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Congenital heart defects

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Previous studies suggest that altered hemodynamics early in development can lead to congenital heart defects. Unfortunately, due to a lack of technology to measure and perturb hemodynamic parameters during cardiac looping in a live preparation, many questions remain unanswered. We have utilized optical coherence tomography (OCT) to measure and optical pacing (OP) to perturb hemodynamics during cardiac looping in a live quail preparation. OP employs infrared pulsed light to noninvasively capture the heart rate to the pulse frequency of the laser without the use of exogenous agents. We hypothesize that altering regurgitant flow early in development leads to congenital heart defects. We increased regurgitant flow with OP in the heart of the embryonic quail at day 2 of development just prior to the formation of cardiac cushions (valve precursors). OCT measured pulsed Doppler traces at the inflow tract of the looping heart and gave a quantitative measurement of regurgitation. Embryos were kept alive until day 3 (mid looping) or day 8 (4 chamber heart) after pacing to look at resultant morphologic changes to the atrioventricular cushions and valves with OCT. Paced embryos had smaller cushions and endothelial mesenchymal transition at day three and defective valves at day eight. The cushion volume decreases scaled with the degree of induced regurgitant flow. These results suggest that abnormal regurgitant flow at the atrioventricular cushions can lead to congenital defects. Understanding the role of hemodynamics in development is an important step towards determining the mechanisms of CHDs and ultimately developing earlier treatment strategies.

Biography

Michael Jenkins is an Assistant Professor at Case Western Reserve University with primary appointment pediatrics and a secondary appointment in biomedical engineering. His research interest includes the development of optical pacing and optical imaging technologies for investigating cardiac development and diseases.

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