NASA Technical Memorandum 102 157

Visual Suppression of the Vestibulo-ocular Reflex During Space Flight

John J. Uri
GE Government Services
Houston, Texas

William E. Thornton, and Sam L. Pool
Lyndon B. Johnson Space Center
Houston, Texas

Thomas P. Moore
Methodist Hospital of Indiana
Indianapolis, Indiana

Contents

Tables

Table | Page

Figures

Figure | Page

Visual suppression of the vestibulo-ocular reflex during space flight

Abstract

Visual suppression of the vestibulo-ocular reflex was studied in 16 subjects on four Space Shuttle missions. Eye movements were recorded by electro-oculography while subjects fixated a head-mounted target during active sinusoidal head oscillation at 0.3 Hz. Adequacy of suppression was evaluated by the number of nystagmus beats, the mean amplitude of each beat, and the cumulative amplitude of nystagmus during two head oscillation cycles. Vestibulo-ocular reflex suppression was unaffected by space flight. Subjects with space motion sickness during flight had significantly more nystagmus beats than unaffected individuals. These susceptible subjects also tended to have more nystagmus beats before flight.

Introduction

A comprehensive investigation of neurologic adaptation to space flight was undertaken as a joint NASA Johnson Space Center Flight Operations Directorate and Medical Sciences Division project. Among other studies, vestibular and optokinetic experiments were performed on Shuttle missions STS-4 through 8 (1982-3) (1). This report presents the results of studies on visual suppression of the horizontal vestibulo-ocular reflex (VOR). Results from a few subjects in this series have been reported elsewhere (2,3,4), but this report presents the complete study. Preflight and in-flight measurements were obtained from 12 subjects, and preflight and postflight data from an additional four subjects.

The VOR compensates for head movement to permit the eyes to maintain foveal fixation on stationary objects in the environment (5). Moving objects in the environment are often tracked with head movements during which the eyes are held stable relative to the head. Under these conditions the VOR must be suppressed to prevent loss of visual fixation on the target (6). Suppression of the VOR is achieved primarily by the visual smooth pursuit system, at least under conditions where that system is effective (e.g., predictable target motion at low-frequency and amplitude) (7), although there is also evidence for direct suppression of the VOR by the cerebellar flocculus (8).

Suppression of the VOR has been well characterized in the terrestrial environment (7-19). A common study method is to have subjects track a target that, during head motion, moves with the same angular velocity as the head. Eye and head movements are recorded and graphically displayed. Usually, amplitude or velocity of eye and head movements are compared and the gain is determined. During either active or passive sinusoidal head oscillations at frequencies below 0.5 Hz, VOR suppression gain is often reported to be less than 0.10 (7,9,12,13,15-19). Another method of assessing visual suppression of the VOR is to measure the total amplitude of nystagmus fast phases produced over a given duration of head oscillation (10).

Subsequent to our studies, VOR suppression has also been examined during space flight (20,21). Using active horizontal head oscillation at 0.25 Hz as the stimulus in one subject on STS-51G, Viéville, *et al* (20) reported in-flight gains of 0.09 and 0.10 on mission days 4 and 7, respectively, and a gain of 0.085 one day after landing. Although no preflight measurements were reported, they concluded that VOR suppression was not affected by space flight.

Benson and Viéville (21) reported preflight and postflight VOR suppression gains of a single Spacelab-1 crewmember. Preflight mean was 0.38, while gain on the first postlanding day was 0.23, significantly ($p=0.05$) lower than preflight. Subsequent postflight values were 0.44. The methodology differed in that passive head oscillation at 1 Hz was the stimulus but with the head axis displaced 1 m from the rotation axis.

Methods

Subjects. The sixteen volunteer subjects for this study were all professional NASA astronauts, fifteen males and one female ranging in age from 32 to 54 years. All had experience in high-performance jet aircraft and three had prior space flight experience. There were no known visual or vestibular abnormalities, other than presbyopia corrected to normal acuity by glasses. Consent was obtained from each subject after explanation and demonstration of the procedure. On three of the flights, a physician crewmember administered the test.

Eye and head position recording. Horizontal eye movements were recorded using conventional electro-oculography (EOG) (22). One cm Ag-AgCl electrodes were located at the lateral canthi with a mid-forehead

Fig. 1 TOP 18%

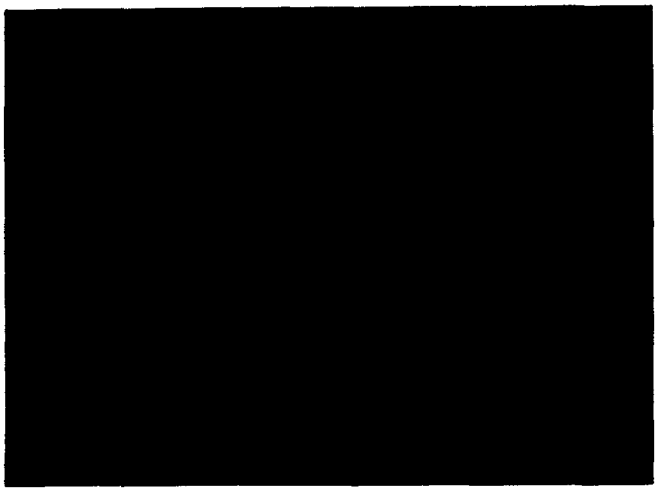

Figure 1.— Subject performing VOR suppression test. The target for visual fixation can be seen at far left, and the pantograph mounting for the head position potentiometer is at upper right.

ground. Amplification and recording had an overall frequency response of 0.05 to 70 Hz (3 dB points). Eye position could be determined to <2% accuracy. EOG was calibrated prior to each study by having the subject fixate five high-intensity light-emitting diode (LED) targets located at visual angles of 0°, ±15° and ±30° (STS-5 and 6), or 0°, ±10° and ±20° (STS-7 and 8).

Head position was recorded by a precision (0.1% error) potentiometer mounted in a pantograph system that allowed ±5 cm translation motion without detectable output. The potentiometer was coupled to the subject's head by a closely-fitting fabric helmet. Overall accuracy was on the order of 2%.

Data were either transmitted from the spacecraft via a DC-100 Hz digital system with eight bit resolution or recorded onboard on a miniature analog magnetic tape system, then transcribed to 100 mm per channel width graphic record at 10 mm sec^{-1}.

Protocol. The target used for VOR suppression studies was a white 5 mm diameter sphere on a 42-cm lightweight boom on the eyes' axis, attached to the head with a cap and chin strap (figure 1). All measurements were recorded with the subject restrained in a crew seat or equivalent. On earth, the subjects were seated with a vertical gravity vector. Subjects were trained to make horizontal sinusoidal head oscillations at an amplitude of ±30 degrees and frequency of 0.3 Hz without external pacing cues. They were requested to fixate on the head-mounted target during at least five head oscillations. The visual background was variable.

Data reduction and analysis. Data were manually reduced from the graphic records. Although most investigators attempt to assign a gain value to VOR suppression, in the large majority of our records no sinusoidal eye movement patterns were evident, and there was insufficient eye movement to attempt to reconstruct a sinusoid. Instead, we used a modified method of Dichgans, *et al* (10) and recorded the number of nystagmus movements (nystagmus frequency) and their mean amplitude during two complete head oscillations and then calculated the total nystagmus amplitude for that duration.

Consistent scheduling of experiments among all subjects was not possible due to in-flight operational constraints. We therefore combined in-flight measurements into two epochs, early (Mission Day [MD] 1-2) and late (MD 3-6). The early epoch corresponds to the period of symptoms of space motion sickness (SMS) in all affected subjects, allowing comparisons between affected and non-affected subjects. Independent t-tests were used to compare preflight and in-flight measurements, as well as SMS susceptible and non-susceptible populations.

Results

Measurements were obtained from 16 subjects on Shuttle flights STS 5 through 8, with a total of 52 records preflight, 35 during space flight, and 15 postflight (table 1). None of the subjects reported any subjective visual or oculomotor disturbances during the flights. The six subjects marked with an asterisk exhibited symptoms of

Table 1.— Schedule of records obtained

Flight/	Subject	Preflight	In-flight Mission Day						Postflight	
			1	2	3	4	5	6	R+0D	R+10D
STS 5	1	2		1		1			1	
	2*	3							1	
STS 6	3	1		1		1			1	
	4*	1		1		1				
	5	1		1		1			1	
	6	2		1		1			1	
STS 7	7	2		1	1					
	8*	2			1	1				1
	9	2		1	1			1		
	10	1			1					
	11*	4	1	1	2	1	1	1		2
STS 8	12	6	1						1	1
	13	5	1			1			1	1
	14*	5	1	1		1				1
	15	6	1				1		1	
	16*	9		1		1				1

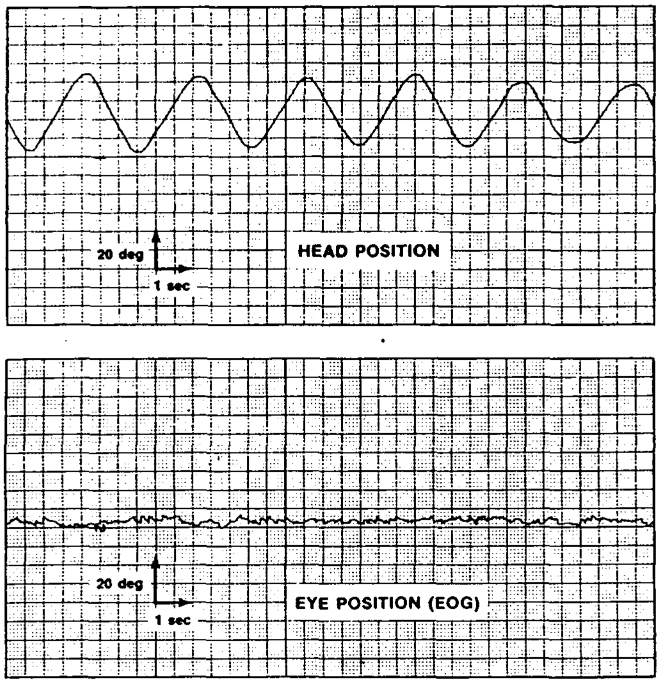

Figure 2.— An example of a typical in-flight recording of VOR suppression.

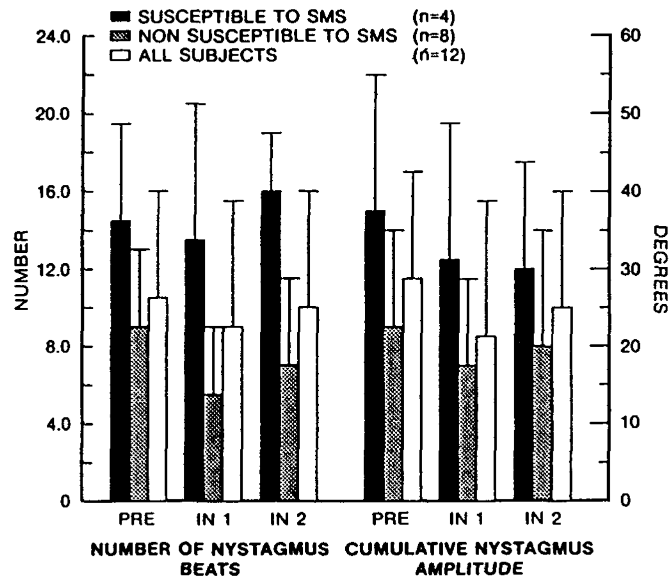

Figure 3.— Preflight and in-flight mean and S.D. of the number of beats of nystagmus and the cumulative nystagmus amplitude during two head oscillation cycles. Differences between SMS and non-SMS populations are also shown. IN1 and IN2 refer to early and late inflight periods, respectively.

SMS on MD 1 and 2; six records were obtained during their symptomatic period. A typical in-flight recording is shown in figure 2.

Twelve of the subjects recorded data during both early and late flight phases. Mean changes from preflight in the number of nystagmus movements and their total amplitude during two complete head oscillations are shown in figure 3. A comparison between SMS and non-SMS populations is also shown.

No significant changes from preflight in either the number of nystagmus movements or their total amplitude were seen during space flight (table 2). However, subjects with SMS had significantly more nystagmus beats than non-susceptible subjects during both in-flight periods (p=0.023 early and p=0.002 late), with no significant difference between the two groups in total nystagmus amplitude. Mean amplitude of each nystagmus movement was not different between the two groups and did not change with space flight (see data in Appendix Table A3).

By pooling all in-flight data, measurements from an additional three subjects were included in a further analysis. In this population of 15 subjects, five with SMS, no significant changes from preflight were seen in nystagmus frequency or total amplitude. During flight, the SMS subjects had greater nystagmus frequency (p=0.004) and total amplitude (p=0.054) than the non-SMS group.

Postflight measurements on the day of landing were obtained from a population of eight subjects, all but one of whom also had in-flight data. Nystagmus frequency was unchanged from preflight, but the total amplitude was decreased (p=0.05).

Table 2.— Summary of statistical results for 12-subject population

Nystagmus frequency	Mean ± SD	p
Pre vs. In-flight 1	10.7 ±5.2 vs. 8.6 ± 6.7	0.466
Pre vs. In-flight 2	10 ± 5.2 vs. 10.9 ± 5.8	0.948
SMS vs. non-SMS		
Preflight	14.7 ± 5.0 vs. 8.8 ± 4.0	0.067
In-flight 1	13.5 ± 7.1 vs. 5.5 ± 3.4	0.023
In-flight2	15.1 ± 3.8 vs. 7.1 ± 4.6	0.002
Nystagmus amplitude	**Mean ± SD**	**p**
Pre vs. In-flight 2	28.1 ± 15.2 vs 22.8 ± 15.8	0.410
Pre vs. In-flight 2	28.1 ± 15.2 vs. 25.7 ± 16.4	0.537
SMS vs. non-SMS		
Preflight	37.9 ± 16.7 vs. 23.3 ± 11.7	0.137
In-flight 1	31.3 ± 16.9 vs. 16.5 ± 11.3	0.095
In-flight 2	34.3 ± 13.5 vs. 19.2 ± 15.3	0.164

When comparing preflight data from SMS susceptible subjects with non-susceptible subjects, nystagmus frequency and total amplitude tended to be greater in the susceptible group, although the differences were not statistically significant ($p=0.067$ and 0.137, respectively). In a retrospective analysis of preflight data from the 16 subjects, individuals with more than 10 beats of nystagmus per two head oscillation cycles were "predicted" to have SMS during space flight, while those with less than 10 beats were "predicted" to be SMS-free (frequency test). Similarly, subjects with 30 degrees or more of cumulative nystagmus amplitude were assigned to the SMS category, and subjects with less than 30 degrees to the non-SMS category (total amplitude test). Overall, 11 of the 16 subjects were correctly assigned by the frequency method and 12 of 16 by the total amplitude method. Specificity and sensitivity of the frequency test were 67% and 80%, and 67% and 70%, for the total amplitude test, respectively.

Discussion

This study found that visual suppression of the VOR was not affected by space flight. In addition, subjects with SMS had a higher frequency of nystagmus during space flight than subjects who did not have SMS. Susceptible subjects also had a tendency toward higher nystagmus frequency before space flight. Postflight, although nystagmus frequency was unchanged from preflight, the total amplitude was decreased.

Most investigators quantify suppression of the VOR by computing gain (7,9,11-19). However, many of the EOG tracings during our VOR suppression studies lacked a sinusoidal pattern, and the limited nystagmus that was present did not permit adequate reconstruction of a sinusoid (see figure 2). In other words, VOR suppression was essentially complete and any gain calculation would have been close to zero.

During incomplete VOR suppression, the observed nystagmus represents errors induced by the VOR that take the eyes off the target and subsequent corrections by saccades to reacquire the target (23). We therefore chose to evaluate the adequacy of VOR suppression by determining the number of beats of nystagmus, the mean amplitude of each beat, and the cumulative amplitude of nystagmus summated over two complete head oscillation cycles.

The clinical and functional significance of our results is unclear. That VOR suppression is unaffected by space flight agrees with the results from the other in-flight study (20). However, VOR suppression may be less complete in individuals with SMS both during their symptomatic period and following recovery. This did not seem to affect any of the crewmembers, all of whom were able to execute without difficulty a variety of tasks requiring good visual and oculomotor performance.

Are the observed differences in VOR suppression a cause or an effect of SMS? On the one hand, retinal image slip due to inappropriate eye movements during head motion can elicit oscillopsia and cause motion sickness-like symptoms (24). Abnormal visual-vestibular interaction in weightlessness has been postulated to be a cause of SMS (1,25,26). However, it is unlikely that the incomplete VOR suppression seen in our study was a cause of SMS. First, the magnitude of the retinal slips during our studies was such that appreciable oscillopsia was unlikely (27). Indeed, in response to detailed questioning, none of the affected crewmembers reported it at any time. And second, the increased nystagmus frequency during VOR suppression persisted even after the affected subjects had recovered from SMS.

On the other hand, the mechanism of SMS is presumed to be of central nervous system (CNS) origin (1,25,26). Although a variety of CNS disturbances have been shown to cause impairment of VOR suppression (10,28,29,30), it is unlikely that any gross pathology was present in any of the subjects in this study. Could more subtle changes, such as altered CNS function caused by the cephalad redistribution of fluids in weightlessness (31) be responsible? This hypothesis is unlikely, since the fluid shift involves all individuals whether affected by SMS or not (32), and other space flight studies of CNS function have not shown any changes (33).

An intriguing result of this study was the finding that subjects who developed SMS in-flight tended to have more nystagmus during preflight VOR suppression tests than non-susceptible subjects. With regard to total nystagmus amplitude, this preflight difference correctly predicted the in-flight SMS status of 75% of the subjects.

Although this is the best single predictor for in-flight SMS that we are aware of, with the exception of previous SMS experience (34,35), caution must be exercised. The preflight differences in nystagmus frequency and total amplitude were not statistically significant; therefore, any predictive capability may be fortuitous. In addition, the lack of a constant visual background during our studies, a factor that may alter the effectiveness of VOR suppression (7), may have introduced variability into the results.

We have shown that space flight per se does not affect visual suppression of the VOR. In subjects who have SMS, suppression may not be as complete as in unaffected individuals. This difference between the two populations may serve as a potential indicator of preflight susceptibility to SMS, although the results in this study need to be repeated with standard visual backgrounds and a larger population. Further study of this phenomenon may shed light on the mechanism of SMS in particular and adaptation to the space flight environment in general.

Acknowledgements

The authors gratefully acknowledge the assistance of the following individuals, among others: Gen. James Abrahamson for fiscal and administrative support, Mr. Aaron Cohen and Dr. William Shumate for making this publication possible, Messrs. Hugh Harrington and Phil Gainer for technical aid, Mr. Henry Whitmore for hardware design and fabrication, Dr. Howard Schneider and Mr. Sid Jones for publication assistance, Dr. Cecil Hallum for statistical evaluation, and especially the crewmembers who participated in this study.

References

1. Thornton, W.E., Moore, T.P., Pool, S.L. and Vanderploeg, J. 1987. Clinical characterization and etiology of Space Motion Sickness. *Aviat Space Environ Med 58 Suppl.*, A1.
2. Thornton, W.E., Moore, T.P., Uri, J.J. and Pool, S.L. 1988. Studies of the vestibulo-ocular reflex on STS 4, 5, and 6. *NASA Technical Memorandum 100 461.*
3. Thornton, W.E., Uri, J.J., Moore, T.P. and Pool, S.L. 1988. Studies of the horizontal vestibulo-ocular reflex on STS 7 and 8. *NASA Technical Memorandum 100 468.*
4. Thornton, W.E., Uri, J.J., Moore, T.P. and Pool, S.L. 1989. Studies of the horizontal vestibulo-ocular reflex in spaceflight. *Arch Otolaryngol Head Neck Surg, 115,* 943.
5. Barnes, G.R., Benson, A.J. and Prior, A.R.J. 1978. Visual-vestibular interaction in the control of eye movement. *Aviat Space Environ Med 49*, 557.
6. Robinson, D.A. 1981. A model of cancellation of the vestibulo-ocular reflex. In: *Functional Basis of Ocular Motility Disorders*, Lennerstrand, G., Zee, D.S. and Keller, E.L. (eds), 5.
7. Koenig, E., Dichgans, J. and Dengler, W. 1986. Fixation suppression of the vestibulo-ocular reflex (VOR) during sinusoidal stimulation in humans as related to the performance of the pursuit system. *Acta Otolaryngol (Stockh) 102*, 423.
8. Schmid, R., Zambarbieri, D. and Magenes, G. 1981. Modifications of vestibular nystagmus produced by fixation of visual and nonvisual targets. *Ann NY Acad Sci 374*, 689.
9. Barr, C.C., Schultheis, L.W. and Robinson, D.A. 1976. Voluntary, non-visual control of the human vestibulo-ocular reflex. *Acta Otolaryngol 81*, 365.
10. Dichgans, J., vonReutern, G.M. and Rommelt, U. 1978. Impaired suppression of vestibular nystagmus by fixation in cerebellar and noncerebellar patients. *Arch Psychiatr Nervenkr 226*, 183.
11. Takahashi, M., Uemura, T. and Fujishiro, T. 1980. Studies of the vestibulo-ocular reflex and visual-vestibular interactions during active head movements. *Acta Otolaryngol 90*, 115.
12. Tomlinson, R.D., Saunders, G.E. and Schwarz, D.W.F. 1980. Analysis of human vestibulo-ocular reflex during active head movements. *Acta Otolaryngol 90*, 184.
13. Barnes, G.R. 1981. Visual factors affecting suppression of the vestibulo-ocular reflex. In: *Functional Basis of Ocular Motility Disorders*, Lennerstrand, G., Zee, D.S. and Keller, E.L. (eds), 387.
14. Jell, R.M., Guedry, F.E. and Hixson, W.C. 1982. The vestibulo-ocular reflex in man during voluntary head oscillation under three visual conditions. *Aviat Space Environ Med 53*, 541.
15. Barnes, G.R. 1983. The effects of retinal target location on suppression of the vestibulo-ocular reflex. *Exp Brain Res 49*, 257.
16. Larsby, B., Hyden, D. and Odkvist, L.M. 1984. Gain and phase characteristics of compensatory eye movements in light and darkness. *Acta Otolaryngol (Stockh) 97*, 223.
17. McKinley, P.A. and Peterson, B.W. 1985. Voluntary modulation of the vestibuloocular reflex in humans and its relation to smooth pursuit. *Exp Brain Res 60*, 454.
18. Jell, R.M., Stockwell, C.W., Turnipseed, G.T. and Guedry, F.E. 1988. The influence of active versus passive head oscillation, and mental set on the human vestibulo-ocular reflex. *Aviat Space Environ Med 59*, 1061.
19. Takahashi, M., Tsujita, N. and Akiyama, I. 1988. Evaluation of the vestibulo-ocular reflex by gaze function. *Acta Otolaryngol (Stockh) 105*, 7.
20. Viéville, T., Clement, G., Lestienne, F. and Berthoz, A. 1985. Adaptive modifications of the optokinetic and vestibulo-ocular reflexes in microgravity. In: *Adaptive Processes in Visual and Oculomotor Systems*, Keller, E.L. and Zee, D.S. (eds), 111.
21. Benson, A.J. and Viéville, T. 1986. European vestibular experiments on the Spacelab-1 mission: 6. Yaw axis vestibulo-ocular reflex. *Exp Brain Res 64*, 279.

22. Barber, H.O. and Stockwell, C.W. 1980. *Manual of Electronystagmography.* CV Mosby, St. Louis.

23. Zee, D.S. 1977. Suppression of vestibular nystagmus. *Ann Neurol 1*, 207.

24. Lackner, J.R. and Graybiel, A. 1983. Etiological factors in space motion sickness. *Aviat Space Environ Med 54*, 675.

25. Leigh, R.J. and Daroff, R.B. 1985. Space motion sickness: Etiological hypotheses and a proposal for diagnostic clinical examination. *Aviat Space Environ Med 56*, 469.

26. Grigoriev, A.I., Egorov, A.D. and Nichiporuk, I.A. 1988. Neurohumoral mechanism of space motion sickness. *Acta Astronautica 17*, 167.

26. Lackner, J.R. and Graybiel, A. 1983. Etiological factors in space motion sickness. *Aviat Space Environ Med 54*, 675.

27. Leigh, R.J., Rushton, D.N., Thurston, S.E., Hertle, R.W. and Yaniglos, S.S. 1988. Effects of retinal image stabilization in acquired nystagmus due to neurologic disease. *Neurol 38*, 122.

28. Halmagyi, G.M. and Gresty, M.A. 1979. Clinical signs of visual-vestibular interaction. *J Neurol Neurosurg Psychiatr 42*, 934.

29. Wennmo, C., Hindfelt, B. and Pyykko, I. 1983. Eye movements in cerebellar and combined cerebellobrainstem diseases. *Ann Otol Rhinol Laryngol 92*, 165.

30. Dayal, V.S., Mai, M. and Tomlinson, R.D. 1988. Vestibulo-ocular (VOR) abnormalities at high rotational frequencies in patients with Meniere's disease. *Otolaryngol Head Neck Surg 98*, 211.

31. Kornilova, L.N., Bodo, G. and Kaspransky, R.R. 1987. Sensory interaction in weightlessness. *Physiologist 30 Suppl*, S85.

32. Thornton, W.E., Moore, T.P. and Pool, S.L. 1987. Fluid shifts in weightlessness. *Aviat Space Environ Med 58 Suppl*, A86.

33. Thornton, W.E., Biggers, W.P., Pool, S.L., Thomas, W.G. and Thagard, N.E. 1985. Electronystagmography and audio potentials during space flight. *Laryngoscope 95*, 924.

34. Homick, J.L., Reschke, M.F. and Vanderploeg, J.M. 1987. Prediction of susceptibility to space motion sickness. In: *The Vestibular System: Neurophysiologic and Clinical Research*, Graham, M.D. and Kemink, J.L. (eds), 39.

35. Davis, J.R., Vanderploeg, J.M., Santy, P.A., Jennings, R.T. and Stewart, D.F. 1988. Space motion sickness during 24 flights of the Space Shuttle. *Aviat Space Environ Med 59*, 1185.

Appendix - Data Tables

Table A1.— Preflight VOR suppression nystagmus data.

	Frequency	Mean amplitude, degrees	Total amplitude, degrees
Subject 1			
L-28 days	22.0	3.6	79.2
L-14 days	11.0	1.5	16.5
Subject 2			
L-35 days	4.0	3.1	12.4
L-28 days	14.0	4.1	57.4
L-14 days	13.0	5.6	72.8
Subject 3			
L-70 days	4.0	2.2	8.8
L-47 days	4.0	1.4	5.6
Subject 4			
L-69 days	8.0	5.0	40.0
L-14 days	5.0	4.7	23.5
Subject 5			
L-47 days	10.0	2.5	25.0
Subject 6			
L-52 days	5.0	4.1	20.5
L-47 days	5.0	3.3	16.5
Subject 7			
L-39 days	4.0	2.1	8.4
L-37 days	5.0	1.4	7.0
Subject 8			
L-46 days	11.0	1.8	19.8
L-39 days	3.0	1.4	4.2
Subject 9			
L-39 days	10.0	1.6	16.0
L-32 days	14.0	2.3	32.2
Subject 10			
L-46 days	8.0	1.4	11.2
Subject 11			
L-46 days	16.0	1.0	16.0
L-44 days	22.0	2.1	46.2
L-37 days	14.0	0.6	8.4
L-33 days	9.0	0.8	7.2
Subject 12			
L-182 days	12.0	1.3	15.6
L-49 days	11.0	2.4	28.6
L-43 days	16.0	2.6	41.6
L-42 days	16.0	2.5	40.0
L-35 days	14.0	1.5	21.0
L-8 days	10.0	2.1	21.0
Subject 13			
L-182 days	12.0	1.7	20.4
L-42 days	8.0	3.4	27.2
L-35 days	6.0	3.5	21.0
L-14 days	11.0	2.2	24.2
L-8 days	8.0	2.2	17.6
Subject 14			
L-182 days	16.0	2.9	46.4
L-51 days	13.0	2.2	28.6
L-49 days	15.0	2.4	36.0
L-8 days	20.0	2.1	42.0
L-6 days	23.0	2.1	48.3
Subject 15			
L-182 days	7.0	3.7	25.9
L-49 days	10.0	3.4	34.0
L-42 days	12.0	7.8	93.6
L-41 days	7.0	3.7	25.9
L-14 days	9.0	3.5	31.5
L-7 days	9.0	2.6	23.4
Subject 16			
L-182 days	16.0	3.3	52.8
L-57 days	31.0	2.4	74.4
L-40 days(#1)	26.0	2.9	75.4
L-40 days(#2)	21.0	4.2	88.2
L-35 days	15.0	3.6	54.0
L-32 days	15.0	3.5	52.5
L-14 days	17.0	3.8	64.6
L-8 days	20.0	2.9	58.0
L-7 days	16.0	2.6	41.6

Table A2.— Frequency of nystagmus beats per two head oscillation cycles

Subject	Preflight	In-flight Mission Day 1	2	3	4	5	6	R+0D
1	16.5		9.0		10.0			11.0
2	10.3							3.2
3	4.0		7.0		6.0			8.0
4	6.5		2.0		13.0			
5	10.0		7.0		3.0			19.0
6	5.0		5.0		6.0			6.0
7	4.5		8.0	5.0				
8	7.0			3.0	5.0			
9	12.0		8.0	17.0			10.0	
10	8.0			16.0				
11	15.3	13.0	8.0	15.0	13.0	13.0	19.0	
12	13.2	13.0						16.0
13	9.0	0.0			7.0			8.0
14	17.4	24.0	16.0		14.0			
15	9.0	0.0				0.0		7.0
16	19.7		18.0		19.0			

Table A3.— Mean amplitude of nystagmus beats, in degrees

Subject	Preflight	In-flight Mission Day 1	2	3	4	5	6	R+0D
1	2.6		2.0		1.7			1.4
2	4.3							2.5
3	1.8		2.6		1.7			1.7
4	4.9		3.2		4.1			
5	2.5		3.4		3.5			1.3
6	3.7		4.8		6.1			3.0
7	1.8		4.4	4.5				
8	1.6			4.5	3.2			
9	2.0		1.6	2.9			3.3	
10	1.4			2.4				
11	1.1	1.5	2.9	1.6	1.8	2.0	1.8	
12	2.1	2.0						1.3
13	2.6	0.0			1.1			1.3
14	2.3	2.4	2.8		1.7			
15	4.1	0.0				0.0		2.2
16	3.2		2.0		2.8			

Table A4.— Cumulative nystagmus amplitude, in degrees

Subject	Preflight	In-flight Mission Day 1	2	3	4	5	6	R+0D
1	42.9		18.0		17.0			15.4
2	44.3							8.0
3	7.2		18.2		10.2			13.6
4	31.9		6.4		53.3			
5	25.0		23.8		10.5			24.7
6	18.5		24.0		36.6			18.0
7	8.1		35.2	22.5				
8	11.2			13.5	16.0			
9	24.0		12.8	49.3			33.0	
10	16.8			38.4				
11	16.8	19.5	23.2	24.0	23.4	26.0	34.2	
12	27.7	26.0						20.8
13	23.4	0.0			7.7			10.4
14	40.0	57.6	44.8		23.8			
15	36.9	0.0				0.0		15.4
16	63.0		36.0		53.2			

REPORT DOCUMENTATION PAGE

1. Report No. TM 102 157	2. Government Accession No.	3. Recipient's Catalog No.
4. Title and Subtitle Visual suppression of the vestibulo-ocular reflex during space flight		5. Report Date August 1989
		6. Performing Organization Code
7. Author(s) John J. Uri, William E. Thornton, Thomas P. Moore, Sam L. Pool		8. Performing Organization Report No. S-598
9. Performing Organization Name and Address Lyndon B. Johnson Space Center Houston, Texas 77058		10. Work Unit No. 073-36-00-00-72
		11. Contract or Grant No.
12. Sponsoring Agency Name and Address National Aeronautics and Space Administration Washington, D.C. 20546		13. Type of Report and Period Covered TM August 1989
		14. Sponsoring Agency Code

15. Supplementary Notes

John J. Uri, GE Government Services; William E. Thornton, Lyndon B. Johnson Space Center; Thomas P. Moore, Methodist Hospital of Indiana; Sam L. Pool, Lyndon B. Johnson Space Center.

16. Abstract

Visual suppression of the vestibulo-ocular reflex was studied in 16 subjects on four Space Shuttle missions. Eye movements were recorded by electro-oculography while subjects fixated a head-mounted target during active sinusoidal head oscillation at 0.3 Hz. Adequacy of suppression was evaluated by the number of nystagmus beats, the mean amplitude of each beat, and the cumulative amplitude of nystagmus during two head oscillation cycles. Vestibulo-ocular reflex suppression was unaffected by space flight. Subjects with space motion sickness during flight had significantly more nystagmus beats than unaffected individuals. These susceptible subjects also tended to have more nystagmus beats before flight.

17. Key Words (Suggested by Author(s)) Electro-oculography Space flight Space motion sickness Vestibulo-ocular reflex VOR suppression		18. Distribution Statement Unclassified - Unlimited Subject Category: 52	
19. Security Classification (of this report) Unclassified	20. Security Classification (of this page) Unclassified	21. No. of pages 12	22. Price

For sale by the National Technical Information Service, Springfield, VA 22161-2171