). The governing equations are the three-dimensional, incompressible, unsteady Navier-Stokes equations written in strong conservation form for mass and momentum. The artificial compressibility method is used by adding a pseudo time derivative of pressure to the equation of continuity. For an arbitrary deformable control volume \(V(t)\), the non-dimensionalized governing equations are

\[
\int_{V(t)} \left( \frac{\partial Q}{\partial t} + \frac{\partial q}{\partial t} \right) dV + \int_{V(t)} \left( \frac{\partial F_x}{\partial x} + \frac{\partial G_y}{\partial y} + \frac{\partial H_z}{\partial z} + \frac{\partial F_x}{\partial x} + \frac{\partial G_y}{\partial y} + \frac{\partial H_z}{\partial z} \right) dV = 0,
\]
where

\[
\mathbf{Q} = \begin{bmatrix} u \\
v \\
w \\
o \end{bmatrix}, \quad \mathbf{q} = \begin{bmatrix} u \\
v \\
w \\
o \end{bmatrix}, \quad \mathbf{F} = \begin{bmatrix} u^2 + p \\
uv \\
uw \\
\lambda u \\
p \\
\lambda w \\
\lambda w \\
\end{bmatrix}, \quad \mathbf{G} = \begin{bmatrix} vu \\
v^2 + p \\
vw \\
\lambda v \\
w \\
\lambda w \\
\lambda w \\
\end{bmatrix}, \quad \mathbf{H} = \begin{bmatrix} wu \\
wv \\
w^2 + p \\
\lambda w \\
\lambda w \\
\lambda w \\
\lambda w \\
\end{bmatrix},
\]

\[
\mathbf{F}_v = -\frac{1}{Re} \begin{bmatrix} 2u_x \\
u_y + v_y \\
u_z + w_z \\
0 \end{bmatrix}, \quad \mathbf{G}_v = -\frac{1}{Re} \begin{bmatrix} v_x + u_x \\
v_x + w_x \\
v_y + w_y \\
0 \end{bmatrix}, \quad \mathbf{H}_v = -\frac{1}{Re} \begin{bmatrix} w_x + u_x \\
w_y + w_y \\
w_x + w_x \\
0 \end{bmatrix}.
\]

In the preceding equations, \( \lambda \) is the pseudo-compressibility coefficient; \( p \) is pressure; \( u, v, \) and \( w \) are velocity components in Cartesian coordinate system \( X, Y, \) and \( Z; t \) denotes physical time while \( \tau \) is pseudo time; and \( Re \) is the Reynolds number. Note that the term \( q \) associated with the pseudo time is designed for an inner-iteration at each physical time step, and will vanish when the divergence of velocity is driven to zero so as to satisfy the equation of continuity. More detailed information is introduced in Liu, et al., (1999).

A volumetric flow rate waveform imposed on inlet as a boundary condition was extracted from Matsuo, et al., (1988), which had been measured in a normal female with Doppler flow-meter catheter (Fig. 3). At inlet a velocity profile was assumed to be fully developed in the form of Womersley solutions while a zero-gradient condition was imposed for pressures; at outlet a zero-gradient condition was for velocity and the pressure was set to be zero. Computational parameters are given in Table 1 in which a reference length and a reference velocity were defined to be the coronary diameter and the time-averaged mean velocity at inlet. Because the flow rate at inlet was not identical to the geometric model of the right coronary artery, a specific tuning method was hereby introduced to well synchronize the vessel movement and deformation with the flow rate waveform based on the CT angiographic images. This was realized based on the fact that most contrast agent injected into the coronary remains to harbor at the inlet during diastole but is flowed away downstream the aorta during systole (Fig. 4).

For an extensive study to investigate how vessel compliance and flow rate waveform influence on coronary hemodynamics, additional computations were carried out with three different flow rate waveforms as well as different vessel stiffness. In addition to the waveform shown in Fig. 3, we constructed other two waveforms (Fig. 5) where \( B \) was extracted from Atbek et al., (1975) and \( C \) was taken to be an 180° phase-delayed as shown in Fig. 3. These

![Fig. 4 The X-ray angiographic images at \( t / T = 0.36 \) (A: diastole) and \( t / T = 0.43 \) (B: systole). A white circle indicates the remaining contrast agent at the coronary inlet or aortic root (A); the contrast agent is invisible at systole (B), which is flowed away downstream the aorta.](image-url)
waveforms were adjusted so that each mean flow rate was equal to 1.65 ml/s. For the vessel stiffness, we employed four cases with the maximal deformation in diameter from 0%, 6%, 12% up to 15% (Fig. 6), based on the studies by Lee and Smith, (2012), Atbek, et al., (1975), Qiu and Tarbell, (2000), and Reddy, et al., (1993). Note that here all the cross-sections were assumed to change with the same rate in the change of diameter at each time instant. All computations were undertaken up to five cycles so as to obtain a stationary stage of the velocities and pressures; and the computed flow fields were then visualized with commercial software of FIELDVIEW (Intelligent Light, USA).

3. Results

3.1 Effects of vessel dynamics and compliance on coronary artery hemodynamics without stenosis

The rate of spatial-averaged diameter change of the coronary model as shown in Fig. 2C is apparently out of phase with the flow rate waveform (Fig. 3). This is due to the contribution of coronary pressure and resistance in distal capillary arteries. During systole, the aortic pressure increases, leading to increasing the coronary pressure and hence expanding right coronary artery while a higher resistance in distal capillary arteries impedes coronary blood flow. During diastole, the coronary pressure decreases with the resistance lowered in distal capillary arteries leads to reducing the diameter of right coronary artery and hence increasing the coronary blood flow (Weissman, et al., 1995; Westerhof, et al., 2006; Zeng, et al., 2008). Velocity vectors and WSSs associated with the three models without
stenosis are first visualized and illustrated in Fig. 7.

It is seen that at the end of diastole the flow migrates toward the outer wall and consequently shows a high WSS region in all the three models. However, these does exist some pronouncing difference among them. Comparing the physiological model (A) to the compliance model (B), we see that obvious stronger migrating flow in the physiological model (A) and hence a higher WSS region at the outer wall though time courses of spatial-averaged WSS of two models are almost the same. On the other hand, no significant difference is observed in the spatial distribution of WSS between physiological (A) and dynamic models (C) though obvious difference is shown in time courses of WSS due to

![Fig. 7 Hemodynamics of normal right coronary arteries: upper panel shows distributions of wall shear stress and velocity vector for three models of physiological model (A), compliance model (B), and dynamic model (C) at $t / T = 0.25$ (end-diastolic phase); middle panel represents the OSI distributions; lower panel shows time courses of spatial-averaged WSS.](image-url)
lower WSS at inner wall of curvature downstream the dynamic model. The difference of WSS observed downstream the models was also reported by the previous study (Zeng, et al., 2008). To evaluate the oscillation feature of coronary hemodynamics we further introduce the oscillatory shear index (OSI), which is defined as:

\[
\text{OSI} = \frac{1}{2} \left( 1 - \frac{\tau_{\text{mean}}}{\tau_{\text{abs}}} \right),
\]

where

\[
\tau_{\text{mean}} = \frac{1}{T} \int_0^T \tau \, dt, \quad \tau_{\text{abs}} = \frac{1}{T} \int_0^T |\tau| \, dt, \quad \tau = \mu \frac{\partial(u-u_g)}{\partial \zeta},
\]

Here \( T \) denotes cardiac duration, \( \mu \) viscosity, \( u \) velocity vector in world coordinate system, \( u_g \) velocity of vessel wall, and \( \zeta \) coordinate of grid perpendicular to vessel wall. As discussed by many conventional studies on relationship between the high OSI location and cardiovascular diseases (Taylor et al., 1998; Torii et al., 2009), we made a specific focus on how vessel dynamics and compliance affect the OSI distribution. There exists a pronounced trend that comparatively higher OSIs are observed on both inner and outer walls in the physiological (A) and dynamic (C) models owning to the vessel movement. However, obviously it is hard to figure out the difference of OSIs between physiological and dynamic models and the compliance model, which is a margin less than 0.01 over a range of OSIs from 0 to 0.5 (Fig. 7).

### 3.2 Effects of vessel dynamics and compliance on coronary artery hemodynamics with stenosis

Then an extended study was undertaken of the effects of vessel dynamics and compliance on coronary artery hemodynamics with a 75% stenosed model (Fig. 8). Interestingly a high WSS region is seen upstream the stenosis while a large recirculation zone is also observed at inner wall downstream the stenosis. Furthermore, the time course of spatial-averaged WSSs shows a pronounced difference between the physiological model (A) and the compliance model (B) particularly at end-diastolic (\( t / T = 0.25 \)) but almost the same trend in case of non-stenosed model. Obviously, the OSIs are remarkably increased in the 75% stenosed model compared with those in the non-stenosed model (Fig. 7). This is because that the stenosis results in accelerating the blood flow upstream the stenosis and a large-scale flow separation downstream the stenosis, respectively. In addition, we find a pronounced difference in the OSI distributions among the three models with 75% stenosis: higher OSIs are observed at the stenosis in the physiological model and dynamic model but are not in the compliance model (Fig. 8). Moreover, the OSIs downstream the stenosis shows higher values in the dynamic model rather than in the physiological model. Figure 9 shows the OSIs distribution along the inner and outer walls, in which two peaks are observed at the stenosis (\( l / L = 0.20 \)) and downstream the stenosis (\( l / L = 0.30 \)) separately. Apparently the existence of the stenosis leads to a significantly different OSI distribution among the physiological model (A), the compliance model (B), and the dynamic model (C), which are not seen in the non-stenosed model. The OSIs in the 75% stenosed model are obviously largely increased showing some variation over a range from 0.01 up to 0.22 with consideration of the coronary artery dynamics and/or compliance (Fig. 9), whereas the range is largely reduced to a margin in less than 0.01 in the non-stenosed model.

### 4. Discussion

In all the cases with consideration of vessel dynamics, a strong flow migration toward the outer wall was observed in the physiological and compliance models particularly at systole when the coronary artery has its maximum centerline velocity, which is observed from end-diastole to early-systole. With respect to the effects of vessel dynamics on coronary artery hemodynamics, our results demonstrate that a distinct difference in OSI distributions between physiological and dynamic models is remarkable in the case of a 75% stenosed model but is a margin in a non-stenosed, normal coronary artery. On one hand, this is because velocities of the blood flow in the coronary arteries are much higher than those owing to the vessel dynamics (Fig. 10). Actually, our computed velocities of the vessel dynamics, which was the centerline velocities of the coronary artery, were approximately one-fourth of time-averaged flow velocity in the coronary artery, which is in reasonable agreement with those by previous studies (Torii et al., 2009; Lee and Smith, 2012). On the other hand, a remarkable increase in the OSIs downstream the stenosis relates to the local large-scale flow separation downstream the stenosis where the blood flow is largely disturbed and easily to be
Fig. 8  Hemodynamics of 75% stenosed coronary arteries: upper panel shows distributions of wall shear stress and velocity vector for three models of physiological model (A), compliance model (B), and dynamic model (C) at $t / T=0.25$ (end-diastolic phase); middle panel represents the OSI distribution; lower panel shows time courses of spatial-averaged WSS.
influenced by the vessel dynamics-induced inertial forces. In some previous work, the computed OSIs show more complicated distribution than that in this study even in a normal coronary artery (Torii et al., 2009), which may be because the complicated geometric models of coronary arteries were used with more realistic spatial curvatures and torsions. Therefore, with consideration of the uncertainty in image digitizing, the vessel outline tracking and the

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Fig. 9 OSI distributions along inner wall (a) and outer wall (b). A, B and C are physiological model, compliance model and dynamic model, respectively.

Fig. 10 Time-varying inlet flow velocities, vessel centerline velocities, and the rates of diameter change in a cardiac cycle. Note that the centerline velocities and the rates of diameter change are spatially averaged across each section. Negative rates of diameter change correspond to contraction of vessel wall whereas positive value means expansion.
calculation of vessel dynamics may have involved some numerical errors more or less in the present models. We further made an extended study on the influence of a possible variation in the vessel compliance of the coronary artery on the coronary artery hemodynamics. The variation in the vessel compliance was within a physiological range from 6% to 15% in a rate of diameter change based on previous studies (Lee and Smith, 2012; Atbek et al., 1975; Qiu and Tarbell, 2000; Reddy, et al., 1993). As shown in Figure 11, our results demonstrate that the effect of compliance-induced influence was indeed a margin compared with that owing to vessel dynamics.

In addition, we investigated the hemodynamic dependency of the flow rate waveform at inlet by introducing two more waveforms as shown in Fig. 5: one was obtained by modifying the original one to have a phase difference of 180 degrees compared to the waveform in Fig. 3, and the other was extracted from the study by Atbek et al., (1975). Our results demonstrate that the effects of vessel movement and deformation on the hemodynamics of the coronary artery with crucial stenosis are important regardless of the inlet flow and vessel compliance.

There are some limitations in our study. First, the only one patient was involved in this study. Because morphologic expression of coronary artery is different from each other (Ballesteros, et al., 2011), the number of patients directly affects the generality of the result. Second, the resolution of CTA images is not very high, which may more or less affect the accuracy of computational model. In particular, the wall deformation owing to vessel compliance is relatively small compared to that of vessel dynamics. Thirdly, the flow volume rate waveform imposed on inlet as a boundary condition is extracted from a literature, which is not a patient-specific one identical to the geometric model of the right coronary artery. In spite of these limitations, we carried out a systematic study of these effects, which thereby confirmed that these limitations would not affect the conclusion reached here. Furthermore, our results show reasonable and consistent agreement with the previous studies by Zeng et al., (2008) and Ramaswamy et al., (2004).

5. Conclusion

We constructed three-dimensional image-based coronary arterial models with consideration of vessel dynamics and compliance and investigated those effects on hemodynamics of human right coronary artery by means of computational analysis. Consequently we found that the vessel dynamics and compliance can extend significant influence on a stenosed coronary artery compared with a non-stenosed coronary artery. Our results therefore point to the importance that the vessel dynamics and compliance in a crucially stenosed coronary artery can be a key issue in influencing the OSI distributions, which should be taken into account.

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Fig. 11 OSI distributions of four models that have deferent values of compliance; Maximum rates of diameter change of A, B, C, and D were adjusted to 0% (rigid), 6%, 12%, and 15%, respectively. The vessel dynamics was also taken into account in the computations.
References


