

Title: Reducing Incapacitating Symptoms during Space Flight – Is Postural Deficiency Syndrome an Applicable Model?

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ABSTRACT

Severe and prolonged unmitigated SAS and SMS related symptoms have been thoroughly described in Astronauts during adaptation periods for orbital flight and post orbital flight. It has recently been shown that there is a strong correlation between these symptoms most often suffered by astronauts to that of the symptoms of patients suffering from Postural Deficiency Syndrome (PDS) on Earth that have been successfully assessed, diagnosed and treated.

International peer-reviewed literature identifies PDS as a trauma induced medical condition which originates from central neural dysregulation of sensory-motor and cognitive controls; these dysfunctions can be accurately identified, measured, and monitored via a specific ocular-vestibular-postural monitoring system along with relevant clinical data. This higher level of understanding is necessary in order to reach the next stage of success for humans living and working in Space.

Central sensory-motor and cognitive controls dysfunction underlie symptoms that can adversely impact and reflect alteration of eye-hand coordination, fine tuned dexterity, body positioning in space, space projection and trajectory control, perception of environment/obstacles, orientation in space and time, sensory motor and cognitive aspects of decision making, sensory-motor/cognitive error proneness. All of these

factors are necessary for Astronaut's mission capabilities, while both carrying out operations in Space and performing the tasks required during and after re-entry.

The objective of this paper is to elucidate how PDS related medical conditions are currently assessed, identified and monitored, and how these methodologies and technologies translate into a potential for better understanding of astronauts' potential incapacitation during space flight operations.

Introduction

In 1961, Soviet scientists were genuinely worried that any prolonged period of weightlessness might be fatal and therefore limited Yuri Gagarin's first space flight to a 108 minute single orbit [1]. Of the many health risks and problems facing astronauts during short and long duration missions, the long term possible harmful effects of weightlessness on the human body is of foremost concern. These effects include lower to upper body fluid shifts; cardiovascular and sensory-motor deconditioning; decrease of bone density, muscle mass and red blood cells; and, changes within the immune system [2]. Sensory-motor control deconditioning is one of the most critical effects to consider as it leads to deconditioning of posture and gait controls during long range missions.

Humans depend on a sophisticated sensory-motor system to sustain proper balance control. This system is calibrated to terrestrial gravity (G1) and relies on the application of gravitational forces to provide a frame of reference in order to supply a necessary data stream to central neural systems (CNS) located in the brain. Key motion sensors include subtle organs located in the vestibular system (inner ear) that function as ultra-sensitive accelerometers for motion and direction. The somatosensory system - which includes tension, pressure, and motion receptors located in the skin, muscles and joints - also assists in spatial awareness and perceptuality through proprioception, while our visual and auditory senses complete the spatial orientation system.

Adaptation to microgravity requires functional re-organization of a hierarchical central command located in the CNS. Three major sources of spatial information – visual, vestibular, and somatosensory are always used simultaneously according to precise neurophysiological paradigms [3]. Current experimental results support the hypothesis that absence of gravity leads to immediate and long term adaptive changes in neural strategies that are used for resolving ambiguous linear accelerations detected by the otolithic system [4]. In the absence of a gravitational reference frame, trivial visual references on Earth such as constant vertical and horizontal spatial references become vital for astronaut's orientation during space flight. Astronauts must rely primarily on visual input for spatial orientation. Thus, impairment of gaze and head stabilization reflexes can lead to overall perceptual distortion, disorientation and reduced performance in tasks relying on a high level of sensory-motor and sensory-cognitive skills, such as piloting a spacecraft or controlling a robotic arm.

Biomedical Concerns in respect to Incapacitation during Space Flight

In the absence of gravity, signals from the central vestibular system, peripheral pressure receptors, and visual sense become misleading, to such a point that immediate perceptual confusion and subsequent disorientation usually occur. Many astronauts suddenly feel as if they are upside-down or may even have difficulty sensing the location of their own arms and legs. This disorientation is described as Space Adaptation Syndrome (SAS) and is widely recognized as the main cause of Space Motion Sickness (SMS) [7]. About 70% of

astronauts will suffer from symptoms of SAS during the first few days of orbital flight [1]. Many astronauts maintain a local “subjective vertical reference” as shown by reports of inversion illusions and visual reorientation illusions. Instability of this “subjective vertical reference” in Microgravity is thought to be a specific trigger for SMS onset [5]. One classical example of SMS is that of cosmonaut Titov in the second manned Soviet mission in 1962 [6]. For a brief period immediately after transition into orbit, Titov felt that he was flying upside down, soon thereafter he described dizziness associated with head movements, and approximately six hours into flight he exhibited motion sickness, the first recorded instance in space flight [1].

The most incapacitating effects of SAS last from the first to the fifth day of weightlessness, and reoccur within the first ten days after landing [7]. Commonly recorded symptoms include dizziness, vertigo, headaches, cold sweating, fatigue, nausea and vomiting (motion sickness) [7]. Consequences may range from simple discomfort to incapacitation that may create potential problems during re-entry and emergency exits from a spacecraft. It is for this reason that no extra-vehicular activities (EVAs), or space-walks, are permitted during the first few days of NASA shuttle flights [7]. A literature review previously published [8] provides a list of the most common symptoms (Table I).

TABLE I: SAS/PFAS

General Signs	SAS/PF AS	Symptoms
Pain	SAS	Headaches, vomiting, digestive spasms
Imbalance (Vestibular Cerebellar)	SAS/PF &AS	Motion sickness, nausea, dizziness, inexplicable falls, poor concentration postural equilibrium disturbance, faintness, illusions and alterations of motor performance such as feelings of heaviness, disorientation when making sudden head movements, inability to move about in the dark, illusions of floor motion during vertical body movements
Neuro- ophthalmologic Coordination	SAS/PF AS	Eye-hand, eye-body, eye-head coordination impairment, postural equilibrium disturbance, disorientation when making sudden head movements, inability to move about in the dark, illusions of floor motion during vertical body movements, illusory sense of surroundings
Proprioceptive	SAS/PF AS	Illusory sense of self, eye-head, eye-hand coordination impairment, postural equilibrium disturbance, dizziness, nausea, illusions and alterations of motor performance such as feelings of heaviness, disorientation when moving suddenly, inability to move about in the dark, illusions of floor motion during vertical body movements.

Articular	SAS/PF AS	Postural equilibrium disturbance, illusions and alterations of motor performance such as feelings of heaviness, limitation in extension amplitude
Neuromuscular	SAS/PF AS	Headaches, eye-head and eye-hand coordination impairment, postural equilibrium disturbance, nausea, illusions and alterations of motor performance such as feelings of heaviness, disorientation when making sudden movements
Neurovascular	SAS/PF AS	Headaches, postural equilibrium disturbance, faintness, dizziness, nausea
Autonomic Neuro-vegetative	SAS	Cold sweating, chills, paleness, dermal goose-bumps

Current research favors a sensory conflict theory as the primary cause of SAS, which can lead to SMS [1]. Conflicting sensory-motor control inputs from visual and tactile senses with inputs coming from the vestibular organs in the inner ear are likely. However, the precise mechanisms by which these conflicts occur are not well-understood and effective therapies or preventative measures for SAS have yet to be developed. Interestingly, symptoms of SAS have not been shown to be reduced on veteran astronauts during subsequent flights [7].

Review of Current Countermeasures

Pharmaceutical

Medications used for treating terrestrial motion sickness; typically scopolamine-dextroamphetamine sulphate (Dexedrine) or promethazine-ephedrine combinations are currently the only method for preventing and treating SMS. These prescription drugs have many adverse side-effects [9], can be highly addictive and are not consistently effective in the treatment or prevention of SMS. All other symptoms of SAS are managed by restricting crew activities in the first few days of space flight.

The following was taken from the *Public Health Agency of Canada's* "Statement on Motion Sickness":

“Common adverse effects of Dexedrine include restlessness, talkativeness and over-stimulation. Other effects that have been observed include changes in sex drive, constipation, diarrhea, dizziness, dry mouth, exaggerated feeling of well-being or depression, headache, heart palpitations, high blood pressure, hives, impotence, loss of appetite, rapid heartbeat, sleeplessness, stomach and intestinal disturbances, tremors, uncontrollable twitching or jerking, unpleasant taste in the mouth, and weight loss. Chronic use may lead to hyperactivity, irritability, personality changes, schizophrenia-

like thoughts and behavior, severe insomnia and skin disease.

Scopolamine hydrochloride causes dry mouth, drowsiness and blurred vision, and there is concern that it may in fact decrease adaptation abilities to motion sickness. Visual problems may increase with continuous use, and can cause confusing states and/or visual hallucinations.

Common adverse effects of using promethazine are severe drowsiness, significant decreases in performance scores, psychomotor function, information processing and alertness. The manufacturer of promethazine describes it as possibly having less impairment than that attributable to the motion sickness itself.”¹

Aside from the undesirable side-effects of motion-sickness drugs, it was shown as early as the Skylab missions that these drugs are relatively ineffective in preventing or treating SMS or any other symptoms of SAS. For example, the Skylab 2 crew carried and took a scopolamine and d-amphetamine combination and was symptom free during the first couple of days of flight. However, the scientist pilot exhibited a slight increase in subjective body warmth on the twentieth day of flight, and cold sweats on day twenty-four. Also on day twenty-four of flight, the pilot reported epigastric awareness, increased body warmth, slight dizziness and cold sweating [10, 16]. Skylab 3 crewmembers carried the same drugs as the Skylab 2 crew; however, they did not take them before flight. The pilot experienced mild symptoms of motion sickness within one hour of insertion into orbit. He took some medication that alleviated his symptoms, although after a few hours his symptoms returned restricting his activities for the day. At eleven hours into the flight, the commander and scientist pilot also reported the onset of motion sickness, shortly afterwards the scientist pilot vomited. Recovery from SMS did not occur until the seventh day of flight [10, 16]. Skylab 4 crewmembers took medication before flight as a precaution, however the pilot experienced nausea and vomiting immediately after insertion into orbit and was not symptom free until after the third day. The commander reported epigastric awareness prior to meals, possibly indicating susceptibility to motion sickness. The scientist pilot remained symptom free. NASA concluded that the drug combinations “were not the ideal anti-motion sickness drugs” [16].

In addition, amphetamines also have a high likelihood of addiction and dependence with prolonged periods of use. In order for astronauts to be effectively cured of SMS, it is clearly desirable to find alternatives to counter such unacceptable effects during Space missions.

¹Committee to Advise on Tropical Medicine and Travel (CATMAT) – Public Health Agency of Canada, Statement on Motion Sickness, Volume 22-13, p.3, July 1st 1996

Physical Countermeasures

Other “traditional” countermeasures against the adverse effects of weightlessness such as exercise; resistive garments and low-body negative pressure (LBNP) appear to be insufficient in practice as they are often too inconvenient for astronauts [10].

Tactile information such as pressure on the soles of the feet can continue to promote a vertical sense. This was shown previously on two cosmonauts during a 196 day MIR mission. The results of this particular study indicated that the application of foot pressure throughout the course of a long duration spaceflight effectively increases neuromuscular activation, suggesting that it may prove useful to explore more sophisticated forms of delivering foot pressure during spaceflight as a countermeasure to SAS/SMS [11]. However, this alone would not be enough as pressure stimulation strictly stimulates the proprioceptive sensory system, and does not address other physiological and cognitive related symptoms being faced by astronauts.

Artificial gravity induced by centrifugation has also been widely studied on Earth as a viable countermeasure. Much of this work, pioneered by Dr. Young [15], has shown strong evidence for positive effects in regards to muscle tonus, cardiovascular deconditioning and bone degeneration. However, while it has been shown that proper centrifugation has little to no adverse effect on vestibular functions, there is little evidence to suggest that it would permanently correct vestibular and proprioceptive disturbances during space flight. It is also unclear as to what effects an astronaut may incur when being subjected to only short, multiple periods of this kind of centrifugation during long duration space flight.

Autogenic feedback training (AFT) is another possible preventative measure against terrestrial and space motion sickness. AFT is designed to sensitize the individual to bodily sensations such as heart rate, respiration, skin temperature and utilize this as feedback to control SMS. Through such training, an individual is to gain a higher level of resistance to motion sickness. A comprehensive literature review recently conducted by Lackner and DiZio [1] found that AFT appears unlikely to have any preventive qualities for space motion sickness.

Sensory-Motor Controls and Adaptation to Microgravity

Correct transduction and integration of signals from all areas of the sensory-motor system is essential for maintenance of stable vision, spatial orientation, and eye-head and hand coordination and postural and locomotion control on Earth. Perception of location and positions are a result of the brain’s ability to integrate visual and auditory signals with vestibular input and proprioceptive information. The input the brain receives from sensors, modified by gravity changes, prompts the CNS to develop a new interpretation, hence a different adjustment strategy. The plasticity of the CNS allows individuals to adapt and compensate under altered sensory stimulus conditions such as those experienced in space flight. In microgravity the suppression of sensory stimulus rearranges the relationship between signals from vestibular, visual, skin, muscle, and membrane and joint receptors. If

this new interpretation does not match fundamental specific calibrated functional patterns in the brain, symptoms of SAS are likely to occur, significantly reducing astronaut's operational efficiency [12].

Vestibular Controls

The peripheral vestibular apparatus in the inner ear consists of two sensory receptors, the semi-circular canals and the otolithic organs. The semicircular canals signal rotary movements of the head. These liquid filled tubular loops act as angular accelerometers arranged in three orthogonal planes. The change in volume of each loop translates movement to neural signals to the brain. The otolithic organs sense linear forces such as gravity acting on the head. These calcium carbonate concretions embedded in gelatinous material act as linear accelerometers for the brain. Neural signals produced under acceleration are integrated in the CNS with signals from proprioceptors reporting the position relationships of the limbs, trunk and neck. Signals from skin pressure receptors, vision and stored cognitive perceptual memory data, are integrated to coordinate movements of the limbs, head and eyes.

Postural reflexes under otolithic control appear to be depressed in flight and return to normal only after several days of recovery post-flight. These symptoms occur even after missions of relatively short duration, where changes in bone and muscle strength are minimal [13]. One possible reason for reaction to weightlessness is described in the Otolith Tilt Translation Reinterpretation (OTTR) hypothesis [7]. It states that the brain learns to reinterpret signals coming from the vestibular system to represent only linear acceleration rather than pitch or roll of the head [14]. Another explanation of these observations considers a gain on otolithic signals that may be reduced, thus leading to a decrease in the ability to sense linear acceleration [18]. Therefore, confusing signals from the inner ear become largely ignored and vision returns as the primary source of posture and gait information [7].

Postural Control – The Fine Postural System

An important function in postural control is the coordination of various muscular activities to maintain proper orientation of the body with respect to gravity. It is provided by a complex regulatory system, the Fine Postural System (FPS). The FPS models the core neurophysiological system that provides balance control to the human body during static and dynamic performance. Known components of the FPS include ocular-motor abilities and vision, vestibular system, proprioception of the lower limbs as well as of the paravertebral muscles and of the eye movements.

The sensory-motor controls, which underlie the FPS, handle through the CNS hierarchy sensory input along with directing motor adjustment and movement throughout the body. This system governs adjustments of postural neuromuscular tonus, vestibular control, and visual-ocular motor control. Hence, the FPS regulates postural tone and precisely adjusts

posture and gait under all circumstances, whether static or dynamic. Its functional core structure includes synergistic and antagonistic sensory motor and cognitive structures that are part of a network diffusely located in various areas of the brain [15].

In summary, the structure of the FPS consists of two main pools of sensors conventionally described as “exosensors” and “endosensors” [19]. These components only control small (fine) postural disturbances that move the body’s axis up to four degrees from the balance reference in a given position. Research has shown that postural balance depends on whether the amplitude of spontaneous movements to or from the balance position (not necessarily vertical) is greater or less than four degrees [16].

Cardiovascular Control

Upon return from spaceflight many astronauts are unable to tolerate upright posture without experiencing symptoms related to fainting. Such a phenomenon may not be explained through postural deficiency *stricto sensu*. The high degree of orthostatic intolerance (OI) observed in astronauts after spaceflight indicates that cardiovascular regulation is altered. Early spaceflight data, as well as studies utilizing 6° head-down tilt bed rest (HDBR) to simulate weightlessness, have shown reduced plasma volume and decreased baroreflex response, post-exposure [17, 18, 19, 20, 21, 22, 23, 24, 25]. The most recent studies, evidence indicates that pre-flight physiology may also play an important role [11, 26, 27, 28] in altered autonomic cardiovascular control with a reduction in parasympathetic regulation as well as possible changes in sympathetic cardiovascular regulation following real or simulated spaceflight.

Investigations of the cardiovascular differences between astronauts who did, or did not, finish a post-flight stand test has revealed pre-flight differences. Compared to finishers, non-finishers had higher parasympathetic tone [32], lower supine and standing peripheral vascular resistance [31] and systolic blood pressure [30, 31] as well as lower standing diastolic blood pressure [31]. Post-flight, finishers had higher catecholamine levels and vasoconstrictor response to standing compared to non-finishers [30, 31]. Although Fritch-Yelle et al. [31] concluded that post-flight presyncope might be due to centrally mediated hypoadrenergic responsiveness; this has recently been challenged [29]. Based on an investigation into the mechanisms leading to syncope in healthy individuals, Evans et al. [33] suggested that presyncopal astronauts might have had similar or higher rates of norepinephrine release since the accumulation time for norepinephrine was shorter due to termination of the stand test. These results indicate that the effect of spaceflight on autonomic responses to post-flight orthostatic stress and the effect pre-flight autonomic status on post-flight OI is not well understood. In a previous study,

A Possible Ground Based Model

Review of Ground-Based Models

Microgravity has offered a unique opportunity to study the role of the vestibular organs as

well as the adaptive mechanisms the brain uses to adjust for altered forces and motion environments. This opportunity was grasped during the 1998 Neurolab (STS-90), where the study of the human vestibular system in Space was a major focus [15].

In the late 1990's, Speers et al. [17] took data from ten astronauts (nine male, one female, mean age of thirty-eight years) who were selected and subjected to a 'Sensory Organization Test' (SOT). They found that changes in postural control following spaceflight are multivariate in nature indicating not only a change in the amount of sway, but change in astronaut coordination as well. Because the coordinative changes varied with sensory conditions, it was shown that they are at least partially explained by changes in sensory processing, which may affect astronaut's perception of spatial orientation. Post-flight testing showed noticeably increased sway, and performance was observed to be much worse when the astronauts were tested with their eyes closed. Their results implied that the contributions of a multivariate combination of somatosensory, visual, and vestibular information for postural control are altered following spaceflight. The multivariate nature of postural control requires a treatment that addresses vestibular function in combination with somatosensory (proprioception), and visual function. To further reinforce the multivariate conclusions of Speers, Paloski and Kuo [17], Bernard Cohen et al. [48] recorded data from four astronauts who were exposed to interaural and head vertical (dorsoventral) linear accelerations on a centrifuge on Earth and in-flight on Neurolab. Their results suggest that a combination of other non-vestibular inputs, including an internal estimate of the body vertical and somatic sensation, were utilized in generating tilt perception [30]

The authors previously hypothesized [8] that since astronauts suffer from a syndrome that alters a multivariate combination of ocular-visual and postural equilibrium along with proprioceptive information, then SAS and PFAS may be of a similar nature to that of PDS. They presented a clinical study of 59 PDS patients and correlated their symptoms to that of the most common symptoms associated with SAS and PFAS. They showed that SAS/PFAS symptomatology meets the definition of PDS, and correlates with that of PDS patients, giving rise to a medical diagnosis as a plausible ground-based model for SAS/PFAS that should be investigated in future research.

Postural Deficiency Syndrome

Postural Deficiency Syndrome (PDS) is conventionally described as a medical condition that includes a composite of symptoms in relationship with variations of the upright position [31]. PDS labeled patients acknowledge, among other signs, balance disorders, postural instability, sensory and cognitive overloads, hypersensitivity, dizziness, pains radiating from or to their body axis, circulatory disorders. As defined, PDS does not correspond to any macroscopic lesion of an anatomically defined system. The severities of PDS symptoms have no apparent relationship to that of the known cause [32, 33].

Table 2 shows the "cardinal" neuromuscular and neurovascular functional signs and other characteristics of symptoms of patients suffering from PDS as defined by Da Cunha in

1987 [38] and more recently by Quercia et al in 2005 [34]. Such signs are conventionally used in trauma medicine to diagnose PDS in clinical posture-related practices.

Table 2: PDS

Signs	Clinical manifestations
Pain	Headache, retro-ocular, thoracic or abdominal, joints and paraspinal
Imbalance	Sickness, nausea, dizziness, inexplicable falls
Ophthalmological	Asthenopia, dim vision, diplopia, directional scotoma, metatopsia
Proprioceptive	Dysmetria, somatoagnosia, errors of appreciation of the body image
Articular	TMJ Syndrome, stiff neck, lumbago, periarthrities, sprains
Neuromuscular	Paresia, defect of driving control of the extremities
Neurovascular	Paresthesia of the extremities, Raynaud's phenomenon
Cardiovascular	Tachycardia, lipothymia
Respiratory	Dyspnoea, fatigue
Otorhinolaryngological	Humming, tinnitus, deafness
Cognitive	Dyslexia, dysgraphia, agoraphobia, defect of orientation, defect of spatial localization right and left, defect of concentration, loss of memory, asthenia, anxiety, depression

Comparison to Astronaut Symptomatology

Prior to Skylab missions where the human physiology in Space first began to be seriously studied, four USSR crewmembers and nine out of the twenty-five Apollo astronauts experienced Space motion-sickness. None of the twelve Apollo lunar astronauts experienced motion sickness, nor did astronauts from the Mercury and Gemini programs reported occurrences of motion sickness. However, other sensory-motor control dysfunctions were reported far more frequently than motion sickness, and postural illusions were experienced immediately after transition into orbit by nearly all astronauts and cosmonauts [7]. In fact, some cosmonauts continued to experience illusions until the g-load that was associated with re-entry appeared. Such illusions induced by rotary motions of the head and/or body movement (sensations of turning and dizziness) were experienced not only in early flight but also recorded over prolonged periods of time [7]. One should note that Mercury and Gemini astronauts were severely restricted in body and head movement due to the small size of the capsules they flew in, and by the helmets that they wore.

Pre-flight and post-flight testing from Apollo 16 crewmen indicated some decrement in postural equilibrium three days following recovery when they were tested with their eyes closed [7].

Crewmen of the eighteen-day Soyuz 9 mission manifested difficulty in maintaining a stable vertical posture that did not normalize until 10 days after flight [7].

Although Skylab crewmen were able to walk immediately after exiting the command module, they did so with noticeable difficulty, tending to use a wide-stance shuffling gait with the upper torso bent slightly forward [10]. During the first several days following post-flight recovery, crew reported the simple act of walking required a conscious effort.

All crewmen reported that rapid head movements post-flight produced a sensation of mild vertigo and any slight head movement while their eyes were closed would induce vertigo and cause loss of balance. NASA Skylab flight surgeons and scientists concluded that Skylab crewmen required about ten days in order to regain their normal postural stability [10].

The most overt change that affects astronauts in space flight, according to Charles Oman, is the immediate response of the neurovestibular area of the sensory-motor control system to changes in gravity level [16]. MIR crewmembers have been recorded as saying that 3D relationships between modules, particularly those with different visual verticals, are difficult to visualize. While crewmembers are able to learn routes, their noticeable lack of direction and surveying abilities is a major concern should fire, power-loss, or depressurization limit their visibility in any way. As the International Space Station, future interplanetary missions, and particularly private industry space travel grow in duration the likelihood of such a situation will continue to grow exponentially.

Orthostatic Syndrome

The primary cause of orthostatic intolerance is a failure to maintain arterial blood pressure that in turn causes a reduction in blood flow in the brain. Although the possibility exists for altered cerebral vascular control, recent evidence suggests that cerebral auto regulation is not affected by space flight [35]. Several other factors can be involved that modify either cardiac output or peripheral vascular resistance. Space flight causes reduced cardiac function and altered heart rate responses, a significant reduction in total blood volume, and possible changes in leg, splanchnic or central vein compliance with blood pooling. Each of these could contribute to the exacerbation of the reduction in cardiac output that occurs on standing [36]. Most current and past research has focused on the interaction of these cardiovascular control factors in relation to post-flight orthostatic intolerance. Most recently, Blaber et al. [11], investigated heart rate variability (HRV) at rest in the supine position and in response to upright posture in 29 astronauts before and after spaceflights lasting up to 16 days. Individuals were separated according to their ability to remain standing without assistance for ten minutes on landing day. Both finishers and non-finishers were found to have an increase in sympathetic activity with stand pre-flight, however; only finishers retained this response post-flight. Pre-flight autonomic responses that might separate finishers and non-finishers were identified. Both groups of astronauts had the expected shift to greater sympathetic over parasympathetic regulation (increased sympathovagal balance, PLO/PHI) of cardiac function from supine to stand on all test days. However, non-finishers had lower values than finishers. That this was unaffected by spaceflight suggests the relationship between sympathetic and parasympathetic modulation

of heart rate was preserved in both groups following short duration spaceflight. Compared to finishers, non-finishers had higher parasympathetic activity.

The mechanism by which higher pre-flight parasympathetic activity might contribute to post-flight orthostatic intolerance is not understood and needs to be investigated further. The interaction of the cardiovascular and postural control centers, particularly in relation to the importance of the skeletal muscle pump in the maintenance of venous return in conditions of impaired vascular control or increased venous pooling has not yet been studied thoroughly. Also, most changes associated with incapacitating SMS have been linked to elevated activity in the sympathetic nervous system, although the influence on heart rate remains uncertain [1]. In our model, modified from Lackner and DiZio [1] and recently presented at [37], we propose that there is a significant relationship between cardiovascular and postural controls [38] which is adversely affected during Space flight. Just as motion sickness and postural disequilibrium have been postulated to be linked and associated with SAS [1], changes in the interaction of postural adjustments and baroreflex controls in post-flight that would lead to reduced venous return and orthostatic tolerance.

Conclusions

This review suggests a new biomedical approach to better understand and more precisely assess central neurophysiological mechanisms and controls underlying incapacitation symptoms during Space flight as seen in SAS, SMS and PFAS. Subsequently, it is possible to mitigate these conditions based on specific neurophysiological assessment criteria, focused on sensory-motor and sensory cognitive controls functional status.

Postural Deficiency Syndrome reflects key central neuroregulatory paradigms. Related assessment and therapeutic methodologies, proven effective in specialized neurotraumatology for two decades, can be easily applied to identify and measure functional sensory-motor and sensory cognitive controls status in specific brain areas during pre-flight, in-flight and post-flight examinations.

In addition, combined with dynamic analysis of the cardiovascular system, it is hypothesized by the authors that this model has the potential to provide key answers to why, how and when SMS related incapacitating symptoms occur as well as to lead to criteria regarding susceptibility to SMS.

The validity of this hypothesis is strengthened by the strong correlation between unmitigated SMS/PFAS and related symptoms in Astronauts as it has been previously published. There is also strong correlation with PDS related symptoms, which can be accurately identified, measured, and monitored via a specific ocular-vestibular-postural monitoring system along with relevant clinical data. The data presented provides strong evidence that advanced biomedical assessment methodologies employed with appropriate technology can lead to better understanding Astronauts' pre-flight and post-flight

biomedical status, necessary to further human exploration in Space on a safe and successful path.

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