

Does the shape of inflow velocity profiles affect hemodynamics in computational coronary artery models?

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Abstract— In this study, the impact of velocity inflow profiles shape on computational hemodynamic models of coronary arteries was investigated. To this purpose, 3D realistic velocity profiles were generated analytically and prescribed as inflow boundary condition and the impact on near-wall and intravascular flow was assessed. The results suggest that the impact of the shape of inflow velocity profiles on simulated coronary hemodynamics is limited to the proximal segment, while the global hemodynamics is poorly affected.

Keywords— Coronary Arteries, Computational hemodynamics, Wall Shear Stress, Helical Flow

I. INTRODUCTION

CORONARY arteries are among the most clinically significant arteries of the human body, and the role of hemodynamics on atherosclerosis initiation and progression is well recognized [1]. In this regard, computational fluid dynamics (CFD) has emerged in recent years as a powerful tool for the exploration of hemodynamics inside coronary arteries, with potential application to diagnostics [2]. However, the paucity of *in vivo* blood velocity data could introduce uncertainties that could weaken the findings of *in silico* studies. In particular, most studies on coronary hemodynamics prescribe idealized (flat, or parabolic) velocity profiles as inflow boundary conditions. This level of idealization clashes with the eccentric shaped velocity profiles with a not negligible presence of in-plane velocity components observed both *in vitro* and *in silico* in the left coronary artery [1,3,4], as a consequence of the presence of bifurcations, branching and geometric complexity. Here, we contribute to define the budget of uncertainty associated with idealizations introduced in computational hemodynamic models of the coronary circulation. In particular, the impact of the shape of velocity profiles applied as inflow boundary condition was investigated with regards to near-wall and intravascular coronary hemodynamics. To this end, physically meaningful 3D velocity profiles were generated analytically and applied as inflow boundary conditions in a realistic model of left anterior descending (LAD) coronary artery.

II. METHODS

A. Computational hemodynamics

A patient-specific model of LAD was reconstructed from two angiographic projections, acquired at Città Della Salute e della Scienza (Turin, Italy), using the commercial software QAngio XA bifurcation (Medis medical imaging systems, Leiden, the Netherlands). After discretization of the reconstructed 3D geometry (mesh cardinality= 3,281,383 tetrahedral elements), the governing equations of fluid motion

were solved under steady-state conditions by using SimVascular, an open-source code based on finite elements method [5].

Walls were assumed as rigid with no-slip boundary condition. Blood was modelled as an incompressible, Newtonian fluid (density = 1060 kg/m³, dynamic viscosity = 0.004 Pa·s). Since *in vivo* measured hemodynamic data were not available, the inlet flow rate and flow split at bifurcations were estimated based upon the hydraulic diameters of inflow and outflow sections as proposed elsewhere [6].

B. Analytical velocity profiles

The estimated flow rate was prescribed at the inflow boundary in terms of velocity profile. In general, velocity profiles have a through-plane (TP) and an in-plane (IP) component. The TP component was prescribed using a generalized form of parabolic velocity profile, adapted to non-circular cross-sections:

$$\mathbf{v}_n(a, \vartheta) = \{[1 - a^2] + ka[a^2 - 1] \cos(\vartheta)\} \mathbf{u}_n \quad (1)$$

where a is the radial coordinate normalized with respect to surface radius, ϑ is the angular coordinate, k is a coefficient regulating the location of the peak velocity value, and \mathbf{u}_n is the unit vector normal to inlet surface.

The IP velocity component was prescribed in terms of two counter rotating vortices by generalizing the analytical solution of steady flow in a curved pipe [7]:

$$\mathbf{v}_r(a, \vartheta) = [1 - a^2]^2 [4 - a^2] \sin(\vartheta) \mathbf{u}_r \quad (2)$$

$$\mathbf{v}_\vartheta(a, \vartheta) = [1 - a^2] [4 - 23a^2 + 7a^4] \cos(\vartheta) \mathbf{u}_\vartheta \quad (3)$$

where \mathbf{u}_r and \mathbf{u}_ϑ are the unit vectors in radial and angular direction, respectively. Here, four different profiles were generated and tested as inflow boundary condition as summarized and displayed in Figure 1.

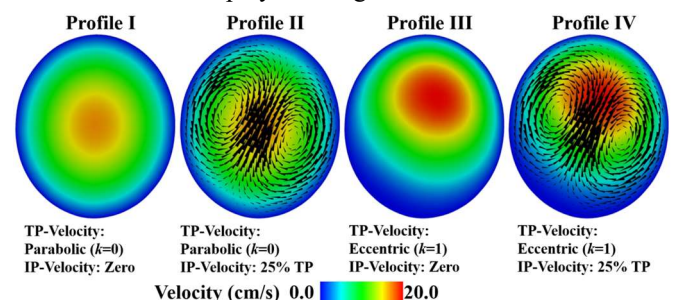


Figure 1. Velocity profiles prescribed as inflow boundary conditions with description of through-plane and in-plane components of velocity. Through-plane (TP) component is represented by the colour map, while in-plane (IP) component was shown in terms of scaled vectors.

Profile I in Figure 1, i.e. the generalized parabolic profile with peak velocity value located on the axis of the vessel, was here considered as the reference condition for comparisons.

C. Hemodynamics descriptors

The influence of the inflow boundary condition on near wall hemodynamics was investigated in terms of wall shear stress magnitude ($|WSS|$).

Motivated by the recently suggested physiological relevance of helical flow in coronary arteries [4], intravascular flow was inspected through the visualization of Local Normalized Helicity (LNH) isosurfaces, computing the local mutual orientation between velocity (\mathbf{v}) and vorticity (\mathbf{w}) vectors, and quantified using two helicity-based descriptors proposed elsewhere [8] in the volume V :

$$h_2 = \frac{1}{V} \int_V |\mathbf{v} \cdot \mathbf{w}| dV \quad (4)$$

$$h_3 = \frac{\int_V \mathbf{v} \cdot \mathbf{w} dV}{\int_V |\mathbf{v} \cdot \mathbf{w}| dV} \quad (5)$$

where h_2 represents the average helicity intensity and h_3 the signed balance of counter-rotating helical flow structures [8].

III. RESULTS

A. Wall shear stress

The distribution of $|WSS|$ at luminal surface is reported in Figure 1. It can be observed that impact of the inflow velocity profile shape is limited to the segment proximal to the inlet section. Conversely, the distal segment presents $|WSS|$ patterns more independent of prescribed inflow velocity profile as differences are negligible across the four investigated profiles.

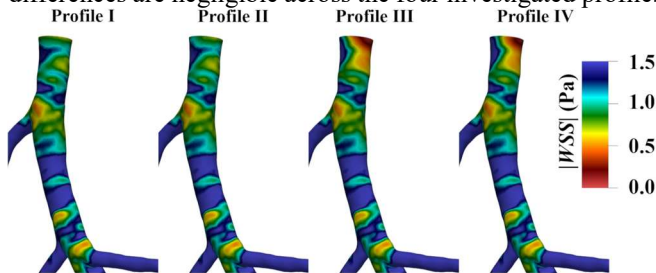


Figure 2. Distribution of $|WSS|$ values at the luminal surface for the four different velocity profiles analysed.

Quantitatively, $|WSS|$ averaged values over the whole luminal surface present differences from the reference profile lower than 1.0% (maximum for profile IV). Focusing on the region proximal to the inlet section (i.e. between inlet and first side branch) the highest difference was observed for the profile IV (6.2%).

B. Helical flow

Figure 3 shows that distinguishable counter-rotating helical flow patterns develop in the LAD, as highlighted by LNH isosurfaces.

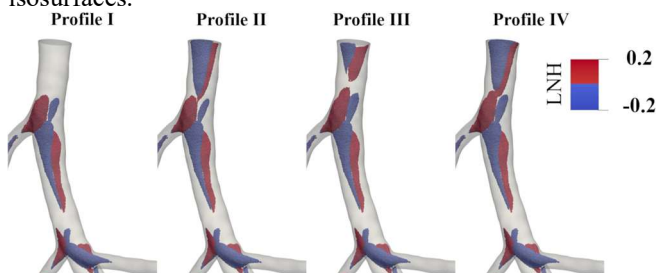


Figure 3. Intravascular fluid structures shown in terms of LNH isosurfaces for the four different velocity profiles analysed (clockwise and counter-clockwise helical structures are coloured in blue and red, respectively).

It can be noticed that the shape of the helical flow patterns is influenced by the shape of the inflow velocity profile only in the proximal segment. This observation is quantitatively confirmed by values of helical flow-based descriptors, reported in Table I. With respect to profile I, the highest difference in the helicity based descriptors was found for profile IV, both for proximal segment (264.8% and 65.2% for h_2 and h_3 respectively) and globally (7.1% and 7.2% for h_2 and h_3 respectively).

TABLE I

Profiles	h_2^{Total} (m/s^2)	$h_2^{Proximal}$ (m/s^2)	h_3^{Total}	$h_3^{Proximal}$
Profile I	6.280	0.890	-0.276	0.040
Profile II	6.654	2.838	-0.262	0.027
Profile III	6.388	1.399	-0.270	0.103
Profile IV	6.724	3.245	-0.256	0.067

Helical flow-based descriptors computed over total model and proximal tract for the four velocity profiles tested as inflow boundary condition.

IV. CONCLUSION

In this exploratory study, we investigated the impact of 3D velocity profiles used as inflow boundary conditions in computational hemodynamic models of coronary arteries. Our findings for the LAD suggest that the imposition of realistic 3D velocity profiles at the inflow section influence the LAD segment proximal to the inflow section of the model. In particular, our results suggest that a combination of features like eccentricity and secondary flows in the inflow velocity profile could impact flow patterns. However, considering the exploratory nature of the study, further analysis (e.g. unsteady-state simulations, other analytical formulations of velocity profiles) will be necessary to definitively assert that a 3D velocity profile does not globally influence patient-specific coronary artery hemodynamics simulations. The possibility to use idealized models instead of *in vivo* measured data would improve and simplify the use of computational hemodynamics as diagnostic tool applied to coronary arteries.

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