Pulmonary hypertension assessed using a fluid mechanics model

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This study uses one-dimensional (1D) fluid dynamics models to predict flow, pressure, shear stress, and wave propagation in pulmonary vasculature networks to gain a better understanding of the progression of pulmonary hypertension (PH) [1,2]. Since the onset of PH is at the capillary level, migrating to small veins, arteries, and eventually the large vessels, our model includes predictions at all three scales. The large arteries and veins are presented by a directed graph extracted from computed tomography images, whereas the small vessels are represented by structured tress with parameters informed by data [3]. The capillary network, modeled using sheets, is coupled to the network of arterioles and venules in a ladderlike structure. In the large vessels, we solve the 1D Navier Stokes equations, while in the network of small arteries and capillaries, we set up and solve linearized equations, which are linked to the large vessels via outflow boundary conditions. The model is calibrated to healthy controls and patients with a progressive increase in disease severity impacting vessel stiffness and vessel dimensions. To differentiate healthy and diseased networks, we study the hemodynamic impacts of changing vessel stiffness, vessel radius, and resistance provided by vessels in the microcirculation. The latter is achieved by modifying scaling factors and vessel stiffness. While the model is in 1D, the network has three dimensions enabling us to predict perfusion in the lung and using the Kullback-Leibler divergence we get a measure of disease severity by comparing flow maps in unobstructed vessels with those in disease network. In summary, this study addresses how the addition of multiple scales improve hemodynamic predictions in disease.

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