Fluid-structure interaction simulation of the brachial artery undergoing flow-mediated dilation

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Flow-mediated dilation (FMD) permits a non-invasive clinical assessment of endothelial dysfunction, a key indication of early atherosclerosis and cardiovascular diseases. This has significant implications with paediatric patients. FMD necessitates the measurement of brachial artery dilation from transient hyperaemia following a period of temporary ischemic occlusion. In addition to arterial diameter changes, the wall shear stress, blood pressure, and wall stiffness vary transiently in FMD, making it a complex fluid-structure interaction (FSI) problem. This work seeks to model the haemodynamic mechanisms associated with FMD utilising the open-source OpenFOAM-extend library[1]. Prior studies have demonstrated the suitability of this library for cardiovascular simulations[2]. Two FSI solvers, based on strong and weak coupling, were implemented for comparison. Both solvers utilise a partitioned approach, where the fluid and structure are solved separately and the information in each domain is exchanged at the FSI interface for each timestep. This is achieved using a dynamic mesh solver based on a discretisation of Laplace's equation. The fluid flow solution is based on the finite volume method (FVM) and the displacement of the solid domain is solved by a Lagrangian FVM solver. The artery wall was modelled as a straight tube with physiological values for the internal diameter, density, wall thickness, Young's modulus, and Poisson's ratio[3]. A Newtonian incompressible fluid was assumed with physiological density and viscosity[4]. The inlet velocity for the fluid domain is specified from an in-vivo hyperaemic condition[5]. The simulation results demonstrate an important variation in the diameter of the arterial vessel during FMD, while haemodynamic wall shear stress and pressure values are also ascertained. These preliminary results are useful for comparing the implementation of strong and weak FSI solvers and for correlating arterial wall displacement with the prescribed in-vivo inlet velocity. Future work will focus on FMD in idealised and patient-specific bifurcation models where ischemic occlusion will be prescribed for the distal branching arteries.

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