Tolerance to Extended Galvanic Vestibular Stimulation: Optimal Exposure for Astronaut Training

Valentina Dilda, Hamish G. MacDougall, and Steven T. Moore

Dilda V, MacDougall HG, Moore ST. ***Tolerance to extended galvanic vestibular stimulation: optimal exposure for astronaut training.*** **Aviat Space Environ Med 2011; 82:770–4.**

Background*:** We have developed an analogue of postflight sensorimotor dysfunction in astronauts using pseudorandom galvanic vestibular stimulation (GVS). To date there has been no study of the effects of extended GVS on human subjects and our aim was to determine optimal exposure for astronaut training based on tolerance to intermittent and continuous galvanic stimulation. ***Methods: There were 60 subjects who were exposed to a total of 10.5 min of intermittent GVS at a peak current of 3.5 mA or 5 mA. A subset of 24 subjects who tolerated the intermittent stimulus were subsequently exposed to 20-min continuous stimulation at 3.5 mA or 5 mA. ***Results*:** During intermittent GVS the large majority of subjects (78.3%) reported no or at most mild motion sickness symptoms, 13.3% reported moderate symptoms, and 8.3% experienced severe nausea and requested termination of the stimulus. During 20-min continuous exposure, 83.3% of subjects reported no or at most mild motion sickness symptoms and 16.7% (all in the 5-mA group) experienced severe nausea. ***Conclusion*:** Based on these results, we propose two basic modes of GVS application to minimize the incidence of motion sickness: intermittent high (5 mA) amplitude, suited to simulation of intensive operator tasks requiring a high-fidelity analogue of postflight sensorimotor dysfunction such as landing or docking maneuvers; and continuous low (3.5 mA) amplitude stimulation, for longer simulation scenarios such as extra vehicular activity. Our results suggest that neither mode of stimulation would induce motion sickness in the large majority of subjects for up to 20 min exposure.

Keywords: microgravity, motion sickness, MSSQ, flight analogue, mission simulation.

ASTRONAUTS RETURNING from spaceflight commonly experience deficits in sensorimotor function. The physiological basis is not well understood, but may represent adaptation of central processing of afferent vestibular input encoding linear acceleration due to the relative absence of gravity in orbit. These changes are maladaptive upon return to Earth and, during readaptation to gravity (a period ranging from a few days for shuttle missions and up to 2 wk after return from the International Space Station), astronaut postural (15), locomotor (1), oculomotor (2,7), fine motor (10,18), and perceptual (5) performance is degraded.

NASA has evaluated sensorimotor risks for future exploration class missions (12), placing a high priority on the development and validation of ground-based analogues of the effects of long-duration microgravity exposure on sensorimotor-dependent tasks such as manned control or supervision of spacecraft during docking and landing maneuvers, emergency egress, and planetary extra vehicular activity (EVA). The current 'gold standard' for replicating the physiological effects of spaceflight on Earth is head-down bed rest, where the subject lays on a bed tilted 6° head down for periods of weeks to months (16). Bed rest is effective at simulating the deconditioning effects of spaceflight on bone, muscle, and cardiovascular system function. However, results from a 21-d bed rest study suggest that microgravity-induced decrements in spatial orientation and otolith-mediated reflex eye movements (11), and postural and locomotor performance (17) observed after spaceflight were not replicated by axial unloading.

Crew preparedness would benefit from a realistic ground-based analogue of microgravity-related sensorimotor deficits. For example, astronaut pilots and commanders perform at a high level during extensive preflight training for orbiter landing in the Vertical Motion Simulator (VMS) at NASA Ames and the Shuttle Training Aircraft (STA - a modified Gulfstream II jet). However, landing performance is clearly degraded after spaceflight relative to preflight simulations; a review of the first 100 shuttle missions found that 20% of landings were above target touchdown speed (10) and an analysis of vertical velocity (sink rate) during actual shuttle landings and preflight training in the STA from STS-43 to STS-108 demonstrated a higher variability postflight, with 10% of landings above the target range of 3.5 $ft \cdot s^{-1}$ (1.1 $m \cdot s^{-1}$), compared to 3% in preflight training (14). The VMS and STA provide a high-fidelity simulation of orbiter flight characteristics. We propose that a critical difference between pre- and postflight landing performance is the negative impact of microgravity exposure on sensorimotor function. Augmenting preflight

From the Human Aerospace Laboratory, Department of Neurology, Mount Sinai School of Medicine, New York, NY, and the School of Psychology, University of Sydney, Australia.

This manuscript was received for review in February 2011. It was accepted for publication in March 2011.

Address correspondence and reprint requests to: Steven T. Moore, Ph.D., Mount Sinai School of Medicine, Department of Neurology, Box 1052, 1 Gustave L. Levy Place, New York, NY 10029; steven.moore@mssm.edu.

DOI: 10.3357/ASEM.3051.2011

simulations with an analogue capable of reproducing postflight sensorimotor deficits has the potential to enhance the effectiveness of crew training, not only for control of spacecraft, but for a wide range of operational tasks on exploration-class missions.

We have developed a technique to replicate the postflight sensorimotor experience of astronauts by disrupting vestibular input in normal subjects by passing small electrical currents between mastoidal surface electrodes (bilateral bipolar galvanic vestibular stimulation - GVS). The pseudorandom current waveform, a sum of sines with dominant frequencies at 0.16, 0.33, 0.43, and 0.61 Hz, was derived in a preliminary study ($N = 4$) comparing anterioposterior sway in normal subjects undergoing GVS during computerized dynamic posturography (6) with postflight astronaut performance on the same device (15). We speculated that postflight postural deficits were the result of changes in central processing of low-frequency otolith information during flight and that modulating otolithic afferent input to the cerebellum with a pseudorandom current waveform may produce similar imbalance. When subsequently applied to a group of healthy subjects ($N = 20$), the GVS analogue accurately replicated the postural instability (6), locomotor impairment, and reduced dynamic visual acuity (9) observed in astronauts after return from shuttle and International Space Station missions. In addition, a study of pilot performance during simulated shuttle landings in the VMS (8) demonstrated that GVS degraded spatial orientation and fine motor control, inducing 'hard' landings (touchdown speed above target) consistent with that observed in actual shuttle landings (10). Subjective validation was provided by seven veteran astronauts (five shuttle, one ISS, one Skylab), who reported that the motor effects and illusory sensations of movement generated by the GVS analogue were remarkably similar to their postlanding experience (13). The ability of GVS to accurately replicate the postural, locomotor, oculomotor, and perceptual difficulties experienced by astronauts suggests a vestibular basis for these postflight sensorimotor deficits.

The aim of this study was to determine optimal GVS exposure for astronaut training based on tolerance to intermittent and continuous galvanic stimulation. We have successfully addressed the issue of irritation or pain at the electrode site by using hypoallergenic electrode gel and large (20-cm^2) electrodes (cut from electrosurgical split grounding plate electrodes), allowing the delivery of higher current amplitudes (up to 5 mA peak), but at lower current densities at the skin, than previous GVS studies (9,20). Here we determined the incidence of autonomic symptoms (analogous to motion sickness) in response to GVS in a large group of healthy subjects as a function of current amplitude and duration of exposure.

METHODS

Subjects

A total of 60 participants were randomly assigned into 2 groups: 3.5 mA [$N = 30$; 17 men, 13 women; mean age 30.3 yr (95% CI 10.8)] and 5 mA [$N = 30$; 15 men, 15 women; age 28.7 yr (CI 10.3)] peak GVS current. Subjects had no prior experience with GVS. Mount Sinai School of Medicine's Institutional Review Board approved the experiments and subjects gave their informed consent and were free to withdraw at any time.

Equipment

Delivery of bilateral bipolar GVS was achieved using a constant-current generator that imposed a constant current amplitude independent of the load (subject) connected. This device consisted of a 9-V battery and a small box containing circuitry under computer control via a USB digital-to-analog converter (12-bit DA 1208LS, Measurement Computing, Middlesboro, MA). Current was delivered to the surface of the subject's skin via leads and large electrodes placed over the mastoid processes. The pseudorandom galvanic stimulus consisted of a sum-of-sines (0.16, 0.33, 0.43, 0.61 Hz) current waveform with peak amplitude set to either 3.5 or 5 mA (6,9).

Procedure and Design

Before starting the experiment each participant completed the Motion Sickness Susceptibility Questionnaire (MSSQ) (4), developed as a predictor of motion sickness susceptibility based on a series of questions regarding motion sickness history. The higher the score the more susceptible the subject to motion sickness; an MSSQ score of 0 indicates insusceptibility (i.e., no childhood or adult history) and the 95th percentile for the adult population is 120 (4). MSSQ scores in the 60 participants ranged from 0 to 85.5. A battery of six cognitive tests (reaction time, dual tasking, Stroop, perspective taking, mental rotation, match to sample) and a manual tracking task were administered to each participant while seated 1 m in front of a computer monitor under three conditions: pre-GVS baseline, during intermittent GVS, and 15 min after GVS exposure. The effects of GVS on cognitive function have been described elsewhere (3); in this paper we focus purely on tolerance to GVS exposure.

During the GVS condition the stimulus was turned on at the beginning of each test and off at completion, with the duration of each exposure averaging 1.1 min (CI 0.37) per task and 10.5 min (CI 0.87) total GVS exposure for subjects completing the test battery. In between tasks, subjects were asked to verbally indicate if headache, nausea, dizziness, cold sweat, or drowsiness were present and any pallor or flushing was noted by the operator on an ordinal scale (absent, mild, moderate, and severe) (6). Motion sickness evaluations were also performed prior to and 15 min after GVS exposure. The experiment was terminated if severe nausea was reported (a feeling that vomiting was imminent) or at any time at the subject's request.

A subset of 24 subjects [16 men, 8 women; age 29.6 yr (CI 3.4)], who reported mild or no symptoms of motion sickness during an average of 10.5 min of GVS exposure in the intermittent experiment, participated in a second experiment of continuous GVS. Subjects were exposed

to a maximum of 20 min GVS at 3.5 mA (N = 12; 8 men, 4 women) or 5 mA (N = 12; 8 men, 4 women) peak current amplitude. Motion sickness symptoms (headache, pallor, nausea, dizziness, flushing, cold sweat, drowsiness) were periodically monitored and recorded on an ordinal scale (absent, mild, moderate, or severe), and the experiment was terminated if severe nausea was reported or at any time at the subject's request. Motion sickness evaluations were also performed prior to application of continuous GVS and 15 min after termination. During GVS exposure subjects were standing and performed computerized dynamic posturography (Equitest, Neurocom, OR) (6). The posturography results are not reported in this paper, which deals exclusively with tolerance of the GVS analogue.

RESULTS

Intermittent Exposure

GVS was well tolerated at both the 3.5-mA and 5-mA levels of exposure. At 3.5 mA the average time of exposure was 10.5 min and 93.3% (28/30) of the subjects completed the experiment (**Table I**). The majority of subjects (25/30) reported no (N = 18) or at most mild (N = 7) motion sickness symptoms (nausea, dizziness, drowsiness, cold sweat, pallor) and 3 subjects reported moderate symptoms (nausea, dizziness, cold sweat, pallor). Two subjects (one man and one woman) asked to interrupt the experiment due to severe nausea following a mean GVS exposure of 3.4 min. In post-GVS testing two subjects reported mild symptoms (nausea, dizziness). None of the subjects experienced headache or flushing. At 5 mA peak current, 90% (27/30) of the subjects completed the experiment with an average of 10.5 min GVS exposure, with 22 subjects reporting no (N = 20) or at most mild (N = 2) symptoms (nausea, dizziness, headache). Five subjects reported moderate symptoms (nausea, dizziness, flushing, pallor, cold sweat) and three female subjects asked to interrupt the experiment due to severe nausea following a mean GVS exposure of 5.5 min. No symptoms were reported in post-GVS testing. Thus, out of the 60 subjects exposed to a cumulative 10.5 min of intermittent GVS, 47 (78.3%) reported no or at most mild motion sickness symptoms, 8 (13.3%) reported moderate symptoms, and 5 subjects (8.3%) experienced severe nausea and did not complete the experiment (Table I).

The MSSQ was not a good predictor of motion sickness susceptibility to intermittent GVS. The MSSQ of the 5 subjects who experienced severe nausea [31.8 (CI 9.4); range 18.5–44.9] did not differ significantly (ANOVA; P = 0.2) from the MSSQ of all 60 subjects [18.8 (CI 4.63)]. Moreover, significant motion sickness symptoms were not reported by 22 subjects with above-average MSSQ scores [35.4 (CI 6.9); range 19–85.4].

Continuous Exposure

Continuous GVS was better tolerated by the 3.5-mA group [MSSQ 11.2 (CI 7.2)] than the 5-mA group [MSSQ 12.2 (CI 8.8)]. In the 3.5-mA group, all 12 subjects completed 20 min of continuous GVS exposure; 11 subjects reported no motion sickness symptoms at all, with only 1 subject (female) reporting mild dizziness. At 5 mA, 75% of the subjects (9/12) completed the 20-min exposure. Of these nine subjects, seven reported no motion sickness symptoms, one (male) reported mild nausea, dizziness, and cold sweat, and one (male) reported moderate dizziness and severe nausea immediately after completion of the 20-min exposure. Three subjects [1 man, 2 women; MSSQ average 32.2 (CI 23.4)] requested early termination of GVS after a mean exposure of 9.8 min due to severe nausea. No motion sickness symptoms were reported by any of the 24 subjects 15 min after GVS exposure. No motion sickness symptoms were reported pre-GVS and headache, pallor, or drowsiness were not reported at any stage.

Thus, out of the 24 subjects exposed to 20-min continuous GVS, 20 (83.3%) reported no or at most mild motion sickness symptoms. Four subjects (16.7%) experienced severe nausea at 5 mA peak current, three of whom terminated exposure early; these four subjects subsequently completed a total of 20 min intermittent 5-mA GVS (10 sessions of 2 min each, with a 1-min break between applications) without reporting any motion sickness symptoms whatsoever. The MSSQ of all 24 subjects

TABLE I. MOTION SICKNESS SYMPTOMS DURING AND 15 min AFTER INTERMITTENT GVS EXPOSURE (MEAN CUMULATIVE EXPOSURE OF 10.5 min) AT 3.5 mA AND 5 mA PEAK CURRENT (30 SUBJECTS PER GROUP).

	3.5 mA						5 mA					
	Per-GVS			Post-GVS			Per-GVS			Post-GVS		
Symptom	Mild	Moderate	Severe	Mild	Moderate	Severe	Mild	Moderate	Severe	Mild	Moderate	Severe
Headache	-	-	-	-	-	-	1	-	-	-	-	-
Pallor	2	1	-	-	-	-	-	1	-	-	-	-
Nausea	5	2	2	1	-	-	2	3	3	-	-	-
Dizziness	7	1	-	1	-	-	1	3	-	-	-	-
Flushing	-	-	-	-	-	-	-	1	-	-	-	-
Cold sweat	2	1	-	-	-	-	-	1	-	-	-	-
Drowsiness	3	-	-	-	-	-	-	-	-	-	-	-
# Subjects reporting	7	3	2	2	0	0	2	5	3	0	0	0

Two subjects at 3.5 mA and three subjects at 5 mA experienced severe nausea and requested early termination of GVS.

[11.7 (CI 7.9); range 0–55.4] was not significantly different (ANOVA $P = 0.06$) to the MSSQ scores of the four subjects who reported severe nausea [27.5 (CI 18.9); range 13.6–55.4]. Five subjects who did not report any motion sickness symptoms had MSSQ scores [24.6 (CI 5.4); range 18.5–34.3] above the group mean.

DISCUSSION

The results of this study demonstrate that extended exposure to GVS is well tolerated at current amplitudes of up to 5 mA. During intermittent GVS, the large majority of subjects (91.7%) had little difficulty completing the 10.5-min exposure and there was no obvious effect of current amplitude (3.5 or 5 mA) on the incidence of motion sickness reports. Thus, in the general population we would predict less than 9% of subjects to be intolerant of intermittent GVS at amplitudes up to 5 mA. For the continuous study we selected from the 78% of subjects who experienced none or at most mild symptoms during intermittent GVS, a group we would expect to be roughly equivalent to the astronaut population in terms of motion sickness insusceptibility. During 20-min continuous galvanic stimulation there was an effect of current amplitude, with almost no motion sickness reported at 3.5 mA (one subject reporting mild dizziness), but a third of subjects at 5 mA peak current (4/12) experienced severe nausea, three of whom requested early termination of the stimulus. However, it was the temporal nature of GVS delivery that appeared to be the critical factor in the development of motion sickness symptoms, rather than the total duration of exposure; the four subjects reporting severe nausea during 20-min continuous 5-mA GVS completed a total of 20 min intermittent 5-mA stimulation (10 × 2-min epochs) without any motion sickness whatsoever. To our knowledge this is the first report of human tolerance to long-duration GVS exposure.

The MSSQ (4) was not a good predictor of susceptibility to GVS-induced symptoms. This suggests that the etiology of autonomic symptoms (motion sickness) induced by changing or unfamiliar inertial environments (such as land, sea, or air transportation) differs from that experienced in response to electrical activation of the vestibular nerve (GVS). It is clearly established that susceptibility to terrestrial motion sickness does not correlate with the occurrence of space motion sickness, which affects approximately 50% of shuttle crewmembers in the first 72 h after orbital injection (19). Moreover, symptoms of space motion sickness differ from the terrestrial variety, with absence of sweating and sudden episodic vomiting with little nausea (19).

The results of this study allow us to suggest guidelines for application of the GVS analogue of postflight sensorimotor dysfunction in astronaut training. We have previously demonstrated the efficacy of 5-mA pseudorandom GVS in replicating postflight imbalance, ataxic gait, reduced visual acuity, and impaired manual control (6,8,9). The sensorimotor effects of GVS are diminished at the lower current amplitude of 3.5 mA, but still induce measurable decrements in performance (21). Thus we propose two basic modes of GVS application to minimize the incidence of motion sickness symptoms: intermittent high (5-mA) amplitude and continuous low (3.5-mA) amplitude stimulation, representing a tradeoff between the fidelity of the sensorimotor analogue and the risk of provoking an adverse autonomic response (both proportional to peak current). Intermittent high amplitude GVS is better suited to simulation of short-duration operator tasks requiring a high-fidelity analogue of postflight sensorimotor dysfunction, such as manual control or supervision of spacecraft and other vehicles. Based on the minimum exposure tolerated by subjects reporting severe nausea during 5-mA continuous stimulation, each GVS interval should be limited to 5 min. This mode was employed in our study of shuttle landing performance in the VMS (8), where each final approach and landing task was around 2 min duration. Longer continuous training epochs may be preferable for scenarios such as EVA, performance of complex repairs, and navigation within a space station; in this case low amplitude (3.5-mA) stimulation would be more suitable, providing a lower fidelity but measurable degradation of sensorimotor function over extended periods. Our results suggest that neither mode of stimulation would induce significant motion sickness symptoms in the large majority of subjects for up to 20 min total exposure.

ACKNOWLEDGMENTS

This project was supported by a grant from the National Space Biomedical Research Institute (NSBRI) through NASA contract NCC 9-58 (Dr. Steven Moore) and the Garnett Passe and Rodney Williams Memorial Foundation (Dr. MacDougall). The authors would like to acknowledge the contribution of John Holden (School of Psychology, University of Sydney) for development of the GVS equipment.

Authors and affiliations: Valentina Dilda, M.S., Ph.D., Hamish G. MacDougall, B.Sc., Ph.D., and Steven T. Moore, M.Eng., Ph.D., Human Aerospace Laboratory, Department of Neurology, Mount Sinai School of Medicine, New York, NY, and Hamish G. MacDougall, B.Sc., Ph.D., School of Psychology, University of Sidney, Sidney, Australia.

REFERENCES

1. Bloomberg JJ, Peters BT, Smith SL, Huebner WP, Reschke MF. Locomotor head-trunk coordination strategies following space flight. J Vestib Res 1997; 7:161–77.
2. Clarke AH, Grigull J, Mueller R, Scherer H. The three-dimensional vestibulo-ocular reflex during prolonged microgravity. Exp Brain Res 2000; 134:322–34.
3. Dilda V, MacDougall HG, Moore ST. Tolerance and cognitive performance during galvanic vestibular stimulation (GVS) [abstract]. In: Proceedings of the 11th ESA Life Science Conference "Life in Space for Life on Earth"; 13-18 June 2010; Trieste, Italy. Paris, France: European Space Agency; 2010:63.
4. Golding JF. Motion sickness susceptibility questionnaire revised and its relationship to other forms of sickness. Brain Res Bull 1998; 47:507–16.
5. Harm DL, Reschke MF, Parker DE. Visual vestibular integration: motion perception reporting. In:Sawin CF, Taylor GR, Smith WL, eds.Extended Duration Orbiter Medical Project Final Report 1989-1995 (NASA/SP-1999-534). Houston: NASA Johnson Space Center; 1999; 5.2:1–12.
6. MacDougall HG, Moore ST, Curthoys IS, Black FO. Modeling postural instability with galvanic vestibular stimulation. Exp Brain Res 2006; 172:208–20.
7. Moore ST, Cohen B, Raphan T, Berthoz A, Clement G. Spatial orientation of optokinetic nystagmus and ocular pursuit during orbital space flight. Exp Brain Res 2005; 160:38–59.

8. Moore ST, Dilda V, MacDougall HG. Galvanic vestibular stimulation as an analogue of spatial disorientation after spaceflight. Aviat Space Environ Med 2011; 82:535–42.
9. Moore ST, MacDougall H, Peters BT, Bloomberg JJ, Curthoys IS, Cohen H. Modeling locomotor dysfunction following spaceflight with galvanic vestibular stimulation. Exp Brain Res 2006; 174:647–59.
10. Moore ST, MacDougall HG, Lesceu X, Speyer JJ, Wuyts F, Clark JB. Head-eye coordination during simulated orbiter landing. Aviat Space Environ Med 2008; 79:888–98.
11. Moore ST, MacDougall HG, Paloski W. Effects of head-down bed rest and artificial gravity on spatial orientation. Exp Brain Res 2010; 204:617–22.
12. NASA. Human Research Program Integrated Research Plan. Houston: NASA JSC; 2010. Available at: http://humanresearch.jsc.nasa.gov/elements/smo/docs/IRP_HRP-47065.pdf.
13. National Space Biomedical Research Institute. Galvanic vestibular stimulation countermeasure demonstrated to astronauts. NSBRI Explorer ; 2006. Available at: http://www.nsbri.org/default/NewsletterArchive/NSBRI-July-2006.pdf.
14. Paloski WH, Oman CM, Bloomberg JJ, Reschke MF, Wood SJ, et al. Risk of sensory-motor performance failures affecting vehicle control during space missions: a review of the evidence. J Gravit Physiol 2008; 15:1–29.
15. Paloski WH, Reschke MF, Black FO. Recovery of postural equilibrium control following space flight (DSO 605). In:Sawin CF, Taylor GR, Smith WL, eds. Extended Duration Orbiter Medical Project Final Report 1989-1995 (NASA/SP-1999-534). Houston: NASA Johnson Space Center; 1999; 5.4:1–16.
16. Pavy-Le Traon A, Heer M, Narici MV, Rittweger J, Vernikos J. From space to Earth: advances in human physiology from 20 years of bed rest studies (1986-2006). Eur J Appl Physiol 2007; 101:143–94.
17. Reschke MF, Bloomberg JJ, Paloski WH, Mulavara AP, Feiveson AH, Harm DL. Postural reflexes, balance control, and functional mobility with long-duration head-down bed rest. Aviat Space Environ Med 2009; 80(5, Suppl.)A45–54.
18. Ross HE, Schwartz E, Emmerson P. The nature of sensorimotor adaptation to altered G-levels: evidence from mass discrimination. Aviat Space Environ Med 1987; 58:A148–52.
19. Thornton WE, Moore TP, Pool SL, Vanderploeg JM. Clinical characterization and etiology of space motion sickness. Aviat Space Environ Med 1987; 58:A1–8.
20. Watson SR, Brizuela AE, Curthoys IS, Colebatch JG, MacDougall HG, Halmagyi GM. Maintained ocular torsion produced by bilateral and unilateral galvanic (DC) vestibular stimulation in humans. Exp Brain Res 1998; 122:453–8.
21. Wood SJ, Black FO, MacDougall HG, Moore ST. Electrotactile feedback of sway position improves postural performance during Galvanic vestibular stimulation. Ann N Y Acad Sci 2009; 1164:492–8.