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A THEORY FOR UNEXPLAINED VASODILATION WITH ELEVATED NORADRENALINE LEVELS IN SPACEFLIGHT.

Abstract

Resting cardiovascular measurements taken in space consistently show vasodilation coupled with a seemingly paradoxical increase in norepinephrine levels compared to resting measurements supine on Earth. The reason for this remains unclear. A common understanding of the adaptation to weightlessness is that a headward fluid shift increases thoracic blood volume and cardiac output. In this scenario, the increased cardiac output should lead to reflex vasodilation, which should lower norepinephrine levels. A baroreflex-induced lowering of heart rate should also occur to prevent an increase in blood pressure, analogous to what happens when going from the supine to the head-down tilt position on Earth. With head down tilt, the additional head-ward fluid shift compared to supine increases stroke volume and leads to decreased heart rate, reflex vasodilation, and reduced norepinephrine levels. This contrasts markedly with measurements from spaceflight where both heart rate and norepinephrine levels are increased rather than decreased.

A possible explanation for this is that the primary event leading to the cardiovascular responses in space is not the fluid shift. Instead, the resting hemodynamic differences between head-down tilt and spaceflight suggest the primary difference is weightlessness-induced vasodilation. The increased cardiac output, heart rate, and norepinephrine levels could be in response to vasodilation that occurs when an individual becomes weightless.

The reason for the vasodilation may be the loss of tissue weight and tissue compressive forces that occurs immediately upon entering weightlessness. Work with numerical modeling shows that the removal of tissue compressive forces helps explain why central venous and peripheral venous pressure drop in space compared to supine values on Earth. Without tissue weight compressing veins, the veins expand, reducing their internal pressure. This same effect may be occurring in the arterial system. The removal of tissue compressive forces may allow arterioles to expand and for capillary beds to open. The net effect would be to reduce total peripheral resistance immediately upon entering weightlessness. This reduction reduces blood pressure leading to baroreflex-mediated increases in sympathetic nerve activity which would increase norepinephrine levels. Heart rate and cardiac output would also increase. Conversely, experiments with Gx centrifugation increased total peripheral resistance, increased aortic pressure and decreased heart rate, inducing the opposite response to weightlessness.

Although the headward fluid shift is often cited as the primary event that initiates the adaptation to weightlessness, the loss of tissue weight and the forces produced by tissue weight may be more significant.