the two subjects who were tested on the evening of the return performed very accurately using only nonvisual cues. Their performance far exceeded that before flight and approached their accuracy for the task with full visual cues. This ability decayed gradually over the week of postflight testing. Dynamic ocular counterrolling, measured during lateral sinusoidal oscillation at 0.6 *g* (0.42 and 0.83 Hz), appeared to be reduced in gain on R + 0.

Discussion. The preliminary nature of these findings makes discussion necessarily speculative. Nevertheless, all of the major findings are consistent with the principal hypothesis: during adaptation to weightlessness the nervous system reinterprets signals from the graviceptors (primarily the otolith organs) to represent fore-aft or left-right linear acceleration, rather than pitch or roll of the head with respect to the vertical. Maintenance of this reinterpretation during the postflight period is maladaptive, resulting in postural instability with eyes closed, increased reliance on visual information for orientation, and improved ability to null lateral linear motion.

Independent refinement of the otolith reinterpretation hypothesis was proposed by Parker *et al.* (*26*) to explain their findings with STS-8 and STS-11 astronauts. Self-motion reports and eye reflexes during roll motion showed primarily linear translation and reduced ocular counterrolling after flight, relative to before launch. The adaptation is presumably not reflected at the more peripheral end organ responses or in fast reflex loops such as the otolith-spinal reflex. One consequence of the presumed linear acceleration sensor reinterpretation in flight is the increased use of local visual cues for spatial orientation and, at least early in the flight, increased attention to tactile and proprioceptive information on both body orientation and sense of body movement.

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References and Notes

1. L. Young, C. Oman, K. Money, D. Watt, *Scientific and Technical Proposal for Vestibular Experiments in Spacelab* (Man-Vehicle Laboratory, Massachusetts Institute of Technology, Cambridge, 1976), described by L. R. Young, in *Space Physiology* [CNES (Cepaudes Editions), Toulouse, 1983]; C. M. Oman, *SAE (Soc. Automot. Eng.) Tech. Pap. 820833* (July 1982).
2. Lead investigator, L.R.Y. Performed by A and B on mission days (MD's) 1, 2, 4, 5, and 7 and by C and D on MD's 3 and 6 (MD 0 was the first day of the mission).
3. J. Dichgans, R. Held, L. R. Young, T. Brandt, *Science* **178**, 1217 (1972).
4. L. R. Young, C. M. Oman, J. Dichgans, *Aviat. Space Environ. Med.* **46**, 264 (1975); J. Dichgans *et al.*, *Acta Otolaryngol.* **78**, 391 (1974).
5. Specially marked soft contact lenses to aid visualization of ocular torsion were adhered with distilled water. Lens preparation by L. Meshel; lenses by Bausch and Lomb and Ocu-ese; fluids by Alcon. Eye examinations by W. Lipsky, L. Meshel, and A. Cavallerano.
6. Lead investigator, C.M.O. Additional details were given by C. Oman, B. Lichtenberg, and K. Money in paper 35, presented at the NATO-AGARD/AMP Symposium on Motion Sickness, Williamsburg, Va., 3 May 1984; O. Bock and C. Oman, *Aviat. Space Env. Med.* **52**, 773 (1982).
7. T. Maitland, *Br. Med. J.* **1**, 171 (1931); J. Reason and J. Brand, *Motion Sickness* (Academic Press, London, 1975), p. 75.
8. Lead investigator, K.E.M.
9. J. Homick, *Acta Astronaut.* **6**, 1259 (1979).
10. J. M. Lentz, C. Hottyman, W. Hixson, F. Guedry, *NAMRL (Nav. Aerosp. Med. Res. Lab., Pensicola, Fla.) Rep. 1243 AP* (1977).
11. Results from tests of adaptation to head movements while wearing reversing prisms for several hours appear more promising, and the tests will be repeated on future missions.
12. Lead investigator, D.G.D.W. Performed in flight by A and B on MD's 0, 1, and 6. Results of the inflight falls as well as hopping and stepping are being analyzed.
13. G. M. Jones and D. G. D. Watt, *J. Physiol. (London)* **219**, 729 (1971).
14. D. G. D. Watt, *J. Neurophysiol.* **39**, 257 (1976).
15. M. Lacour, C. Xerri, M. Hugon, *J. Physiol. (Paris)* **74**, 427 (1978).
16. R. Greenwood and A. Hopkins, *J. Physiol. (London)* **254**, 507 (1976); R. Greenwood and A. Hopkins, *Brain* **99**, 375 (1976).
17. S. B. Backman and D. G. D. Watt, paper presented at the Society for Neuroscience Annual Meeting, Atlanta, Ga., November 1979; D. G. D. Watt and S. B. Backman, paper presented at the Canadian Federation of Biological Societies Annual Meeting, St. John's, Newfoundland, June 1980.
18. D. G. D. Watt and L. Tomi, paper presented at the Society for Neuroscience Annual Meeting, Boston, Mass., November 1983.
19. Lead investigator, K.E.M. Performed on MD's 1, 6, and 7.
20. Lead investigator, L.R.Y. with R. Kenyon, A. Arrott, S. Modestino, R. Renshaw, and M. Shelhamer.
21. J. L. Homick and M. F. Reschke, *Acta Otolaryngol.* **83**, 455 (1977); M. F. Reschke, D. J. Anderson, J. L. Homick, *Science* **225**, 212 (1984).
22. J. T. Baker, A. E. Nicogossian, G. W. Hoffer, R. L. Johnson, J. Hordinsky, in "Biomedical results from Skylab," *NASA Spec. Publ. SP-377* (1977), p. 131.
23. S. E. Asch and H. A. Witkin, *J. Exp. Psychol.* **38**, 455 (1948).
24. R. von Baumgarten *et al.*, *Science* **225**, 208 (1984).
25. G. M. Jones and L. R. Young, *Acta Otolaryngol.* **85**, 45 (1978).
26. D. Parker, M. Reschke, A. Arrott, J. Homick, B. Lichtenberg, *DSO-0433* (Space Biomedical Research Institute, NASA–Johnson Space Center, Houston, Texas, 1983); *STS-11* (Space Biomedical Research Institute, NASA–Johnson Space Center, Houston, Texas, 1984).
27. We gratefully acknowledge the outstanding cooperation of the Spacelab 1 science crew and support from NASA (NAS 9-15343), the Defense and Civil Institute of Environmental Medicine (Canada), and the Medical Research Council (Canada). We are grateful for the guidance and assistance of G. M. Jones, F. Guedry, A. Weiss, R. Donahue, and especially R. Clark and G. Salinas. We also thank W. Mayer, project manager, and the staff of the Laboratory for Space Experiments at the MIT Center for Space Research. A. Arrott directed the sled protocols and R. Renshaw coordinated the MIT activities in the Baseline Data Collection Facility (BDCF); S. Modestino ran the rails and the rod and frame tests, R. Kenyon the posture platform, and M. Shelhamer the rotating dome experiment. BDCF tests were performed 152, 122, 65, 44, and 10 days before flight, on the day of landing, and 1, 2, 4, and 6 days after return. Several eye movement experiments require further data processing and are not covered in this report. The horizontal vestibulo-ocular reflex was measured during angular oscillation, post rotatory-nystagmus and pitch down nystagmus dumping. Ocular torsion was recorded during eccentric *z*-axis sinusoidal angular oscillation performed at high and low frequencies.

27 March 1984; accepted 17 May 1984

Effects of Rectilinear Acceleration and Optokinetic and Caloric Stimulations in Space

Abstract. *During the flight of Spacelab 1 the crew performed a number of experiments to explore changes in vestibular function and visual-vestibular interactions on exposure to microgravity. Measurements were made on the threshold for detection of linear oscillation, vestibulo-ocular reflexes elicited by angular and linear movements, oculomotor and posture responses to optokinetic stimulations, and responses to caloric stimulation. Tests were also conducted on the ground, during the 4 months before and on days 1 to 6 after flight. The most significant result was that caloric nystagmus of the same direction as on the earth could also be evoked in the weightless environment.*

During the European vestibular experiments on the Spacelab 1 mission (experiment 1ES201) crew members performed experiments to explore the effects of exposure to microgravity on vestibular function and visual-vestibular interaction. Tests were also conducted on the ground during the 4 months before and on days 1 to 6 after the flight.

The flight hardware included the vestibular helmet and a collapsible seat and backrest, the body restraint system (BRS) in which the subject was secured by a harness in the yogi position. The helmet was equipped with electro-oculography (EOG) amplifiers and a CCD camera with infrared illumination (EMIR) in front of the right eye. The EMIR device allowed eye movements to be computed in real time for an *x-y*

display on the ground. A television monitor in front of the left eye could be used to provide optokinetic stimuli as well as a movable target cross, which was also used for EOG and EMIR calibration. Caloric stimulation of both ears was provided by insufflation of heated or cooled air at a preselected and controlled temperature.

Caloric nystagmus in microgravity. Since the last century it has been known that irrigation of the ear canals with water at a temperature lower or higher than body temperature causes nystagmus—involuntary rapid eye movements (*1*). According to Barany (*2*), the temperature change in the temporal bone causes circulation of the fluid within the membranous labyrinth by thermal convection. This circulation of endolymph in the semicircular canals in turn deflects the cupulae and thus stimulates the sensory cells in the ampullae.

Several authors doubted that thermal convection is sufficient to explain caloric nystagmus, citing (i) a less than satisfactory relation between nystagmus strength and position of the canals (*3*), (ii) maintenance of a response even after experimental plugging of the canals or after labyrinthectomy (*4*), or (iii) insufficient pressure differences on both sides of the cupula (*5*). With the microgravity in space it is possible to test the convection hypothesis directly, since in weightlessness thermal convection cannot take place.

Caloric tests were performed on astronauts C and D during flight as well as before and after flight. Each test consisted of static and dynamic (linear oscillations of the subject in the x-axis, 0.2 g, 0.3 Hz) phases. The stimulus conditions (*6*) involved simultaneous cooling of one ear and heating of the other.

On mission days 0 and 2, caloric nystagmus of both tested crew members was detectable but insufficient to be evaluated quantitatively. On mission days 7 and 8, a well-defined nystagmus slow-phase velocity of 19° per second for D and 5° per second for C was elicited, with the direction of the quick phase toward the warmer ear (Fig. 1). When the thermal stimulus was reversed, the direction of nystagmus for both subjects reversed within a few seconds. It is noteworthy that the response evoked in flight for different temperature settings did not differ significantly in magnitude from those before and after flight (Fig. 2), although the head position on the sled in the ground tests was not conventional for maximal response.

The presence of caloric nystagmus in microgravity demonstrates that mechanisms other than thermal convection must be responsible. It appears possible that caloric nystagmus on the ground is partially or fully caused by the same mechanisms (that is, without thermal convection). Further experimentation is needed to resolve this important question. The identity of nystagmus direction (toward the warmer ear), and the similarity of nystagmus strength at different temperature settings in orbit and before and after flight on the ground, suggest a common mechanism for caloric nystagmus. However, any common explanation must account for the observation that the direction of caloric nystagmus reverses with head inversion. The suppression of caloric nystagmus during the early phase of spaceflight is well correlated with its suppression in parabolic weightless flight. Although this could also have been caused by the drug taken by one of our two subjects to prevent motion sickness, suppression both early in microgravity and at certain positions on the ground as well as reversal in an upside-down position could be caused by changes in otolith activity.

Threshold. Threshold measurements for linear oscillatory motion at 0.3 Hz in the x, y, and z body axes were made both in flight and on the ground. For pre- and postflight tests, we used a small horizon-

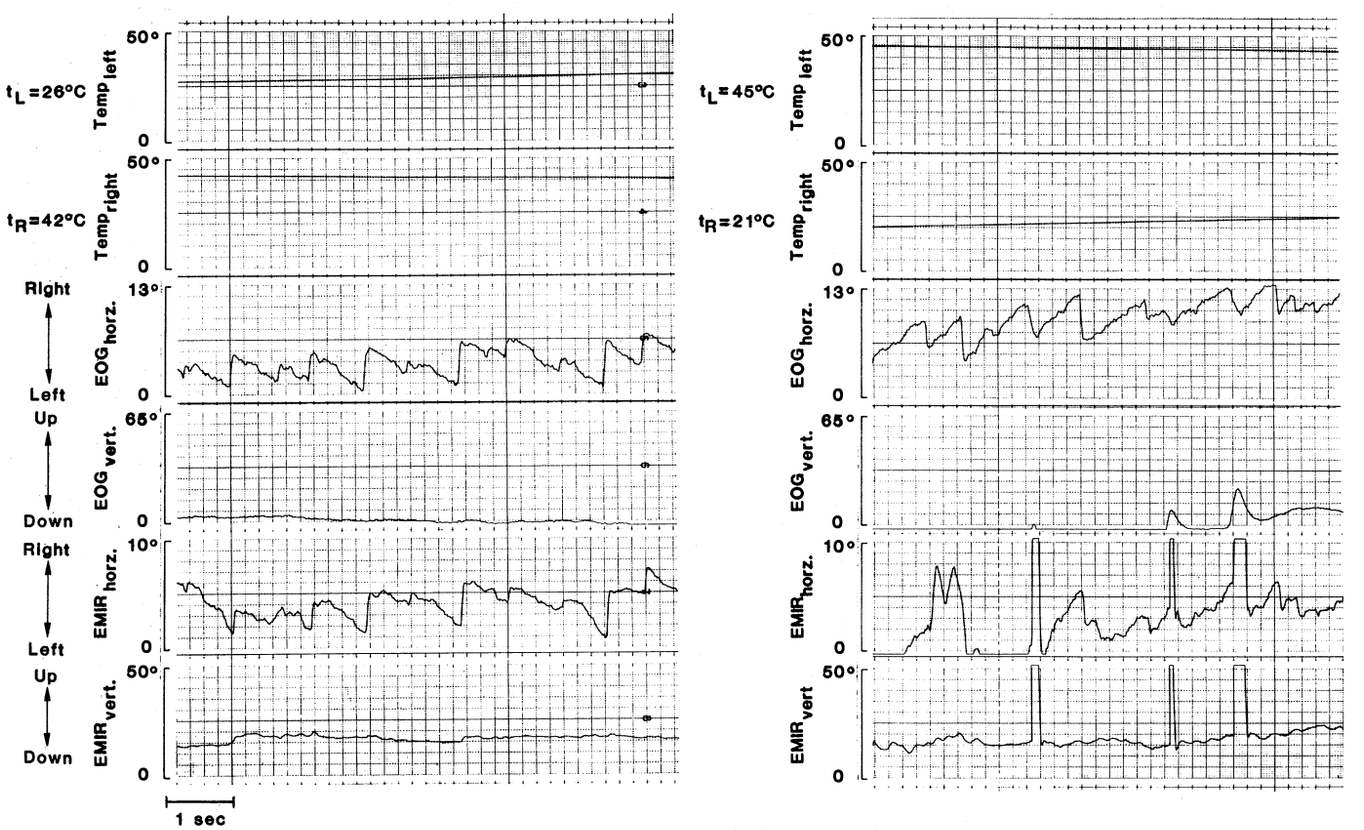

Fig. 1. Effect of inflight caloric stimulation of subject C on day 7.

tal linear oscillator with air bearings to minimize adventitious motion cues. The continuous oscillatory stimulus increased or decreased in intensity by 0.5 dB per half cycle according to the subject's responses. Two test procedures were employed. In mode 1 the subject indicated, by means of a key switch, when he could detect the oscillatory motion; in mode 2 he had to signal the direction of the perceived motion. In flight an attempt was made to replicate the ground-based procedure by the use of the body restraint system. The subject was moved in an approximately sinusoidal manner at 0.3 Hz by the other crew member.

Baseline measures were reasonably stable, with z-axis thresholds (subject lying on his back) consistently ($P < 0.001$) higher than those obtained with x- and y-axis stimuli. The mean thresholds of all four subjects before and after flight are shown in Fig. 3. Note the strong reduction in the z-axis on day R + 1. Analysis of variance indicated that the responses of subjects C and D differed significantly from those of A and B (who were on a different shift), particularly in the immedate postflight period. Significant findings in C and D are an elevation ($P < 0.05$) of x- and y-axis thresholds on day R + 1 (tested 14 hours after landing) with return to preflight levels by R + 2, and no change in z threshold. In contrast, A and B exhibited a reduction ($P = 0.01$) in y- and z-axis thresholds on R + 1 with return to baseline on R + 2 and P + 6, while z-axis threshold were elevated ($P = 0.05$) on R + 4. The data obtained with procedure 2 were used to assemble frequency of detection ogives, from which thresholds were computed. The z-axis thresholds exceeded those for x and y (*7*). In general, these values were correlated with those obtained by procedure 1 and, in particular, confirmed the reduction of z-axis threshold in A and B on R + 1.

The inflight threshold data are at present fragmentary. Preliminary values obtained with procedure 1 on days 4 and 6 are: x-axis: 6.8, 4.7, and 3.5; y-axis: 8.3, 5.9, and 7.0; and z-axis: 9.1×10^{-2} m/sec^2. Each of these values is higher than those obtained before flight. The elevation of threshold in flight and immediately after flight is in accord with data obtained after flight for one member of the crew of STS-8 (*8*) and indicates that one facet of the adaptation to microgravity is a reduction in the gain of the sensory system processing signals from vestibular and somesthetic gravireceptors.

Angular vestibulo-ocular reflex. Angular stimulation was achieved with a hand-driven oscillation of the test subject at approximately 0.3 and 1 Hz. The x- and y-axis of his head was aligned with the rotation axis, which was either coaxial with the head (centric mode) or 1 m distant (eccentric mode). In the latter orientation the vestibular stimulus was not only a changing angular acceleration but also a changing tangential and radial acceleration. Further tests were conducted in the z eccentric mode with the subject fixating on a head-fixed visual target. Eye movements were recorded in darkness by EMIR, closed circuit television, and two-channel EOG. Turntable angular velocity was also recorded.

Data obtained during the preflight test sessions (120, 64, 43, and 11 days before flight) failed to show any significant difference between the vestibulo-ocular reflex (VOR) responses evoked by centric and eccentric oscillation in darkness, although, as was to be expected (*9*), the VOR gain at 1 Hz (mean, 0.65; range, 0.51 to 0.79) was higher than at 0.3 Hz (mean, 0.33; range, 0.20 to 0.46). The values obtained 1, 2, 4, and 6 days after flight were comparable to the preflight

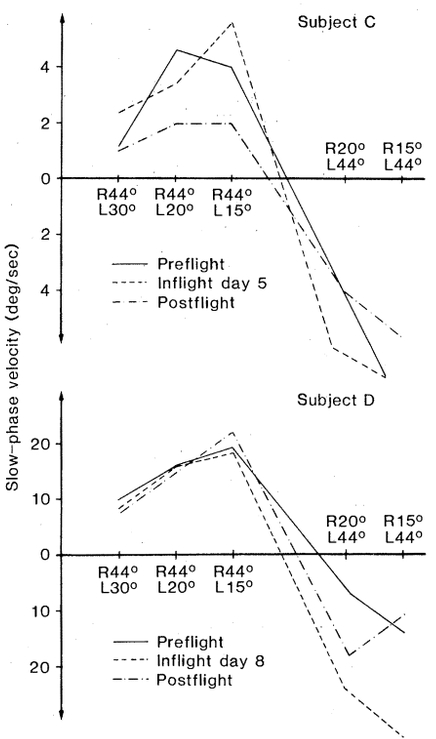

Fig. 2. Slow-phase velocity of caloric nystagmus before, during, and after flight for crew members C and D at different temperature settings. [Inflight tests on mission day 1 (for D) and mission day 2 (for C) produced only inconsistent nystagmus and are not included on the figure.]

measures, the mean VOR gain being 0.58 (range, 0.50 to 0.62) at 1 Hz and 0.32 (range, 0.22 to 0.55) at 0.3 Hz. The responses obtained in darkness on the first day after flight were within the range of preflight values. In contrast, the gain of the VOR at 1 Hz, when reduced by the presence of a single collimated fixation target (subtending approximately 5 mrad at the subject's right eye), was significantly ($P < 0.05$) lower on R + 1 than on any other pre- or postflight day. The mean suppressed VOR gain before flight was 0.38 (range, 0.32 to 0.44) and after flight was 0.23, 0.37, 0.37, and 0.36 on days R + 1, 2, 4, and 6, respectively (*10*).

If this observation is confirmed by future experiments, it suggests that one aspect of adaptation to microgravity is an increased dependence on visual as opposed to vestibular mechanisms in the stabilization of the retinal image during head movement.

It is extremely difficult on the ground to assess the exact contribution of the otoliths to the vertical VOR. However, microgravity is a privileged situation because the static otolithic component is absent. The hypothesis is that in space the gain will be lower and phase will be changed. On Spacelab 1 this experiment was conducted during the oscillopsia test. The subject was asked to shake his head, with eyes open, at 1 Hz in pitch, roll, and yaw successively. Head movement was measured with linear accelerometers and eye movements recorded by EOG and EMIR. When the subject reported no oscillopsia with his eyes open, it was assumed that head and eye movements were nearly the same (gain of VOR ~1). Head oscillation was then repeated at the oscillopsia threshold with the eyes closed. By comparing the values of eye movements for eyes open and eyes closed during similar head movements, we obtained an approximate value for the VOR gain in microgravity.

The subject was asked to imagine the target fixed to the Spacelab wall. Two methods of analysis were used to determine the VOR gain: (i) the peak-to-peak values of the EOG were compared between head oscillations of comparable amplitude and frequency, and (ii) the amplitudes of the eye displacements were computed from the reconstructed sinusoidal component for 20 cycles after elimination of saccades. Both methods showed a significant difference in the gain of the pitch VOR between ground tests [gain, 0.9; standard deviation (S.D.), 0.1] and tests in flight on days 5 and 7 (gain, 0.75; S.D., 0.15) for subjects

C and D. The phase analysis corroborates this apparent otolithic contribution to the vertical VOR. Whereas on the ground the phase error was small (mean, 1°; S.D., 0.3°), in microgravity compensatory eye movement caused remarkable phase lag (mean, 20°; S.D., 8°).

These preliminary results suggest that on the ground, the otoliths make a significant contribution to the gain of the VOR in the pitch axis. The observation, in space, that head movements in pitch are particularly nauseating may be explained by the lack of gravity-related otolithic components which normally contribute to the visual vestibular stabilization of gaze in this axis.

Ocular counterrolling tests. Two color photographs of the subject's left eye were made 15 and 20 seconds after positioning during all pre- and postflight test sessions at body tilt angles ranging from 0° (upright position) to 90° left and right. The angular deviation of the eye in roll, relative to the canthi, was determined from the projected image of the color transparencies. After flight, subjects B, C, and D showed significantly less torsion than before flight when tilted to the left (Fig. 4). There was also increased asymmetry between left and right tilt after flight. These findings seem to agree with counterrotation data after the STS-8 flight (*9*). Measurements on A did not reveal major differences between pre- and postflight values. The average standard error for all measurements is ±0.55°, using twice the standard deviation, with a mean of 1.71°.

Luminous line test. Luminous line tests were performed before and after flight tests by comparing the indicated subjective vertical (SV) with the true vertical at different tilt angles of the body. The most significant change occurred in subject A, whose deviation of the SV from the true vertical at 45° body tilt was almost 20° after flight. In addition, all subjects exhibited considerably more bilateral asymmetry when comparing SV at left and right body tilts.

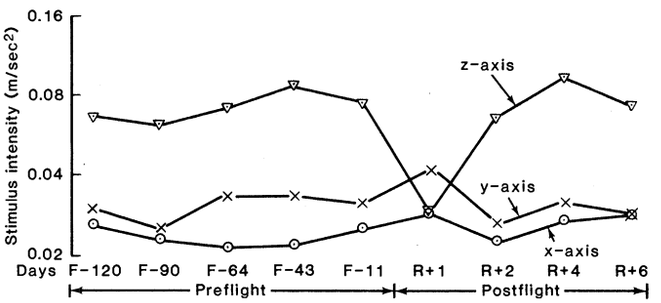

Fig. 3. Mean thresholds of four subjects for detection of horizontal linear oscillation at 0.3 Hz, mode 1: detection of motion, mean of upper and lower values.

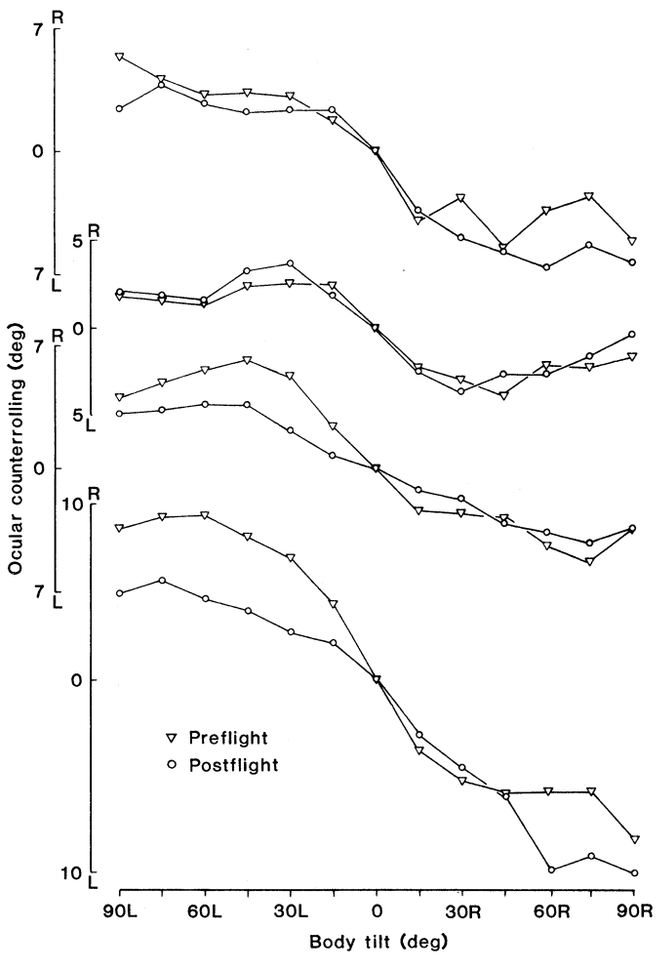

Fig. 4. Mean values of ocular counterrolling plotted against body tilt for four crew members before and after flight.

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References and Notes

1. B. Baginsky, *Du Bois' Archiv* (1881), p. 201.
2. R. Barany, *Physiologie und Pathologie des Bogengang-Apparates beim Menschen* (Franz Deuticke-Verlag, Leipzig, 1907).
3. A. C. Coats and S. Y. Smith, *Acta Oto-Laryngol.* **63**, 515 (1967).
4. B. E. Gernandt, M. Igarashi, H. W. Ades, *Exp. Neurol.* **14**, 249 (1966).
5. A. F. Ludin, *Opusc. Med.* **8**, 4 (1963).

6. Temperature settings during the caloric experiments in flight were 37°C in each ear at the start of the experiment. Thereafter, the temperature settings in the right and left ears, respectively, were 44° and 30°; 44° and 20°; 44° and 15°; 20° and 44°; and 15° and 44°. For each pair of values the static phase was 2 minutes and the dynamic phase 30 seconds.
7. G. M. Jones and L. R. Young, *Acta Oto-Laryngol.* **85**, 45 (1978).
8. D. Parker, personal communication.
9. A. J. Benson, in *Life Sciences Research in Space* (ESA SP-130, European Space Agency, Paris, 1977), pp. 101–108.
10. Inflight measurements of angular vestibulo-ocular reflex are not yet available.
11. These experiments were sponsored by the German Department of Research and Technology and by the French Centre Nationale d'Etudes Spaciales.

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Vestibulospinal Reflexes as a Function of Microgravity

Abstract. *The idea that sustained microgravity will result in altered otolith input requiring a modification of postural control was explored by using the pathway that links the otolith organs and spinal motoneurons. Two related methods were used. First, the Hoffmann reflex was used to measure at specific times the excitability of the soleus-spinal motoneuron pool during a brief unexpected linear acceleration. Second, extensive dynamic postural testing with a moving platform was done before and after flight. The Hoffmann reflex amplitude, reflecting otolith-modulated motoneuron sensitivity, was low in flight after adaptation, and its postflight potentiation may have been dependent on rate of adaptation. The strength of inflight motion sickness symptoms was related to postflight Hoffmann reflex amplitude. Dynamic posture tests showed significant deviations from the results obtained before flight. The strategy used for balance on the moving platform was modified, and the behavior of the subjects suggested a decrease in awareness of the direction and magnitude of the motion.*

Data from previous manned spaceflights suggest that significant alterations occur in vestibular, neuromuscular, and related sensory system function on exposure to microgravity (*1*). The observed changes may be a function of adaptation induced by altered otolith input. The purpose of this Spacelab 1 experiment was to investigate this adaptation as it occurred in flight and after flight, and to relate the observed changes to mechanisms underlying space motion sickness.

To explore this concept, we used the anatomic pathway that links the otolith organs and spinal motoneurons. The overall sensitivity of the spinal motoneurons was tested by two related methods: (i) electrical excitation of neural tissue and recording of vestibulospinal reflexes in conjunction with a brief linear acceleration (*2*) and (ii) measurements of dynamic postural ataxia.

Vestibulospinal reflex mechanisms. Excitation of the neural tissue was accomplished by the classical Hoffmann reflex or H reflex technique (*3*). The H reflex was obtained through elicitation of a monosynaptic reflex recorded from the soleus muscle by electrical stimulation of large group I afferent fibers in the popliteal nerve. A needle electrode, which served as the cathode, was inserted in the popliteal fossa at a permanently marked (tattoo) location on the right leg. The anode, a plate electrode, was secured over the patella. A 1-msec constant current pulse limited to a maximum of 20 mA was delivered through an isolation unit under computer control. A differential bipolar electrode configuration was used to record the reflex from the soleus muscle. The reflex was a two-part response: a direct orthodromic muscle response (M wave) with a latency of 5 to 10 msec that was followed 15 to 20 msec later by the monosynaptic H reflex. Because the M wave represented a direct muscle response, it was used as a control during vestibular stimulation. The H reflex amplitude reflected the sensitivity of the spinal motoneurons as set by the descending postural control signals.

Vestibular stimulation during preflight and postflight testing was provided by dropping the subject over a short distance (10 to 14 cm), using a quick-release helicopter cargo hook. In flight, we used the Canadian "hop and drop station" (*4*) and calibrated bungee cords to pull the subject to the floor of Spacelab. All drops were under computer control (*5*).

For each drop, the subject was shocked three times. The first shock, used to condition the neural tissue, was followed 3 seconds later by a shock that elicited a control response, and the third (3 to 5 seconds later) was delivered at predetermined delay times during the drop. Before and after flight these drop-to-shock delay times ranged in 10-msec increments from a shock that was coincident with the computer command to drop, to a shock that was delayed 80 msec. In flight, eight shock-to-drop delays ranging from 0 to 70 msec in 10-msec increments were used. Approximately four responses (*6*) were collected at each viewpoint during each test period (that is, on F − 10, F + 24 hours, R + 0, and so on). This method permitted tracking of the H wave amplitude as a function of both time (shock delay) and acceleration across test days.

For analysis, the H reflex response to each drop was normalized as a difference percentage with respect to the control H reflex. To obtain a stable reference response, the control H reflex responses were averaged for each crew member for all preflight, inflight, and postflight test periods. The preflight drop H reflex responses for each drop-to-shock delay were then averaged ($N = 180$) for each crew member.

The H reflex results for a single crew member (subject A) are presented in Fig. 1. Drop H reflex amplitude expressed as a difference from the control H reflex is plotted as a function of drop-to-shock delay and test period. The drop-to-shock delay times were adjusted to account for a 20-msec delay between the computer command to drop and the time of the actual fall. Data for this crew member are representive of general trends found with the shape of the drop-to-shock H reflex potentiation curve but not of peak amplitude changes found between subjects. Before flight there was an initial potentiation of the H reflex in response to the drop for shocks delayed 20 to 40 msec. Peak amplitudes for this subject occurred with drop-to-shock delays of 80 msec. Inflight H reflex peak amplitudes recorded at a mission elapsed time of 24 hours showed a gain analogous to the preflight values. By mission day (MD) 7, the H reflex amplitude was low and showed little or no change as a function of drop-to-shock delay times.

After flight, a general potentiation was recorded. The amplitude for subject A on R + 0 showed an approximately 30-fold gain. The R + 1 and R + 2 values indicated amplitudes lower than that obtained on R + 0. However, on R + 4 and R + 6 a potentiation greater than that recorded on R + 0 occurred. This observation may have been due to an intervening parabolic flight on R + 3. A two-way analysis of variance for repeated measures on these data showed a significant difference as a function of test day ($P < 0.001$). The M wave amplitude changes were not significant when subjected to the same two-way analysis of variance ($P < 0.50$), confirming that a constant electrical stimulus was applied and that H reflex amplitude changes represent the state of excitement within the spinal motoneuron pool.