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THE IMPACT OF HYDROSTATIC PRESSURE ON MUSCLE OXYGENATION DURING LOW-LEVEL RHYTHMIC PLANTARFLEXION EXERCISE

Abstract

[Background] Physical exercise has proven to be effective in mitigating physiological de-conditioning during spaceflight and developing more time-efficient training methodologies for the crew on long-duration missions is important. Oxygenation is essential for muscular contraction, and muscle ischemia leads to inflammatory responses, muscle pain and injury, vascular remodeling, and angiogenesis. However, the impact of gravity (i.e. hydrostatic pressure) on muscle oxygenation during exercise has not been well studied. Therefore, the present study investigated the effect of different hydrostatic pressure on muscle oxygenation in the gastrocnemius muscle, one of the main anti-gravity muscles, during low-level plantarflexion exercise.

[Methods and results] Healthy participants (n=11, 2 females; mean age 273 years) performed a 7 minutes rhythmic (2-second contraction and 4-second relaxation) plantarflexion exercise at 10% of maximum voluntary contraction, with legs above the level of heart (LA position; negative hydrostatic pressure), straight at the level of heart (LS position; zero hydrostatic pressure), and below the level of heart (LB position; positive hydrostatic pressure). Gastrocnemius muscle tissue oxygen saturation (StO2) and blood flow in the superficial femoral artery were assessed by near-infrared spectroscopy and ultrasound during exercise. Exercise-induced hyperemia was defined as the percentage increase in blood flow from rest to post-exercise. In each leg position, a pneumatic cuff was inflated in the upper thigh at 220mmHg for 5 minutes to establish severe ischemic status. To assess micro- and macrovascular function in dependence on hydrostatic pressure, reactive hyperemia and flow-mediated dilatation of the superficial femoral artery after cuff occlusion were assessed in LA and LB positions. Although StO2 was similar at rest in the 3 leg positions (LA, 635% LS, 669% LB, 6810%; P=0.309), it was lower in the leg position with lower hydrostatic pressure (LA, 4215% LS, 619% LB, 674%; P<0.001). Especially, in the LA position, StO2 at the end of the exercise was similar to that in severe ischemia (3313%; P=0.074). Exercise-induced hyperemia was lower in the leg position with lower hydrostatic pressure (LA, 22381% LS, 22980% LB, 328134%; P=0.044). Reactive hyperemia area under the curve of blood flow was lower (480100ml/min vs. 559115ml/min; P=0.05) and flow-mediated dilatation of the superficial femoral artery tended to be smaller (3.72.8% vs. 5.02.1%; P=0.184) in the LA position than in the LB position.

[Conclusion] Lower hydrostatic pressure leads to reduced muscle oxygenation even with low-levels of short duration exercise. This may be related to low exercise-induced hyperemia and micro- and macrovascular responses.