Abstract — Optimal human performance depends upon integrated sensorimotor and cognitive functions, both of which are known to be exquisitely sensitive to loss of sleep. Under the microgravity conditions of space flight, adaptation of both sensorimotor (especially vestibular) and cognitive functions (especially orientation) must occur quickly—and be maintained—despite any concurrent disruptions of sleep that may be caused by microgravity itself, or by the uncomfortable sleeping conditions of the spacecraft. It is the three-way interaction between sleep quality, general work efficiency, and sensorimotor integration that is the subject of this paper and the focus of new work in our laboratory. To record sleep under field conditions including microgravity, we utilize a novel system called the Nightcap that we have developed and extensively tested on normal and sleep-disordered subjects. To perturb the vestibular system in ground-based studies, we utilize a variety of experimental conditions including optokinetic stimulation and both minifying and reversing oculovestibular paradigms that have been extensively studied in relation to plasticity of the vestibulo-ocular reflex. Using these techniques we will test the hypothesis that vestibular adaptation both provokes and is enhanced by REM sleep under both ground-based and space conditions. In this paper we describe preliminary results of some of our studies. © 1998 Elsevier Science Inc.

Keywords — sleep; vestibular adaptation; REM sleep; microgravity.

Introduction

The functions of sleep remain unknown. The compelling idea that sleep subserves neuronal plasticity was first clearly articulated by Giuseppe Moruzzi (1). This hypothesis has been tested in many ways, including the systematic perturbation of the vestibular system. The close interrelationship between the vestibular nuclei and the sleep inducing structures of the pontine brain stem has been extensively studied by Moruzzi’s colleagues, especially Ottavio Pompeiano (2). It is the purpose of this paper to discuss the confluence of these two lines of thought and research in a new program of work on human sleep in space and to present the results of the first studies conducted within this paradigm.

The basic idea is that the sudden exposure to microgravity conditions of space flight functionally decouples the vestibular otolith organ receptors and eliminates tonic and phasic microgravity postural adjustments. This compels the organism to reprogram the vestibular system so that it can work in an exclusively inertial mode. This monumental reorganization constitutes a natural challenge to the plasticity of the brain. If sleep—and especially REM sleep—is involved in enhancing plasticity, it would be desirable to monitor changes of sleep in space. To this end, we have designed the Nightcap, a simple, portable, and self applied measurement system which
is compatible with the behavioral and operational constraints of space flight (3).

In this paper, we review the rationale for selecting the vestibular adaptation model and the evidence concerning its effects on sleep under normal gravity conditions. Then we show how the Nightcap could be used to assess the effects on sleep of sudden and prolonged exposure to microgravity and compare this approach to the traditional sleep laboratory methods that have been used until now in attempts to document sleep in space.

This promising approach is in an early stage of development. To indicate how useful it may be, we present some preliminary data from a ground-based experiment in which subjects slept after exposure to optokinetic stimulation in a rotating drum. Its description here is offered in the spirit of Giuseppe Moruzzi's visionary approach to the study of sleep function. When he first proposed his plasticity hypothesis, he could not have dreamed that it might soon be tested in human beings hurtling through space.

A Conceptual Framework

In the ultimate paradigm of our interest, a human being is exposed to the microgravity conditions of space. This immediately provokes vestibular reprogramming, a process which may affect both sleep (via changes in the excitability of pontine sleep control neurones) and psychomotor learning that is essential to adaptation to space may then both alter and be altered by sleep (since plasticity is both mediated and modulated by the same neurones that are critical in determining the absolute and relative amounts of NREM and REM sleep).

The distinction between these two phases of sleep is best appreciated by recognizing that during REM, the EEG and motor pattern generators of the upper brain are reactivated to waking levels, but motor output is blocked by active inhibition of all motoneurones except those which move the eyes.

The three main phenomena of interest in this paper and the pairwise links between them are illustrated in Figure 1. We will argue that there is a strong tie between the vestibular system and sleep on the one hand and between sleep and performance on the other.

The Vestibular System and Microgravity

It is obvious that all normal sensorimotor integration depends upon the vestibular system. Whenever the head moves, the eyes must be appropriately repositioned to maintain fixation on a target or to fixate on a new one. This vestibulo-ocular reflex (VOR) was first described by Lorente de No (4) and has been extensively studied (5). One robust feature of the VOR is its plasticity (6). When subjects are exposed to novel conditions (for example, microgravity or distortions of visual input), the VOR must be recalibrated. This adaptation can take hours or days and is learned with variable speed by different experimental measurement via a target acquisi-

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Figure 1. The three main phenomena of interest in this paper and the pairwise links between them.
tion task is a potent paradigm for measuring adaptation to microgravity.

It is not surprising that astronauts commonly experience dysfunctional states upon exposure to microgravity and require extensive training and variable periods of adaptation to acquire comfort and psychomotor competence in microgravity. Motion sickness, nausea, and malaise commonly hinder performance during the early days of space flights, even in carefully selected and highly trained subjects. It is typical for astronauts to treat these symptoms with promethazine, but it is not known whether this drug im-

Even when vestibulo-cerebellar adaptation has occurred, astronauts may experience disorientation of an explicit type (for example, unwittingly viewing an instrument panel upside-down resulting in left-right confusion). For this reason, major portions of the physiological research effort of the manned space flight program have been appropriately dedicated to understanding and countering these effects. We intend to link our studies to this knowledge base and to build upon it.

The Vestibular System and Sleep

Located in the pontine brainstem, the vestibular nuclei are adjacent to and integral with the reticular neuronal machinery that controls the human sleep-wake cycle (2,4,7). The vestibular nuclei receive inputs encoding head position from the vestibular end organ in the labyrinth, and they integrate them with signals encoding limb and trunk position from peripheral proprioceptors in the limbs and trunk. The vestibular neurones are also reciprocally innervated by the oculomotor neurones (cranial nerves III, IV, VI) and the pontine reticular pre-motor neurones. These in turn control them and initiate appropriate postural adjustments when reflex conditions or task demands change (see the schematic model in Figure 2). Other chapters in this volume document the link between the vestibular nuclei and the autonomic nervous system.

This massive brain-stem integrative network could be said to be the body's own navigational system. As we all know from our own everyday experience with sleep, the network is significantly state-dependent: its sensitivity, gain, and operating properties change with changes in central state. A commonplace example is the relief of motion sickness by drowsiness and sleep. Indeed sleep is so often experienced as a response to motion sickness as to suggest that it may be an integral part of adaptation to sudden changes in gravitational vectors.

The state dependent changes in the integrative network of the brainstem are regulated by three well-known processes. One is simple disfacilitation of the reticular system. This reduces

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navigational network. We resist the temptation to digress here to a literature review of the many bases of this hypothesis and simply point out that (1) many new learning challenges entail increases in REM and are impaired by REM deprivation, and (2) new learning challenges that involve vestibular adaptation may be particularly REM-sleep evocative and REM-sleep loss sensitive. As we indicate below, it is now feasible to track vestibular adaptation and sleep in parallel under the prolonged microgravity conditions.

A final point closes this section. REM sleep can be immediately triggered (in the short term) by cholinergic microstimulation of the pontine reticular formation. In addition, the threshold for REM sleep can be lowered (in the long term) by cholinergic microstimulation of the caudal peribrachial complex in immediate proximity to the vestibular complex (see Figure 2). These findings indicate (1) that acute vestibular adaptation may be (in part) REM-sleep mediated and (2) that the REM sleep mediation of vestibular plasticity may be (in part) cholinergically modulated. It thus follows that pharmacological or behavioral interventions that enhance REM sleep might enhance vestibular adaptation. Indeed, it is our understanding that the anticholinergic agent scopolamine (which would be expected to block REM) has not proven useful in helping humans adapt to space. To the best of our knowledge, the REM sleep–vestibular adaptation link that we propose to study has not yet been made explicit in space physiology and medicine. We hope to correct this oversight by testing several novel and important hypotheses.

Sleep and Psychomotor Performance

As anyone who has ever experienced a night of impaired sleep knows, the ability to attend to,
Sleep and Vestibular Adaptation

process, and retain information is adversely affected by poor sleep. The literature on this subject, pioneered by Wilkinson's group in the Human Performance Laboratory in Cambridge, England, has been extensively confirmed by subsequent research (10).

Recently a more exciting proposal has been advanced: not only does sleep enhance performance by preventing attentional lapses (a protective function), it actually serves to promote the retention or consolidation of previously learned material (a conservative function). This second, stronger form of the theory is related to the hypothesis that vestibular-mediated plasticity alters (and is altered by) REM sleep.

Sleep, Dreams, and Vestibular Functions

One of the most remarkable features of dreams is the sense of weightlessness that accompanies oneiric flight. This unusual dream sensation is so pleasurable that it is often sought by practitioners of lucid dreaming. But it has also prompted speculation regarding its possible physiological basis in the altered neurophysiology of REM sleep. In this section, we discuss data linking sleep and dreaming to vestibular function.

Sleep Responses to Vestibular Learning with Distorting Glasses

When an individual turns his head, his eyes automatically move in the opposite direction to maintain stability of the visual field. This corrective ocular movement is under the control of the vestibular system and is known as the vestibulo-ocular reflex (VOR). Under various conditions, these neuronal networks can learn to behave differently. One of the best studied paradigms of vestibular learning involves the use of distorting glasses (see reference 5 for a review). These glasses contain lenses that reverse left and right, reverse up and down, magnify or "minify" (reduce to a smaller size) the retinal image of visual space.

In consultation with Dr. Jacob Bloomberg and Dr. James Lackner, we have confirmed our previous section. It is supported by the preliminary findings of Karni and Sagi (11,12), which indicate that new visual discriminative learning is retained if and only if sleeping subjects enter REM.

If neocortically mediated visual learning also proves to be REM sleep dependent, then the vestibular adaptation concept would have relevance not only to the space context but to plasticity enhancement in any context. Microgravity might then be viewed as a particularly potent test of the hypothesis that vestibular-mediated plasticity alters (and is altered by) REM sleep. It is this concept that we will explore utilizing the Nightcap recording system.

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proximity of the vestibular nucleus to sleep centers in the pontine brainstem, the initiation of eye movements during sleep, and the frequent occurrence of themes of movement and even flight in dream reports all argue for the existence of functional interactions between these brain regions ([8,13,14,15], but see [17]).

Second, some of the early studies of visual learning showed increases in REM sleep ([16,18,19]). Third, under conditions of sleep deprivation, subjects in our laboratory have reported that attempts at normal target acquisition (that is, without any distorting lenses) resulted in pronounced under-shoots of their ballistic eye movements. Taken together, these provide a rationale for studying the relationship between sleep, the visuo-oculomotor system, and the VOR under normal conditions as well as under conditions of modified vision (for example, minifying lenses), sleep deprivation, and varied vestibular load (for example, microgravity). In this paper we focus mainly on visuo-oculomotor effects.

Sleep and Posture

In our early time-lapse photographic and video studies, we established the strong temporal correlation between major posture shifts and sleep stage transitions ([20,21]). This finding was the foundation of our movement-based approach to sleep stage detection now instantiated in the Nightcap. Under normal gravity conditions, all humans make, on average, two major posture shifts per 90-minute sleep cycle: one tends to limb and head movements are observed ([21]). It is not known whether either the major posture shifts or the head and the limb movements are gravity sensitive. Indirect evidence comes from astronaut reports of bizarre sleep postures in space and of persistent limb elevations on awakening from postflight sleep. Thus, gravity and microgravity may exert differential effects upon sleep posture, and these may in turn affect the quality and quantity of sleep and even of dreaming.

Sleep and the Fictive Movement of Dreams

Since formulating the Activation-Synthesis Hypothesis of Dreaming in 1977, our group has developed a set of quantitative probes that measure formal aspects of dream cognition, including the illusion of movement. Our early work ([22]) showed that dreaming subjects perceived themselves to be constantly moving through the dream space, a finding that we have recently confirmed and extended ([23]). In this and other recent work, we have shown that these dream features are REM-sleep based and that this correlation can be detected with the Nightcap ([23,24]).

One particularly interesting feature of dreamed movement (which we call “fictive” because it is illusory) is its “vestibular” content ([22]). This feature is prominent in reports and involves sensations of floating, swimming, sailing, flying, spinning, twitching, or turning, which dreamers generally regard as exciting or pleasurable. “Vestibular” dream content is increased when subjects sleep in a swinging hammock ([25]). To our knowledge, this dream feature has never been quantified and therefore never measured in subjects before and after exposure to those shifts in vestibular input as occur in microgravity.

As the vestibular system is initially perturbed by entry into microgravity, is the illusion of dreamed movement changed? Can this change be tracked as adaptation occurs? What new baselines are established under prolonged exposure? Finally, what is the sequence of events that allows us to measure vestibular adaptation via its effects upon the subjective experience of fictive movement in REM sleep dreaming.

Measuring Sleep and Vigilance in the Field

This section will focus on two features of our Nightcap System of particular relevance to this paper: 1) its ability to reliably predict sleep stages and 2) its ability to detect lapses of vigil-
lance that occur at sleep onset and which may interfere with performance during waking. Although we focus mainly on sleep itself, it is important to appreciate the potential of the approach to detect the microsleeps that interrupt waking. We therefore point out that work in progress in our laboratory (26) shows clearly that our eyelid sensor’s output falls dramatically at sleep onset when EEG alpha synchrony is suppressed and hypnogogic reverie commences. We believe that it is feasible and desirable to study the eye sensor not only in sleep but also in target tracking tasks.

How Does the Nightcap Predict Sleep Stage?

The history of the development of a 2-channel state detection system is provided in previous publications (21,30). The physical appearance of the device is shown in Figure 3. The Nightcap is a wallet sized (10 cm × 7 cm × 2 cm) device weighing only 150 g and capable of recording 10 nights’ data on a single 9V alkaline battery. Its internal memory can store data for up to 30 nights.

Since both eyelid and head movements generate 10mV signals, they are easily detected without electronic amplification. Furthermore, because the movements are digitized at the acquisition stage, they can be stored in Nightcap memory as events per minute. This eliminates the need for the large amounts of memory re-

How Do Nightcap Data Compare with Polygraphically Recorded Sleep?

We have now completed two studies comparing Nightcap measures with polygraphic (sleep lab) measures in normal and sleep-disordered subjects (see Figure 4). Under conditions of gravity, there was an 87% agreement be-

Figure 3. The Nightcap senses signals from a multipolar mercury switch on the head and a piezoelectric film on the eyelid and stores movement data in dynamic RAM. The data is subsequently down-loaded to a Macintosh computer that identifies REM–nonREM–wake states according to a simple set of rules: 1) If there are counts in both channels it scores wake. 2) If there are counts in neither channel it scores NREM sleep. 3) If there are eyelid counts but no head counts, it scores REM. A sample record is shown in Figure 4.
between the Nightcap and the polygraph in the scoring of 1-min epochs (27,28,29).

The head movement channel of the Nightcap is roughly equivalent to the movement-artifact sensing function of the EMG, and movement time is thus a good index of the brief awakenings that often interrupt sleep. Under microgravity conditions, we will determine if head movement is still a sensitive or selective index of such awakenings.

The eyelid sensor is a piezoelectric film applied to the lid that is sensitive to underlying eye movements; the corneal bulge distorts the lid every time the eye moves. Nightcap eyelid sensor counts correlate well with REMs identified by EOG (28).

How Does the Nightcap Detect Sleep Onset and Microsleeps?

An unexpected property of the eyelid movement sensor is its exquisite sensitivity to sleep onset and microsleeps. We have recently found (26,32) that eyelid movements, which are very frequent in waking, drop precipitously at sleep onset in conjunction with (1) the suppression of alpha waves and (2) the subjective experience of hypnogogic mentation. The reason for this exquisite sensitivity is grounded in eyelid neurophysiology (see Figure 5).

The eyelid is raised by the levator palpebrae (LP) muscle as indicated in Figure 5B. The eyelid is controlled by brain stem neurones that project directly to the levator palpebrae muscle. The LP muscle is thus monosynaptically innervated by axonal branches of oculomotor neurones in the caudal central division of the oculomotor nucleus (31). It is probably for this reason that the eyelid piezo film is as good a vigilance detector as it is a REM sleep detector!

The result is that a single system raises the lid and the eye with each upward gaze. Like the extrinsic ocular muscles, the LP muscle contains fast twitch fibers that mediate the coordinate movements of eye and lid during upward gaze displacement. But the LP also contains slow twitch fibers of the sort found in skeletal anti-gravity muscles that maintain posture (31). These

![Figure 4. A typical night of sleep recorded simultaneously with nightcap and sleep lab polysomnography (sample output and analysis). Top trace: histogram of Nightcap-detected eye movements; second trace: hypnogram representing the manually scored polysomnograph record; third trace: hypnogram of computer-scored Nightcap data; Fourth trace: histogram plotting Nightcap-detected body movements. On both hypnograms, the top level represents wake, the second (darkened) level represents REM, and the third level is nonREM. Periodic movements are not shown on the hypnograms. The lower axis indicates the time of night.](image-url)
slow twitch fibers actively hold the lid open via tonic activation signals from the brainstem (31).

It is known (as shown in Figure 2) that the pontine reticular formation neurons are premotor controllers of the oculomotor nuclei (for example, VI in Figure 2 and III in Figure 5B) and hence of the LP muscle. It is this highly specific, fine-tuned system that the Nightcap eyelid sensor monitors. Naturally, any decrease in output of the RF, as occurs at sleep onset and with attentional lapses, will be reflected in 1) an immediate decrease in eyelid sensor counts, 2) EEG changes, and 3) cognitive and performance decrements.

It is for these reasons that we see the eyelid sensor as possibly useful in providing measures of alertness as well as sleep. But we also note, in passing, the possible value of the eyelid sen-

Figure 5. Schematic sagittal section of head showing position of nightcap head and eyelid sensors. (A) Note that head movement is integrated with eye position (and posture, not shown) by means of head position signals arising in the labyrinth impinging on the vestibular neurons VIII and transmitted to neck muscles (XI). (B) Note that the eyelid is innervated by fibers from the oculomotor neurons (III), which also innervate the superior rectus muscle. For comparison to the circuitry for Figure 2, the relative positions of the abduceus nucleus and nerve (VI) are also shown.
or in vestibulo-ocular reflex monitoring. Many scientists now measure this function using D.C. electro-oculography, whose output could be compared with our eyelid sensor in collaborative studies with a view to developing a multipurpose headgear system.

How is the Nightcap System Actually Used?

In addition to the physical economies already enumerated, the Nightcap (Figure 3) is extremely user-friendly. In its current configuration, subjects simply tie a bandanna kerchief around their heads before retiring. The head movement sensor is sewn into the bandanna and is self-positioning when the subject aligns the previously attached eye sensor with the center of the left eye. A plastic film is peeled away from the sensor, and it is self-applied to the left eyelid to which it adheres firmly overnight. The eye sensor is removed in the morning and discarded.

On retiring before sleep, the subject puts on the bandanna and sensors, and then turns the Nightcap recorder on. A flashing light signals if the battery needs to be replaced. On waking, the subject shuts off the recorder and removes the Nightcap headgear. The time, effort, and discomfort involved are minimal, another major advantage over standard recording techniques.

Sleep in Space

Although sleep is notoriously fugitive in extreme conditions such as high altitudes, it has been difficult to obtain satisfactory data from humans exposed to microgravity. Standard recording techniques require time-consuming electrode attachment, are themselves sleep inhibiting, and involve complex analog signal processing. In this section, we provide an overview of the published data.

Table 1 summarizes many of the sleep studies performed to date in space. Reviews of sleep

<table>
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<tr>
<th>Reference</th>
<th>Mission</th>
<th># Days for mission</th>
<th>N and species</th>
<th>PSG†</th>
<th>First sleep</th>
<th>PSG‡</th>
<th>Pre-flight sleep</th>
<th>PSG‡</th>
<th>In-flight sleep</th>
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<th>Subjective sleep assessment</th>
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<td>Adey et al.</td>
<td>Gemini 7</td>
<td>?</td>
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<td>Berry (33)</td>
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*Species coded as follows: hu = human, ma = macaque (Macaca nemestrina), rh = rhesus monkey.
†Number or "+" refers to polysomnography (PSG) studies (EEG, EOG, and EMG) reported to have taken place. When only certain channels specified, EE = EEG, EO = EOG, EM = EMG.
‡Sleep and wake states not differentiated in reports on EEG and EOG findings.
§Cited in Gundel et al. (37).
in space among Apollo (33) and Space Shuttle (34) astronauts have documented frequent subjective sleep difficulty and use of sleep medication, but do not report any polysomnographic data. Lack of EOG prevented unequivocal measurement of REM in early EEG studies on the Gemini 7 astronaut (35, 36). A recent polysomnographic study of space sleep in one subject (37) documented circadian shifting, redistribution of slow wave sleep, shortened REM latency, and decreased REM during the second REM-NREM cycle but, again, did not record during the initial nights in space.

REM during sleep in space, extending throughout the missions (38, 39). In addition, EOG recording of the Biosatellite III monkey revealed eye movement abnormalities in the early part of the flight (40), and similar phenomena may occur in humans (41).

Among U.S. studies, only Frost and colleagues (42) recorded a large number of polysomnograph (PSG) nights in space on replicate subjects. In three Skylab missions lasting 28, 59, and 84 days, this group monitored sleep EEG during pre-flight (3 nights), in-flight (12 to 20 nights), and post-flight (3 nights) periods in one crew member per mission. Again, no in-flight recordings were made on the first two nights in space.

The only change in REM sleep consistently found over all three missions was an increase in REM time and a decrease in REM latency which occurred late in the post-flight period (42). In-flight average REM percent was either slightly lower (especially late in the missions) or unchanged as compared to pre-flight baseline. Since the post-flight REM elevation occurred late in the post-flight period and was sustained over more than one night, these authors hypothesized that it was not a rebound effect due to previous REM deprivation but, rather, was a result of re-adaptation to normal gravity. They pointed out that REM effects during adaptation to microgravity may have been missed since sleep during the first 3 nights in space was not recorded. Other sleep changes observed on all three missions were an increase in stage 3 during flight and a decrease in stage 4 post-flight. Sleep quality did not show consistent in-flight decrement compared to pre-flight baselines as measured by sleep latency, total sleep time, number of awakenings, or subjective reports. The subject studied on the 84-day mission experienced sleep difficulties during the first half of his flight, while the occasional insomnia reported by all 3 men was attributed to work schedules rather than to microgravity effects.

Among U.S. studies, only Quadens and Green (43) report any PSG data with EOG for the first sleep period in space. These authors were able to record EMG and EOG in a single Spacelab 1 astronaut during the first two sleep epochs in post-flight recovery phase. They document increases in 1) REM percent, 2) eye movements, and 3) the relative occurrence of fast (>1/s) versus slow (0.5 to 1.0/s) eye movements during the first sleep epoch in space (3 h) when compared to either the second sleep epoch in space (6 h), pre-flight baseline nights, or post-flight recovery nights. By the second sleep epoch in space, these sleep parameters (as well as sleep duration) had returned to baseline levels. On the second day after return to earth, the elevation in relative occurrence of fast versus slow eye movements (but not in overall eye movement frequency) recurred. These authors interpret these eye movement elevations as reflecting the learning and integration of new information related to the return to normal gravity.

Optokinetic Stimulation and Sleepiness

As a first step in our efforts to establish a causal link between visuo-oculomotor stimulation and sleep, we exposed human subjects to optokinetic stimulation (OKS) of sufficient strength to cause both subjective drowsiness and nausea and then measured the time taken to fall asleep immediately thereafter and on the nights following stimulation. Measures were derived from Nightcap parameters in both the sleep latency tests (MSLT) and the home-based nocturnal sleep assessments—and are reported in full elsewhere (44).

To produce optokinetic stimulation of the vestibular system, we placed the subjects in a stationary chair in the center of a vertically
striped rotating drum for 10 min. The control condition was 10 min spent reading in a quiet room. The experimental procedure was successful in producing motion sickness of moderate severity using Graybiel's criteria (45-47). The motion sickness scores in the experimental condition were ten times greater than in the control condition, a highly significant difference [\(M = 9.07\) versus \(M = 0.82\), \(F(1,13) = 33.83, P < 0.001\]. The subjects were also significantly more likely to awaken in the frames of Graybiel's drowsiness subscale scores [\(M = 2.12\) versus \(M = 0.51\), \(F(1,12) = 20.49, P < 0.001\]. So the stimulation was effectively nauseogenic and soporific.

We were therefore surprised to detect only an insignificant trend to go to sleep more rapidly following the rotating drum exposure than after reading [\(M = 12.7\) min versus \(M = 13.6\) min] \((F = 3.53, P = 0.085)\]. The number of times that the subjects fell asleep while taking the MSLT was not different after stimulation, and there were no differences in any variable measured in the nocturnal sleep data (sleep onset latency, REM sleep latency, total sleep duration, REM sleep latency, total REM sleep, total NREM sleep, number of REM periods, and REM density). These results indicate that moderate motion sickness of short duration does not result in a significant increase in objective sleep tendency or in changes in nocturnal sleep parameters. Why might this be? We speculate that it is because we were using a threshold-level stimulus that was neither strong enough nor long enough to influence the sympathetic nervous system (for example, sweating), which probably worked against the soporific drive of the vestibular stimulation. On this view, it might be hypothesized that the sleep response to OKS competes with a potent arousal effect. Finally, it must be emphasized that our stimulation method did not influence the vestibular system directly, nor was any learning involved, so this experiment cannot be considered a definitive test of our central hypothesis.

This experiment was worthwhile for two reasons. First, it demonstrated that short-term optokinetic stimulation, which is moderately nauseogenic and produces significant subjective drowsiness, does not increase objective sleep tendency. Second, it shows that the valuable and versatile sleep latency test can be administered under field conditions by using the Nightcap instead of expensive and constraining sleep lab technology. We intend to take advantage of both of these findings in our future work.

**Conclusion**

The modern reality of space flight presents behavioral neuroscience with unique challenges and opportunities to reexamine and experimentally explore the three-way interaction between sleep, the vestibular system and performance. This paper has reviewed some of the literature relevant to this task, suggested a few practical ways of developing an investigative program, and presented preliminary data showing that optokinetic stimulation that produces subjective drowsiness may not enhance objective sleep.

REFERENCES

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