An Integrated Model of the Cardiovascular and Central Nervous Systems for Analysis of Microgravity Induced Fluid Redistribution

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Objective and Motivation

A recognized side effect of prolonged microgravity (mg) exposure is visual impairment and intracranial pressure (VIP) syndrome [1]. Though there is limited medical understanding of this phenomenon, it is hypothesized that cephalic shift of the cerebrospinal fluid (CSF) and blood in mg may be a contributor. Computational models can be used to provide insight into the origins of VIP (1, 2). In order to further investigate this phenomenon, NASA’s Digital Astronaut Project (DAP), in collaboration with some of the world’s leading experts in ocular biomechanics, is developing an integrated fluid physics-based computational model of the human body. The model is divided into the eye, the central nervous system (CNS), and the cardiovascular system (CVS). This poster summarizes our current progress on the models can be used to provide insight into the origins of VIIP [1, 2]. In order to reflect the complex interaction of the cerebrospinal fluid (CSF) and blood in mg may be a contributor to the pathogenesis of this condition. The general structure of the model:

- Includes blood, cerebrospinal fluid (CSF), tissue, interstitial fluid, pulmonary circulation and organs
- Captures direct flow between compartments, as well as transfer of fluid between capillaries and tissue by filtration
- Includes compliant interactions between adjacent compartments
- Incorporates functions to allow the inclusion of the lymphatic system and the sympathetic nervous system (SNS) functions
- Includes a series of differential equations to describe the pressure dynamics of the system in accordance with the laws of conservation:

\[ \text{flow in} – \text{flow out} = \text{rate of volume change} \]

In addition, the following assumptions are applied:

- All fluids are assumed to be incompressible and isothermal
- Pressure-driven flows are laminar and governed by the hydrodynamic equation:

\[ Q_i = \frac{P_i - P_j}{R_{ij}} \]

- Fluid filtration between the capillary and the interstitial spaces is governed by the Starling-Landis equation:

\[ \text{filtration} = K_{ff}(P_i - P_j) - \left(\alpha_i(n_i - n_j)\right) \]

- Compliance between compartments depends on the change in pressure differences between compartments in the form of:

\[ \frac{dP_{ij}}{dt} = C_{ij}(P_{ij} - P_{ij}) \]

- Revokes the Kellie-Monroe Doctrine in order to take into account the influence of extracranial physiology on ICP dynamics.
- Hydrostatic pressure variation can be approximated through an ad hoc variation of resistances in the upper and lower portions of the body.

Verification and Validation

The initial verification test challenged the model in a time-dependent mode by specifying steady-state values that were offset by 10% from Lakin et al.’s [3] mean values to perturb the system. Since the system returned to the baseline mean values, our model successfully conserved all conservation parameters.

In addition, we performed two validation cases as outlined by Lakin et al. [3] for the situations of:

1. Postural change
2. Spaceflight

In order to leverage this model as a foundation for an integrated systems model to simulate the CNS and CVS for VIP research, we will:

- Formally incorporate hydrostatic pressure variation into the matrix equation
- Modify system parameters such as tissue and flow properties, flows and pressures to reflect the most current VIP research and to define a reasonable physiological envelope that encompasses the human body
- Analyse the model with sensitivity tests to identify the most significant parameters
- Test the model against and train the model with independent studies in the CNS and CVS
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Results and Discussions

The model successfully simulated a pulsatile cardiac cycle that was comparable to [3]. As can be seen in Figure 2, there are minor discrepancies in the period and amplitude of our prediction of heart outflow and Lakin et al.’s function, as digitized from the paper. However, when compared with literature values [4], our simulated systolic and diastolic pressures in the central and intracranial arteries were within physiological limits of 120/80 mmHg and 100/65, respectively.

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References