INCREASED CEREBRAL BLOOD FLOW RELATED TO HIGH ALTITUDE EXPOSURE – MRI ASSESSMENT OF AIRCREW IN THE HYPOBARIC ENVIRONMENT

A AUGMENTÉ LE DÉBIT SANGUIN CÉRÉBRAL LIÉ À L'EXPOSITION DE HAUTE ALTITUDE – ÉVALUATION DE MRI DE L'ÉQUIPAGE DANS L'ENVIRONNEMENT HYPOBARE

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Introduction: Subcortical white matter (WM) injury and global decreased fractional anisotropy are associated with repetitive exposure to non-hypoxic hypobaric conditions. A single hypobaric hypoxic occupational exposure to 7,620 m (5.45 psi) induces magnetic resonance imaging (MRI) changes that reflect transient brain injury.

Methods: Subjects underwent 3T MRI brain examinations 24 h pre-exposure and 24 and 72 h post-exposure. 90 U.S. Air Force aircrew trainees undergoing occupational altitude chamber training and 65 age-matched control subjects not exposed to hypobaria were evaluated. MRI protocol included fluid-attenuated inversion recovery images, magnetization-prepared rapid gradient-echo sequences, and arterial spin labeling perfusion imaging. Statistical analyses were performed with a linear mixed model for within group comparisons and with a generalized additive model for intergroup comparisons.

Results: WM cerebral blood flow (CBF) at 24 h post-exposure increased by 6% (p=0.004) while gray matter (GM) CBF increased by 5% (p=0.006). At 72 h post-exposure, CBF remained significantly elevated (WM 6%, p=0.021; GM 6%, p=0.037). No significant change in CBF was observed in the control subjects (WM p = 0.154; GM p = 0.563). Exposure vs. control group comparison was statistically significant, WM p=0.002; GM p=0.054. There were no WM fluid- attenuated inversion recovery or gradient echo changes.

Discussion: Results demonstrate up-regulation of both GM and WM CBF 24 h post exposure to hypobaric training that persists at 72 h. This reflects an increased metabolic demand and suggests a transient cerebral injury has occurred, equated to a concussive-like injury. Repetitive hypobaric exposure prior to an adequate recovery period, a "double-hit" process, may characterize an underlying basis for previously reported subcortical WM injury.

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