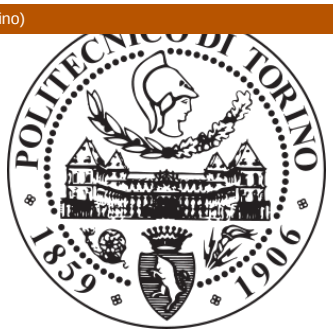

Idealized models of arterial bifurcation to investigate diseased coronary hemodynamics

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***Abstract*— Hemodynamics is recognized as a main factor of the onset and progression of atherosclerotic lesions at coronary bifurcations. In this study, computational fluid dynamics (CFD) is applied to investigate the impact that the distal angle, a peculiar anatomical feature, has on the hemodynamics of population-based models of diseased coronary bifurcation. Both near-wall (WSS) and intravascular flow features (helical flow) are analysed and related to the geometry of the bifurcation. Results show that the bifurcation angle moderately influences local hemodynamics. Additionally, the complex interplay between anatomy and intricate fluid structures in bifurcations is highlighted.**

***Keywords*—coronary bifurcation, computational fluid dynamics, wall shear stress, helical flow.**

I. Introduction

ATHEROSCLEROTIC lesions at coronary bifurcations account for 15-20% of total coronary lesions [1]. The so-called hemodynamic hypothesis suggests that local hemodynamics is a main factor of the onset and progression of lesions at coronary bifurcations [2]. Based on this hypothesis, atherosclerotic plaque formation at specific locations, such as the lateral walls opposite to the carina, has been attributed to the unique local flow conditions, in particular the low and oscillatory wall shear stress (WSS) [2]. This peculiar local hemodynamics is majorly determined by the underlying anatomical features of the bifurcation, like distal bifurcation angle, curvature of the vessel, and degree of stenosis [2,3].

The aim of this study is to investigate the impact of peculiar anatomical features of a diseased coronary bifurcation on local hemodynamics. In order to do so CFD simulations were performed in population-based, idealized coronary bifurcation models. At this stage, the hemodynamic impact of the bifurcation distal angle is investigated. Both near-wall (WSS) and intravascular flow features (helical flow) were considered and related to the geometry of the bifurcation.

II. Methods

An idealized, parametric bifurcation model representative of a diseased left anterior descending (LAD) coronary artery with its diagonal branch is created using the open source software pyFormex (nongnu.org/pyformex) (Fig. 1). The model has a 3.30 mm proximal main branch (MB) diameter. The diameters of the distal MB and of the side branch (SB) are defined according to Finet's law [4], and they are set equal to 2.77 mm and 2.10 mm, respectively. The presence of 60% stenosis is considered at the proximal MB, and at distal MB and SB. The lesion is 12 mm long and eccentric, with plaque located at the inner arc of the vessel (Fig. 1B). The model curvature, taking into account the presence of the heart, is equal to 0.018 mm^{-1}. The distal angle (α) is varied within the physiological range [5], to investigate its impact on local hemodynamics. In particular, three bifurcation models with distal angle of 40°, 55°, and 70°, respectively, are created. The bifurcation models are discretized with a hybrid mesh of cardinality ~2,500,000. The governing equations of motion are solved using the finite volume method-based code Fluent (ANSYS Inc., USA). Unsteady-state condition is simulated by imposing a pulsatile flow tracing representative of a human LAD (mean flow-rate equal to 40 mL/min) at the inflow section. A flow-split of 65%-35%, calculated using the relation proposed by van der Giessen et al. [6], is imposed at the MB and SB outlets, respectively. The arterial wall is assumed as rigid, no-slip condition is applied at vessel wall. The blood is modelled as an incompressible, non-Newtonian fluid using the Carreau model.

Near-wall hemodynamics is evaluated in terms of relative residence time (RRT), a hemodynamic descriptor able to identify regions of low and oscillatory WSS [7]. The fraction of the luminal area exposed to RRT>4Pa^{-1} is calculated in the whole model and in the daughter branches. Intravascular fluid structures are investigated in terms of helical flow. Helicity intensity *h* is calculated integrating the absolute value of the

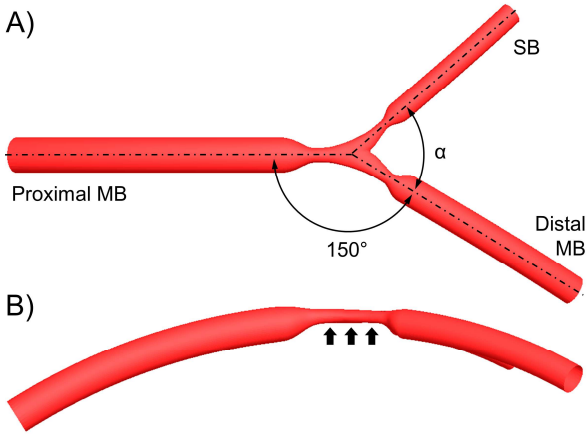

Figure 1 – Parametric coronary bifurcation model of the diseased left descending coronary artery with its first diagonal branch: top (A) and lateral (B) view. The black arrows at the plaque location in B) indicate the plaque eccentricity.

helicity density (i.e., the internal product of velocity and vorticity vectors) over the volumetric fluid domain, averaged over the cardiac cycle [7].

III. Results and Discussion

RRT mainly capture flow disturbances at both distal MB and SB (Fig. 2A), in agreement with previous observations. Qualitatively, RRT seems to be minimally influenced by the distal bifurcation angle, as also confirmed by the fraction of the luminal surface exposed to low and oscillatory WSS (RRT>4 Pa^{-1}), reported in Table I. When considering the branches separately, the luminal surface exposed to high RRT decreases in the distal MB and increases in the SB for larger branch angles, albeit moderately.

The complexity of the hemodynamics establishing within the bifurcation can be appreciated by visualizing local normalized helicity (LNH) isosurfaces. In all cases, well-distinguished helical counter-rotating flow structures (left-handed, blue color; right-handed, red color; Fig. 2B) characterize the intravascular hemodynamics. For all cases, helical flow structures originate in the bifurcation region and develop into the daughter branches, elongating more downstream in the MB than in the SB. The quantitative assessment of helicity intensity confirms that the total amount of helical flow in the three cases is similar (Table I). Helical flow topology is independent from the bifurcation angle, being mainly affected by the stenosis degree and the curvature of the vessels. The relationship between bifurcation angle and the hemodynamic risk of disease is documented. Previous investigations showed that larger angles increase the area exposed to low and oscillatory WSS around the carina [8]. It has also been observed that wider angle may induce plaque proliferation at the bifurcation region and lead to more severe stenosis in bifurcation area and SB ostium, and consequent higher risk of SB occlusion [8]. Moreover, it has been also noticed that the bifurcation angle affects intervention strategy selection, especially stenting procedure. The bifurcation angle is reported to be an independent predictor of SB occlusion after MB stenting, based on 1,171 patients [8], with larger angles associated to higher pressure drop and flow resistance. Our results in a stenosed bifurcation confirm these observations, even if moderately.

IV. Conclusion

In this study, the influence of bifurcation angle on near-wall and intravascular flow quantities is evaluated. CFD simulations show that the bifurcation angle moderately influences local hemodynamics, with differences that are probably not clinically meaningful. The proposed approach, even if preliminary, provides a controlled benchmark to investigate the effect of various geometrical features, overcoming the current limitation of previous studies, which rely on 2D *in vivo* measurements, leading to not conclusive results. Our findings also highlight the complex interplay between anatomy and intricate fluid structures in bifurcations. Additional analyses will be conducted by varying the curvature and stenosis grade of the bifurcation.

TABLE I
Summary of computed hemodynamic descriptors.

	Relative Area (RRT > 4 Pa^{-1})			*h* (m/s^2)
	Whole Model	*Distal MB*	*SB*	
40°	1.18%	5.81%	3.83%	3.52
55°	1.19%	4.54%	5.70%	3.60
70°	1.25%	4.27%	6.98%	3.73

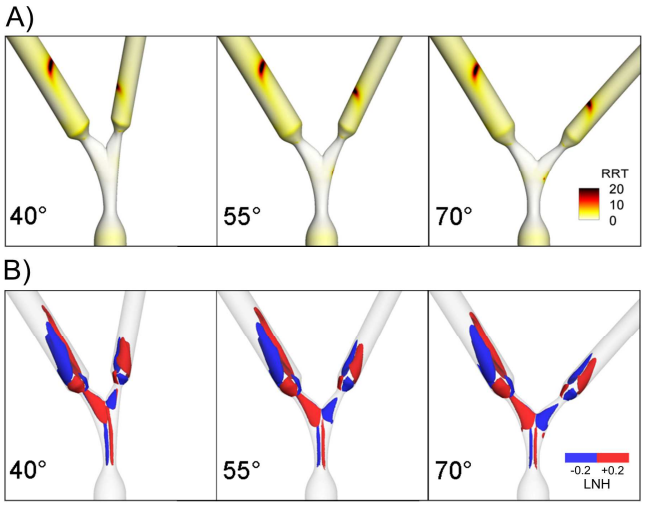

Figure 2 – Hemodynamics results for the three coronary bifurcation models: A) contour plots of relative residence time (RRT, Pa^{-1}); B) isosurfaces of local normalized helicity (LNH). Positive and negative values of LNH indicate counter-rotating flow structures.

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