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Coupled 0D-1D CFD Modeling of Right Heart and Pulmonary Artery Morphometry Tree MELODY DONG, Bioengineering - Stanford Univ, WEIGUANG YANG, JEFFREY A. FEINSTEIN, Pediatric Cardiology - Stanford Univ, ALISON MARSDEN, Bioengineering, Pediatric Cardiology - Stanford Univ
— Pulmonary arterial hypertension (PAH) is characterized by elevated pulmonary artery (PA) pressure and remodeling of the distal PAs resulting in right ventricular (RV) dysfunction and failure. It is hypothesized that patients with untreated ventricular septal defects (VSD) may develop PAH due to elevated flows and pressures in the PAs. Wall shear stress (WSS), due to elevated flows, and circumferential stress, due to elevated pressures, are known to play a role in vascular mechanobiology. Thus, simulating VSD hemodynamics and wall mechanics may facilitate our understanding of mechanical stimuli leading to PAH initiation and progression. Although 3D CFD models can capture detailed hemodynamics in the proximal PAs, they cannot easily model hemodynamics and wave propagation in the distal PAs, where remodeling occurs. To improve current PA models, we will present a new method that couples distal PA hemodynamics with RV function. Our model couples a 0D lumped parameter model of the RV to a 1D model of the PA tree, based on human PA morphometry data, to characterize RV performance and WSS changes in the PA tree. We will compare a VSD 0D-1D model and a 0D-3D model coupled to a mathematical morphometry tree model to quantify WSS in the entire PA vascular tree.

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