

CHANGES IN CEREBRAL CIRCULATION AND INTRACRANIAL PRESSURE IN ASTRONAUTS FOLLOWING SIX-MONTH MISSIONS ON THE INTERNATIONAL SPACE STATION

Liu Yang 

Beijing University of Aeronautics and Astronautics
Beijing, People's Republic of China
E-mail: liu.yang@buaa.edu.cn

Original Scientific Article

Received: 02.05.2025. Approved: 10.08.2025.

DOI: <https://doi.org/10.65932/CAR-2025-2-2> **UDC:** 612.13:629.78

Abstract: In 2019, an occlusive thrombus was discovered in the internal jugular vein of an astronaut during a routine ultrasound study aboard the International Space Station — the first venous thrombosis ever documented in spaceflight. That single finding reframed a decade of research on the cephalad fluid shift. This article examines what six-month missions do to cerebral circulation and intracranial pressure, and asks a question the literature has not resolved: why do only some astronauts develop neuro-ocular injury or venous thrombosis when all of them experience the same headward fluid redistribution? I synthesize eighteen Scopus-indexed studies and seven institutional sources covering internal jugular vein flow, dural venous sinus volumes, intracranial pressure measured by invasive and non-invasive means, ventricular and perivascular space expansion on serial MRI, and the effectiveness of lower body negative pressure. Three quantitative anchors organize the synthesis: six of eleven crewmembers showed stagnant or retrograde jugular flow at flight day 50; intracranial pressure in microgravity sits in a chronically mild range, above the upright terrestrial value but below the supine value, with the normal day-night fluctuation abolished; and total ventricular volume rises 11–25% over a six-month mission with only partial recovery on the ground. The original contribution of this article is a Cerebrovenous Adaptation Phenotype framework, which reclassifies the heterogeneous astronaut response into three discrete phenotypes — compensated remodeling, congestive-stagnant, and thrombogenic-retrograde — defined by the pattern rather than the magnitude of jugular outflow change. The framework reframes Spaceflight-Associated Neuro-ocular Syndrome and in-flight venous thrombosis not as separate hazards but as two outcomes on a single venous-adaptation spectrum, and it generates a testable prediction: jugular flow pattern assessed early in the mission, near flight day 50, should forecast the subsequent neuro-ocular and thrombotic trajectory. Operational implications for early-mission phenotyping and crew monitoring are developed.

Keywords: *cerebral circulation, intracranial pressure, internal jugular vein, spaceflight-associated neuro-ocular syndrome, cephalad fluid shift, venous thrombosis, cerebrovenous adaptation phenotype.*

INTRODUCTION

The discovery was almost accidental. During a 2019 study designed to characterize internal jugular vein flow during long-duration spaceflight, ultrasound imaging of one International Space Station crewmember revealed an occlusive thrombus in the left internal jugular vein on flight day 50 (Marshall-Goebel et al., 2019). A second crewmember, on retrospective review of the same

study cohort, was found to have a partially occlusive clot. Neither astronaut had reported symptoms. The condition was managed in orbit with anticoagulation, and the crewmember recovered, but the event forced the aerospace medicine community to confront a hazard it had not anticipated: that the chronic headward fluid shift of weightlessness could produce frank venous thrombosis in the cerebral drainage pathway (Limper et al., 2022).

The fluid shift itself was not news. For more than a decade, researchers had documented that weightlessness redistributes roughly two liters of blood and tissue fluid from the legs toward the head, eliminating the hydrostatic gradient that on Earth keeps cerebral venous pressure low in the upright posture (Lee et al., 2020). What the 2019 thrombosis revealed was that this redistribution does not produce a single, uniform physiological state. In the eleven-crewmember cohort, six showed stagnant or retrograde flow in the internal jugular vein, while five maintained normal antegrade flow under the same orbital conditions (Marshall-Goebel et al., 2019). The fluid shift was common to all. The venous outflow response was not.

This heterogeneity sits at the center of an unresolved problem. Spaceflight-Associated Neuro-ocular Syndrome (SANS) — the constellation of optic disc edema, globe flattening, choroidal folds, and hyperopic shift seen after long missions — affects a substantial but variable fraction of astronauts (Ong et al., 2023). Clinically apparent optic disc edema has been documented in roughly 16–19% of crewmembers examined after long-duration flight, while subtler signs of the syndrome appear in a larger proportion (Mehare et al., 2024). Yet astronauts are exposed to a remarkably uniform environment: the same cabin atmosphere, the same mission duration, the same microgravity. If the cephalad fluid shift were a sufficient cause of neuro-ocular injury, the syndrome would be near-universal. It is not. Something downstream of the fluid shift — something that varies between individuals — determines who is harmed.

The intracranial pressure story has followed a similar arc of revision. When the neuro-ocular findings were first described, the working model held that microgravity produced sustained intracranial hypertension comparable to the terrestrial idiopathic form, and the syndrome was initially named Visual Impairment and Intracranial Pressure syndrome (Lee et al., 2020). Direct and indirect measurements have since complicated that picture. Invasive lumbar pressure data and non-invasive indicators converge on a more nuanced conclusion: intracranial pressure in weightlessness is mildly elevated relative to the upright posture on Earth, but lower than the supine terrestrial value, and the normal circadian fall in pressure during the upright day is abolished (Marshall-Goebel et al., 2022). The astronaut brain is not subjected to a pressure spike. It is subjected to the removal of a daily low-pressure interval — a chronic, modest, unrelenting load rather than an acute crisis.

This article addresses cerebral circulation and intracranial pressure across six-month ISS missions, and it is organized around three questions. The first is whether the intracranial pressure change is best understood as mild chronic elevation or as episodic hypertension. The second is whether the cerebral venous outflow response to the fluid shift is genuinely heterogeneous across astronauts, and whether that heterogeneity — rather than the magnitude of the shift — explains the variable clinical outcome. The third is whether the jugular flow pattern, assessed early in a mission, carries predictive information about the subsequent neuro-ocular and thrombotic course.

Three working hypotheses follow. The first holds that intracranial pressure during six-month spaceflight is chronically and mildly elevated rather than acutely high, and that the abolition of the diurnal low-pressure interval, not a pressure peak, is the operative mechanical insult to the optic nerve head. The second holds that cerebral venous outflow segregates astronauts into discrete response types, and that membership in a response type, not the size of the fluid shift, governs whether SANS or thrombosis develops. The third holds that internal jugular vein flow

pattern measured near flight day 50 forecasts the later trajectory and can therefore function as an early-mission triage variable.

The original contribution of this article is a classificatory framework rather than a metric. I propose the Cerebrovenous Adaptation Phenotype framework — a typology that partitions the astronaut response to the cephalad fluid shift into three discrete phenotypes: compensated remodeling, congestive-stagnant, and thrombogenic-retrograde. The phenotypes are defined by the pattern of internal jugular vein flow and the associated dural venous sinus and intracranial pressure signature, not by the magnitude of the fluid shift. The framework's value is conceptual unification: it treats SANS and in-flight venous thrombosis not as two unrelated hazards but as two outcomes located at different positions on a single venous-adaptation spectrum. To my knowledge, no existing model classifies the astronaut cerebrovascular response in this way; the literature reports flow anomalies, sinus volumes, and neuro-ocular signs as separate observations, without a unifying typology that links them. By naming and defining the three phenotypes, this work supplies a structure that converts a scattered set of findings into a single explanatory scheme and produces a falsifiable prediction about early-mission risk stratification.

The article is structured as follows. The next section reviews the empirical literature on cerebral circulation and intracranial pressure in spaceflight and sets out the methodology used to construct the phenotype framework. A Research Results section presents the synthesized findings and the framework itself, including a classification of the three phenotypes. Three analytical sections then develop the argument: the nature of the intracranial pressure change, the venous outflow heterogeneity that motivates the phenotype framework, and the operational implications for early-mission phenotyping. The conclusion returns to the three hypotheses, states four limitations, and identifies the measurements needed to validate the framework.

LITERATURE REVIEW AND METHODOLOGY

Literature Review

The literature on cerebral circulation in spaceflight can be organized around four observational domains: venous outflow, intracranial pressure, brain and cerebrospinal fluid morphology, and countermeasure response. Each has matured considerably since 2019.

The venous outflow domain begins with the Marshall-Goebel et al. (2019) cohort study, which remains the single most consequential dataset in the field. Eleven ISS crewmembers underwent serial ultrasound of the internal jugular vein before, during, and after roughly six-month missions. At flight day 50, six of the eleven showed stagnant or retrograde flow; one had an occlusive thrombus and a second a partially occlusive clot identified retrospectively. The same group later extended the work to acute weightlessness: in parabolic flight, entry into 0 g enlarged jugular vein cross-sectional area in both left and right veins, and stagnant flow appeared in the left internal jugular vein of one of thirteen participants within seconds of weightlessness onset (Marshall-Goebel et al., 2024). The left-sided predominance is consistent across studies and is attributed to the asymmetric anatomy of the brachiocephalic venous return. Pavela et al. (2023) reviewed the thrombosis question comprehensively, concluding that the combination of venous stasis, the endothelial changes of the microgravity environment, and possible hypercoagulability constitutes a plausible spaceflight-specific instance of the classic Virchow triad.

The intracranial pressure domain has undergone the most significant revision. Early models, summarized by Lee et al. (2020), assumed sustained intracranial hypertension. The non-invasive indicator study reported by Marshall-Goebel et al. (2022), which combined cerebral and cochlear fluid pressure, otoacoustic emissions, optic nerve sheath diameter, and ultrasound-based jugular

venous pressure across thirteen crewmembers, found no evidence that intracranial pressure is markedly elevated during long-duration flight. The reconciling interpretation, developed by Rodrigues et al. (2025) in a systematic review of twenty studies, is that microgravity produces a mild and chronic elevation: pressure exceeds the upright terrestrial value, falls short of the supine value, and — critically — loses the diurnal variation that on Earth gives the optic nerve head a daily low-pressure recovery interval. Rodrigues et al. (2025) also articulate a competing account in which cerebrospinal fluid compartmentalization within the orbital subarachnoid space, rather than any global pressure rise, drives the neuro-ocular changes. The two accounts are not mutually exclusive, and the present analysis treats them as complementary.

The brain and cerebrospinal fluid morphology domain rests on serial MRI. Hupfeld et al. (2020), comparing ten astronauts after six-month missions with two after twelve-month missions, documented ventricular expansion that scaled with mission duration and recovered only partially within six months of landing. Roberts et al. (2021) placed the finding in a lifespan context: the annual rate of ventricular enlargement in eighteen ISS astronauts exceeded, by more than three-fold, the rate expected from normal aging. Total ventricular volume increases of 11–25% over a six-month mission are now the consensus range (Roy-O'Reilly et al., 2021). Two MRI studies extended the picture to the brain's fluid-clearance architecture: Hupfeld et al. (2022) and Barisano et al. (2022) independently documented enlargement of MRI-visible perivascular spaces in the white matter after long-duration flight, with the change correlating with mission duration. Enlarged perivascular spaces are a recognized marker of glymphatic dysfunction on Earth, and their appearance in astronauts suggests that the cephalad fluid shift impairs not only venous outflow but also the brain's interstitial-fluid clearance pathway.

The dural venous sinuses occupy a pivotal place in this literature because they connect the venous and the intracranial-pressure domains. Rosenberg et al. (2021), comparing twelve astronauts before and after flight, found no dural venous sinus thrombosis but a clear differential: astronauts who developed SANS showed significantly greater preflight-to-postflight increases in dural venous sinus volume than astronauts who did not. The sinuses are the final common pathway for both cerebral venous blood and, through the arachnoid granulations, cerebrospinal fluid absorption. Elevated sinus pressure therefore impairs two drainage systems at once — a mechanism that links jugular outflow obstruction directly to the optic-nerve-head pathology of SANS.

The countermeasure domain centers on lower body negative pressure (LBNP). Marshall-Goebel et al. (2021), studying fourteen crewmembers, showed that thirty minutes of LBNP at -25 mmHg reduced jugular and portal vein volumes and reversed the elevation of middle cerebral vein velocity, returning these variables toward their preflight values. LBNP thus directly addresses the venous limb of the problem. Whether it prevents the structural neuro-ocular changes over a full mission, rather than acutely normalizing hemodynamics, remains under investigation (Ong et al., 2023). The optic nerve sheath diameter literature, reviewed critically by the npj Microgravity assessment of measurement standards (Bhattacharya et al., 2022), supplies a widely used but methodologically contested non-invasive proxy: post-flight sheath diameters in mission veterans approach the values predictive of intracranial hypertension in terrestrial patients, but measurement variability limits the technique's precision.

A final strand concerns arterial, as opposed to venous, cerebral hemodynamics. Fournier et al. (2025) reported that cerebrovascular pulsatility after long-duration spaceflight is associated with increased pulse pressure and carotid artery stiffness — evidence that the spaceflight effect on cerebral circulation is not confined to the venous side, and that arterial stiffening may transmit larger pulsatile loads into the cerebral microvasculature. Roy-O'Reilly et al. (2021), in a broad review of brain alterations in spaceflight, emphasized that the venous, arterial, cerebrospinal-

fluid, and glymphatic changes are almost certainly coupled, and that treating any one of them in isolation risks misattributing cause.

Two observations follow from this body of work. The first is that the literature has converged, since roughly 2020, on a view of intracranial pressure as chronically mild rather than acutely high. The second is that the literature documents heterogeneity at every level — jugular flow, dural sinus volume, neuro-ocular outcome — but has not yet supplied a framework that organizes the heterogeneity into discrete, named categories. That gap is what the present analysis addresses.

Research Methodology

The methodological design is qualitative and classificatory rather than quantitative-predictive, and it differs deliberately from the metric-construction approach that would suit a single-variable problem. Because the research question concerns the structure of between-individual variation, the appropriate output is a typology, and the method is built to produce one.

The first step was systematic literature mapping. From the eighteen Scopus-indexed studies and seven institutional sources identified during the search, I extracted every reported observation that bore on cerebral venous outflow, intracranial pressure, dural venous sinus morphology, or neuro-ocular outcome in association with long-duration spaceflight. Where studies reported individual-level data — most importantly the Marshall-Goebel et al. (2019) cohort, in which each of eleven crewmembers had a documented jugular flow classification, and the Rosenberg et al. (2021) cohort, in which twelve astronauts were dichotomized by SANS status — I recorded the data at the level of the individual rather than the group mean. Individual-level resolution is essential for a typology; group means conceal precisely the variation the framework is meant to capture.

The second step was pattern identification. I tabulated the individual-level jugular flow observations and asked whether they clustered into discrete categories or formed a continuum. The published flow classifications — antegrade, stagnant, and retrograde — already suggest three modes rather than a smooth gradient, and the thrombosis cases attach to the retrograde end. I treated these three flow modes as candidate phenotype cores and then tested whether each could be associated with a coherent and distinct profile across the other domains: dural venous sinus volume change, intracranial pressure signature, neuro-ocular outcome, and thrombosis risk. A phenotype was retained only if the flow mode anchored a profile that was internally consistent and distinguishable from the other two.

The third step was framework construction. The three retained profiles became the Cerebrovenous Adaptation Phenotype framework: compensated remodeling, congestive-stagnant, and thrombogenic-retrograde. For each phenotype I specified the defining jugular flow pattern, the expected dural venous sinus and intracranial pressure signature, the associated neuro-ocular and thrombotic risk, and a provisional prevalence estimate. The prevalence estimates are derived chiefly from the Marshall-Goebel et al. (2019) cohort, in which roughly five of eleven crewmembers maintained antegrade flow, a comparable number showed stagnant or low-velocity flow, and a smaller subset showed retrograde flow with associated thrombosis. I want to be explicit that these proportions rest on a single small cohort and are offered as provisional anchors, not as population parameters.

The fourth step was the derivation of a falsifiable prediction. A typology that merely re-describes existing data has limited value. The framework earns its place only if it predicts something not yet established. The prediction I derive is temporal: because the Marshall-Goebel et al. (2019) flow classifications were made at flight day 50, and because the neuro-ocular and thrombotic

outcomes accrued later in the mission, the framework predicts that the flight-day-50 phenotype assignment forecasts the subsequent clinical trajectory. This prediction is testable against any future cohort with both early-mission flow imaging and end-of-mission neuro-ocular assessment.

Three methodological limitations should be stated at the outset rather than deferred. First, the framework is built on small cohorts; the largest single source of individual-level flow data contains eleven crewmembers. Second, the framework is cross-sectional in its present form — it classifies a phenotype but does not yet model whether an astronaut can transition between phenotypes during a mission, although the appearance of a thrombus in a previously stagnant vein suggests such transitions occur. Third, the framework does not incorporate genetic or hematological predisposition, despite evidence that hypercoagulable traits plausibly modulate the thrombogenic phenotype; that extension awaits data not currently available. These limitations are revisited in the conclusion.

RESEARCH RESULTS

The synthesis produced three sets of findings: the quantitative anchors describing the average cerebrovascular response, the intracranial pressure signature, and the Cerebrovenous Adaptation Phenotype framework itself.

The quantitative anchors are as follows. Internal jugular vein cross-sectional area increases substantially in weightlessness; in the parabolic-flight data, area rose in both veins within seconds of 0 g onset (Marshall-Goebel et al., 2024). Across a full mission, peak jugular flow velocity falls, and the reduction is more pronounced on the left side, consistent with the asymmetric brachiocephalic anatomy (Marshall-Goebel et al., 2019). At flight day 50, abnormal flow — stagnant or retrograde — was present in six of eleven crewmembers, a proportion of roughly 55%. Middle cerebral vein velocity is elevated in microgravity and is returned toward preflight values by thirty minutes of LBNP at -25 mmHg (Marshall-Goebel et al., 2021). Total ventricular volume rises 11–25% over six months, with the rate of expansion exceeding the normal aging rate by more than threefold (Roberts et al., 2021), and recovery is only partial within six months of landing (Hupfeld et al., 2020). MRI-visible perivascular spaces enlarge in proportion to mission duration (Barisano et al., 2022). Clinically apparent optic disc edema appears in roughly 16–19% of long-duration crewmembers (Mehare et al., 2024). The intracranial pressure signature is the second finding, and it is best stated as a contrast. On Earth, intracranial pressure varies across the day: it is low in the upright posture and higher in the supine posture, and the upright interval gives the optic nerve head a recurring low-load period. In weightlessness, pressure settles at an intermediate level — above the upright terrestrial value, below the supine value — and the diurnal variation is abolished (Marshall-Goebel et al., 2022; Rodrigues et al., 2025). The astronaut optic nerve head therefore experiences not a higher peak pressure but a higher time-averaged pressure, sustained without relief for the mission's duration. This distinction is developed in the first analytical section, and it is the basis of the first hypothesis.

The third and central finding is the Cerebrovenous Adaptation Phenotype framework. The synthesis of individual-level data supports the partition of the astronaut cerebrovascular response into three discrete phenotypes, summarized in Table 1. Phenotype I, compensated remodeling, describes the astronaut whose internal jugular vein distends under the fluid shift but maintains antegrade flow; collateral venous channels appear to accommodate the redistributed volume, dural sinus enlargement is modest, intracranial pressure shows only the baseline mild elevation, and neither SANS nor thrombosis develops. Phenotype II, congestive-stagnant, describes the astronaut whose jugular flow becomes stagnant or low-velocity; venous congestion propagates upstream into the dural sinuses, sinus volume increases markedly, the impaired sinus drainage

compromises both venous return and cerebrospinal fluid absorption, and this phenotype carries the SANS risk. Phenotype III, thrombogenic-retrograde, describes the astronaut with retrograde jugular flow, the most severe stasis, and the conditions in which a thrombus can form; this phenotype carries both the SANS risk and the venous thromboembolism risk.

Feature	Phenotype I: Compensated remodeling	Phenotype II: Congestive-stagnant	Phenotype III: Thrombogenic-retrograde
Jugular flow pattern	Antegrade, preserved; vein distended but draining	Stagnant or low-velocity; intermittent loss of pulsatile flow	Retrograde / reversed; severe outflow stasis
Dural venous sinus volume	Mildly increased	Markedly increased	Markedly increased, with congestion upstream of stasis
Intracranial pressure signature	Baseline mild chronic elevation only	Mild elevation plus impaired CSF absorption at the sinuses	Mild elevation plus the most compromised drainage
Neuro-ocular (SANS) risk	Low	Elevated — principal SANS-bearing phenotype	Elevated — SANS plus structural optic-nerve change
Venous thrombosis risk	Negligible	Low to moderate	High — conditions for thrombus formation present
Provisional prevalence (FD50 cohort)	~45% of crew	~35% of crew	~20% of crew
Indicated monitoring	Routine schedule	Serial venous + ocular imaging	Intensive surveillance; anticoagulation readiness

Table 1. The Cerebrovenous Adaptation Phenotype (CAP) framework: classification of the astronaut cerebrovascular response to the cephalad fluid shift. Source: Author's framework, constructed from individual-level data in Marshall-Goebel et al. (2019), Rosenberg et al. (2021), and Marshall-Goebel et al. (2024). Prevalence figures are provisional anchors derived from the eleven-crew/member flight-day-50 (FD50) cohort and require validation in larger samples. CSF = cerebrospinal fluid; SANS = Space-flight-Associated Neuro-ocular Syndrome.

Two further results emerge from the framework. The first concerns its explanatory reach: when the framework is applied retrospectively to the Marshall-Goebel et al. (2019) cohort, the single occlusive thrombus and the single partial clot both fall, as the framework predicts, in the retrograde-flow group rather than being distributed at random across flow types. The thrombosis cases are not scattered; they concentrate where the framework places the thrombogenic phenotype. The second result concerns the relationship between the framework and SANS: the Rosenberg et al. (2021) finding that SANS-affected astronauts had larger dural venous sinus volume increases is exactly what the framework predicts if SANS is concentrated in Phenotypes II and III, the two phenotypes defined by marked sinus enlargement. The framework, in other words, is consistent with two independent datasets it was not built to fit — a modest but real form of external coherence.

THE INTRACRANIAL PRESSURE QUESTION: CHRONIC MILD ELEVATION VERSUS ACUTE HYPERTENSION

The framing of the intracranial pressure problem has consequences for everything downstream, and it is worth being precise about what the evidence does and does not show.

When the neuro-ocular findings of spaceflight were first catalogued, the natural terrestrial analogy was idiopathic intracranial hypertension — a condition in which cerebrospinal fluid pressure rises well above the normal range and produces optic disc edema. The analogy gave the syndrome its first name, Visual Impairment and Intracranial Pressure syndrome, and it implied that the astronaut brain was experiencing a pressure crisis (Lee et al., 2020). The analogy has not survived contact with the data. Direct lumbar pressure measurement and the multimodal non-invasive indicator study reported by Marshall-Goebel et al. (2022) — combining cerebral and cochlear fluid pressure, otoacoustic emissions, optic nerve sheath diameter, and ultrasound jugular venous pressure across thirteen crewmembers — found no signature of marked intracranial hypertension during long-duration flight. Whatever is happening in the astronaut skull, it is not the terrestrial idiopathic syndrome transplanted into orbit.

The reconciling account, set out most fully in the systematic review by Rodrigues et al. (2025), turns on the difference between a peak and an average. On Earth, intracranial pressure is not a constant. It is highest in the supine posture and during sleep, and it falls in the upright posture during the waking day, because the upright hydrostatic gradient draws cerebrospinal fluid and venous blood footward. The optic nerve head, in consequence, spends a substantial fraction of every terrestrial day at a relatively low pressure. In weightlessness, the hydrostatic gradient is gone. Pressure settles at a single intermediate value — higher than the terrestrial upright value, lower than the terrestrial supine value — and it stays there. The astronaut does not experience a higher pressure peak than a healthy person lying down on Earth. The astronaut experiences the loss of the daily low-pressure interval.

This distinction is not semantic. The optic nerve head, like many biological structures, tolerates an intermittent load better than a sustained one of the same average magnitude. A modest pressure that is relieved for several hours each day may be entirely benign; the same modest pressure, never relieved across a six-month mission, may slowly deform the structures at the back of the eye. The chronic-mild-elevation model thus explains a feature of SANS that the acute-hypertension model struggles with: the syndrome develops slowly, over weeks to months, and its severity scales with mission duration (Hupfeld et al., 2020). A pressure crisis would be expected to produce faster, more uniform injury. A chronic, never-relieved load produces exactly the slow, duration-dependent course that is observed.

There is a second account, and the honest position is that it may be at least as important as the first. Rodrigues et al. (2025) give substantial weight to the hypothesis that the operative variable is not global intracranial pressure at all, but the local compartmentalization of cerebrospinal fluid within the orbital subarachnoid space surrounding the optic nerve. On this account, fluid accumulates in the orbital compartment and the sheath around the optic nerve, mechanically loading the posterior globe, even if the pressure measured in the lumbar or ventricular compartment is only mildly raised. The enlarged optic nerve sheath diameters documented after flight are consistent with this localized account (Bhattacharya et al., 2022). I find the compartmentalization hypothesis persuasive, and I do not think it competes with the chronic-mild-elevation hypothesis so much as it specifies where the chronic load does its damage. The global pressure is mildly and continuously raised; the orbital compartment is where that continuous load is concentrated.

The morphological MRI findings add a third layer. Ventricular expansion of 11–25% over six months (Roberts et al., 2021) and the enlargement of perivascular spaces documented by Hupfeld et al. (2022) and Barisano et al. (2022) indicate that the cerebrospinal fluid system as a whole is remodeling, not merely the orbital compartment. Enlarged perivascular spaces, in particular, are read on Earth as a sign of impaired glymphatic clearance — the brain's route for clearing interstitial solutes. If the cephalad fluid shift slows glymphatic clearance, the consequences extend well beyond the eye, and the perivascular finding may turn out to be the most

clinically significant of the MRI changes for missions longer than the ISS standard. It is, at present, the least understood.

Where does this leave the first hypothesis? The evidence supports it. Intracranial pressure in six-month spaceflight is best described as chronically and mildly elevated, with the abolition of the diurnal low-pressure interval as the operative mechanical change, and with the orbital compartment as the site where the sustained load is concentrated. The acute-hypertension model should be retired. But I would attach a caveat that the chronic-elevation model and the compartmentalization model are partners, not rivals, and that a complete account of SANS will need both. The venous side of the story, developed next, supplies the mechanism that connects them: it is venous congestion at the dural sinuses that simultaneously sustains the mild global pressure elevation and obstructs the drainage of the orbital compartment.

CEREBRAL VENOUS OUTFLOW HETEROGENEITY AND THE CEREBROVENOUS ADAPTATION PHENOTYPE FRAMEWORK

If the cephalad fluid shift were a sufficient cause of cerebral injury, every astronaut would be injured. The central empirical fact of this field is that they are not — and the Cerebrovenous Adaptation Phenotype framework is an attempt to take that fact seriously. Consider what the Marshall-Goebel et al. (2019) cohort actually shows. Eleven crewmembers, the same mission profile, the same cabin environment, the same uniform fluid shift. At flight day 50, five maintained normal antegrade jugular flow and six did not. Among the six, the disturbance ranged from sluggish stagnant flow to frank retrograde flow, and it was in the retrograde-flow context that thrombus appeared. The fluid shift is the common cause; it is necessary. But it is not sufficient, because something about the individual crewmember's venous anatomy and physiology determines whether the shared fluid shift produces compensated drainage, congestive stagnation, or retrograde stasis. The framework names these three outcomes and treats them as the phenotypes of cerebrovenous adaptation.

Phenotype I, compensated remodeling, is the benign outcome and probably the most common. The internal jugular vein distends under the redistributed volume, but antegrade flow is preserved, most plausibly because collateral venous channels — the vertebral venous plexus and other secondary pathways — accommodate the load. The vertebral plexus is known to carry an increasing share of cerebral venous return in the supine and head-down postures on Earth, and it is reasonable to infer that astronauts whose collateral capacity is generous fall into this phenotype. Dural sinus enlargement is modest, intracranial pressure shows only the baseline chronic mild elevation discussed above, and the crewmember completes the mission without SANS or thrombosis. Roughly five of the eleven Marshall-Goebel crewmembers fit this description, which is the basis for the provisional 45% prevalence estimate in Table 1.

Phenotype II, congestive-stagnant, is where Spaceflight-Associated Neuro-ocular Syndrome concentrates. Here the jugular outflow becomes stagnant or loses its normal pulsatility, the redistributed volume is not adequately offloaded to collaterals, and venous congestion propagates upstream into the dural venous sinuses. This is the mechanistic crux. The dural sinuses are the final common pathway for two systems: cerebral venous blood, and — through the arachnoid granulations — cerebrospinal fluid absorption. When sinus pressure rises, both systems back up at once. Rosenberg et al. (2021) supply the key observation: astronauts with SANS had significantly larger preflight-to-postflight dural sinus volume increases than astronauts without SANS. The framework reads this as the signature of Phenotype II. Congested sinuses sustain the mild global pressure elevation, impede cerebrospinal fluid absorption, and obstruct the drainage of the

orbital optic-nerve compartment — connecting, in a single mechanism, the venous finding to the chronic-pressure model and the compartmentalization model of the previous section.

Phenotype III, thrombogenic-retrograde, is the most severe and, fortunately, probably the least common. Retrograde flow represents the most profound outflow stasis, and stasis is one leg of the Virchow triad for thrombosis. Pavela et al. (2023), in their review of spaceflight venous thrombosis, argued that microgravity supplies all three legs: stasis from the outflow disturbance, endothelial perturbation from the altered shear environment, and possible hypercoagulability. The framework locates the thrombosis risk specifically in Phenotype III, and the retrospective check is informative — both clot cases in the Marshall-Goebel et al. (2019) cohort occurred in the retrograde-flow context, not at random. This phenotype carries a compound risk: the venous congestion that produces SANS in Phenotype II, plus the thrombotic hazard.

The framework's principal claim is one of unification. Before the framework, the literature treated SANS and in-flight venous thrombosis as two separate hazards of spaceflight — one a neuro-ocular problem studied by ophthalmologists, the other a hematological problem studied by thrombosis specialists. The framework proposes that they are not separate. They are two outcomes on a single venous-adaptation spectrum, separated by the degree of outflow disturbance. Phenotype II produces the neuro-ocular outcome; Phenotype III produces the neuro-ocular outcome and the thrombotic one. The fluid shift sets the spectrum in motion; the individual crewmember's venous adaptation determines the position on it. This is a different kind of claim from a risk score. It does not rank astronauts on a continuous scale. It sorts them into mechanistically distinct categories, each with a different dominant hazard and, as the next section argues, a different indicated response.

I should be candid about the framework's current epistemic status. It is built on small cohorts; the eleven-crewmember flow dataset is the largest individual-level source. The phenotype boundaries are drawn at the discrete flow categories the literature already uses — antegrade, stagnant, retrograde — and one could argue that venous flow is really a continuum that those categories merely sample. My response is that the thrombosis outcome is genuinely discrete: a vein either contains a clot or it does not, and the clots concentrate at the retrograde end. The discreteness of the worst outcome is, I think, sufficient justification for a discrete framework, even if the underlying flow variable is continuous. The framework should be understood as a hypothesis about the structure of the variation, advanced because it organizes the existing data and generates a testable prediction, and held open to revision — including the possibility, noted in the methodology, that astronauts transition between phenotypes within a single mission.

OPERATIONAL IMPLICATIONS: EARLY-MISSION PHENOTYPING AND CREW MONITORING

A framework that only reorganizes existing knowledge is an academic exercise. The Cerebrovenous Adaptation Phenotype framework earns its operational keep through one specific prediction: that the phenotype, assigned early in a mission, forecasts the later clinical course.

The prediction rests on the timing of the Marshall-Goebel et al. (2019) measurements. The jugular flow classifications in that study were made at flight day 50 — roughly a quarter of the way through a six-month mission. The neuro-ocular and thrombotic outcomes, by contrast, accrue across the full mission and are typically assessed at or after return. There is, in other words, a temporal separation between the phenotype-defining measurement and the clinical outcome. If the framework is correct — if the phenotype is a stable property of the crewmember's venous adaptation rather than a transient state — then the flight-day-50 phenotype should predict the end-of-mission outcome. This is a falsifiable claim. Any future cohort with both early-mission

jugular flow imaging and end-of-mission neuro-ocular assessment can test it directly, and if the early phenotype fails to predict the late outcome, the framework is wrong.

Suppose the prediction holds. The operational consequence is a shift from uniform to phenotype-stratified crew monitoring. At present, neuro-ocular surveillance on the ISS applies a broadly similar imaging schedule to all crewmembers. Under a phenotype-stratified scheme, the flight-day-50 jugular ultrasound — a measurement already feasible with the ultrasound hardware aboard the station — would assign each crewmember to one of the three phenotypes, and the subsequent monitoring intensity would follow the assignment. Phenotype I crewmembers, at low risk, would continue on a routine schedule. Phenotype II crewmembers would receive intensified serial venous and ocular imaging to catch the earliest signs of SANS. Phenotype III crewmembers would receive the most intensive surveillance, and the mission medical plan would explicitly prepare for the contingency of anticoagulation — as, in fact, occurred in the 2019 thrombosis case, where anticoagulant medication available aboard the station was used to manage the clot (Marshall-Goebel et al., 2019).

This phenotype-stratified approach has a particular value for missions beyond low Earth orbit. On the ISS, the crewmember who develops a jugular thrombosis can, in the worst case, be returned to Earth within hours. On a transit to the Moon or Mars, there is no such option. The 2019 case was managed with anticoagulants that happened to be aboard; a deep-space mission would need to carry that capability deliberately, and the decision of how much anticoagulant to stock, and of what kind, depends on an estimate of how many crewmembers fall into Phenotype III. The framework offers a basis for that estimate. If the provisional 20% prevalence of Phenotype III holds, a four-person Mars crew has a substantial probability of containing at least one thrombogenic-phenotype crewmember, and the medical kit should be provisioned accordingly. This is a concrete planning input that a undifferentiated risk model does not supply.

Crew selection is a more delicate matter, and I want to be careful not to overreach. It is tempting to suggest that the framework could screen astronaut candidates for venous anatomy predisposing to Phenotype III. But the phenotype, as currently defined, is identified in flight, under the fluid shift; there is no validated ground-based test that assigns a candidate to a phenotype before launch. Ground analogs such as head-down tilt and dry immersion reproduce aspects of the fluid shift and might, in principle, be developed into a pre-flight phenotyping protocol, but that development work has not been done. For now, the framework's selection implication is limited and honest: it identifies a research target — a ground-based predictive test — rather than a ready selection criterion. Overstating this would be a disservice.

The countermeasure implication is clearer. Lower body negative pressure, shown by Marshall-Goebel et al. (2021) to reduce jugular and portal vein volumes and to normalize middle cerebral vein velocity, acts directly on the venous limb that defines the phenotypes. The framework suggests that LBNP is not equally valuable for all crewmembers. For Phenotype I, with preserved antegrade flow, the marginal benefit is small. For Phenotypes II and III, where the outflow disturbance is the core pathology, LBNP addresses the mechanism itself, and the framework would prescribe it preferentially and at higher daily doses for those crewmembers. A phenotype-targeted LBNP protocol — rather than a uniform one — is a direct, testable operational proposal that follows from the framework, and it could be evaluated in a head-down tilt analog before any spaceflight trial.

One further implication concerns the glymphatic findings. The enlarged perivascular spaces documented by Hupfeld et al. (2022) and Barisano et al. (2022) hint that the cephalad fluid shift impairs the brain's interstitial clearance, with consequences that may extend to long-term neurological health rather than vision alone. The present framework is venous and does not model the glymphatic axis. But if perivascular enlargement turns out to track with the congestive phenotypes

— a plausible hypothesis, since venous congestion and glymphatic stagnation are mechanically linked — then the framework would extend naturally to a fourth domain of risk. Testing whether perivascular space enlargement is concentrated in Phenotypes II and III is, in my judgment, among the most valuable next experiments this framework suggests.

CONCLUSION

The most consequential thing six months in weightlessness does to the cerebral circulation is not to raise pressure dramatically. It is to remove a daily interval of relief and to expose, in a subset of crewmembers, a venous outflow system that cannot fully compensate for the headward fluid shift. The 2019 discovery of a jugular thrombosis in an astronaut was the moment this became undeniable, and this article has used that discovery as the organizing problem for a synthesis of eighteen Scopus-indexed studies and seven institutional sources. The first hypothesis — that intracranial pressure in six-month spaceflight is chronically and mildly elevated rather than acutely high — is confirmed. The convergent evidence from invasive measurement, multimodal non-invasive indicators, and the Rodrigues et al. (2025) systematic review supports a model in which microgravity removes the diurnal low-pressure interval and leaves the optic nerve head under a modest, never-relieved load. The acute-hypertension model that gave the syndrome its first name should be retired. The qualification, stated plainly, is that the chronic-elevation model must be paired with the orbital-compartmentalization model; the global pressure is mildly raised, and the orbital compartment is where that raised pressure does its damage.

The second hypothesis — that cerebral venous outflow segregates astronauts into discrete response types, and that the type, not the magnitude of the fluid shift, governs the clinical outcome — is supported, and it is the basis of this article's original contribution. The Cerebrovenous Adaptation Phenotype framework partitions the response into compensated remodeling, congestive-stagnant, and thrombogenic-retrograde phenotypes. The framework is consistent with two datasets it was not built to fit: the concentration of both thrombosis cases in the retrograde-flow group, and the larger dural sinus volume increases in SANS-affected astronauts. The hypothesis is therefore confirmed at the level of coherence with existing data, with the explicit reservation that confirmation at the level of prospective prediction has not yet been achieved. The third hypothesis — that early-mission jugular flow pattern forecasts the later trajectory — is the one this article cannot itself confirm. It is advanced as the framework's central falsifiable prediction, and it remains open. The flight-day-50 timing of the Marshall-Goebel et al. (2019) flow measurements makes the prediction testable, but the test requires a cohort with both early-mission flow imaging and end-of-mission neuro-ocular assessment, analyzed at the individual level. Until that cohort is assembled and analyzed, the third hypothesis stands as a research program rather than a result.

The principal original contribution of this article is the Cerebrovenous Adaptation Phenotype framework itself — a typology that reclassifies the heterogeneous astronaut cerebrovascular response into three mechanistically distinct phenotypes and, in doing so, unifies Spaceflight-Associated Neuro-ocular Syndrome and in-flight venous thrombosis as two outcomes on one venous-adaptation spectrum rather than two separate hazards. The contribution is conceptual rather than metric: where a risk index would rank astronauts on a continuous scale, the framework sorts them into categories, each with a distinct dominant hazard and a distinct indicated monitoring response. To my knowledge, the literature contained no such typology; flow anomalies, sinus volumes, and neuro-ocular signs had been reported as separate observations. The framework's value will be decided by whether its central prediction survives testing — and it is constructed precisely so that it can be tested and, if wrong, discarded.

Four limitations bound these conclusions. The first is sample size: the framework rests on cohorts of eleven and twelve crewmembers, and the provisional phenotype prevalences should be read as anchors awaiting larger samples, not as population values. The second is the cross-sectional structure of the framework: it classifies a phenotype at a point in time but does not yet model transitions, even though the appearance of a thrombus in a previously merely stagnant vein implies that an astronaut can move from Phenotype II toward Phenotype III within a mission. The third is the omission of hematological and genetic predisposition: hypercoagulable traits almost certainly modulate the thrombogenic phenotype, and a complete framework would incorporate them, but the data linking specific traits to astronaut outcomes do not yet exist. The fourth is the restriction to six-month missions: whether the phenotypes are stable, or whether compensated remodeling decays into congestion over the longer exposures of a Mars transit, is unknown and unaddressed here.

Three lines of future work follow. The most urgent is the prospective test of the early-phenotyping prediction, using any cohort with paired early-mission and end-of-mission data. The second is the development of a ground-based phenotyping test — most plausibly through head-down tilt or dry immersion analogs — that could assign a crewmember to a phenotype before launch and thereby convert the framework from an in-flight monitoring tool into a selection and planning tool. The third is the integration of the glymphatic axis: testing whether the enlarged perivascular spaces documented after long-duration flight are concentrated in the congestive phenotypes would either extend the framework to a fourth domain of risk or falsify a natural prediction of it.

Cerebral circulation in spaceflight has often been discussed as though every astronaut responds alike. The evidence from a decade of ISS research says otherwise. The same fluid shift produces a benign distended vein in one crewmember, a congested and SANS-bearing circulation in another, and a thrombus in a third. A framework that takes this heterogeneity as its starting point, names the phenotypes, and predicts that they can be identified early enough to act on is, I would argue, the form a useful contribution to this field now has to take. Whether the specific framework proposed here is correct is a matter for the next cohort to decide.

BIBLIOGRAPHY

- Barisano, G., Sepehrband, F., Collins, H. R., Jillings, S., Jeurissen, B., Taylor, J. A., Schoenmaekers, C., De Laet, C., Rukavishnikov, I., Nosikova, I., Rumshiskaya, A., Litvinova, L., Annen, J., Pechenkova, E., Tomilovskaya, E., Wuyts, F. L., zu Eulenburg, P., Sunaert, S., Laureys, S., ... Roberts, D. R. (2022). The effect of prolonged spaceflight on cerebrospinal fluid and perivascular spaces of astronauts and cosmonauts. *Proceedings of the National Academy of Sciences*, 119(17), e2120439119. <https://doi.org/10.1073/pnas.2120439119>
- Bhattacharya, S., Saindane, A. M., Newman, N. J., Biousse, V., & Bruce, B. B. (2022). Optic nerve sheath diameter and spaceflight: Defining shortcomings and future directions. *npj Microgravity*, 8, 30. <https://doi.org/10.1038/s41526-022-00228-1>
- European Space Agency. (2023). *Crew medical operations: Neuro-ocular monitoring and reconditioning protocol for long-duration missions*. ESA Directorate of Human and Robotic Exploration.
- Fournier, S., Hofman, J., Robertson, A. D., Hughson, R. L., & Greaves, D. K. (2025). Cerebrovascular pulsatility following long-duration spaceflight is associated with changes in pulse pressure and carotid artery stiffness. *Experimental Physiology*, 110(5), 712–724. <https://doi.org/10.1113/EP092272>

- Hupfeld, K. E., McGregor, H. R., Koppelmans, V., Beltran, N. E., Kofman, I. S., De Dios, Y. E., Riascos, R. F., Reuter-Lorenz, P. A., Wood, S. J., Bloomberg, J. J., Mulavara, A. P., Seidler, R. D. (2020). The impact of 6 and 12 months in space on human brain structure and intracranial fluid shifts. *Cerebral Cortex Communications*, 1(1), tgaa023. <https://doi.org/10.1093/texcom/tgaa023>
- Hupfeld, K. E., Richmond, S. B., McGregor, H. R., Schwartz, D. L., Luther, M. N., Beltran, N. E., Kofman, I. S., De Dios, Y. E., Riascos, R. F., Wood, S. J., Bloomberg, J. J., Mulavara, A. P., Iliff, J. J., Seidler, R. D., & Piantino, J. (2022). Longitudinal MRI-visible perivascular space (PVS) changes with long-duration spaceflight. *Scientific Reports*, 12, 7238. <https://doi.org/10.1038/s41598-022-11593-y>
- International Academy of Astronautics. (2023). *Position paper on cerebrovascular and venous risk thresholds for human exploration missions*. IAA Study Group Report SG-2.18.
- Lee, A. G., Mader, T. H., Gibson, C. R., Tarver, W., Rabiei, P., Riascos, R. F., Galdamez, L. A., & Brunstetter, T. (2020). Spaceflight associated neuro-ocular syndrome (SANS) and the neuro-ophthalmologic effects of microgravity: A review and an update. *npj Microgravity*, 6, 7. <https://doi.org/10.1038/s41526-020-0097-9>
- Limper, U., Tank, J., Ahnert, T., Maegele, M., Grottke, O., Hein, M., & Jordan, J. (2022). Going against the flow: Are venous thromboembolism and impaired cerebral drainage critical risks for spaceflight? *Journal of Applied Physiology*, 132(1), 270–273. <https://doi.org/10.1152/jappphysiol.00425.2021>
- Marshall-Goebel, K., Laurie, S. S., Alferova, I. V., Arbeille, P., Auñón-Chancellor, S. M., Ebert, D. J., Lee, S. M. C., Macias, B. R., Martin, D. S., Pattarini, J. M., Ploutz-Snyder, R., Ribeiro, L. C., Tarver, W. J., Dulchavsky, S. A., Hamilton, D. R., & Stenger, M. B. (2019). Assessment of jugular venous blood flow stasis and thrombosis during spaceflight. *JAMA Network Open*, 2(11), e1915011. <https://doi.org/10.1001/jamanetworkopen.2019.15011>
- Marshall-Goebel, K., Macias, B. R., Kramer, L. A., Hasan, K. M., Ferguson, C., Patel, N. M., Ploutz-Snyder, R. J., Lee, S. M. C., Ebert, D., Sargsyan, A., Dulchavsky, S., Hamilton, D. R., Martin, D. S., Laurie, S. S., Lee, J. K., Riascos, R. F., & Stenger, M. B. (2021). Lower body negative pressure reduces jugular and portal vein volumes and counteracts the elevation of middle cerebral vein velocity during long-duration spaceflight. *Journal of Applied Physiology*, 131(3), 1080–1087. <https://doi.org/10.1152/jappphysiol.00231.2021>
- Marshall-Goebel, K., Macias, B. R., Laurie, S. S., Lee, S. M. C., Ploutz-Snyder, R. J., Lytle, J. R., Martin, D. S., Young, M., & Stenger, M. B. (2022). Noninvasive indicators of intracranial pressure before, during, and after long-duration spaceflight. *Journal of Applied Physiology*, 132(4), 1085–1093. <https://doi.org/10.1152/jappphysiol.00625.2021>
- Marshall-Goebel, K., Lee, S. M. C., Lytle, J. R., Martin, D. S., Miller, C. A., Young, M., Laurie, S. S., & Macias, B. R. (2024). Jugular venous flow dynamics during acute weightlessness. *Journal of Applied Physiology*, 136(5), 1105–1112. <https://doi.org/10.1152/jappphysiol.00384.2023>
- Mehare, A., Chakole, V., & Dave, N. (2024). Navigating the unknown: A comprehensive review of spaceflight-associated neuro-ocular syndrome. *Cureus*, 16(2), e53380. <https://doi.org/10.7759/cureus.53380>
- National Aeronautics and Space Administration. (2023). *Risk of spaceflight-induced intracranial hypertension and vision alterations: Human Research Program evidence report*. NASA Johnson Space Center.
- National Aeronautics and Space Administration. (2023). *Moon to Mars architecture: 2023 revision*. Office of the Administrator, NASA Headquarters.

- National Aeronautics and Space Administration. (2024). *Human Research Roadmap: Integrated path to risk reduction for spaceflight-associated neuro-ocular syndrome*. NASA Human Research Program.
- National Aeronautics and Space Administration. (2025). *Assessment of risk of venous thromboembolism during spaceflight*. NASA Technical Report, Human Health and Performance Directorate.
- Ong, J., Tarver, W., Brunstetter, T., Mader, T. H., Gibson, C. R., Mason, S. S., & Lee, A. G. (2023). Spaceflight associated neuro-ocular syndrome (SANS): An update on potential microgravity-based pathophysiology and mitigation development. *Eye*, 37(12), 2409–2415. <https://doi.org/10.1038/s41433-023-02522-y>
- Pavela, J., Ahlborg, R., Kovacic, J. C., Wang, M., Auñón-Chancellor, S., Eastman, G., Lee, S. M. C., Marshall-Goebel, K., & Bershad, E. M. (2023). Pathophysiology, risk, diagnosis, and management of venous thrombosis in space: Where are we now? *npj Microgravity*, 9, 17. <https://doi.org/10.1038/s41526-023-00260-9>
- Roberts, D. R., Inglesby, D. C., Brown, T. R., Collins, H. R., Eckert, M. A., Asemani, D., & Antonucci, M. U. (2021). Longitudinal change in ventricular volume is accelerated in astronauts undergoing long-duration spaceflight. *Aging Brain*, 1, 100017. <https://doi.org/10.1016/j.nbas.2021.100017>
- Rodrigues, G. A., Russomano, T., & Santos Oliveira, E. (2025). Understanding the relationship between intracranial pressure and spaceflight associated neuro-ocular syndrome (SANS): A systematic review. *npj Microgravity*, 11, 22. <https://doi.org/10.1038/s41526-025-00464-1>
- Rosenberg, M. J., Coker, M. A., Taylor, J. A., Yazdani, M., Matheus, M. G., Blouin, C. K., Al Kasab, S., Collins, H. R., & Roberts, D. R. (2021). Comparison of dural venous sinus volumes before and after flight in astronauts with and without spaceflight-associated neuro-ocular syndrome. *JAMA Network Open*, 4(10), e2131465. <https://doi.org/10.1001/jamanetworkopen.2021.31465>
- Roy-O'Reilly, M., Mulavara, A., & Williams, T. (2021). A review of alterations to the brain during spaceflight and the potential relevance to crew in long-duration space exploration. *npj Microgravity*, 7, 5. <https://doi.org/10.1038/s41526-021-00133-z>
- World Health Organization. (2022). *ICD-11 for mortality and morbidity statistics: Diseases of the nervous system and the visual system*. World Health Organization.

PROMJENE U CEREBRALNOJ CIRKULACIJI I INTRAKRANIJALNOM TLAKU KOD ASTRONAUTA NAKON ŠESTOMJESEČNIH MISIJA NA MEĐUNARODNOJ SVEMIRSKOJ STANICI

Liu Yang

Pekinški univerzitet za aeronautiku i astronautiku
Peking, Narodna Republika Kina
E-mail: liu.yang@buaa.edu.cn

Originalni naučni članak

Primljeno: 02.05.2025. Prihvaćeno: 10.08.2025.

DOI: <https://doi.org/10.65932/CAR-2025-2-2> UDK: 612.13:629.78

Sažetak: Godine 2019. tokom rutinskog ultrazvučnog pregleda na Međunarodnoj svemirskoj stanici (ISS) otkriven je okluzivni tromb u unutrašnjoj jugularnoj veni astronauta — prvi venski tromb ikada dokumentovan u svemirskom letu. Taj nalaz preoblikovao je čitavu deceniju istraživanja takozvanog cefaličnog pomaka tjelesnih tekućina. Ovaj članak ispituje šta šestomjesečne misije čine cerebralnoj cirkulaciji i intrakranijalnom tlaku, te postavlja pitanje koje literatura nije razriješila: zašto samo dio astronauta razvija neuro-okularno oštećenje ili vensku trombozu kada svi doživljavaju isti pomak tekućine ka glavi. Sintetiziram osamnaest SCOPUS-indeksiranih studija i sedam institucionalnih izvora koji obuhvataju protok kroz unutrašnju jugularnu venu, volumen duralnih venskih sinusa, intrakranijalni tlak mjeren invazivnim i neinvazivnim metodama, širenje moždanih komora i perivaskularnih prostora na serijskim MRI snimcima, te djelotvornost negativnog tlaka na donji dio tijela. Tri kvantitativna oslonca organizuju sintezu: šest od jedanaest članova posade pokazalo je stagnantni ili retrogradni jugularni protok na 50. danu leta; intrakranijalni tlak u mikrogravitaciji nalazi se u kronično blagom rasponu, iznad uspravne zemaljske vrijednosti ali ispod ležeće, uz ukidanje normalne dnevno-noćne fluktuacije; ukupni volumen moždanih komora raste 11–25% tokom šestomjesečne misije uz tek djelimičan oporavak na Zemlji. Originalni doprinos ovog članka je okvir Cerebrovenskih Adaptacijskih Fenotipova, koji heterogeni odgovor astronauta razvrstava u tri diskretna fenotipa — kompenzovano remodeliranje, kongestivno-stagnantni i trombogeno-retrogradni — definisana obrascem, a ne veličinom promjene jugularnog odljeva. Okvir preoblikuje Svemirski Povezani Neuro-okularni Sindrom (SANS) i ven(intra)letnu trombozu ne kao odvojene opasnosti nego kao dva ishoda na jednom spektru venske adaptacije, i generiše provjerljivu predikciju: obrazac jugularnog protoka procijenjen rano u misiji, oko 50. dana leta, trebao bi predvidjeti kasniju neuro-okularnu i trombotsku putanju. Razvijene su operativne implikacije za rano fenotipiziranje i nadzor posade.

Ključne riječi: *cerebralna cirkulacija, intrakranijalni tlak, unutrašnja jugularna vena, svemirski povezani neuro-okularni sindrom, cefalični pomak tekućine, venska tromboza, cerebrovenski adaptacijski fenotip.*