Author Response: Optic Nerve Sheath Distention as a Protective Mechanism Against the Visual Impairment and Intracranial Pressure Syndrome in Astronauts

We thank Wostyn and De Deyn for their excellent response1 to our recent correspondence “Choroidal Folds in Astronauts”2 published in IJOVS and for their insightful ideas regarding the potential role of the optic nerve sheath (ONS) in the production of disc swelling, globe flattening, and choroidal folds observed during and after long-duration space flight (LDSF). Two basic mechanisms, or perhaps a combination of the two, are hypothesized to account for elevated cerebrospinal fluid (CSF) pressure within the orbital subarachnoid space (SAS) that may be responsible for these anatomic changes.3–6

The first mechanism is based on the notion that a rise in intracranial pressure (ICP) occurs during space flight as a result of a microgravity (MG)-induced cephalad fluid shift that produces venous stasis in the head and neck. This stasis may lead to impairment of CSF outflow from the brain, cerebral venous congestion, and a resultant increase in ICP which may then be transmitted down the ONSs to the intraorbital SAS.3–7

The second mechanism proposes that these changes may result from a local, MG-induced rise in ONS pressure within the orbit with or without a rise in ICP.3–6,8–11 The intracanalicular portion of the optic nerve in this setting might be a “bottleneck,” leading to reduced outflow of CSF from the distended ONS.3,11 A similar mechanism may occur in terrestrial intracanalicular or posterior orbital apex nerve tumors (e.g., sheath meningiomas) where the visual acuity remains normal, the visual field might show only an enlarged blind spot, and the optic disc may show chronic edema for years without the development of optic atrophy or significant visual loss over time.

The end result of either mechanism is a rise in CSF pressure within the SAS of the orbit that may cause ONS distention and a concurrent anteriorly directed force that indents the posterior globe. As the authors point out, this posterior globe flattening would account for the axial shortening, hyperopic shift, and choroidal folds documented in many astronauts. Additionally, this same increase in ONS pressure could compress the ON within the orbit and, in conjunction with its effects across the lamina cribrosa, may produce stasis of axoplasmic flow with resultant optic disc swelling.5,12

It should be noted that ON sheath expansion and globe flattening have been documented by in-flight ultrasound only 10 days into a space mission.5 Therefore, whatever the specific mechanism, these anatomic changes occur rather quickly following exposure to MG. Furthermore, posterior globe flattening has been documented for more than 7 years following LDSF suggesting that these changes may be permanent (C. Robert Gibson, personal communication, 2017). It is possible that increased ONS pressure may act in conjunction with metabolic toxins within the ONS to structurally remodel the posterior sclera.5,10 We would also like to mention that the National Aeronautics and Space Administration has recently changed the nomenclature for this syndrome from visual impairment intracranial pressure syndrome, as stated by the authors, to space flight–associated neuro-ocular syndrome (SANS) as this is thought to be a more appropriate descriptive term.13

The authors propose that the ONS response to the rise in CSF pressure within the orbital SAS may impact the susceptibility of an astronaut to the anatomic changes of SANS. This ONS response to pressure could indeed help to explain the large spectrum of anatomic change noted in astronauts during and after nearly identical MG exposure on the ISS. They point out the work of Hansen and Helmke, who used intrathecal infusion to demonstrate that the extent of ONS dilation, as measured by ultrasonography, was directly correlated with increasing CSF pressure within the sheath until a saturation point was reached at which no further dilation occurred.14 Presumably, at this saturation point, the ONS assumes a degree of rigidity that prevents further expansion and causes a more direct transfer of increased CSF pressure to the posterior globe and ON. The authors point out that as this compensatory mechanism reaches its limit even small increases in CSF volume may result in prominent increases in CSF pressure within the ONS. Therefore, a low saturation point may anatomically predispose an astronaut to a more prominent increase in pressure. In contrast, a degree of protection may be associated with a higher saturation point. In this scenario, sufficient ONS elasticity may result in continued expansion of the ONS with less of a SAS pressure increase. Thus, variations in elasticity within the structure of the ONS may result in dissimilar degrees of anatomic change to the globe and ON during spaceflight.

Hansen et al. also documented that following ONS dilation there can be a permanent expansion or resetting of the ONS.15 The long-term persistence of varying degrees of post mission globe flattening and associated refractive changes in astronauts during MG exposure suggests a similar permanent resetting of the posterior globe contour that may be partially determined by the elastic properties of the ONS. Further work is necessary and ongoing to determine the basic mechanism causing SANS as we prepare for possible longer duration space flights including a potential manned mission to Mars.

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