

A MODEL OF THE EXERCISE RESPONSE OF CORONARY BLOOD FLOW, WITH AN APPLICATION TO THE CORONARY STEAL PHENOMENON

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Introduction. An array of complex, interacting, and sometimes competing mechanisms contribute to the control of coronary blood flow [1, 2]. These act by adjusting the resistance to flow in coronary vessels, and include feedforward neural control of coronary microvascular tone, and local feedback control, which matches oxygen supply to myocardial oxygen demand. Failures in these control systems can result in myocardial ischemia.

Due to the spatially-local nature of the dominant feedback system, the coronary flow response to exercise is, in essence, spatially greedy. This means that maintaining perfusion pressure, and thus blood supply, in a given region of the myocardium may be impossible due to vasodilation elsewhere in the coronary tree.

Specific Motivation. The example which interests us is *coronary steal* in the presence of a coronary stenosis [2], a phenomenon characterised as follows. Under resting conditions, all regions of the myocardium are sufficiently perfused, but upon a stimulus triggering vasodilation throughout the coronary tree, such as exercise or pharmacological vasodilator administration, the region downstream of the coronary stenosis experiences ischemia. The reason is that at rest, the coronary microvasculature downstream of the stenosis is highly vasodilated, thus compensating for the heightened contribution of the stenosis to the serial resistance. This state is functional at rest, but if, as in exercise, the resistance decreases in the remainder of the coronary tree, an inability of the stenosed branch to further reduce its resistance may result in it receiving insufficient blood flow.

Modelling. Understanding coronary steal is important for assessing the functional significance of coronary stenoses, and multiscale blood flow modelling provides an ideal tool for its study. Our model uses a multidomain method, in which we represent the heart by a dynamic, electrical circuit analogue lumped parameter network (LPN), the aorta and large coronary arteries are included as a three-dimensional domain, derived from human data, and where the incompressible Navier-Stokes equations are used to characterise the

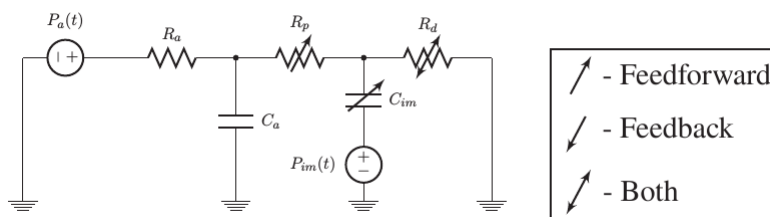


Figure 1: Coronary LPN model, with feedforward and feedback control targets. Respectively, P_a and P_{im} are the 3D domain outlet and intramyocardial pressures; R_a , R_p and R_d are the large epicardial, small conduit and microvascular resistances; C_a and C_{im} are the epicardial and microvascular compliances [3].

blood flow. Lastly, the coronary microvasculature within each perfusion territory is represented by a coronary LPN; see Figure 1. The LPNs provide physiological boundary conditions for the 3D domain. Resistances and compliances within the LPN for each perfusion territory are dynamically adjusted by ODEs which model coronary flow control in response to exercise [3].

Experiments. We use the model to investigate coronary steal in two cases. The first is that of exercise, in which the cardiac output increases, along with central arterial pressure. Thus, the myocardial oxygen demand increases, and the control ODEs adjust the coronary resistances in an attempt to compensate. In the second case, we investigate coronary steal due to vasodilatory drugs such as dipyridamole; here, the microvasculature vasodilates, without any significant change in myocardial oxygen demand or central arterial pressure.

Results. Results are in preparation; we expect to show that our model can reproduce the coronary steal phenomenon, providing a tool for its investigation with, for example, a range of patient coronary geometries. It will be ready for integration into models of the wider cardiovascular system, providing the capability for general cardiovascular haemodynamic simulation with (patho)physiologically realistic coronary flow control.

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