Single ventricle (SV) anomalies account for one-fourth of all cases of congenital heart disease. The existing three-hybrid staged surgical approach serving as a palliative treatment for this anomaly entails multiple complications and achieves a survival rate of only 50%. To reduce trauma associated with the second stage of the hybrid procedure, the hybrid comprehensive stage 2 (HCS2) operation was introduced in 2014 at Arnold Palmer Hospital in Orlando as a novel palliation alternative for a select subset of SV patients with adequate antegrade aortic flow. It avoids dissection of the pulmonary arteries by introducing a stented intrapulmonary baffle and avoids reconstruction of the aortic arch by maintaining patency of the ductus arteriosus. This dissertation aims to provide better insight on the postoperative hemodynamics of HCS2 patients. A multi-scale Computational Fluid Dynamics (CFD) analysis of a synthetic, patient-derived HCS2 geometry based on unsteady laminar flow conditions and a non-Newtonian blood model is utilized to quantify the resultant hemodynamics. The 3-D CFD model is coupled to a 0-D lumped parameter model of the peripheral circulation that supplies the boundary conditions necessary to run the CFD analyses of the HCS2.

Based on clinical parameters suggesting the baffle related narrowing to be at minimum 10mm and the pressure gradient not surpassing 20mmHg, hemodynamic analysis reveals that for even a 7.23mm narrowing the average pressure drop across the baffle is 0.53mmHg. A peak pressure drop of 2.96mmHg was computed over the investigated range of clearances over the pulmonary baffle. Vortex shedding presents no concerns as the distance between the baffle and the aortic arch is much smaller compared to the length required for full vortices to form. Uneven contour distribution of the wall shear stress was observed due to the bend presented by the baffle that strongly affects the velocity profile in the lumen across the pulmonary trunk and into the ductus arteriosus. Moreover, an oxygen transport model was derived, and the results showed a consistency with published data of Glenn patients. Particle residence time was also reported to identify any blood recirculation or flow stagnation that may lead to platelet activation leading to clot formation rate.

The study provides a range of main pulmonary artery geometries that, following multi-scale CFD analysis, present no concerns regarding excessive pressure gradients or vortex formation. Moreover, the model identifies locations of potentially problematic hemodynamics that could be mitigated by shape optimization of the reconstruction.