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AN IMPROVED CORONARY MODEL FOR ONE-DIMENSIONAL PRESSURE-FLOW ANALYSIS

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NONINVASIVE ASSESSMENT OF CORONARY ARTERY DISEASE

One-dimensional modelling of the cardiovascular system has been carried out extensively in recent years, for gaining a better understanding of pressure-flow propagation. Predictions from such models exhibit many of the features of the systemic and coronary arteries [1].

Estimation of fractional flow reserve (FFR) from computed tomography coronary angiograms (CTCA) can be a useful guide in clinical decision-making for revascularization of specific lesions. FFR appears as one of the most accurate index to assess the functional severity of coronary stenoses [2]. If resistance is minimal and constant, then pressure can be used as a surrogate of flow during maximal hyperæmia (Poiseuille's Law) [3]. Estimation of FFR from CTCA data can provide a noninvasive method for identifying ischæmia-causing stenosis [4]. The diagnostic performance of this new method relies on the accurate calculation of coronary flow and pressure from acquired CTCA scans, using computational fluid dynamics (CFD), and requires the construction of a realistic physiological model.

CORONARY MODEL

The average coronary arterial network is derived from anatomical measurements [5, 6]. While all of the major arteries are incorporated, in terms of function, only a small number of side branches are included. The average model is then used as a basis for one individual's coronary anatomy, and adapted using measurements obtained from CTCA. The

one-dimensional coronary model is used in a closed loop system and coupled with zero-dimensional components, representing the heart, the microcirculation, auto-regulatory mechanisms and systemic effects.

RESULTS

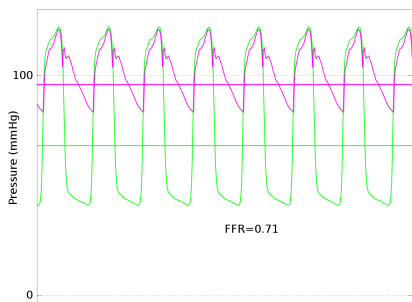


Figure 1: Phasic and mean aortic and distal coronary pressures for a 90% stenosis.

Preliminary results are presented using the baseline model, for stenoses of varying degree of severity in the left circumflex artery. Although flow and resistive losses depend on geometric factors that cannot be modelled using a 1D approach, we include within a stenosed segment semi-empirical equations to determine the pressure drop. FFR is independent of changes in heart rate and systemic blood pressure and is not altered by conditions known to increase the base-line myocardial flow. The numerical simulations presented do not currently include a model of hyperemic blood flow, to account for the effect of

adenosine on reducing the peripheral resistance of the microcirculation downstream. Future work will focus towards the validation of a comprehensive 1D model of the coronary tree, with the inclusion of patient-specific data.

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