LETTER

The buffering capacity of the brain and optic nerve against spaceflight-associated neuro-ocular syndrome

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We read with great interest and enthusiasm the article by Van Ombergen et al. (1) published in PNAS. We would like to congratulate the authors for doing this prospective study with findings of great importance to the field of space health research, and we appreciate the opportunity to make a comment.

The authors found an increased ventricular cerebrospinal fluid (CSF) volume after spaceflight in supratentorial ventricular structures (i.e., lateral and the third ventricles) and further hypothesize that the impact on the supratentorial regions is an indicator for the structural compliance and CSF reserve capacity of these ventricles acting as buffers in healthy adults (1). According to the authors, this might be considered a preliminary coping mechanism in space that acts as an intermediate overflow zone for the reduced CSF resorption. They further suggest a potential link between the brain ventricular and CSF changes and symptoms of spaceflight-associated neuro-ocular syndrome (SANS), in the sense that a larger compensatory reserve capacity in CSF space, for example by a greater compliance and elasticity of the ventricles, may protect against ophthalmic changes in astronauts. We fully agree with this notion. We recently proposed a similar compensatory mechanism at the level of the optic nerve sheath (2, 3). We hypothesized that astronauts with less-compliant optic nerve sheaths may be more likely to develop optic disc swelling, given that a greater degree of optic nerve sheath rigidity that prevents further expansion may result in more CSF pressure increase in the subarachnoid space surrounding the optic nerve (2, 3). According to this hypothesis, microgravity-induced ocular changes may be partially determined by the elastic properties of the optic nerve sheath (4). As such, it would be interesting to evaluate the optic nerve sheath response to alterations in CSF pressure as a predictive biomarker for optic disc edema in astronauts (2).

It has also been questioned whether the optic canal acts as a buffer against the transmission of CSF pressure to the lamina cribrosa (5). In a retrospective analysis with magnetic resonance imaging (MRI), Bidot et al. (5) found that larger optic canals were associated with more severe papilledema and poor visual function in 69 patients with idiopathic intracranial hypertension (IIH). One possible explanation suggested by the authors was that narrower optic canals limit CSF flow into the perioptic CSF compartment, whereas larger optic canals facilitate the transmission of the CSF pressure to the lamina cribrosa, resulting in worse papilledema (5). Accordingly, they demonstrated using MRI that the optic canal was always larger on the side of the worst papilledema in a series of 8 patients with IIH and very asymmetric papilledema (6). Therefore, examination of the optic canal size in astronauts would be very informative and could lead to further elucidation of the possible mechanisms underlying SANS (7, 8).

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