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Simulating pulse-wave hemodynamics under the effects of vasoactivity

One-dimensional, pulse-wave propagation models are able to replicate hemodynamic waveforms that are representative of measured data. While these models are a potential tool in the era of digital twins, few models have considered the role of smooth muscle vasoactivity and its effects on blood pressure and flow. This is especially important for understanding cerebrovascular function, especially in diseases like dementia and Alzheimer's, where cerebral vasoactivity is known to be a cause and consequence of altered mechanical stimuli. Thus, there is a need for new computational models that explicitly account for vascular tone during hemodynamic simulation.

Here, we implement a relatively simplistic exponential model of the proximal vasculature pressure-area relationship, given by

$$p(x,t) = p_0\left[e^{\beta_{ECM}\left(\sqrt{\frac{A}{A_0}}-1\right)}+\frac{k}{k_{ref}}e^{\beta_{SMC}\left(\frac{1}{1-k}\sqrt{\frac{A}{A_0}}-1\right)}\right]$$

which incorporates extracellular matrix stiffness, β_{ECM}, vascular smooth muscle cell stiffness, β_{SMC}, the degree of vasomotor tone k in comparison to some reference tone k_{ref}, and the reference pressure, p_0. The strain is measured by the relationship between current and reference area, A and A_0, respectively. We couple this vasoactive large vessel model to the structured tree boundary condition, which represents the microvascular beds. To differentiate between proximal and small vessel vasoconstriction, we also introduce a vasodilation factor in the structured tree that controls microvascular radii. We analyze the model using global sensitivity analysis, and provide insight into the distinct contributions of large and small vessel vasoactivity in an idealized systemic arterial network. Our results show that microvascular vasoconstriction is more impactful that proximal vessel vasotone, but that stress-strain behavior in the large vessels can be modulated divergently depending on the relative magnitudes of ECM and smooth muscle stiffness. This study lays the foundation for future studies investigating the effects of vasoactivity on hemodynamic outcomes.