Arterial adaptations in microgravity contribute to orthostatic tolerance

The removal of gravitational stress with spaceflight induces a number of adaptations within the cardiovascular system. However, such microgravity-induced adaptations are thought to be maladaptive on return to Earth. Understanding the interactions between the cardiovascular system and gravitational stress and the resulting problem of orthostatic intolerance among astronauts is the focus of the study of Tuday et al. (6) in this issue of the Journal of Applied Physiology. It has previously been shown that postflight orthostatic intolerance is related to a diminished capacity of the sympathetic nervous system to elevate peripheral vascular resistance, which could occur through altered function of the afferent limb of the baroreceptors, central integration, sympathetic efferents, or end-organ responsiveness to sympathetic stimulation (1, 7). The present study of Tuday et al. (6) further indicates that there is a stiffening of the aorta (i.e., decreased aortic compliance) in astronauts who can tolerate orthostatic stress on return to Earth, whereas astronauts who are orthostatically intolerant had no change or a slight decrease in aortic stiffness. Such an increase in aortic stiffness would displace more blood volume to the venous side of the circulation and allow for greater support of cardiac output in the astronauts able to withstand the postflight orthostatic stress.

To further investigate this phenomenon of aortic stiffening induced by microgravity in humans, Tuday et al. (6) turned to a ground-based animal model of microgravity, the hindlimb-unweighted (HLU) rat. Hindlimb unweighting in the rat elicits many of the hemodynamic alterations that characterize the cardiovascular deconditioning that occurs in humans after exposure to microgravity, including hypovolemia (3), resting and exercising tachycardia (5), altered tissue perfusion (5, 9), and arterial hypotension during head-up tilt (4, 8). Using measures of pulse wave velocity to assess vascular stiffness, the authors (6) found a greater pulse wave velocity through the thoracic aorta of HLU rats, indicating a stiffening of the aorta in this group of animals. These in vivo studies of aortic stiffness were then confirmed with in vitro assessment of mechanical stiffness, where thoracic aortic stiffness was greater in HLU rats.

Collectively, the data from both humans and animals indicate that the mechanical stiffness of the thoracic aorta increases with actual and simulated microgravity. Interestingly, hindlimb unweighting did not alter the stiffness of the abdominal aorta. The increase in vascular stiffness of the thoracic aorta and the lack of change in stiffness of the abdominal aorta are likely related to the headward fluid shift that occurs during the head-down position in HLU rats. Such observations highlight the potential importance of fluid and pressure changes for inducing adaptations within the cardiovascular system. Thus increases in arterial stiffness associated with increasing arterial pressure in the thoracic aorta appear to promote venous return and beneficially serve to enhance orthostatic tolerance in astronauts and perhaps in HLU rats. However, other adaptations that take place in the arteria circulation with actual or simulated microgravity may serve to adversely affect orthostatic tolerance. These include a diminished ability to vasocostrict in peripheral arteries (2) and a loss of capacity to elevate peripheral vascular resistance (1, 5, 7, 9).

In summary, the study of Tuday et al. (6) is important for several reasons. First, it provides the initial observation that associates increases in aortic stiffness with orthostatic tolerance among astronauts. Second, the study highlights the complexity of vascular adaptations that occur with actual and simulated microgravity, some of which may be beneficial for withstanding orthostatic stress on return to Earth from space and others that may contribute to the problem of orthostatic intolerance among astronauts.

REFERENCES

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