GUEST EDITORIAL

SPACE AND THE VESTIBULAR SYSTEM:
WHAT HAS BEEN LEARNED?

In the 35 years since the dawn of the space age, the view of vestibular function in weightlessness has changed considerably. At first it was considered likely that the unusual agravic condition of space flight would "functionally deaffent" the otolith organs, and, consequently, completely disrupt spatial orientation and produce intolerable motion sickness. In fact, except for some early Soviet reports of "space sickness," the operational problem appeared minor. Not until the Apollo program, in the late 1960s, did the problem of space sickness—or "space adaptation syndrome"—clearly emerge as an operational issue recognized by both space programs. Despite the seeming relationship between substantial astronaut movement and space sickness, the involvement of the vestibular system in its etiology remained in question. Meanwhile basic research on vestibular reactions to space flight was limited to simple behavioral observations on fish and higher animals, a single frog centrifugation, and some postflight otoconia examination of rats that may have been damaged by the landing.

In the past few years, however, vestibular research has moved onto center stage in Space Life Sciences. The speculations discussed at Ashton Greybiel's Pensacola symposium on "The Role of the Vestibular Organs in the Explorations of Space" over 25 years ago have largely been tested in a series of human and animal orbital experiments. To mark this rapid progress, a special symposium was organized at the 1992 Barany Society meeting in Prague—one which might have been termed "The Role of Space in the Exploration of the Vestibular System." By way of introduction and summary, we developed a personal review of the state of the art—what we know and what we don't know about space vestibular function. This editorial repeats the main points of that summary—to introduce the main points to those in the vestibular community not conversant with space research. Many colleagues generously contributed their critique of this summary (see Acknowledgments), but the responsibility for the judgements rests with the author. This is not a review article, but an editorial, and references are deliberately not included.

1. Space Motion Sickness (SMS)

WE KNOW that most space travellers (about two-thirds) will experience symptoms ranging from headache and stomach awareness to nausea and vomiting, beginning shortly after entry to orbit. Although the symptoms are sometimes brought on by a view of an unusual scene (like an inverted crew mate, or seeing the earth at the top of the window), the problem is normally brought on by head movements, particularly in the pitch plane (and therefore justifies the name space "motion" sickness). The occurrence of spatial orientation illusions, including the inversion of the subjective visual vertical, have been associated with space sickness; but do not appear to correlate very well. As more detailed data becomes available, it appears that no significant difference in space sickness occurs between men and women. Symptoms are no significantly reduced on a refight, although some benefit and strategies for dealing with postural instability may carry over to subsequent flights. The current favorite drug treatment is intramuscular injection of promethazine, rather than the use of scopolamine or other prophylactic medications. Its effectiveness and side effects are still under investigation.

WE DON'T KNOW a reliable and validated predictor of space sickness, although...
measurements of the subjective vertical and of disconjugate eye torsion have correlated with past incidents of SMS. (One of the difficulties is that we have never measured SMS susceptibility, but only occurrence, since head movements among other activities are neither controlled nor adequately measured.) The influence of fluid shift toward the head during weightlessness is not known regarding SMS, nor is the possible influence of otolith organ asymmetry in possibly inappropriate compensation understood. The generalization of the habituation to weightlessness is not known. For example, one cannot predict the SMS problems that would exist in an artificial gravity rotating spacecraft. Finally, the sensory-motor conflict theory of motion sickness needs development and validation to cover all sources of SMS.

2. Vestibulo-Ocular Reflexes (VOR)

WE KNOW that the gain of the VOR for rotational head movements can change in microgravity, even during short parabolic flights; but the nature and time course of the changes is not fully known. Some evidence supports an initial increase in gain (and reduction in head movement threshold) in weightlessness, followed by a prolonged inhibition following several days in space. The time constants of decay of postrotational nystagmus appear to be shortened on orbit, reflecting a possible loss of some “velocity storage.” Vertical eye movements, including pitch VOR and vertical pursuit, seem to be more disturbed by weightlessness than are horizontal movements. Spontaneous nystagmus appears and disappears periodically during long flights. The importance of gravity in the “dumping” of postrotational nystagmus has been established, but the explanation for the dumping of subjective responses in 0-g is unproven. Caloric responses exhibit a nonconvective component (beating toward the warm ear), but the mechanism for it has not been established. Some oscillosia has been reported on entry.

WE DON’T KNOW precisely how the VOR gains vary, or if they are even adaptive; nor is the relationship of these gains in weightlessness to oscillosia during and following entry established. Similarly, the details of changes in optokinetic nystagmus thresholds and range remain to be determined definitively.

3. Perception of the “Vertical” in 0-g

WE KNOW that some egocentric “vertical” reference is maintained by everyone in space, although there are major intrasubject differences. Some people are strongly “field dependent” and rely on the internal floor, ceiling, and walls for a reference; others establish the local work surface as a vertical plane; others are strongly influenced by localized pressure, especially on the feet; while still others seem to carry along a reference frame tied to their own trunk. The perception of the local subjective vertical is more variable in flight, and pointing experiments generally show a tendency to point low in the dark. Although the threshold for detection of linear acceleration may be slightly lowered in weightlessness, the detection is subject to frequent errors at first, and again after return to earth. During the return, the acceleration levels are strongly overestimated, and a feeling of “heaviness” is commonly reported.

WE DON’T KNOW how or why the egocentric vertical is maintained. Nor is it fully understood why a sensation of free-fall is absent in orbit, or why astronauts do not associate each linear acceleration with a change in body orientation. Finally, an association between the various orientation perceptual styles and either underlying physiological processes or space sickness susceptibility has not been established.

Postural Instability

WE KNOW that the initial awkwardness in locomoting inside a spacecraft quickly disappears and that this learned behavior carries over to subsequent flights. Postflight, how-
ever, most astronauts experience some instability and difficulty walking or standing, especially with eyes closed. The recovery is mostly rapid, over several hours, but some subtler effects last a week or more. The narrower “cone of stability” post flight is associated with rigid standing in darkness, a wide stance when walking, and a tendency to round corners wide. Increased reliance upon visual cues, and a possible recalibration of the joint angle proprioceptive system, produces both postural instability and false illusions during active movements after landing.

WE DON’T KNOW whether the 1-g motor control programs in standing and walking are stored or re-learned. The specific contribution of otolith organ cues to posture post flight is not well understood. In particular, we don’t know the extent to which the putative CNS reinterpretation of otolith signals to represent acceleration rather than tilt contributes to the problem. We know that muscle atrophy occurs in weightlessness, and that a shift from slow to fast motor fiber dominance occurs—but the extent that this contributes to post flight posture problems is undetermined. Similarly, the tendency toward fainting while standing (orthostatic hypotension), which is well documented following return for many astronauts, has not been quantitatively tied to posture. It is not possible, based upon current knowledge, to determine the postural state of astronauts on the surface of Mars after a year-long flight.

5. End Organ Changes

WE KNOW that the semicircular canal afferent signals function in microgravity, as do otolith afferents. Otoconial formation is probably normal, although an early report showed some damage.

WE DON’T KNOW whether the semicircular canal afferent responses reflect real changes in end-organ time constants, or if the otolith organs produce hypersensitive responses at the eighth nerve level. Potentially important increases in peripheral type II hair cell synapse development in microgravity needs confirmation.

The role of the efferent system in adaptation to weightlessness, and in the apparent change in peripheral time constant remains to be determined. Finally, the differences between the unloading of the gravity bias on the saccular and on the utricular otolithic membranes have yet to be explored.

6. Multisensory Interaction

WE KNOW that both static and dynamic visual scene information becomes increasingly dominant for most subjects in weightlessness. However, visually induced motion (vection) is often not saturated in weightlessness, despite the absence of nonconflicting gravity reference information. Frequently the weighting attached to localized tactile cues is also increased.

WE DON’T KNOW, however, whether changes in the basic reflexes (vestibulo-ocular, ocular counterrolling, optokinetic, vestibulocollic) are related to the changes in weighting of visual and tactile cues associated with perception. The extent to which linearvection might be more influenced than circularvection is unknown, although it would be expected to be more affected by weightlessness. Finally, we don’t know whether the body centered “idiotropic vertical,” which contributes to orientation independent of sensory inputs, undergoes any changes in weightlessness.

1. What is Needed in Order to Answer the Open Questions

Many of the ongoing human and animal experiments are designed to make progress toward answering the “unknowns,” but some major new facilities and programs are needed. A variable gravity animal centrifuge, of the type being designed for inclusion with Space Station Freedom, is of absolute necessity for progress in nearly all of the space physiology disciplines. For vestibular research it can help
answer questions about the time course of changes in the otolith system subjected to differing g levels at different times of flight, as well as to house “1-g controls” for the microgravity space animals. A human linear accelerator, or sled, along with a versatile rotating chair, is required as a facility for regular in-flight neurovestibular study. The human and animal experimental programs for ever-increasing durations on orbit should be carefully coordinated, with incremental human exposure accompanied by long-duration animal exposure. Finally, an artificial gravity research facility, capable of supporting astronauts in space at different g-levels, and at different radii and rotation rates, is a prerequisite to understanding the vestibular as well as other physiological and human factor issues of artificial gravity.

Acknowledgments—Comments from the following colleagues were influential in shaping the final version of this editorial: I Curthoys, S Diamond, F Guedry, M Igarashi, I Kozlovskaya, L Kornilova, C Markham, H Mittelstaedt, K Money, C Oman, D Parker, and M Reschke. The preparation was supported by NASA Contract NAS9-15343 and Grant NAG2-443.