34th Annual International Conference of the IEEE EMBS
San Diego, California USA, 28 August - 1 September, 2012

Characterization of Physiological Flow in Arterial Bifurcation Lesions

Marjan Molavi Zarandi, Rosaire Mongrain, Olivier F. Bertrand

Abstract—**Bifurcation lesions account for approximately 15-20% of all percutaneous coronary interventions. Coronary artery bifurcation lesions have always represented a major challenge for interventional cardiologists. A part of this challenge is related to the variety of coronary lesions located at bifurcations. Hemodynamics factors and biomechanical forces play key roles in atherosclerosis initiation and progression. Wall Shear Stress (WSS) is the most important flow related factor in the initiation and development of lesions. In this paper a numerical analysis of pulsatile blood flow in stenosed (constricted) coronary artery bifurcation is performed. Two-dimensional models of moderate stenoses of 50% in coronary bifurcations are reconstructed to represent the seven possible situations that the Medina lesion classification can assess. Our computational results provided with detailed numerical analysis on the blood flow pattern and WSS distribution that may be involved in a better understanding of the regions of lesion initiation and progression. It was shown that the worst bifurcation diseases as assessed by the Medina anatomical classification are not the worst lesions based on the hemodynamical parameters.**

I. Introduction

From clinical practice, it is known that coronary artery bifurcations are regions where the flow is strongly perturbed, and is prone to the development of atherosclerotic lesions. As a definition, bifurcation lesion is a coronary artery narrowing that may involve the proximal main vessel, the distal main vessel, and the side branch [1]. Bifurcation lesions have always represented a major challenge for percutaneous treatment [2-5]. Part of this challenge is related to the variety of coronary lesions located at a bifurcation which present wide range of anatomical morphologies. Currently, there are seven different coronary bifurcation lesion classifications in the literature [6-13].

Marjan Molavi Zarandi, Department of Mechanical Engineering , McGill University, Montreal, Quebec, H3A 2K6, Canada
marjan.molavizarandi@mail.mcgill.ca
Rosaire Mongrain, Department of Mechanical Engineering ,McGill University, Montreal, Quebec, H3A 2K6, Canada
rosaire.mongrain@mcgill.ca
Olivier F. Bertrand, Faculty of Medicine, Laval University, Quebec City, G1V 4G5, Canada
olivier.bertrand@criucpq.ulaval.ca

These classifications characterize bifurcation lesions based on the presence or absence of stenosis within the three vessels of the bifurcation.

In all these classifications, different lesion types are named using numbers or letters. Most of the classifications are difficult to remember [6-11]. The first attempt to overcome some of the limitations of previous classifications and simplify these classifications was successfully made by Medina *et al*. [12]. In this classification, the bifurcation is divided into three segments: the Main Branch Proximal (MBP), the Main Branch Distal (MBD), and the Side Branch (SB). Any narrowing with critical stenosis of 50% and above in any segment receives the binary value 1; otherwise a binary value 0 is assigned starting from left to right. The three suffixes are separated by commas. In that context, the Medina classification is essentially an anatomical classification.

From clinical practice, it is known that atherosclerosis lesions initiate at the regions where the flow is strongly perturbed. Bifurcations, curvatures and junctions are the regions which are more prone to the initiation and progression of atherosclerotic lesions [13-16]. Hemodynamic factors play a major role in the initiation and progression of atherosclerosis [17-19]. Studies show that arterial regions exposed to low and non-uniform shear stress are more prone to atherosclerotic lesions development [20-22]. To date, there has been no report studying flow conditions in bifurcation lesions associated with the Medina lesion classification. Using computational fluid dynamics (CFD) analysis we studied flow conditions and wall shear stress (WSS) distributions in bifurcations types associated with the Medina lesion classification.

II. Material and Methods

A comprehensive hemodynamic analysis was carried out to obtain the velocity field and associated WSS distribution in a coronary artery bifurcation. The simulation was conducted using COMSOL which is dedicated for various physics and engineering applications amongst CFD analysis. The computational domain was also meshed in this software. The combination of both momentum and continuity equations for transient and Newtonian model of the blood flow were analyzed using the same software. A geometrical model of coronary artery bifurcation was considered to simulate the bifurcation between Left Main Coronary Artery

(LMCA) and Left Anterior Descending artery (LAD). Fig. 1 shows the geometrical model of two dimensional angiographic projections of the bifurcation (Main Branch Proximal (MBP), Main Branch Distal (MBD) and Side Branch (SB)) as well as the lesion locations associated with the Medina lesion classification [12].

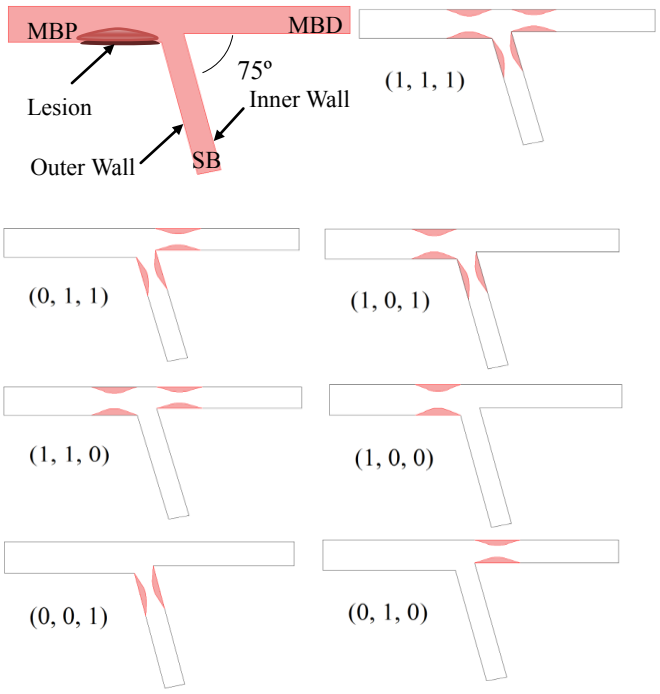


Figure 1. Geometrical model and Medina lesion classification of coronary artery bifurcation.

The parent vessel diameter was 4 *mm* and daughter vessels diameters were 3.4 *mm* and 2.7 *mm* respectively [23]. The lesions length is 6 *mm*. The angle between the centerline LMCA and LAD as well as the angle between the centerline MBD and SB (75˚) corresponds to physiological anatomical range [24]. For the study of blood flow in the arteries, we assumed that blood can be represented by an incompressible fluid which is governed by the momentum equation:

$$\rho(\frac{\partial u}{\partial t} + u.\nabla u) = -\nabla p + \nabla .\tau \qquad (1)$$

and the continuity equation:

$$\frac{\partial \rho}{\partial t} + \nabla .\rho u = 0 \qquad (2)$$

where, ρ denotes the density of the fluid ($kg\ m^{-3}$), u the velocity vector ($m\ s^{-1}$), p the pressure (Pa) and τ the stress tensor which is dependent on the viscosity and shear rate.

Wall shear stress was determined as the product of viscosity (μ) and the shear rate ($\dot{\gamma}$). The shear rate , in two dimensions is defined according to "(3)":

$$\dot{\gamma} = [2\{(\frac{\partial u}{\partial x})^2 + (\frac{\partial v}{\partial y})^2\} + (\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x})^2]^{\frac{1}{2}} \qquad (3)$$

where u, v are the velocity vectors, respectively.

To solve the governing equations, a set of boundary conditions is required. In this analysis, the maximum velocity (V_{max}) varies between 15 *cm/s* and 25 *cm/s* for a coronary artery of 4 *mm* in diameter. The flow was considered laminar and fully developed throughout the study section. Fig. 2 shows the variation of the blood flow rate in the left coronary artery during the cardiac cycle [25].

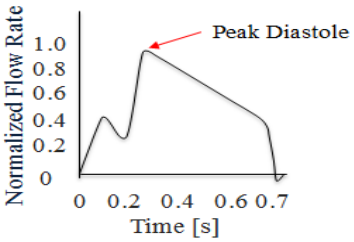
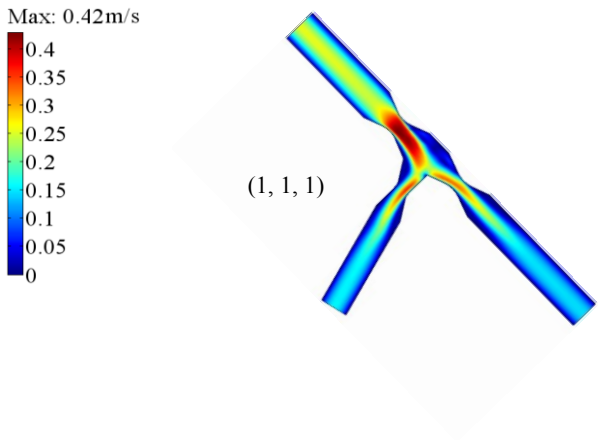


Figure 2. Normalized flow rate waveform for the left main coronary artery.

Pulsatile flow was considered, where the inflow velocity was based on a time-dependent flow rate obtained from Figure 2. The waveform has a period T =0.7 s, where 0 <t (sec) <0.2 is systolic and 0.2 <t (sec) <0.7 is diastolic phase. At the walls, the velocity obeyed the no-slip condition. At the outlet of the daughter vessels, a traction-free condition was assumed as the boundary conditions. The Newtonian blood properties in this model are blood viscosity η=0.0035 *Pa.s*, and blood density ρ =1060 Kg/m^3 respectively [26].

III. Results

Numerical analysis of pulsatile blood flow in stenosed coronary artery bifurcation was carried out. Transient simulations were performed and the corresponding velocity field and shear stress distributions were investigated. In Fig. 3, the seven representative snapshots of the magnitude of the velocity field for 50% stenoses are shown at peak diastole (t=0.27 s) for bifurcation types (1, 1, 1), (0, 1, 1), (1, 0, 1), (1, 1, 0), (1, 0, 0) (0, 1, 0) and (0, 0, 1) associated with the Medina lesion classification.



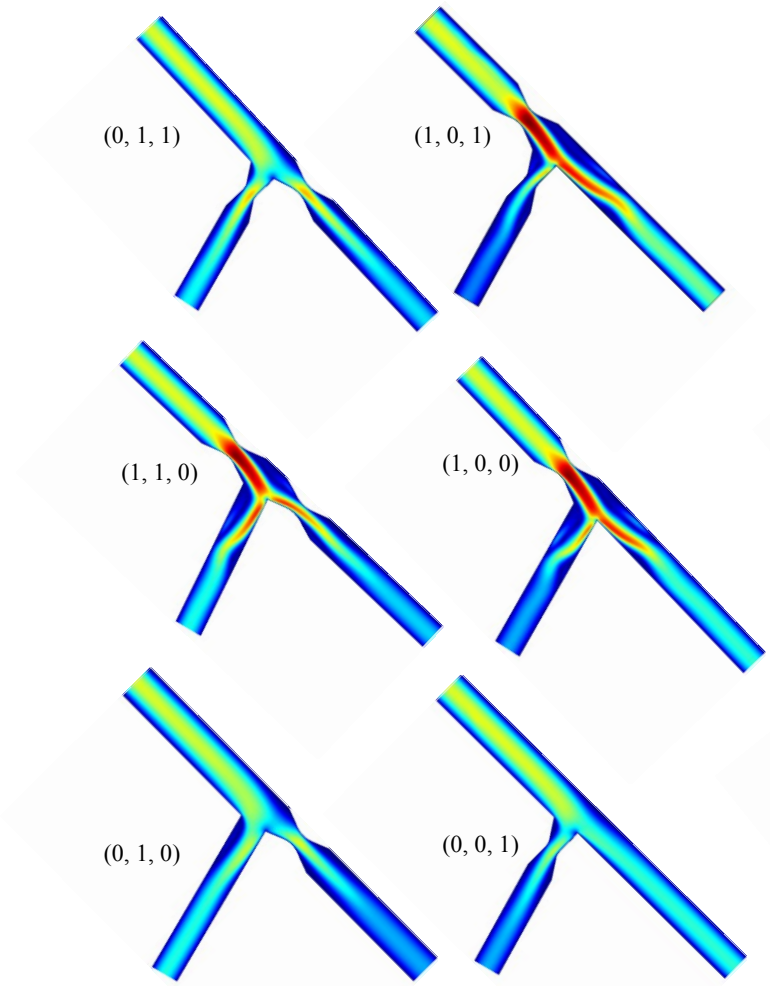


Figure 3. Maps of the velocity fields in 50% stenosed bifurcations at peak diastole.

The simulation results presented in Fig. 3 shows a considerably different velocity profile of the blood flow in the downstream of stenotic bifurcations and demonstrated a significant influence of the bifurcation types on the blood flow pattern, such as flow separation and recirculation zones. The flow pattern in SB is directly affected by flow alterations in MBP and MBD.

Wall shear stress is recognized one of the most important factors in developing of atherosclerosis and arterial branches are found more prone in formation of arterial plaques. Although atherosclerosis affects the entire vascular system, it creates different flow and WSS distributions in SB and MBD in different bifurcation types. Therefore, the WSS distribution is studied for bifurcation types (1, 1, 1), (0, 1, 1), (1, 0, 1), (1, 1, 0), (1, 0, 0), (0, 1, 0) and (0, 0, 1) associated with the Medina lesion classification.

At peak diastolic point of a cardiac cycle, the WSS fields for bifurcation lesions are presented in Fig. 4.

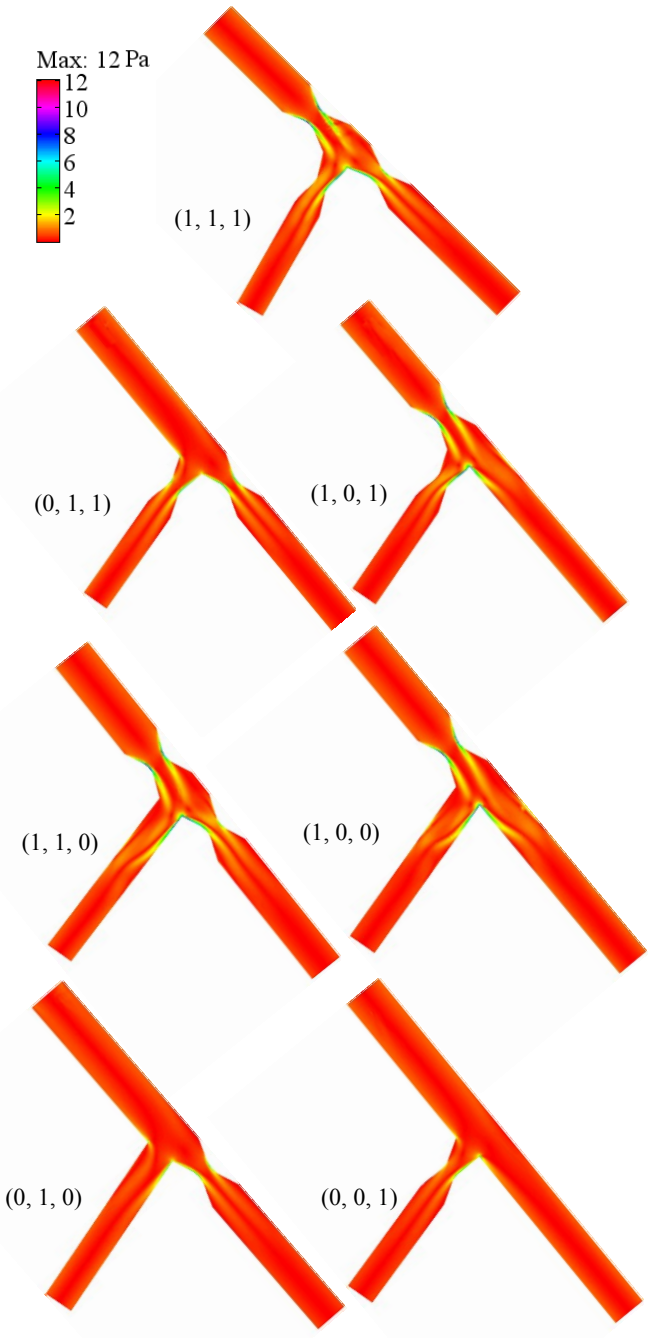


Figure 4. Wall Shear Stress fields in 50% stenosed bifurcations at peak diastole.

The distribution pattern shows that, at the MBP and upstream the flow divider, WSS is uniform along the arterial wall. The peak values of the WSS are at the top of stenosis and at the carina of bifurcations. The WSS value at the inner and outer walls peaks at the center of stenosis and reaches the minimum in the post-stenosis region. The WSS values recover gradually and then remain constant downstream stenosis along the arterial wall. To assess mean WSS along the entire walls at peak diastolic point of a cardiac cycle, the average WSS values along the inner and outer walls of SB and downstream the lesions are computed and the results are represented in Fig. 5.

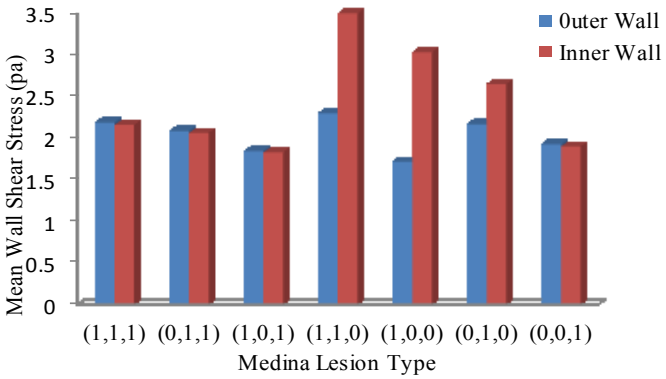


Figure 5. Mean WSS on the outer and inner walls of SB at peak diastole.

From mean WSS values presented in Figure 5, it can be observed that the lowest magnitude of WSS in the outer wall of SB is related to lesion types (1, 0, 1) and (1, 0, 0). Amongst bifurcation types with lesion in their SB, the lowest mean WSS value on the outer wall is related to bifurcation type (1, 0, 1). The larger value of the WSS at peak diastole occurs on the SB inner wall downstream the carina which results in higher mean value of WSS along the entire inner wall in bifurcations with no lesion in their SB ((1, 1, 0) (1, 0, 0) (0, 1, 0)).

IV. CONCLUSION

The current work on two-dimensional axisymmetric stenosis models of a stenotic coronary bifurcation has demonstrated detailed numerical results for time dependent velocity and WSS distributions. Accurate predictions of WSS distribution on the arterial walls and downstream the stenosis is particularly useful in the understanding of the effects of disturbed flow on endothelial cells and blood elements near the wall which may be involved in lesion initiation and progression. In the present study, variations of WSS were studied and shown for seven bifurcation lesion types. Examination of the WSS distribution in bifurcation lesions showed that on the outer walls of SB, the lowest WSS magnitudes are related to the (1, 0, 1) and (1, 0, 0) types of Medina lesion classification. The larger value of the WSS at peak diastole occurs on the SB inner wall downstream the carina. The results indicate that bifurcation types with no lesion in their SB ((1, 1, 0) (1, 0, 0) (0, 1, 0)), exhibit lower WSS values on the outer walls compare to inner walls of the SB.

RFERENCES

[1] I. H. Tanboga, M. Ekinci, T. Isik, M. Kurt, A. Kaya and S. Sevimli,"Reproducibility of Syntax Score: From Core Lab to Real World," *J. Interv. Cardiol*., vol. 24, no.4, pp. 302-306,2011.

[2] F. Zhang, L. Dong and J. Ge,"Simple versus complex stenting strategy for coronary artery bifurcation lesions in the drug-eluting stent era: a meta-analysis of randomised trials," *Heart*, pp. 1676–1681, 2009.

[3] M. Pan, J. Suárez de Lezo, A. Medina, M. Romero, E. Hernández, J. Segura, J. R Castroviejo., D. Pavlovic, F. Melian, A. Ramírez and J. C. Castillo, "Simple and complex stent strategies for bifurcated coronary arterial stenosis involving the side branch origin," *Am. J. Cardiol*., vol. 83, pp. 1320-1325, 1999.

[4] E. Jørgensen, and S. Helqvist, "Stent treatment of coronary artery bifurcation lesions," *Eur. Heart*. J., vol. 28, pp. 383–385, 2007.

[5] I. Iakovou and L. Ge, "Colombo A: Contemporary stent treatment of coronary bifurcations," *J. Am. Coll*. Cardiol., vol. 46, pp. 1446-1555, 2005.

[5] J. Popma, M. Leon and E. J. Topol, "Atlas of Interventional Cardiology," Philadelphia, Pa: Saunders. 1994.

[7] A. M. Spokojny and T. M., Sanborn,"The bifurcation lesion In: Ellis SG, Holmes DR., editors. Strategic Approaches in Coronary Intervention. Baltimore," MD: Williams and Wilkins. 1996, pp. 288.

[8] T. Lefèvre, Y. Louvard, M. C. Morice, P. Dumas, C. Loubeyre, A. Benslimane, R. K. Premchand, N. Guillard and J. F. Piéchaud, "Stenting of bifurcation lesions: classification, treatments, and results," *Catheter Cardiovasc. Interv*., vol. 49, pp. 274-283, 2000.

[9] R. D. Safian, Bifurcation lesions In: Safian R D, Freed M., editors. Manual of Interventional Cardiology. Royal Oak, MI: Physicians' Press, 2001, pp. 221-236.

[10] G. Sianos, M. A. Morel and A. P. Kappetein, "The SYNTAX score: an angiographic tool grading the complexity of coronary artery disease," *Eurointervention*, vol. 1, pp. 219-22, 2005.

[11] M. R. Movahed and C.T. Stinis, "A new proposed simplified classification of coronary artery bifurcation lesions and bifurcation interventional techniques," *J. Invasive Cardiol*., vol. 18, no. 5, pp. 199-204, 2006.

[12] A. Medina, J. Suarez de Lezo and M. A. Pan, "New classification of coronary bifurcation lesions," *Rev. Esp. Cardiol*.,vol. 59, pp. 183, 2006.

[13] T. Asakura and T. Karino, "Flow patterns and spatial distribution of atherosclerotic lesions in human coronary arteries," *Cir. Res*., vol. 66, pp. 1045-1066, 1990.

[14] B. I Tropea, S. Glagov, and C. K. Zarins, 1997, "Hemodynamics and Atherosclerosis," Futura publishing company. Inc., Armonk, NY.

[15] C. G. Caro, J. M. Fitz-Gerald and R. C. Schroter, "Arterial wall shear and distribution of early atheroma in man," *Nature*, vol. 223, pp. 1159-1161, 1969.

[16] A. M. Malek, S. Alper, S.L. Izumo, "Hemodynamic shear stress and its role in atherosclerosis," *Am. Med. Ass*., vol. 282, pp. 2035-2042, 1999.

[17] P. H. Stone, A.U. Coskun, Y. Yeghiazarians, S. Kinlay, J. J. Popma, R. E. Kuntz and C. L. Feldman, "Prediction of sites of coronary atherosclerosis progression: in vivo profiling of endothelial shear stress, lumen, and outer vessel wall characteristics to predict vascular behavior," *Curr. Opin. Cardiol*., vol. 8, pp. 458-470, 2003.

[18] M. A. J. Gimbrone, J. N. Topper, T. Nagel, K. R. Anderson and G. Garcia- Cardena, "Endothelial dysfunction, hemodynamic forces, and atherogenesis," *Ann. N. Y. Acad. Sci*. vol. 902, pp. 230-239, 2000.

[19] K. S. Cunningham and A. I. Gotlieb,"The role of shear stress in the pathogenesis of atherosclerosis*," Lab. Invest*., vol. 85, pp. 9-23, 2005.

[20] S. Fabregues, K. Baijens, R. Rieu and P. Bergeron, "Hemodynamics of endovascular prostheses," *J. Biomech*., vol.31, no.1, pp. 45–54. 1998.

[21] J. A. E. Spaan, J. J. Piek, J. I. E. Hoffman and S. Maria, "Physiological basis of clinically used coronary hemodynamic indices," *Circulation*, vol. 113, pp. 446-455, 2006.

[22] D. N. Ku, D. P. Giddens, C. K. Zarins and S. Glagov, "Pulsatile flow and atherosclerosis in the human carotid bifurcation. Positive correlation between plaque location and low oscillating shear stress," *Arteriosclerosis*, vol. 5, pp. 293-302, 1985.

[23] Z. Kaimkhani, M. Ali and A. M. Faruqui, "Coronary artery diameter in a cohort of adult Pakistani population," *J. Pak. Med. Assoc*., vol. 54, no. 5, pp. 258-261, 2004.

[24] C. Godino, R. Al-Lamee, C. La Rosa, N. Morici, A. Latib, A. Ielasi, C. Di Mario, G. M. Sangiorgi and A. Colombo, "Coronary left main and non-left main bifurcation angles: how are the angles modified by different bifurcation stenting techniques?," *J. Interv. Cardiol*., vol. 23, no. 4, pp. 382-393, 2010.

[25] I. Faik, R. Mongrain, R. L. Leask, J. Rodes-Cabau, E. Larose and O. F. Bertrand, "Time-dependent 3D simulations of the hemodynamics in a stented coronary artery," *Biomed. Mater*., vol. 2, pp. 28-37. 2007.

[26] N. Benard, R. Perault and D. Coisne, "Computational approach to estimating the effects of blood properties on changes in intra-stent flow. *Ann. Biomed. Eng*., vol. 3, no. 8, pp. 1259–1271, 2006.