the eye. However, in long-duration space flight–induced increased ICP, paravenous outflow may fail to match ocular paravascular arterial inflow due to the reduction of the normal TLCPD. Ocular paravenous outflow may be completely impeded if there is reversal of the TLCPD. This may result in glymphatic stasis predominantly within the prelamellar region of the optic nerve head, which could contribute to the optic disc edema seen in astronauts. The accumulation of toxic metabolites due to glymphatic stasis then may cause further disc swelling.

Although this mechanism is speculative, it is important to note that Denniston et al (6) provided support for the importance of the ocular glymphatic system in the pathogenesis of papilledema secondary to idiopathic intracranial hypertension (IIH). Using spectral domain optical coherence tomography in a cohort of patients with IIH, they demonstrated a number of structural differences not seen in healthy controls, including the presence of perivascular “black holes.” This was observed both in the optic nerve head and within the retinal nerve fiber layer, and the authors hypothesized that this represents dilated perivascular glymphatic channels. There seemed to be a relationship between the degree of papilledema and the extent of these changes. In healthy controls, the authors did not detect these features. Given these findings, we believe that our proposed theory deserves further study and may ultimately contribute to the field of space health research.

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REFERENCES

Why a One-Way Ticket to Mars May Result in One-Way Directional Glymphatic Flow to the Eye: Response

We greatly appreciate the opportunity to respond to the comments of Dr. Wostyn et al. Their theory focuses on a glymphatic flow imbalance mechanism at the optic nerve head that may, at least partially, explain the development of optic disc swelling in astronauts during long-duration space flight. This hypothesis would still be dependent on a space-flight induced rise in cerebrospinal fluid (CSF) pressure by some mechanism within the subarachnoid space (SAS) surrounding the optic nerve. Perhaps the effects of increased intracranial pressure, sequestration of CSF within the SAS of the optic nerve or a combination of the 2, acting in conjunction with the authors’ 2 proposed mechanisms, may produce varying degrees of optic disc swelling. It also is possible that the contribution of this mechanism to disc swelling is a relatively constant low magnitude finding that occurs with even a small change in the translaminal pressure difference.

Continued examination of all space flight crew members, including those without clinically apparent optic disc swelling, may provide additional insight regarding the relative contribution of glymphatic stasis. More measurements of opening pressure on lumbar puncture are needed in astronauts completing long-duration space flights. These data, in conjunction with...
future preflight, in flight and postflight analysis of the optic discs and choroid by optical coherence tomography as well as ultrasound and MRI examination of the globe and optic nerve sheaths, may provide important information for analysis.

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Contiguous Silicone Oil Migration From the Vitreous Cavity to the Optic Tract

We would like to applaud Boren et al (1) for their article “Retrolaminar migration of silicone oil.” We evaluated a patient who adds to the spectrum of neuroimaging findings of this disorder.

A 92-year-old man with a longstanding history of glaucoma underwent vitrectomy with silicone oil endotamponade in his right eye for a retinal detachment 3 years previously. Because of transient right hand weakness and gait ataxia, brain MRI was performed. This showed hyperdense material involving the right optic nerve, optic chiasm, and right optic tract (Fig. 1). Automated visual fields showed changes consistent with advanced glaucoma, but a homonymous defect was not detected.

We are unaware of previous reports of optic tract involvement with silicone oil. Perimetry showed that it did not cause homonymous visual field loss, supportive of the 2 asymptomatic cases reported by Boren et al. Given that the mechanism of intracranial silicone oil migration is not yet fully understood, future post-mortem histopathological examination of the eyes and brain will be essential in better understanding this disorder.

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