Head-down tilt bed rest

It is well known that exposure to microgravity in astronauts leads to a plethora of physiological responses such as headward fluid shift, body unloading, and cardiovascular deconditioning. When astronauts return to Earth, some encounter problems related to orthostatic intolerance. Impaired cerebral autoregulation (CA), which could be compromised by the effects of microgravity, has been proposed as one of the mechanisms responsible for orthostatic intolerance. CA is a homeostatic mechanism that maintains cerebral blood flow for any variations in cerebral perfusion pressure by adapting the vascular tone and cerebral vessel diameter. The ground-based models of microgravity are useful tools for determining the gravitational impact of spaceflight on the human body. The headdown tilt bed rest (HDTBR), where the subject remains in the supine position at -6 degrees for periods ranging from few days to several weeks is the most commonly used ground-based model of microgravity for cardiovascular deconditioning. head-down bed rest (HDBR) is able to replicate cephalic fluid shift, immobilization, confinement, and inactivity. The dry immersion (DI) model is another approach where the subject remains immersed in thermoneutral water covered with an elastic waterproof fabric separating the subject from the water. Regarding DI, this analog imitates the absence of any supporting structure for the body, centralization of body fluids, immobilization, and hypokinesia observed during spaceflight. However, little is known about the impact of microgravity on CA. Here, we review the fundamental principles and the different mechanisms involved in CA. We also consider the different approaches in order to assess CA. Finally, we focus on the effects of short- and long-term spaceflight on CA and compare these findings with two specific analogs to microgravity: HDBR and DI¹⁾.

Astronauts who undergo prolonged periods of spaceflight may develop a unique constellation of neuro-ocular findings termed Spaceflight Associated Neuro-Ocular Syndrome (SANS). SANS is a disorder that is unique to spaceflight and has no terrestrial equivalent. The prevalence of SANS increases with increasing spaceflight duration and although there have been residual, structural, ocular changes noted, no irreversible or permanent visual loss has occurred after SANS, with the longest spaceflight to date being 14 months. These microgravity-induced findings are being actively investigated by the United States National Aeronautics Space Administration (NASA) and SANS is a potential obstacle to future longer duration, manned, deep space flight missions. The pathophysiology of SANS remains incompletely understood but continues to be a subject of intense study by NASA and others. The study of SANS is of course partially limited by the small sample size of humans undergoing spaceflight. Therefore, identifying a terrestrial experimental model of SANS is imperative to facilitate its study and for testing of preventative measures and treatments. Head-down tilt bed rest (HDTBR) on Earth has emerged as one promising possibility. In this paper, we review the HDTBR as an analog for SANS pathogenesis; the clinical and imaging overlap between SANS and HDTBR ².

Laurie et al. exposed healthy subjects (n = 24) to strict 6° head-down tilt bed rest (HDTBR), an analog of weightlessness that generates a sustained headward fluid shift, and monitored for ocular changes similar to findings that develop in Spaceflight associated neuro-ocular syndrome (SANS). Two-thirds of the subjects received daily 30-min exposure to artificial gravity (AG, 1 g at center of mass, ~0.3 g at eye level) during HDTBR by either continuous (cAG, n = 8) or intermittent (iAG, n = 8) short-arm centrifugation to investigate whether this intervention would attenuate headward fluid shift-induced

ocular changes. Optical coherence tomography images were acquired to quantify changes in peripapillary total retinal thickness (TRT), retinal nerve fiber layer thickness, and choroidal thickness, and to detect chorioretinal folds. Intraocular pressure (IOP), optical biometry, and standard automated perimetry data were collected. TRT increased by 35.9 μ m (95% CI, 19.9-51.9 μ m, p < 0.0001), 36.5 μ m (95% CI, 4.7-68.2 μ m, p = 0.01), and 27.6 μ m (95% CI, 8.8-46.3 μ m, p = 0.0005) at HDTBR day 58 in the control, cAG, and iAG groups, respectively. Chorioretinal folds developed in six subjects across the groups, despite small increases in IOP. Visual function outcomes did not change. These findings validate strict HDTBR without elevated ambient CO2 as a model for investigating SANS and suggest that a fluid shift reversal of longer duration and/or greater magnitude at the eye may be required to prevent or mitigate SANS ³.

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