



Pulse Wave 1D Algorithms: Computational Models of Fear and Aerobic Exercise to Investigate Large Artery Production of Nitric Oxide

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We simulated blood flow in the major vasculature of the head, trunk, and limbs to investigate the hemodynamic effects of acute cardiovascular stress (i.e., fear and aerobic exercise). Vessel dimensions were based on modern high-resolution medical imaging data for humans with a focus on active young adults. Utilizing a network of reduced-order transmission line elements (0.5 cm or smaller arterial segments), together with peripheral three-element Windkessel models, we solved for instantaneous changes in flow rates and pressures in response to stress-adjusted cardiac output, peripheral resistance, and arterial compliance. Wave propagation was initiated by applying an external force mimicking the myocardial contraction of the left ventricle. To quantify the effects of fear and exercise on arterial wall shear stress (WSS)—a key physiological regulator of endothelial nitric oxide synthase, we developed a sequential mathematical model in which local one-dimensional pressure gradients inform the Womersley flow equations and subsequent reconstruction of transient velocity profiles, thus providing WSS changes over the cardiac cycle. Compared to the resting state, time-averaged WSS increased by 50% in the brachial arteries (BAs) and by 60% in the femoral arteries (FAs) in response to fear. Likewise, WSS increased by 110% in the BAs and by 410% in the FAs during moderate aerobic exercise. Furthermore, we demonstrate the breakdown of the Poiseuille flow assumption in conduit arteries (commonly used in clinical applications) via quantification of the oscillatory shear index. Our updated anatomical database is publicly available (DOI: 10.5281/zenodo.4630326) and allows for location-specific assessment of hemodynamics under active physiologic conditions. These computationally efficient models may also be coupled with the mass transport equations to predict the propagation of vasoactive agents through the vascular system, which is the focus of our current work.



ABOUT the SPEAKER

Joseph Muskat is a 5th year PhD candidate at the Weldon School of Biomedical Engineering at Purdue University co-advised by Drs. Craig Goergen and Vitaliy Rayz. He obtained his B.S. in biomedical engineering from Purdue University, where he developed mathematical models for simulating low-frequency thrombolysis under Dr. Charles Babbs and established imaging-derived topographic prototyping of x-ray phantoms with Stryker Corporation. His current work focuses on reduced-order computational modeling of cardiovascular stress to evaluate potential evolutionary benefits of the circle of Willis, the hemodynamic effects of aerobic exercise, and endothelial-derived nitric oxide.

Monday, March 21 at noon
1003 Engineering Centers (Tong Auditorium)