0D-1D multiscale cardiovascular modelling for spaceflight applications

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The human cardiovascular system is optimized to work under Earth's gravitational load (1g). Therefore, alterations in gravity elicit a range of hemodynamic changes primarily driven and ruled by fluid dynamics processes, starting from hydrostatic pressure variations that lead to blood redistribution throughout the cardiovascular system. In microgravity (0g), these changes are responsible for cardiovascular deconditioning, venous thromboembolism, and increased intracranial pressure (ICP) [1]. Thus, the development of effective in-flight countermeasures is essential for astronaut health and safety. Among these, lower body negative pressure (LBNP) is one of the most extensively studied, due to its ability to mimic the 1g environment by pooling blood towards the legs [2].

To date, the hemodynamic characterization is restricted to a small set of measurable variables due to the limited availability of experimental data and the constraints of ground-based analogs. In this context, the computational approach represents a valuable tool. In particular, multiscale modeling enables the investigation of cardiovascular hemodynamics at different levels of detail while preserving the systemic perspective, which is typically lacking in 3D models. In this work, we refined our validated model [3], capable of reproducing acute 0g-induced effects, to make it suitable for assessing the effectiveness of LBNP as an in-flight countermeasure. Our model combines a 1D description of the main systemic and coronary arteries with a 0D representation of the peripheral, venous, cardiopulmonary, and cerebro-ocular circulations. The model includes short-term regulation mechanisms, venous collapse, and accounts for gravity and postural transitions.

The model reproduces acute 0g-induced effects, including central hypervolemia, decreased heart rate (HR), and increased ICP. LBNP induces a hypovolemic stimulus that counteracts central congestion associated with 0g. In particular, a -40 mmHg LBNP stimulus produces an increase in HR comparable to that observed during standing at 1g (78 bpm) and leads to a slight reduction in ICP. However, the different mechanisms driving fluid redistribution – gravity in standing posture and external negative pressure in LBNP – result in distinct distributions of pressures and volumes throughout the cardiovascular system.

In conclusion, the proposed fluid dynamics-based model proves to be a powerful tool for investigating microgravity-induced cardiovascular alterations and evaluating efficacy and potential improvements of current spaceflight countermeasures.

References

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