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ACUTE CARDIOVASCULAR RESPONSE TO GRAVITY CHANGES: A MULTISCALE MATHEMATICAL MODEL FOR MICROGRAVITY AND HYPERGRAVITY APPLICATIONS

Abstract

In recent years, several studies revealed that long-term human spaceflights induce many cardiovascular alterations (from blood volume decrease to cardiac atrophy), which seriously threaten the health of the astronauts. Among all the hazards of the space environment, continuous exposure to gravity changes (from micro- to hyper-gravity) plays a crucial role in the cardiovascular response to such a hostile environment. However, hemodynamic characterization is still incomplete, and no definitive data are available to date. In this context, numerical modeling provides a useful tool for evaluating cardiovascular and cerebral adaptation to spaceflight environments.

To investigate the acute cardiovascular response to gravity changes (from 0g to 2.5g), we propose a multiscale 0D-1D cardiovascular-cerebral-ocular model combining a closed loop central-systemic cardiovascular 0D-1D model with two 0D cerebrovascular and ocular models. The 0D-1D cardiovascular model includes the 1D description of the arterial tree and the 0D representation of the peripheral microcirculation, the venous return, and the cardio-pulmonary circulation, and accounts for the intramyocardial and intrathoracic pressures, as well as the venous collapse. The 0D cerebrovascular model describes the circle of Willis, distal and capillary-venous circulations, the cerebrospinal fluid and the intracranial pressure (ICP). Finally, the 0D ocular model defines the intraocular pressure (IOP) and the ocular globe volume. The overall model is equipped with short-term regulation mechanisms (baroreflex, cardiopulmonary, cerebral and CO_2 reactivity), and explicitly accounts for gravity and posture changes.

Our analysis reveals that: (i) as gravity increases from 0g to 2.5g a worsening of several cardiac and mechano-energetic parameters is observed: heart rate (HR) increases from approximately 70 bpm to 120 bpm, while stroke volume (SV), ejection fraction (EF) and cardiac output (CO) decrease respectively by about 65%, 25% and 40%. Despite the increased HR, diminished SV results in reduced cardiac output. Furthermore, these parameters show a non-linear behaviour with g; (ii) in microgravity (0g-1g), ICP and IOP rise due to a head-ward fluid shift caused by the absence of gravitational forces, while in hypergravity (1g-2.5g) they exhibit a significant reduction. Conversely, cerebral blood flow (CBF) remains relatively stable within the range of 0g-1g, but decreases rapidly with higher g values, suggesting an inability of cerebral autoregulation to maintain an adequate CBF.

In conclusion, in the short-term, the 0g condition is less demanding, yet it exposes distal cerebral regions to heightened pulsatility, potentially resulting in hypertensive and hypoperfusive events. Conversely, hypergravity conditions induce strong orthostatic stress, decreasing cerebral perfusion and reducing exercise tolerance.