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GEOMETRIC MULTISCALE MODELLING FOR THE FUNCTIONAL CHARACTERIZATION OF CORONARY BIFURCATION LESIONS

Catherine Pagiatakis[1] and Rosaire Mongrain[1]

[1] Department of Mechanical Engineering, McGill University, 817 Sherbrooke Street West
Montreal, Quebec H3A 0C3
catherine.pagiatakis@mail.mcgill.ca, rosaire.mongrain@mcgill.ca

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INTRODUCTION: Atherosclerosis is an inflammatory response whereby the lumen of a vessel becomes gradually stenosed. In the coronary arteries, when blood flow is critically reduced due to a stenosis, ischemia is induced. For isolated lesions, the idea that the dynamics and functionality of a stenosis (whether or not it induces ischemia) is influenced by both the morphology of the stenosis and the dynamics of the supplied myocardium has been widely accepted. Conversely, the dynamics and consequent functionality of coronary bifurcation lesions are still not well understood. Computational studies have shown that the flow ratio between the daughter vessels is affected nonlinearly by the configuration topology (number and severity of lesions) [1]; however, the downstream myocardial dynamics were neglected. In this study, it is hypothesized that the dynamics of the myocardial beds being supplied by the diseased bifurcation influence the upstream bifurcation dynamics and thus the functional impact of the bifurcation lesion configuration. Therefore, the objective is to study different synthetic coronary bifurcation lesion topologies in a geometric multiscale (MS) framework that couples the 3D model of diseased bifurcation to a lumped-parameter model (LPM) of the rest of the cardiovascular system. The effect of bifurcation lesion topology and relative stenosis severity on the functionality of the lesion configuration is sought.

METHODS: Synthetic 3D geometries of the left main coronary artery (LMCA) bifurcation into the left anterior descending artery (LAD) and the left circumflex artery (LCX) based on a previous study are used [1]. Seven bifurcation lesion topologies, based on the Medina classification, with axis-symmetric stenoses are considered. The severity of the lesions is varied between a mild case (20% area reduction) and a severe case (85% area reduction). A closed-loop, electrical analog LPM is used to model the global dynamics of the left and right heart, the systemic and pulmonary circulation and the dynamics of the myocardium downstream the 3D bifurcation geometries over a full cardiac cycle (based on existing models [2]). Transient flow simulations using the incompressible and Newtonian Navier-Stokes equations will be executed for the 3D bifurcation lesion geometries using the commercial software package ANSYS Fluent (Canonsburg, PA, USA). A partitioned, geometric MS approach based on the work of Quarteroni [3] is utilized to prescribe proper boundary conditions in the 3D model, by coupling the Navier-Stokes equations to the nonlinear system of ordinary differential equations that describes the dynamics of the LPM. Since the effect of the bifurcation lesion topology on the flow rate is sought, the MS algorithm applies average pressures at all the boundaries of the 3D model. The pressures are calculated by the LPM at each time step by forcing its solution with average flow rates obtained from the 3D model at the previous time step.

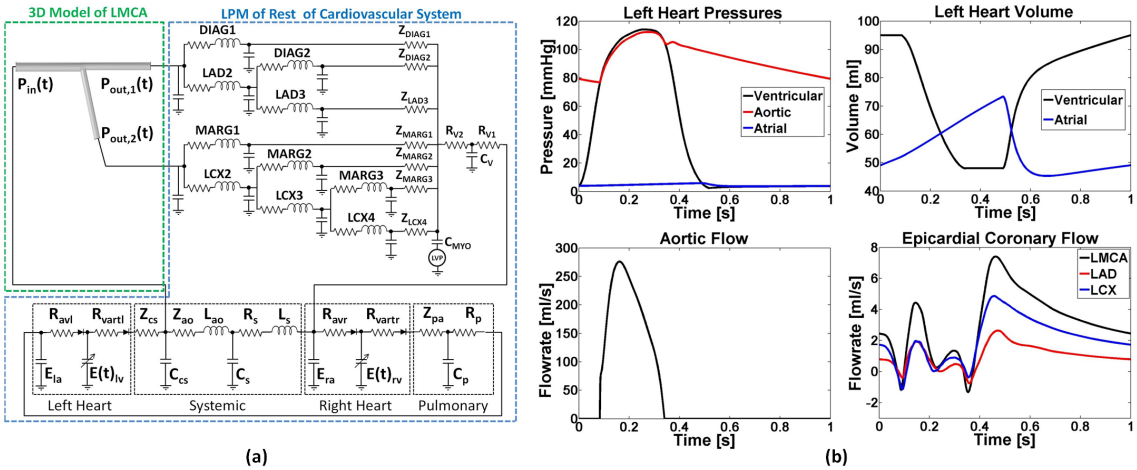

Figure 1: (a) MS model of LMCA bifurcation (b) pressure and flow results from the full LPM corresponding to (a)

RESULTS: The developed MS model is displayed in Figure 1a. The left and right hearts are modelled using time-varying elastances for the ventricles, constant elastances for the atria and diodes and linear resistances for the valves. In the coronary circulation each epicardial artery is represented by a 3-element Windkessel model and the terminal arteries are coupled to a resistance representing the microcirculation. The compression of the ventricles is taken into account by a pressure generator with the same phase and magnitude as the left ventricle. An in-house C++ code was written to simulate the full LPM equivalent to the presented MS model. The calculated pressure and flows display physiological behavior over the cardiac cycle (Figure 1b). The C++ code will be used directly in Fluent through a user-defined function in order to implement the MS algorithm for the synthetic bifurcation lesion geometries. Examples of the generated geometries of the diseased bifurcation are provided in Figure 2. The MS model will provide accurate flow and pressure fields within the 3D geometries.

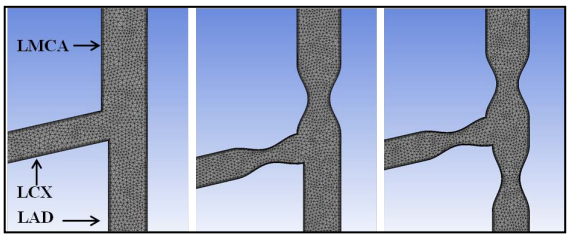

Figure 2: 3D synthetic lesion configurations (50% area reduction) at the LMCA bifurcation.

CONCLUSION: With the MS framework, both local and global haemodynamic interactions between the lesions could be elucidated (as a function of lesion topology and stenosis severity) which allows for the functional characterization of the various configurations and the distinction between globally versus locally critical configurations. Consequently, the presented model is expected to provide a functional perspective on bifurcation lesion dynamics which is essential in their diagnosis and treatment.

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