

Abstract Submitted
for the DFD14 Meeting of
The American Physical Society

Quantification of avian embryonic cardiac outflow hemodynamics through 3D-0D coupling STEPHANIE LINDSEY, Cornell University; INRIA Paris-Rocquencourt, IRENE VIGNON-CLEMENTEL, INRIA Paris-Rocquencourt, JONATHAN BUTCHER, Cornell University — Outflow malformations account for over 20% of CHDs in the US. While the etiology of these malformations is poorly understood, most can be traced back to perturbations in the patterning of the pharyngeal arch arteries (PAAs), the precursors to the great vessels. Here, we examine the effects of normal and aberrant PAA flow, through the use of two computational models. A 0D electric analog model allows for rapid computation and global tuning of the embryo's vasculature relative to the arches. A second 3D-0D model replaces the electric analog representation of the arches with a 3D reconstruction, thereby leading to more extensive pressure and flow characterization. We obtain 3D arch artery reconstructions from nano-CT stacks and couple them to 0D outlets. In contrast to standard boundary conditions, such coupling maintains the physiologically desired cranial-caudal flow split in control embryos and predicts how this will change with vessel occlusion. We use flow inputs from Doppler velocity tracings to compute 0D and 3D-0D pulsatile hemodynamic simulations in HH18 (day 3), HH24 (day 4), and HH26 (day 5) geometries. We then calculate flow distributions and wall shear stress maps for control embryos. From here, we modify HH18 geometries to simulate varying levels of PAA occlusion. Pulsatile simulations are run in each geometry and results compared to that of controls. Results serve as a basis for examining flow-mediated growth and adaptation in cardiac outflow morphogenesis.

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Date submitted: 01 Aug 2014

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