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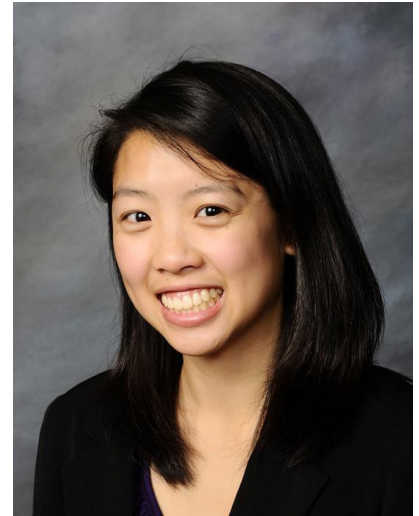
Patient-Specific Modeling of Tetralogy of Fallot

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Tetralogy of Fallot (TOF) is one of the more complicated types of congenital heart defects. TOF affects 3-5% of all infants born with congenital heart defects and is a leading cause of cyanosis, which occurs when insufficient amounts of oxygen are transported to the rest of body. Four major defects contribute to cyanosis in patients with TOF. The first defect is an overriding aorta, which is a shift of the base of the aorta towards the pulmonary artery. The second defect is a ventricular septal defect, or a hole in the wall between the two ventricles. The third defect is right ventricular hypertrophy which is caused by overworking the ventricles in order to pump sufficient blood to the lungs. The final and fourth defect is pulmonary valve stenosis which is a narrowing of the opening of the valve that connects the heart

to arteries going to the lungs. The primary treatment of TOF is the repair of the pulmonary valve stenosis. This repair involves either valvuloplasty (where a catheter placed into the heart opens up the blocked area and partially or completely destroys the valve) or surgical resection of the valve (where open-heart surgery is used to repair or replace the valve). These repairs typically have high initial-success rates, but can lead to long-term complications such as pulmonary regurgitation where blood flows backward into the ventricle instead of forward through to the lungs. There is a need to more precisely evaluate the necessity for reparative surgery based on the severity of stenosis. This study investigates the hemodynamic effects of pulmonary stenosis in order to further our understanding of the disease and its effects in TOF.



A 3D patient-specific computer model of the post-treatment pulmonary arteries was constructed from MRI data obtained from a patient with a history of pulmonic stenosis, using the custom software package SimVascular (simvascular.org). Four varying severity levels of pulmonary valve stenosis were virtually recreated by reducing the area of the main pulmonary artery inlet at the valve. To simplify the complexity of blood flow in the arteries, steady-flow and rigid-wall assumptions were made. Incompressible Navier-Stokes computational fluid dynamic simulations were then performed based on standard

cardiac models. Pressure and flow boundary conditions were prescribed at the inlet and resistance boundary conditions at the outlet based on clinical measurements in order to simulate the necessary pressures required for normal body function, the patient's blood flow rate, and the resistance to flow from smaller arterioles downstream of the valve.

The simulations showed increased pressure differences between the inlet and outlet. This demonstrated a physiological need for higher pressure in the ventricles to pump blood to the rest of the arteries during stenosis. A higher velocity jet at the main pulmonary artery and greater wall shear stress at the site of stenosis were also observed. Because velocity from the main pulmonary artery can be quantified with non-invasive imaging techniques, an index relating the velocity jet to severity of stenosis and

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treatment of the defect could help clinicians better diagnose and provide treatment plans for patients with TOF. Furthermore, the relation to wall shear stress could indicate localized areas in

the artery that are being affected the most by the defect. These results provide a preliminary quantitative

assessment of the downstream effects of pulmonary stenosis. Since current treatments of TOF and the resulting effects are not well understood, a quantitative understanding of the hemodynamics of pulmonary stenosis would provide less

ambiguous treatment options for patients. More in depth simulations could also reveal long-term effects of different treatment options of TOF and would further clarify our understanding of pulmonary stenosis with regards to timing of interventions in patients with TOF.



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