

UNILATERAL LOSS OF SPONTANEOUS VENOUS PULSATIONS IN AN ASTRONAUT

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Abstract:

Spontaneous venous pulsations seen on the optic nerve head (optic disc) are presumed to be caused by fluctuations in the pressure gradient between the intraocular and retrolaminar venous systems. The disappearance of previously documented spontaneous venous pulsations is a well-recognized clinical sign usually associated with a rise in intracranial pressure and a concomitant bilateral elevation of pressure in the subarachnoid space surrounding the optic nerves. In this correspondence we report the unilateral loss of spontaneous venous pulsations in an astronaut 5 months into a long duration space flight. We documented a normal lumbar puncture opening pressure 8 days post mission. The spontaneous venous pulsations were also documented to be absent 21 months following return to Earth.. We hypothesize that these changes may have resulted from a chronic unilateral rise in optic nerve sheath pressure caused by a microgravity-induced optic nerve sheath compartment syndrome.

Changes in Spontaneous Venous Pulsations During Long-Duration Space Flight

Introduction:

Spontaneous venous pulsations (SVPs) are pulsatile, sometimes subtle, changes in the diameter of one or more retinal veins just before they exit the eye to form the central retinal vein (CRV).¹ The disappearance of previously documented SVPs is a well known clinical sign usually associated with elevated intracranial pressure (ICP). Cessation of SVPs is thought to occur when a rise in ICP is propagated bilaterally down the subarachnoid space (SAS) surrounding the optic nerves (ON), resulting in a concurrent rise in central retinal venous outflow pressure equal to or greater than the intraocular venous pressure. In this report we document the unilateral loss of SVPs in an astronaut 5 months into a 6 month space mission. We documented a normal lumbar puncture opening pressure 8 days post mission. The SVPs were also absent 21 months following return to Earth. We hypothesize that this unusual finding may have resulted from a unilateral optic nerve sheath compartment syndrome brought about by cephalad fluid changes associated with long-duration space flight.

Case report:

A 57 year old male astronaut flew on his first 6 month space mission in 2003 and his most recent in 2011-12. His first pre mission fundus examination in 2003 was normal bilaterally (OU) but his post mission examination revealed choroidal folds and a cotton wool spot (CWS) in his right eye (OD). The left (OS) ocular fundus was normal. His follow up pre-flight examination, performed in 2011, documented normal fundi, subtle choroidal folds OD by optical coherence tomography (OCT), as well as bilateral ON sheath distention and mild globe flattening OD by magnetic resonance imaging (MRI). These findings were reported previously in the medical literature.² Examination of the optic discs with a 78 D hand-held lens at the slit lamp documented that SVPs were present OU pre mission. Five months into the second mission, remotely guided video funduscopy on the International Space Station (ISS) documented mild disc edema and choroidal folds OD with a normal disc OS. Although remote video examination could not achieve the same degree of resolution as the pre-mission 78 D lens examination, SVPs appeared to be absent OD but were prominent OS. Two days post mission Frisen grade 1 disc edema with trace SVPs were documented by 78D lens assessment OD as were choroidal folds. The left fundus was normal with prominent SVPs. Six days postflight, MRI documented a moderate increase in both optic nerve sheath diameters (ONSDs), OS > OD, compared with preflight and there was globe flattening OU. There was a prominent peripapillary retinal nerve fiber layer (RNFL) thickening by spectral domain OCT on the right with a normal thickness on the left. Eight days post mission the astronaut underwent a lumbar puncture (LP) in the lateral decubitus

position at which time the opening pressure was 18 cm H₂O. Intraocular pressures (IOPs) averaged 12 mm Hg OU during the mission as compared to pre and post flight measurements of 10 mm Hg. Fifty-two days following the mission his right optic disc swelling had resolved as documented by 78 D lens assessment and OCT. Subtle SVPs were now documented OD, whereas SVPs were still prominent OS. His last dilated fundus examination and OCT, performed approximately 21 months following completion of the mission, documented normal peripapillary RNFLs OU but persistent choroidal folds OD. At this time, high resolution Heidelberg Spectralis OCT video and 78 D lens assessment revealed that SVPs were absent OD but prominent OS. His last MRI, performed nearly 7 months postflight, was unchanged compared to the previous study with continued bilateral ONSD, OS>OD.

Discussion:

The specific mechanism of SVP formation is poorly understood. However, SVPs are generally thought to arise from normal cyclical venous pressure changes that occur within the eye and retrolaminar region. Blood flows from the retinal capillaries into the retinal veins because there is a relatively higher pressure in the retinal capillary bed compared with the lower intraocular retinal venous pressure (RVP).¹ Ocular outflow pressure is defined as the RVP at the point where the central retinal vein (CRV) exits the eye. The outflow pressure is determined by the magnitude of the retrolaminar venous pressure within the optic nerve and must be lower than the intraocular RVP in order for blood to flow out of the eye.^{1,3} The CRV exits the ON to enter the SAS about 10 mm posterior to the globe and therefore is subject to changes in cerebrospinal fluid (CSF) pressure.^{4,5} The classic theory of SVPs assumes that as the intraocular pulse pressure is normally 1 mmHg higher than the retrolaminar venous pulse pressure during systole and 1 mmHg lower during diastole, blood flow from the eye increases during systole and ceases during diastole.⁶ Thus, because the RVP is always greater than the IOP⁷ and venous drainage from the capillaries to the CRV remains constant, the sudden transient increase in flow at the point of venous outflow during systole may temporarily decrease the volume of blood in a small segment of the vein at the disc, resulting in a segmental venous collapse.¹ During diastole the flow of venous blood exiting the eye ceases because the retrolaminar venous pressure is higher than the intraocular venous pressure. Thus, the venous blood volume quickly increases in the previously collapsed segment and the vein expands. As emphasized by Levine, the length of the pulsating venous segment is short because the pulsation is dampened by the physical properties of the vein, blood and surrounding structures.⁸ Therefore, due to small but abrupt changes in the venous pulse pressure between the intraocular space and the retrolaminar CRV we are able to visibly detect pulsations at the ON head in patients with normal ICPs. The SVPs are eliminated when the downstream pressure in the CRV at the lamina cribrosa rises above that of the maximum intraocular venous pressure produced during systole. When this scenario occurs, the venous pressure thrust of systole cannot overcome the higher downstream pressure in the CRV and the vein retains its normal diameter throughout the cardiac cycle. An alternative theory suggests that central retinal vein collapse occurs in time

with IOP and ICP diastole. This hypothesis suggests that ICP pulse pressure dominates the timing of venous pulsations.⁹

Theoretically, any clinical condition that causes a sufficient rise in CRV pressure can abolish SVPs. Most commonly, this results from a rise in ICP from space-occupying intracranial masses or idiopathic intracranial hypertension (IIH). In these settings, elevated CSF pressure around the brain, regardless of the cause, is transmitted down both optic nerve sheaths toward the globe. As noted above, from the lamina cribrosa, the CRV drains within the central portion of the ON but exits the nerve approximately 10 mm posterior to the globe. At this point, the vein traverses the SAS and the stage is set for an elevated SAS pressure to directly impact the pressure within the CRV.^{4,5} There is generally thought to be homogeneity of pressures throughout the SAS of the brain and ONs. Thus, abolition of previously noted SVPs generally is seen bilaterally because presumably both ON sheath pressures are affected equally from a rise in ICP.

Asymmetrical disc swelling, globe flattening, and choroidal folds recently have been documented in astronauts following long duration space flight.^{2,10} These changes have been hypothesized to result from a microgravity induced ON sheath compartment syndrome that may result in asymmetrical elevation of pressure within the ON sheath of some astronauts during the flight.^{2,10-12} This may be caused by the gradual sequestration of CSF within the ON sheath compartment caused by cephalad fluid shifts during long duration space flight.^{2,10} It is interesting to note that in the astronaut we describe, a postflight MRI documented a moderate increase in the ON sheath diameters of both eyes, OS > OD, with bilateral globe flattening in conjunction with right sided disc swelling and loss of SVPs. Without knowing the concurrent pressures or flow characteristics of the ON sheath compartments, it is difficult to determine precisely why prominent SVPs would continue on the left side while being abolished on the right. However, these data suggest that local anatomic factors such as nerve sheath compliance may play a role in determining the degree of pressure elevation within each nerve sheath compartment.¹³ This is also consistent with the finding of normal ICP by lumbar puncture at the time there were only trace SVPs in the right eye.

In our current report we documented bilateral SVPs on Earth prior to this astronaut's second space mission, suggesting normal SAS pressures within the ON sheaths. However, 5 months into the mission we documented mild disc swelling with absent SVPs on the right, despite prominent SVPs and a normal disc on the left. The disappearance of right SVPs with concurrent disc swelling and its contrast with the normal appearance of the left disc during a space mission, strongly suggests that microgravity exposure led to a prominent increase in the right ON sheath pressure. Twenty-one months following completion of the space mission, although the disc swelling had resolved, SVPs were clearly absent in the right eye, but present and obvious on the left. A post mission LP opening pressure of 18 cm H₂O implies normal ICP in the presence of these clinical findings, but in flight increased ICP cannot be completely excluded as an alternative hypothesis. Although our study

was impacted by some variability in examination techniques the fact that the right eye SVPs were present preflight and clearly absent 21 months post mission suggests that, in this astronaut, some residual pressure elevation from an ON sheath compartment syndrome may have persisted long after the mission ended. This may help to explain continued asymmetrical ON sheath induced globe flattening and associated hyperopic shifts noted in some astronauts years following space missions.^{2,10} Further work is necessary to determine if our hypothesis of local cephalad fluid shift versus increased ICP or both or some other mechanism are at play in the etiology of these ocular findings in our astronauts during long duration space flight.

References

1. Jacks AS, Miller NR. Spontaneous retinal venous pulsation: aetiology and significance. *J Neurol Neurosurg Psychiatry*. 2003;74:7-9.
2. Mader TH, Gibson CR, Pass AF, Lee AG, Killer HE, Hansen H-C, Dervay JP, Barratt MR, Tarver WJ, Sargsyan AE, Kramer LA, Riascos R, Bedi DG, Pettit DR. Optic disc edema in an astronaut after repeat long-duration space flight. *J Neuro-Ophthalmol*. 2013;0:1-7.
3. Baurmann M. Ueber die Entschung und klinicke Bedeutung des Netzhoutvenenpulses. *Ber Zusammenkunft Dtsch Ophthalmol Ges* 1925;45:53-9.
4. Fry WE, Variations in the intraneural course of the central retinal vein. *Arch Ophthalmol*. 1930;4:180-7.
5. Fry WE. The pathology of papilloedema: an examination of forty eyes with special reference to compression of the central vein of the retina. *Am J Ophthalmol*. 1931;14:874-83.
6. Dardenne G, Dereymaeker A, Lacheron JM. Cerebrospinal fluid pressure and pulsatility: an experimental study of circulatory and respiratory influences in normal and hydrocephalic dogs. *Invest Neurol* 1969;2:193-216.
7. Attariwala F, Giels CP, Glucksberg MR. The influence of elevated intraocular pressure on vascular pressures in the cat retina. *Invest Ophthalmol Vis Sci* 1994;35:1019-25.
8. Levin BE. The clinical significance of spontaneous pulsations of the retinal vein. *Arch Neurol* 1978;35:37-40.
9. Morgan WH, Lind CRP, Kain S, Fatebee N, Bala A, and Yu D. Retinal vein pulsation is in phase with intracranial pressure and not intraocular pressure. *Invest Ophthalmol Vis Sci* 2012;53:4676-81.
10. Mader TH, Gibson CR, Pass AF, Kramer LA, Lee AG, Fogarty J, Tarver WJ, Dervay JP, Hamilton DR, Sargsyan A, Phillips JL, Tran D, Lipsky W, Choi J, Stern C, Kuyumjian R, Polk JD. Optic disc edema, globe flattening, choroidal fold, and hyperopic shifts observed in astronauts after long-duration space flight. *Ophthalmol* 2011;118:2058-69.
11. Killer HE, Jaggi GP, Flammer J, Miller NR, Huber AR, Mironov A. Cerebral spinal fluid dynamics between the intracranial and the subarachnoid space of the optic nerve. Is it always bidirectional? *Brain* 2007;130:514-520.
12. Killer HE, Jaggi GP, Miller NR. Papilledema revisited: Is its pathophysiology really understood? *Clin Experiment Ophthalmol* 20 09:37:444-7.
13. Hansen H-C, Lagreze W, Krueger O, Helmke K. Dependence of the optic nerve sheath diameter on acutely applied subarachnoidal pressure – an experimental ultrasound study. *Acta Ophthalmol*. 2011;1-5.