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Uncertainty of 1-D Fluid Models in Patients with Pulmonary Hypertension

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Pulmonary Arterial Hypertension (PAH) is defined as abnormally high mean blood pressure (> 25mmHg) in the proximal pulmonary artery. This is coupled with remodeling of arterial walls, involving changes in arterial wall cellular composition, and the distal vascular network, such as microvascular rarefaction. A lumped effect of vascular remodeling can be characterized as an increase in arterial stiffness and the total pulmonary vascular resistance (>3 HRU). This leads to changes in the mean and pulsatile components of the arterial blood pressure, which in turn determine the right ventricular afterload.

Our current work is based on a 1-D fluid dynamical network model that uses either Windkessel or structure tree models to represent the vascular beds. We also believe that arterial viscoelastic behavior can be accurately identified, though currently we have assumed a linear elastic model. The model is capable of simulating the physiological pressure and flow, which can be validated using real patient data, available from our clinical collaborators at Golden Jubilee National Hospital, Glasgow. However, the clinical data are subject to inaccuracies in measurements due to invasive and non-invasive techniques, e.g. catheterization, MRI, and CT scans. This leads to uncertainties in pressure and flow recordings, and the geometric measurements of large vessels. Since both construction of the model and its validation are based on real data, the measurement errors which propagate throughout the model may lead to an augmented effect on the model output. This makes the process of validation a challenging task, which can only be dealt by quantifying the uncertainty in model simulations in a systematic way involving methods of error propagation. Thus, the focus of this work is prediction as well as uncertainty quantification of physiological parameters that have strong influence on blood pressure, flow, and vessel cross-sectional area in the pulmonary vasculature. This will be done by extending common physiological and mathematical relationships, such as Poiseuille's, Murray's, and Stoke's law, to obtain nominal parameter values corresponding to error bounds around measurements of fundamental quantities, such as vascular dimensions and pulse pressure. Since these mathematical relationships explicitly depend on these fundamental quantities, their values can become highly variable if appropriate bounds are not set on measurement errors. This approach would allow us to determine the sensitivity of simulated blood pressure and flow in the main pulmonary artery to the inaccuracies in the geometric and hemodynamic data. Finally, this will also increase our understanding of the processes involved in cardiovascular modeling when there is a relatively high error propagation.