Chapter 7

# ANGULAR VELOCITIES, ANGULAR ACCELERATIONS, AND CORIOLIS ACCELERATIONS

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This chapter deals with the rotary motions generated by man and by machine that may be encountered in the exploration of space and the effects of such motions on the human organism. Under all conditions, active rotary motions will fall within the parameters of such movements executed by man under normal terrestrial conditions. These active rotations are characterized by short arcs, and the associated angular accelerations, although transient, may, in the case of head movements, far exceed the accelerations generated by conveyance or laboratory devices. It is also noteworthy that these active rotary motions produce their effects chiefly by stimulating sensory receptor systems with subsequent "amplification" in the central nervous system. The receptor systems in the semicircular canals deserve special attention, partly because these end organs are uniquely structured to respond to transient angular accelerations, and partly because they are gravity-independent.

Except in unprogramed situations, passive rotary motions to which man may be exposed are significant chiefly for the long duration of the angular velocities and not for the associated brief angular accelerations. In the generation of artificial gravity (by rotating part of a space vehicle or space station), spin-up or spin-down would rarely occur, and rotation at a constant angular velocity would be maintained for long periods. In this circumstance, active rotation of the head would generate Coriolis accelerations,[1] constituting abnormal patterns of vestibular stimulation that might elicit vestibular side effects. These side effects comprise reflex phenomena and widespread delayed epiphenomena (best known as motion sickness). Even in the weightless part, although head movements generate normal accelerative stimuli, the resulting sensory input encounters an abnormal integrative pattern (resulting from loss of stimulation of the otolithic receptors by gravity), and vestibular side effects may be elicited; thus, both vestibular organs may be involved by unnatural stimulatory conditions in space exploration.

Although all major sensory systems must be taken into account, their unfavorable influence, under conditions met in the exploration of space, are unequal. The organs of equilibrium, the semicircular canal and otolith organs, pose the essential problem. Much of the discussion which follows, therefore, will deal with the vestibular system, the mechanisms involved in the elicitation

[1] In this chapter Coriolis acceleration is defined as the "added acceleration" generated either by simultaneous exposure to angular velocities about two axes or to one linear and one angular velocity; it is left to the reader to determine from the context which one is applicable or if both are applicable.



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of side effects (especially motion sickness), and the operational problems engendered.[2]

## MAN'S BIOLOGICALLY EFFECTIVE FORCE ENVIRONMENT

In this section, the concept of man's biologically effective force (BEF) environment is briefly developed in relation to life activities under terrestrial conditions and to the unique force environments in space exploration. The need for a common basis in discussing and comparing these forces is best exemplified in comparing "life" in a weightless spacecraft and life on Earth. The purpose here is not to attempt a comprehensive analysis and synthesis, which would involve a great undertaking and even then be incomplete, but to set forth major guidelines which point to further exploitation for practical or theoretical purposes.

Man's gravitoinertial force environment has its genesis in gravity due to a central field factor in the inertial forces generated by the motions of machine or man, or both in combination (Fig. 1). Under ordinary living conditions on Earth, gravitational force may be regarded as a constant and the only force of sufficient magnitude to affect total body weight significantly. It is the force to which man has become adapted throughout evolutionary development and to which he is accustomed through experience. The addition of mutually perpendicular lines to the vector representing gravitational upright forms the spatial frame of Earth reference. When man is exposed in conveyances and devices that generate accelerations or change his position with respect to the gravitational or gravitoinertial vertical, he is subjected to unnatural stimulatory conditions that may range far beyond physiologic limits. These accelerations generate an external force field that, along with gravity, comprises the total *external* force field.

The inertial forces generated by the active motions of the body or its parts may be regarded as "immanent" forces inasmuch as they do not contribute to the external force field, but are combined with it. These immanent forces are of small magnitude or short duration, deriving their significance partly because of being associated with motions that change the position of the body with regard to the other components in the force environment, and partly because these forces are sufficient to stimulate specialized sensory receptors that provide information about body statics and dynamics. Combined gravitational, inertial, and immanent accelerative forces constitute a complex, dynamic pattern that varies as a function of time.

Although the equivalence of gravitational and inertial mass is the unifying principle underlying the gravitoinertial force concept, this simplicity gives way to great complexity when taking into account the structural and functional characteristics of the body. Not only does the body lack uniformity, but also a state of mechanical equilibrium in all parts of the body is never reached.

The agravitoinertial forces are far more difficult to identify and measure in terms of a common unit than the accelerative forces. They assume great importance in a weightless environment, and a dichotomy may be drawn between agravitoinertial forces of mechanical and nonmechanical origin. With the latter, further distinctions are possible, ranging between forces so great at one extreme that absence of gravitational force is of no practical consequence, and the other extreme when its influence is felt. All these mechanical forces are generated by tensions and compressions and, with gravitoinertial forces, contribute to bodily deformations and stimulation of nonvestibular mechanoreceptors.

### Weightlessness

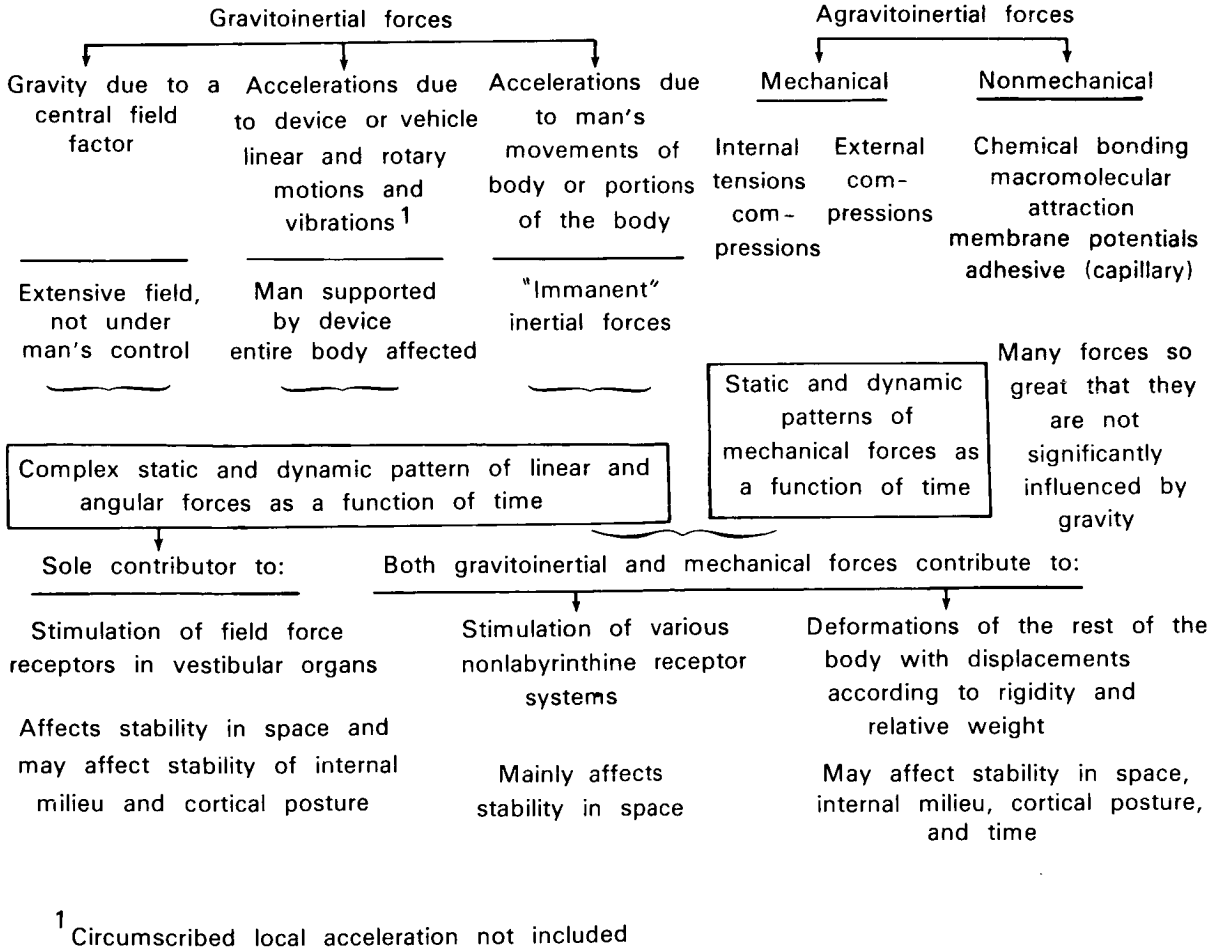
**Under natural terrestrial conditions, the force of gravity due to a central field factor is only part of our BEF environment; hence, it is important to distinguish between weightlessness per se and man active in a weightless spacecraft. This difference, which may be very great, will be determined mainly by the role played by mechanical forces that are effective in countering the zero-gravity state.**

[2] It is a pleasure to acknowledge my debt to Professor V. Polaev who provided an annotated bibliography covering key reports in the relevant Soviet literature.

Figure 2 illustrates an effort to analyze the BEF environment in a weightless spacecraft. Gravitoinertial forces are generated mainly by man's motions unless a device capable of generating accelerations is used. Immanent accelerations generated in the course of man's work and housekeeping activities contribute little to his "apparent weight" but are important since they stimulate sensory receptors (vestibular and nonvestibular mechanoreceptors) and thus contribute to the flow of information to the nervous system.

The preservation of man's well-being in a weightless spacecraft is heavily dependent upon agravitoinertial forces, which are of mechanical or nonmechanical origin. An analysis of the latter should be made in terms of their effectiveness at different organizational levels in the body. A table might be prepared indicating the forces operant at different levels; e.g. molecular, intracellular, cellular, and tissue levels. Cytochemical reactions involve forces (thermodynamic, bioelectric, and chemical) so great that they are gravity-independent. Agravitoinertial *mechanical* forces stimulate mechanoreceptors serving touch, pressure, and kinesthesis but cannot stimulate vestibular receptors.

The analysis is carried one step farther in Figure 3. Presumably, with the crew at rest and with head fixed, there would be physiologic deafferentation of the otolith apparatus, with consequent loss of its tonic discharge but retention of a spontaneous or resting discharge analogous to the difference between eyes open and closed. There would be no corresponding effect on the




[1] Circumscribed local acceleration not included

FIGURE 1.—Man's biological force environment under terrestrial conditions.

semicircular canals. Rotations of the head would provide stimulus to the canals the same as under terrestrial conditions, but the transient linear accelerations generated might or might not constitute an adequate stimulus to the otolith apparatus, and, if adequate, the information would be neither useful for orientation to the upright nor concordant with the canalicular input. Thus, among vestibular receptors and nonvestibular mechanoreceptors, the canals alone are stimulated essentially the same with natural movements of the head (body) under terrestrial and weightless conditions. The otolith organs encounter the unique stimulatory condition with the lifting of the stimulus due to nullification of gravity. Reference is made to reports that deal with the problem in a different manner or approach [32, 33, 51, 92, 122, 138].

## Rotating Environment

In a slowly rotating room constrained to rotate about the Earth-vertical axis, a person carrying out various activities is subjected to complex changes in the gravitoinertial force environment. Not only must the forces acting at man's center of gravity be taken into account, but also the sep-

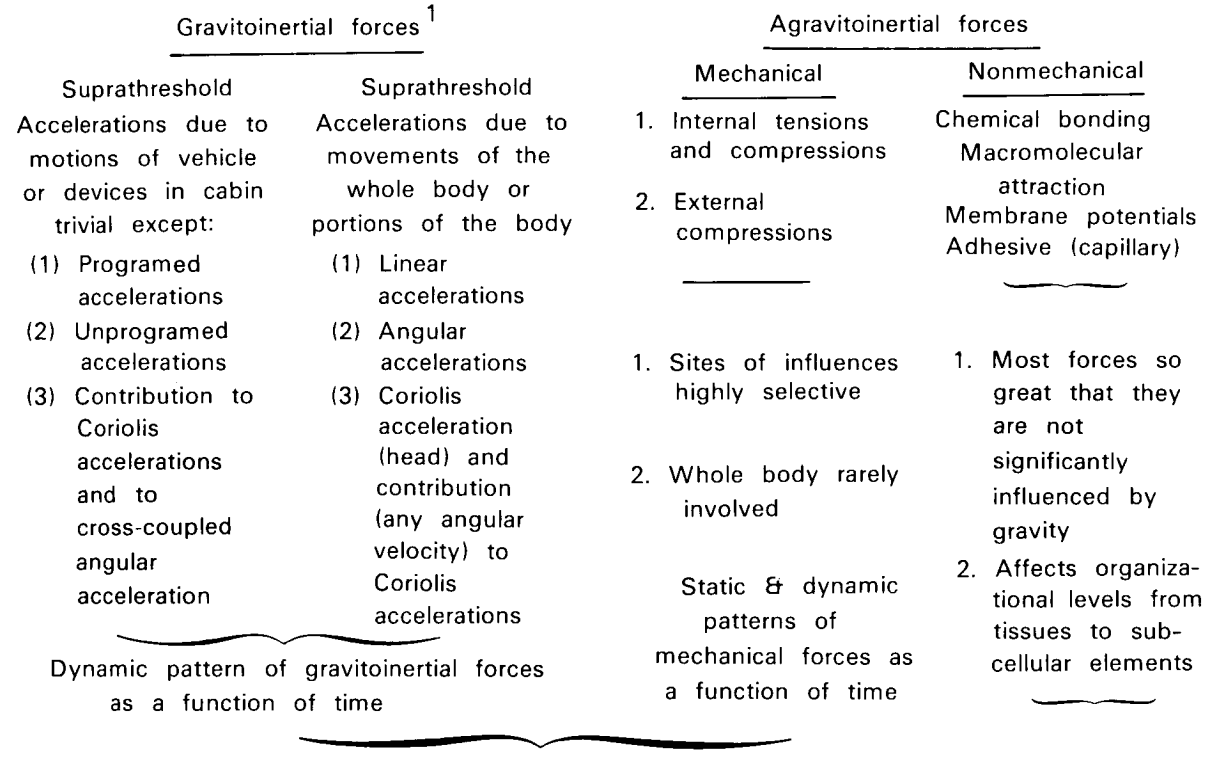



FIGURE 2.—Man's biological force environment in near-weightlessness. (Comparisons must be made with terrestrial conditions taking account of (1) body configurations and motions, and (2) the maintenance of homeostasis in terms of gravity-dependence.)

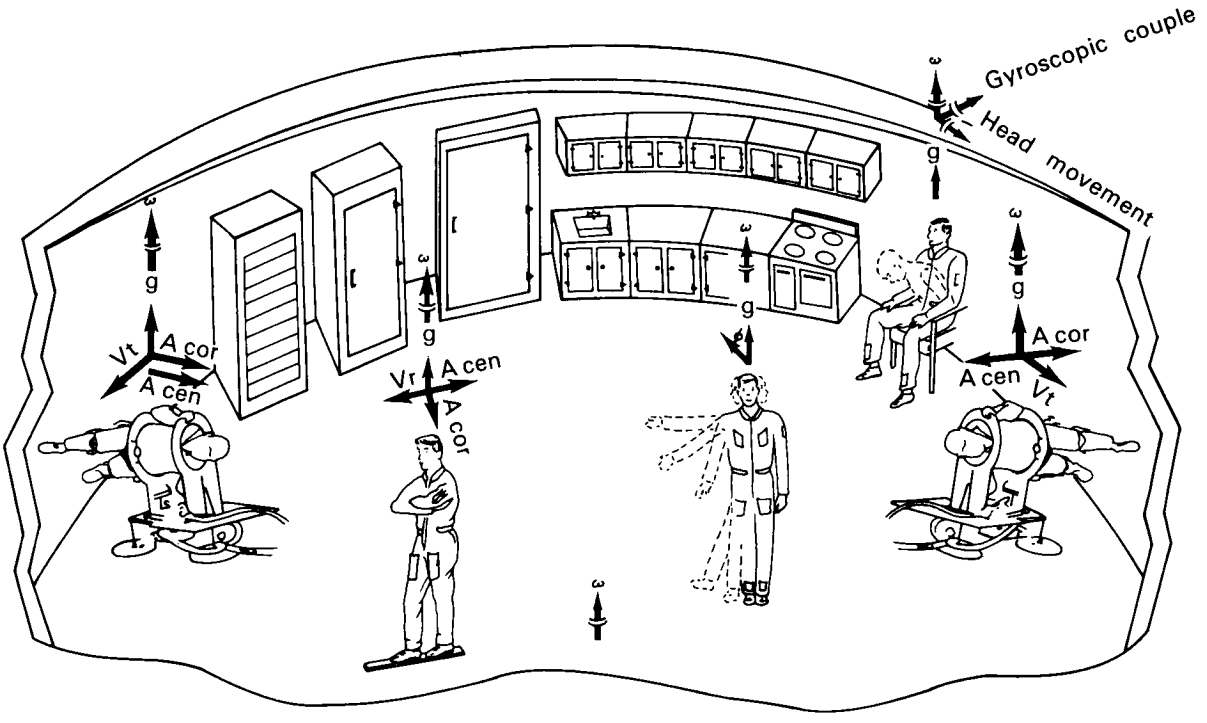
| | | | Resting discharge | | | | | | Physiological stimulus conditions | | | | | | Artificial stimulus conditions | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Genesis | Maint. neural 2nd neur | Maint. conh beyond 2nd n. | Modulated by oto. apparatus | Tonus | Affects R-L V. balance | Inc. susc. to CNS funct. dist. | Stimulus | Sensory | Mod. oto. output | Mod. by oto. output | Affects R-L vestib. balance | Inc. susc. to CNS dist. | Coriolis acc. | Angular acc. | Linear acc. & constant rotation within critical range |
| Canalicular system gravity-independent | Earth | Biochem. gravity-independent | Yes | Yes | Yes | Probably | Yes | ? | Inertial angular & Coriolis acc. | Phasic | Yes | Yes | No | Yes | Abn. responses reflex phenom motion sickness | Response thresholds 0.04°/sec² | May constitute an adequate stimulus |
| | Spacecraft | Biochem. gravity-independent | Yes | Yes | Yes | Probably | Yes | ? | Same | Phasic | Yes | Yes & may affect behavioral resp. | ? | Yes | Abn. responses reflex phenom. motion sickness | Response thresholds may well be diff. | Should be differences (Quant.) at least imp. to investigate |

| | | | | Physiological stimulus conditions | | | | Artificial stimulus conditions | |
|---|---|---|---|---|---|---|---|---|---|
| | | Presence | Genesis | Stimulus due to gravity: Proof | Stimulus due to gravity: Sensory input | Stimulus due to change in position of head in gravitational field: During change | Stimulus due to change in position of head in gravitational field: Head fixed in new position | Stimulus due to immanent inertial linear acc. | Response thresholds to stimulation | Suprathreshold levels of stimulation |
| Otolithic systems gravity-dependent | Earth | Inferred | Chem–el. act. | Ocular counterroll, oculogravic illusion A&E & percept. upright vary as function of g-loading | Tonic modulates reflex activity and canalicular influence | Dynamics not investigated in systematic fashion | Few systematic studies. Servation decreases with large departures from upright | Probably less imp. than canals for maintaining postural upright in man | Vary with departures from grav. upright or gravitoinertial upright | Establishment of gravitoinertial vertical other than in long body axis characterized by a "lag" and a "magnitude" effect (overcompensation at levels greater than 1.2 g units) |
| | Spacecraft | Demonstrated in parabolic flight | Gravity independent | ? No A&E C-roll percept. upright<br>A&E Mueller & Aubert phenomena | No input | Not possible | | Mainly transients<br>Should be measured<br>Stimulus thresholds may be affected by loss of g loading<br>Probably effective in stimulating oto. system | Has not been investicated carefully but there are reports of "increased sensitivity"<br>Behavioral significance in terms of gravitoinertial upright necessarily different | Absence of the earth's standard of gravity will have effect of exaggerating influence of vectors not in accord with body or visual upright. Possible effects on orientation and susceptibility to motion sickness |

FIGURE 3.—Certain differences between life on Earth and in weightless spacecraft with reference to the canalicular and otolithic systems.

arate consequences of head and limb motions with and without whole body motions.

Figure 4 illustrates the forces acting on the subject's mass when he is recumbent and seated. Centrifugal force derives from $r\omega^2$ where $r$ = radius and $\omega$ = angular velocity. The angle $\phi$ represents the change in direction of the gravitoinertial upright from the gravitational upright. With the subject moving with or against the direction of rotation or toward or away from the center of rotation, Coriolis forces must be taken into account as well as the changes in centripetal force. The fundamental law relating the time rate of change of a vector, measured by an observer in space rotating with respect to the reference space, may be expressed mathematically by the vector equation:




g - Acceleration of gravity
A cen - Centripetal acceleration
A cor - Coriolis acceleration
Vt - Tangential velocity
Vr - Radial velocity
ω - Angular velocity of rotating room
ø - Angle between gravitoinertial and gravitational upright

FIGURE 4.—Responses to the force environment in a rotating room. Crewmen 1 and 2, in articulated molds supported by air-bearing devices, are "walking on the wall," simulating the orientation in a rotating spacecraft. Crewman 2, walking in the direction of rotation becomes somewhat heavier because his angular velocity, hence, centripetal acceleration, is increased and sums with the Coriolis accelerations generated. Crewman 1, walking opposite the direction of rotation becomes somewhat lighter because his centripetal accelerations are decreased and Coriolis accelerations must be subtracted. Crewman 3, walking toward the periphery of the room is exposed to increasing levels of centripetal acceleration and constant levels of Coriolis accelerations. Crewman 4, standing, is demonstrating two phenomena: first, as he moves his arm or leg sideways, a tendency to veer backward, the so-called "giant-hand" effect; second, as he makes (rotates) his head move in the plane of the room's rotation, Coriolis accelerations and illusions are not generated, a so-called "free movement;" third, the angle phi ($\phi$) indicates the change in direction of the gravitational upright due to centripetal force. Crewman 5 is making a head movement out of the plane of rotation which does generate Coriolis accelerations producing characteristic illusions described. (Drawing courtesy of Dr. D. B. Cramer)

$$\left(\frac{d\bar{V}}{dt}\right)_r = \left(\frac{d\bar{V}}{dt}\right)_m + (\bar{\omega}rm \times \bar{V}) \qquad (1)$$

where

$(d\bar{V}/dt)_r$ = change in velocity vector with respect to the reference space,
$(d\bar{V}/dt)_m$ = change of velocity with respect to moving space,
$(\omega rm \times \bar{V})$ = change of velocity vector due to rotation of moving space.

To a subject in the rotating environment, this acceleration or force vector may manifest itself in two ways. First, it adds to the apparent weight of a body moving in the direction of rotation and subtracts from the apparent weight when it moves against the direction of rotation. Second, when a body moves toward the center of rotation, the linear Coriolis force is exerted in the direction of rotation at right angles to the body's motion; when moving away from the center of rotation, the force is opposite the direction of rotation. A motion parallel to the axis of rotation will generate no Coriolis acceleration. The value of Coriolis acceleration in g-units for a body moving perpendicularly to the axis of rotation in a spinning system may be determined by:

$$F\ (\text{Coriolis}) = 0.00651\ VN \qquad (2)$$

where

$V$ = velocity of body relative to rotating vehicle in feet per second,
$N$ = vehicle rate of rotation in revolutions per minute.

For any motion not exactly perpendicular to the axis of rotation, the component of the velocity that is perpendicular is used to determine the Coriolis force; hence, the value must be multiplied by the sine of the angle between the angular rotation rate vector and the velocity vector. Figure 5 illustrates the Coriolis force in g-units for various rates of movement perpendicular to the axis of rotation at different rates of the room's rotation. The combined Coriolis and centripetal forces influence ataxia exhibited by subjects. It is apparent from Figures 4 and 5 that a person walking against the direction of spin will experience a slight decrease of apparent weight and a slight increase when walking with the direction of rotation. Also, moving toward the center, the linear Coriolis force would be in the direction of spin, and walking toward the periphery, the direction would be reversed.

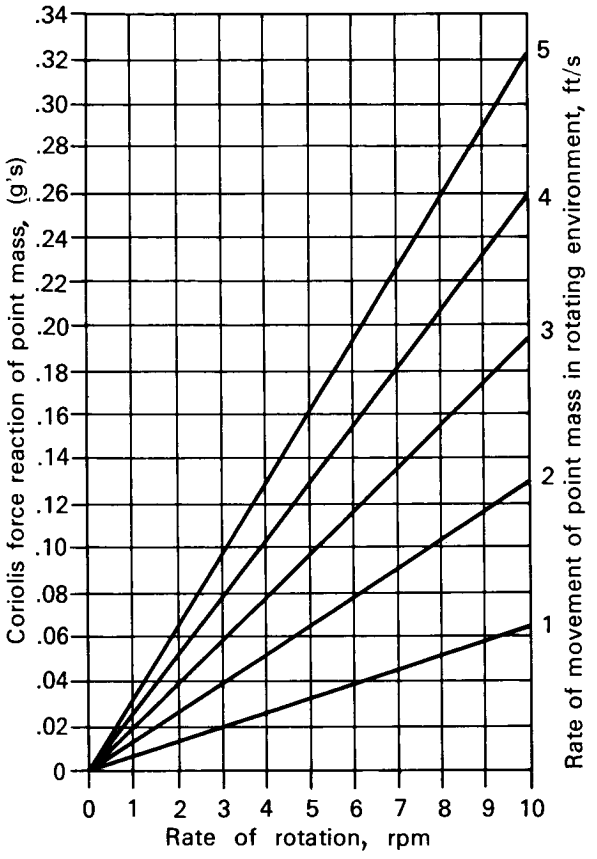



FIGURE 5.—Coriolis force reaction of a mass moving in a rotating environment. Force in pounds = Earth gravity weight × g units

The above analysis is oversimplified because the motions of limbs and head need not conform to motions of the center of gravity; moreover, the motion of the center of gravity itself is complex, although motions normal to the Earth horizontal would not generate a Coriolis acceleration.

Consideration must also be given to the angular Coriolis accelerations which constitute an abnormal stimulatory pattern where the semicircular canals are concerned. When angular Coriolis accelerations are above threshold, reflex vestibular disturbances result and may be followed by motion

sickness, making them of great practical importance in dealing with rotating environments. Angular Coriolis accelerations per se are independent of the distance from the center of rotation of the room and, indeed, of the level of G-loading within the ranges to be encountered in rotating environments.

## Mathematical Analysis of Coriolis Accelerations

Stone and Letko [127][3] have developed equations for the effects of angular acceleration and angular velocity about two axes.

### *Definitions and Symbols*

Nodding — a rotation of the head about the $y$-axis (Fig. 6).

Turning — a rotation ot the head about the $z$-axis (Fig. 6).

Rolling — a rotation of the head about the $x$-axis (Fig. 6).

$\alpha_{G_\theta}$ angular nodding acceleration (component of angular acceleration about the $y$-axis)

$\alpha_{G_\psi}$ angular turning acceleration (component of angular acceleration about the $z$-axis)

$\alpha_{G_\phi}$ angular rolling acceleration (component of angular acceleration about the $x$-axis)

$$\omega_{G_\theta} = \int \alpha_{G_\theta}\, dt$$

$$\omega_{G_\psi} = \int \alpha_{G_\psi}\, dt$$

$$\omega_{G_\phi} = \int \alpha_{G_\phi}\, dt$$

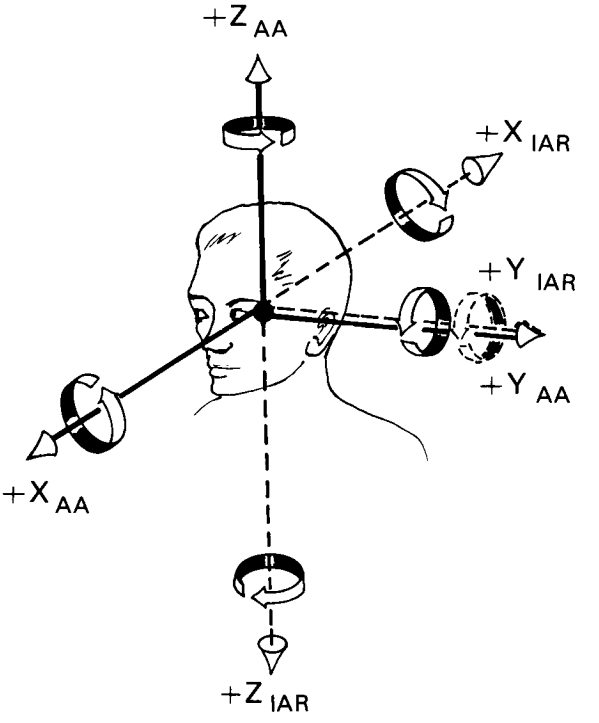



FIGURE 6.—Right-hand rule applied to the True reference system (angular accelerations) and the inertial (reaction) force system. Note that the two systems have opposite signs except in $y$ axis—facing the page (True) and facing the left side of the body (inertial).

$\omega_{h_\theta}$ nodding velocity—a fore and aft motion of the head at the neck or from the whole body [4]

$\omega_{h_\psi}$ turning velocity—a motion about the neck or long-body axis [4]

$\omega_{h_\phi}$ rolling velocity—a sideways motion of the head or from the body [4]

[3] Their original publication has been reworked by Professor J. L. Patterson, Jr., with the help of Stone, and reproduced here with their kind permission.

[4] These are angular head motions which may be from motions at the neck and shoulders or from body bending, and similar.

$\omega_V$ vehicle rotational velocity

$\omega_{h_x}$ total angular velocity of head about rolling axis ($x$-axis)

$\omega_{h_y}$ total angular velocity of head about nodding axis ($y$-axis)

$\omega_{h_z}$ total angular velocity of head about turning axis ($z$-axis)

$t$ time

$\theta_G$ $\iint \alpha_{G_\theta}\, dt^2$

$\psi_G =$ $\iint \alpha_{G_\psi}\, dt^2$

$\phi_G =$ $\iint \alpha_{G_\phi}\, dt^2$

$\theta_n$ nodding displacement of the head (about the $y$-axis)

$\psi_n$ turning displacement of the head (about the $z$-axis)

$\phi_n$ rolling displacement of the head (about the $x$-axis)

$\phi_e, \theta_e, \psi_e$ Euler angular displacement using this order of rotation. (Euler angles relate one set of axes to another set and are used in the classical method of this transformation.)

$\theta_{sc}$ backward tilt of semicircular canals from $X_bY_b$ plane

$\psi_{sc}$ rotation of semicircular canals from $X_bY_b$

$X, Y, Z$ inertial space axes

$x_b, y_b, z_b,$ body axes

Subscripts:

*lr, ll* right and left lateral canals, respectively

pr, pl right and left posterior canals, respectively

*ar, al* right and left anterior canals, respectively

Head Axes Used by Stone and Letko

$x$ is (+) facing forward out of the nose.
$y$ is (+) extending out of the right ear.
$z$ is (+) extending downward (caudad) from the head.

Note: These head axes are part of a rectangular coordinate system. The negative of each axis has the sense and direction opposite the positive.

Semicircular Canal Axes

Equations (21–26) and (27–32) relate to the axes of the semicircular canals themselves. Each acceleration vector in these equations represents an angular acceleration vector that is perpendicular to the plane of the canal in question. The angles in these equations represent the transformation of head axes into canal axes. A dot over a symbol indicates its first derivative with respect to time.

The equations for the *total angular velocities experienced by the head* are the sum of the various angular velocities acting:

$$\omega_{h_x} = \omega_{h_\phi} + w_V \cos\theta_e \cos\psi_e \quad (3)$$

$$\omega_{h_y} = \omega_{h_\theta} - w_V \cos\theta_e \sin\psi_e \quad (4)$$

$$\omega_{h_z} = \omega_{h_\psi} + w_V \sin\theta_e \quad (5)$$

where $w_V$, the angular or rotational velocity of the vehicle, is assumed to be constant.

The differentiation of Equations (3–5) with respect to time gives the *angular accelerations experienced by the moving head*, where $\dot{\omega}_{h_x}$, $\dot{\omega}_{h_y}$, and $\dot{\omega}_{h_z}$ are the angular accelerations of the head in inertial space (the accelerations which will stimulate the semicircular canals) and $\dot{\omega}_{h_\phi}$, $\dot{\omega}_{h_\theta}$, and $\dot{\omega}_{h_\psi}$ are the angular accelerations of the head in the rotating frame of reference.

$$\dot{\omega}_{h_x} = \dot{\omega}_{h_\phi} - w_V (\sin\theta_e \cos\psi_e \dot{\theta}_e + \cos\theta_e \sin\psi_e \dot{\psi}_e) \quad (6)$$

$$\dot{\omega}_{h_y} = \dot{\omega}_{h_\theta} - w_V (\cos\theta_e \cos\psi_e \dot{\psi}_e - \sin\theta_e \sin\psi_e \dot{\theta}_e \quad (7)$$

$$\dot{\omega}_{h_z} = \dot{\omega}_{h_\psi} + w_V \cos\theta_e \dot{\theta}_e \quad (8)$$

Applying the principles of classical mechanics, the rates of change of the Euler angles in Equations (6–8) may be calculated from the following:

$$\dot{\phi}_c = \left(\omega_{h_\phi} \cos\psi_e - \omega_{h_\theta} \sin\psi_e \frac{1}{\cos\theta_e}\right) \quad (9)$$

$$\dot{\theta}_e = (\omega_{h_\phi} \sin\psi_e + \omega_{h_\theta} \cos\psi_e) \quad (10)$$

$$\dot{\psi}_e = \omega_{h_\psi} - \tan\theta_e(\omega_{h_\phi} \cos\psi_e - \omega_{h_\theta} \sin\psi_e) \quad (11)$$

A substitution of Equations (9–11) into Equations (6–8) then gives the *general expressions for the total angular acceleration* that would be experienced *for any orientation of the head and for any head motion* when *in a vehicle rotating at constant angular velocity:*

$$\dot{\omega}_{h_x} = \dot{\omega}_{h_\phi} - w_V(\omega_{h_\theta} \sin\theta_e + \omega_{h_\psi} \cos\theta_e \sin\psi_e) \quad (12)$$

$$\dot{\omega}_{h_y} = \dot{\omega}_{h_\theta} - w_V(\omega_{h_\psi} \cos\theta_e \cos\psi_e - \omega_{h_\phi} \sin\theta_e) \quad (13)$$

$$\dot{\omega}_{h_z} = \dot{\omega}_{h_\psi} + w_V(\omega_{h_\theta} \cos\theta_e \cos\psi_e + \omega_{h_\phi} \cos\theta_e \sin\psi_e) \quad (14)$$

When the vehicle is not rotating ($w_V = 0$) the equations simplify to:

$$\dot{\omega}_{h_x} = \dot{\omega}_{h_\phi} \quad (15)$$

$$\dot{\omega}_{h_y} = \dot{\omega}_{h_\theta} \quad (16)$$

$$\dot{\omega}_{h_z} = \dot{\omega}_{h_\psi} \quad (17)$$

When Equations (15–17) are subtracted from Equations (12–14), a set of expressions results for the accelerations caused by the rotation of the vehicle. These accelerations are termed by Stone and Letko [127] the *cross-coupled angular accelerations* or Coriolis accelerations which are given by the Equations:

$$\alpha_{G_\theta} = -w_V(\omega_{h_\theta} \sin\theta_e + \omega_{h_\psi} \cos\theta_e \sin\psi_e) \quad (18)$$

$$\alpha_{G_\theta} = w_V(\omega_{h_\theta} \sin\theta_e - \omega_{h_\psi} \cos\theta_e \cos\psi_e) \quad (19)$$

$$\alpha_{G_\psi} = w_V(\omega_{h_\theta} \cos\theta_e \cos\psi_e + \omega_{h_\theta} \cos\theta_e \sin\psi_e) \quad (20)$$

A number of investigators concerned with the physics of stimulation of the semicircular canals have believed that the Coriolis accelerations are those which primarily cause the disturbing symptoms and signs during rotation of a vehicle. It should be noted that the instantaneous angular velocities ($\omega$) in these equations are not necessarily associated with a condition of angular acceleration, but can be part of a constant angular velocity profile. Thus, Coriolis accelerations ($\alpha$) can result from the cross-coupling effects of constant angular velocities about more than one axis—an extremely important principle.

### *Coriolis Acceleration*

At the beginning of the last century, a French engineer and mathematician, G. G. de Coriolis, carried out a mathematical analysis of an apparent force generated when a body is moving in a linear path in a rotating frame. Such a situation obtains when a mass is moving on the surface of a rotating carousel or when a projectile is fired from a gun on the surface of the rotating earth. This has been discussed previously in the introductory section and will not be considered in detail here. The point should be emphasized, however, that there is increasing use in physics, engineering, and physiology, of the term "Coriolis" force or acceleration where there is rotation of a body about more than one axis simultaneously.

### *Effective Components of Coriolis Accelerations*

The effective component of the Coriolis acceleration which applies to each of the six semicircular canals can be derived from Equations (18–20). These derivations, kindly provided by Stone (personal communication), are:

$$\Delta\dot{\omega}_{sc_{lr}} = \alpha_{G_\phi} \sin\theta_{sc} + \alpha_{G_\psi} \cos\theta_{sc} \quad (21)$$

$$\Delta\dot{\omega}_{sc_{ll}} = \alpha_{G_\phi} \sin\theta_{sc} + \alpha_{G_\psi} \cos\theta_{sc} \quad (22)$$

$$\Delta\dot{\omega}_{sc_{ar}} = \alpha_{G_\theta} \cos\psi_{sc} - \alpha_{G_\phi} \cos\theta_{sc} \sin\psi_{sc} + \alpha_{G_\psi} \sin\theta_{sc} \sin\psi_{sc} \quad (23)$$

$$\Delta\dot{\omega}_{sc_{al}} = \alpha_{G_\theta}\cos\psi_{sc} - \alpha_{G_\phi}\cos\theta_{sc}\sin\psi_{sc} + \alpha_{G_\psi}\sin\theta_{sc}\sin\psi_{sc} \quad (24)$$

$$\Delta\dot{\omega}_{sc_{pr}} = \alpha_{G_\phi}\cos\theta_{sc}\cos\psi_{sc} + \alpha_{G_\theta}\sin\psi_{sc} - \alpha_{G_\psi}\sin\theta_{sc}\cos\psi_{sc} \quad (25)$$

$$\Delta\dot{\omega}_{sc_{pl}} = \alpha_{G_\phi}\cos\theta_{sc}\cos\psi_{sc} - \alpha_{G_\theta}\sin\psi_{sc} - \alpha_{G_\psi}\sin\theta_{sc}\cos\psi_{sc} \quad (26)$$

### *Total Angular Acceleration Experienced by Each Semicircular Canal*

The total angular acceleration experienced by each of the six semicircular canals is given by the following equations, which include the cross-coupled components of angular acceleration. It will assist visualization of these relationships if Figure 12 (p. 260, in the next section of this chapter) is consulted, which shows orientation of the semicircular canals within the cranium.

$$\dot{\omega}_{sc_{lr}} = \dot{\omega}_{h_x}\sin\theta_{sc} + \dot{\omega}_{h_z}\cos\theta_{sc} \quad (27)$$

$$\dot{\omega}_{sc_{ll}} = \dot{\omega}_{h_x}\sin\theta_{sc} + \dot{\omega}_{h_z}\cos\theta_{sc} \quad (28)$$

$$\dot{\omega}_{sc_{ar}} = \dot{\omega}_{h_y}\cos\psi_{sc} - \dot{\omega}_{h_x}\cos\theta_{sc}\sin\psi_{sc} + \dot{\omega}_{h_z}\sin\theta_{sc}\sin\psi_{sc} \quad (29)$$

$$\dot{\omega}_{sc_{al}} = \dot{\omega}_{h_y}\cos\psi_{sc} - \dot{\omega}_{h_x}\cos\theta_{sc}\sin\psi_{sc} + \dot{\omega}_{h_z}\sin\theta_{sc}\sin\psi_{sc} \quad (30)$$

$$\dot{\omega}_{sc_{pr}} = \dot{\omega}_{h_x}\cos\theta_{sc}\cos\psi_{sc} + \dot{\omega}_{h_y}\sin\psi_{sc} - \dot{\omega}_{h_z}\sin\theta_{sc}\cos\psi_{sc} \quad (31)$$

$$\dot{\omega}_{sc_{pl}} = \dot{\omega}_{h_x}\cos\theta_{sc}\cos\psi_{sc} - \dot{\omega}_{h_y}\sin\psi_{sc} - \dot{\omega}_{h_z}\sin\theta_{sc}\cos\psi_{sc} \quad (32)$$

### *Application of the Equations for Angular Accelerations*

The equations presented enable calculation of instantaneous angular accelerations. Since inertial, viscous, and other damping properties provide the semicircular canal system with various delays, which can be expressed as time constants, the canals do not represent transducers whose output of nerve impulses at any moment is directly proportional to the instantaneous angular acceleration experienced by each canal. There may well be, and probably are, moments when the afferent nerve traffic from the cupula of a given canal is proportional to the instantaneous angular acceleration, or some instances where the momentary physical conditions combine to produce a response proportional to the instantaneous angular velocity. These moments would be exceptional, according to expectations.

Apparently, a continuous readout of the solutions to these equations is actually needed for the solution of a number of physiological problems. Accelerometers and computing circuits presently available make it technically feasible to provide continuous recording of angular velocity and angular acceleration in the axes of the accelerometers, together with the necessary transformations to render the data applicable to the plane of each semicircular canal. In this way, the antecedent history of the major variables could be studied in relation to the subjective and objective physiological phenomena. Anatomic differences among different subjects, it is true, will introduce some, usually small, error in the assumed positions of the semicircular canals within a given subject's head; yet it is probable that such on-line computation of these functions would quite likely make a major contribution toward understanding of the responses both of physical models and of biological systems, including man.

## THE VESTIBULAR SYSTEM

### The End Organs

The labyrinth of the human inner ear comprises the cochlea (organ of hearing), otolith organs, and semicircular canals—collectively termed the vestibular organs. These are paired end organs with similar histologic features, a common blood supply, a shared secondary lymph circulation, and with afferent and efferent nerve fibers comprising the acoustic nerve. These sensory organs are situated in hollowed-out channels in the petrous portion of the temporal bone (Fig. 7 [2]), and, within the bony labyrinth, the membranous labyrinth is surrounded by perilymph and filled with endolymph. Thus, the sensory receptor mechanisms are protected from the effects of superimposed body weight by the bony labyrinth and, by virtue of the contained fluids, receive additional protection from impact accelerations.

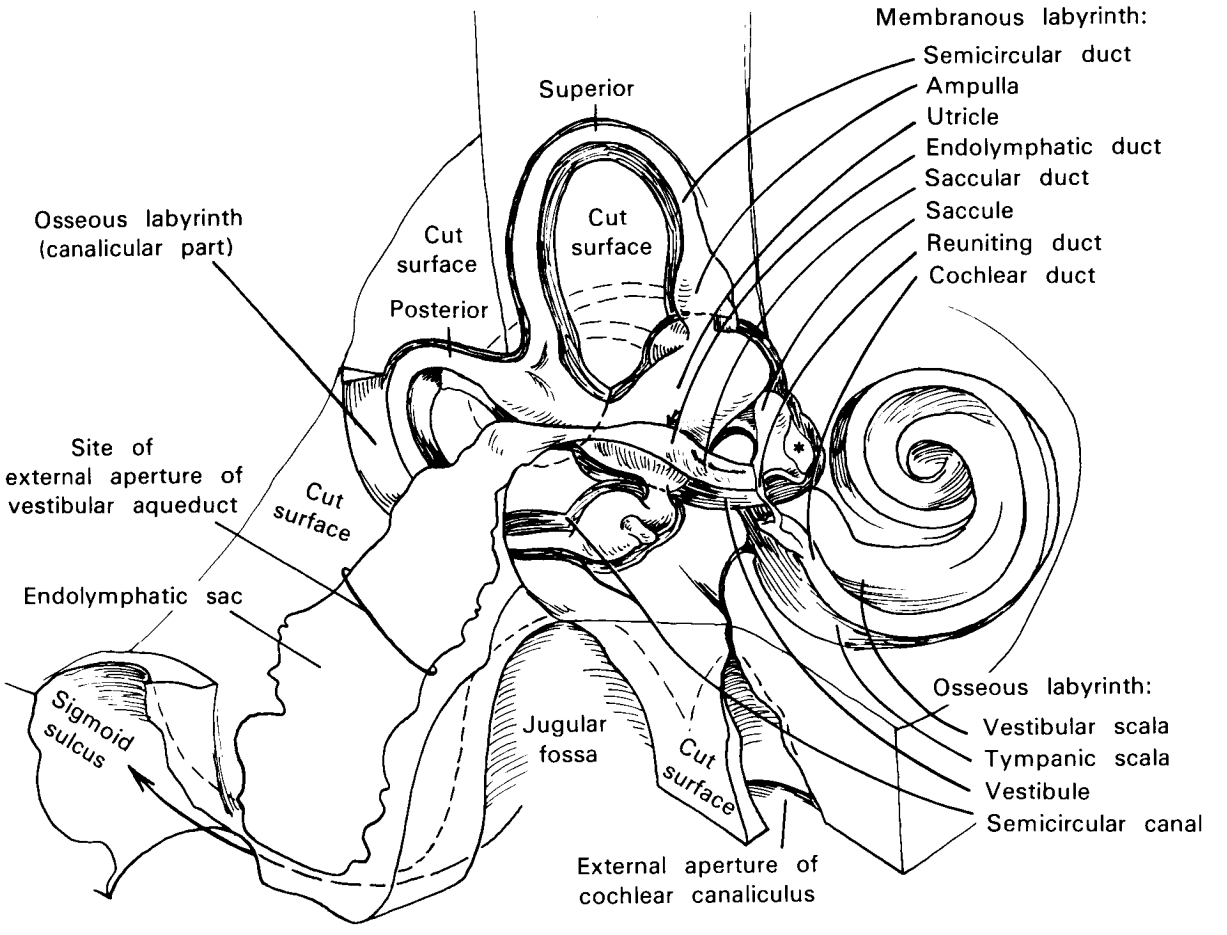



FIGURE 7.—Reconstruction of the membranous labyrinth and related anatomy. (From Ref. [2])

*Otolith Organs*

The four otolith organs appear as thickened portions on the inner walls of the paired utricle and saccule (Fig. 8) that are termed macular plates. A cross section of the saccular macula of a squirrel monkey and a sketch of the zonal structure are shown in Figure 9 [61]. The otolithic membrane contains otoconia (concretions of calcium carbonate crystals with a specific weight of about 2.71) which are embedded in a gelatinous material. It is noteworthy that this membrane comprises the only tissue within the bony labyrinth that differs considerably from the specific gravity of the lymph fluids. The hairlike projections of the sensory cells protrude into the cupular membrane on which the otolith membrane rests.

Figure 10 was drawn from electronmicrographs of the sensory epithelium of the utricular macula of the squirrel monkey [125]. Two types of hair cells, each with two types of cilia, are depicted. Each cell has 60 to 70 sterocilia and one kinocilium laid out in strict geometrical arrangement. In different regions of the macula, the kinocilia (which play the major role in the energy transfer) are polarized in different directions; hence, a shearing force in one plane will result in kinocilia moving in different directions with reference to the kinociliar pole. The result is mechanical deformation of the cilia which, in turn, causes chemical changes, resulting in the generation of bioelectricity (nerve action potentials). This apparatus may be termed the cilia-otolith mechanism.

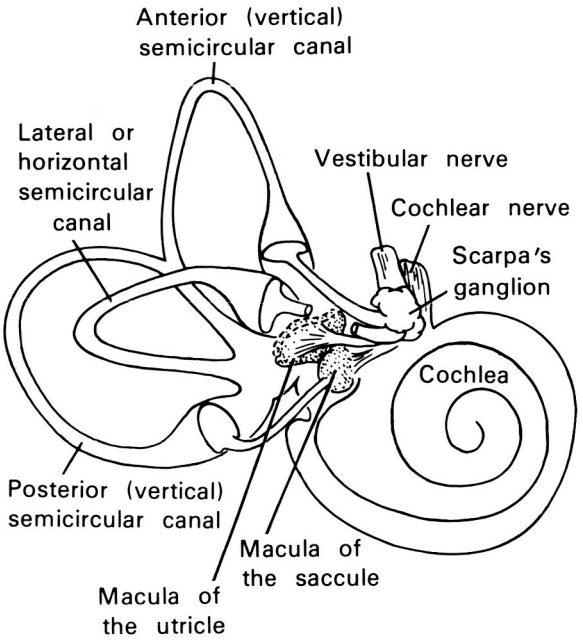
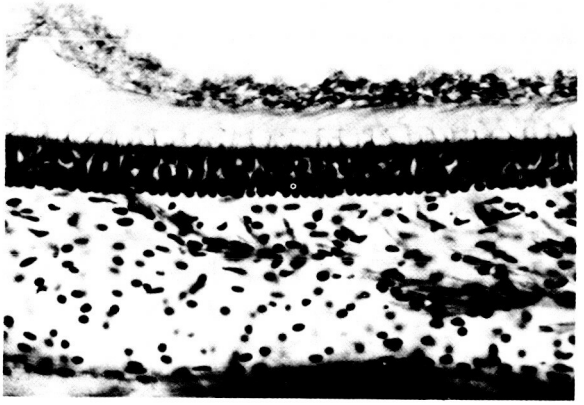



FIGURE 8.—Labyrinth of the left ear viewed from the medial aspect.

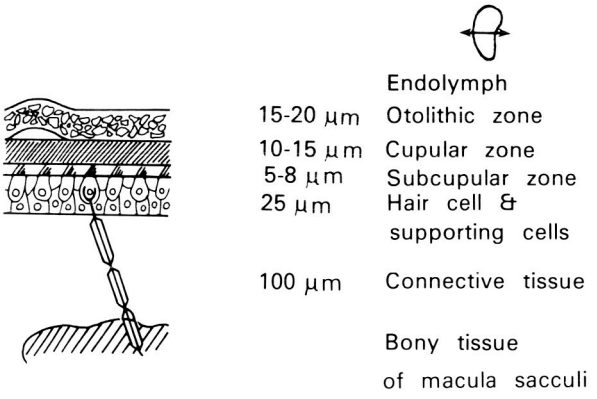
### *Semicircular Canals*

The mechanoreceptors in the two vestibular organs are similar, but the gross structure of the semicircular canals bears little resemblance to that of the otolith organs (Fig. 7). The three canals in each human labyrinth are mutually perpendicular, and each so-called semicircular canal actually forms a complete circuit by virtue of its connections with the utricle, near which the duct expands into what is called the ampulla. A section through the ampulla of an exceptionally well-preserved human specimen[5] is shown in Figure 11. The crista is a transverse ridge of tissue covered with the sensory epithelium containing hair cells (similar to those in the maculae) whose cilia extend into the cupula. The kinocilia in the hair cells are uniformly polarized; in the horizontal canals they are toward the utricle (utricular pole) and in the vertical canals toward the opposite pole. The cupula, a meshwork (presumably of collagen fibers), extends to the roof of the ampulla (not shown in Fig. 11), completing a fluid-tight gate across the ampulla, hinged

[5] Kindly provided by Professor Makoto Igarashi, Department of Otolaryngology, Baylor University College of Medicine, Texas Medical Center, Houston.



a A view of macula sacculi from a squirrel monkey. Zonal structure is clearly seen.




b Schematic of the zonal structures in the squirrel monkey.

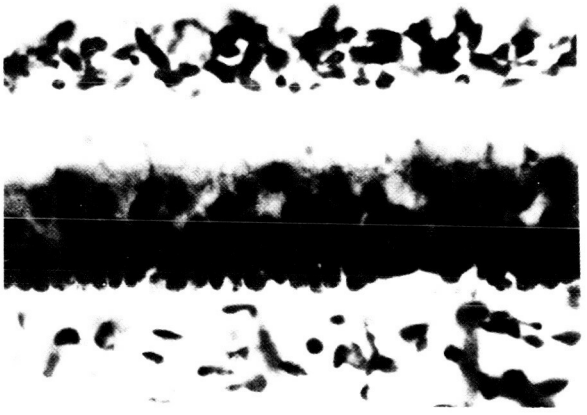


FIGURE 9.—Cross section of macula saccular of a squirrel monkey with zonal structure as inset. (Modified from Ref. [61])

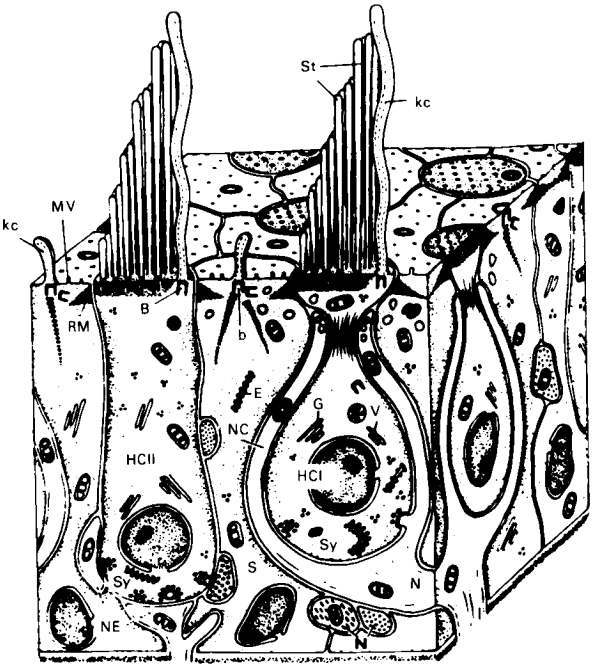



FIGURE 10.—Schematic of an area from a vestibular sensory epithelium with the two types of haircells (HCI and HCII), KC, Kinocilia; St. sterocilia. (From Ref. [125])

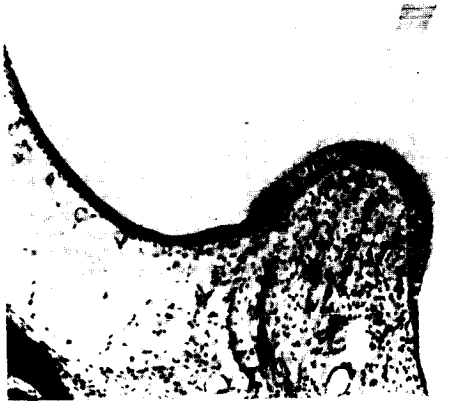


FIGURE 11.—Cross section of normal human ampulla (posterior semicircular canal) showing the crista with its sensory epithelium surmounted by the cupula.

at the crista, and free to move back and forth in response to movements of the endolymph. This apparatus constitutes the cupula-endolymph mechanism. On rotation of the head, the endolymph lags behind the movement of the bony canal, thus displacing the cupula in a direction counter to the rotary motion. The cupula-endolymph system, responding only to impulse angular accelerations in the plane of the canal, has been likened to a fluid-filled torus, with the cupula responding to movements of the endolymph in the manner of a spring-mass system with viscous damping.

Head motions, under natural conditions, generate a high angular acceleration with the onset of rotation, transient in character, followed by a very brief period of rotation approaching constant velocity, and ending with another transient acceleration of opposite sign. Although the acceleration and deceleration magnitude may be different, the time-integral of angular acceleration at the onset and offset is equal (area under the curves). Thus, it is thought that under most natural conditions the end organ responds as an integrating accelerometer.

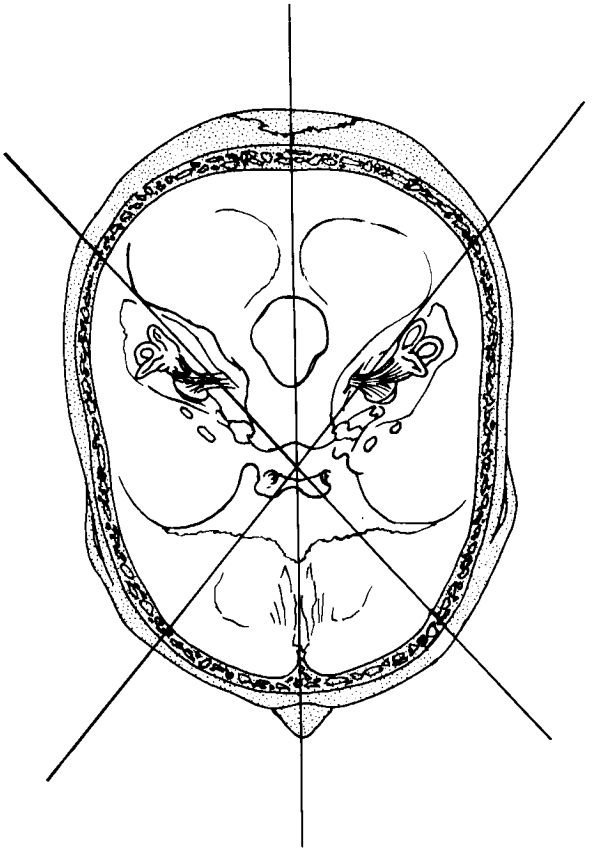


FIGURE 12.—Orientation of semicircular canals (enlarged) viewed in the skull from above.

Orientation of the six semicircular canals with reference to the head is shown in Figure 12. Although the three canals on one side lie approxi-

mately in mutually perpendicular planes, only the horizontal canals lie close to one of the coordinate planes of the skull, the superior and posterior canals deviating by 45° from the sagittal and frontal planes. Thus, rotary motions in the horizontal plane generating impulse angular accelerations would stimulate the horizontal pair of canals (although not maximally) with the subject's head upright; but, with head tilted forward about 25°, near-maximal stimulation would result. Rotation in the sagittal and frontal planes would generate angular accelerations in planes almost 45° from the planes of the vertical (superior and posterior) canals.

## Functional Neurology

The reflex character of the vestibular system differs markedly from the predominantly sensory character of the auditory system; auditory pathways to the cortex have been almost fully delineated, whereas a corresponding vestibular pathway has not been identified. The vestibular system functions automatically, mainly through motor effector mechanisms, which accounts for it not being placed in the same category as somatic sensory systems and that the vestibular organs are termed special sense organs rather than organs of special sense. The great differences in structure and modes of stimulation of the two end organs indicate that they serve different functions by providing different information; yet, when we leave the periphery, their identity is lost when we use the combining term "vestibular." Added to this vagueness is the need to take into account the differences between vestibular influences under natural and unnatural stimulatory conditions. Under certain abnormal stimulatory conditions, it would seem that "preferential pathways" are open to vestibular activity. A very brief condensation of the neurology of the vestibular system based on anatomical, physiological, and behavioral studies follows. References are given to detailed reports on these important but exceedingly complex aspects of the vestibular system.

### *Anatomical Aspects*

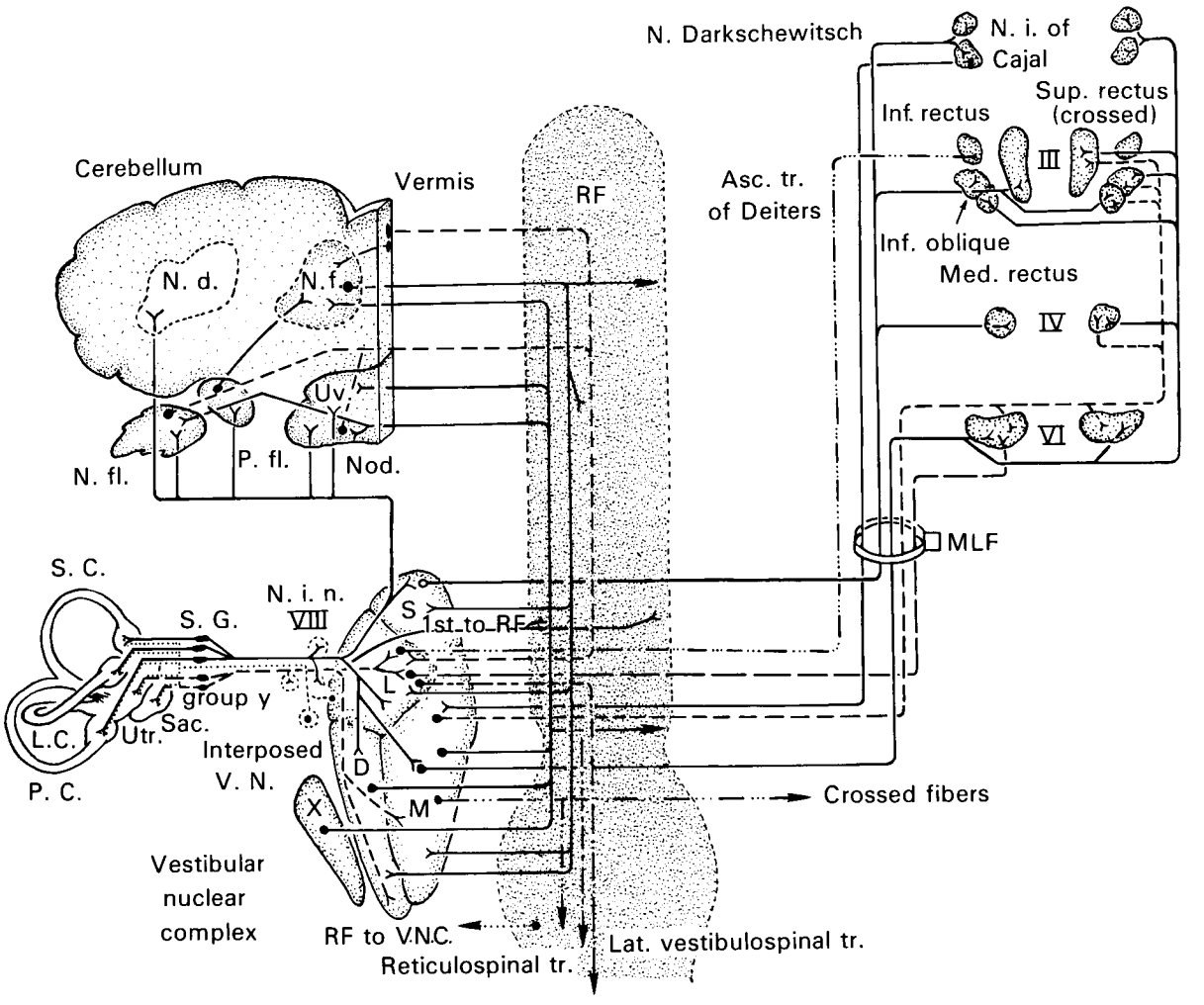
Morphological studies using classical techniques are definitive in nature, have important functional implications, and derive a great advantage from the high relevance to man of studies conducted on animals. Prior to the fourth edition (1952) of Rasmussen's *The Principal Nervous Pathways* [111], important contributions were made by Cajal, Lorente de No', Retzius, Burlet, Camis, and Vilstrup, among others. During the past 20 years, extensive morphologic studies have been carried out in a number of countries; the details may be found in the literature [14, 23, 112, 113, 133].

Figure 13 shows the main connections, revealed by classic anatomical techniques, comprising the reflex vestibular system. The vestibular nerve carries primary afferent fibers to the vestibular nuclear complex, cerebellum, and reticular formation and return efferent fibers to the mechanoreceptors in cristae and maculae. The vestibular nuclear complex (a term introduced by Brodal and associates [14]) comprises not only subdivisions within the confines of the four classical nuclei (superior, medial, lateral, and descending), but also small cell groups (known as *f*, *g*, *i*, *x*, *y*, *z*, *Sv*) and the interstitial nucleus of the vestibular nerve.

Cerebellar connections extend beyond the archicerebellum or classical vestibulocerebellum and include much of the vermis but not the hemispheres. Only a few fibers have been traced to the (pontine) reticular formation, but the absence of discrete nuclei may account for part of the sparsity. In general, sites of termination of primary fibers are sites of origin of secondary fibers that not only consolidate interrelations among the three major recipients of primary fibers but also ascend, descend, and cross the neuraxis.

*The vestibular nerve.* This nerve is the smaller division of the acoustic nerve coursing from the internal auditory meatus to the cerebellopontine angle where it enters the dorsolateral aspect of the brain stem, medial and somewhat ventral to the cochlear part of the VIIIth nerve.

*Efferent vestibular system.* Rasmussen, whose schema [111] did not show efferent fibers to the end organs, led the way in their discovery [112] and participated in defining their origin, course, and termination [123]. Efferent fibers arise in the lateral vestibular nucleus (Fig. 13) and, according to Rossi and Cortesina [121], in the nearby interposed vestibular nucleus. They leave




Abbreviations

III, IV, and VI: cranial motor nerve nuclei
D: descending vestibular nucleus
group y: small cell group
Interposed V. N.: Interposed nucleus of the vestibular nerve
L: lateral vestibular nucleus (Deiters')
L. C.: lateral semicircular canal
M.: medial vestibular nucleus
MLF: medial longitudinal fasciculus (ascending)
N. d.: dentate nucleus
N. f.: fastigial nucleus
Nod.: nodulus
Nin. VIII: interstitial nuclei (above and below afferents from cristae) of the vestibular nerve
N. i.: interstitial nucleus of Cajal
P. C.: posterior semicircular canal
P. fl.: paraflocculus
RF: reticular formation
S.: superior vestibular nucleus
Sac.: saccule
S. C.: superior semicircular canal
S. G.: Scarpa's (vestibular) ganglion
Utr.: utricle
U. V.: uvula
V. N. C.: vestibular nuclear complex
X.: small-celled group x

FIGURE 13.—Schema of the reflex vestibular system showing: (1) sites of origin and termination of first- and second-order neurons, (2) efferent fibers of the vestibular nerve, and (3) third- (or higher) order fastigial fibers. Note absence of pathways to the cerebral cortex.

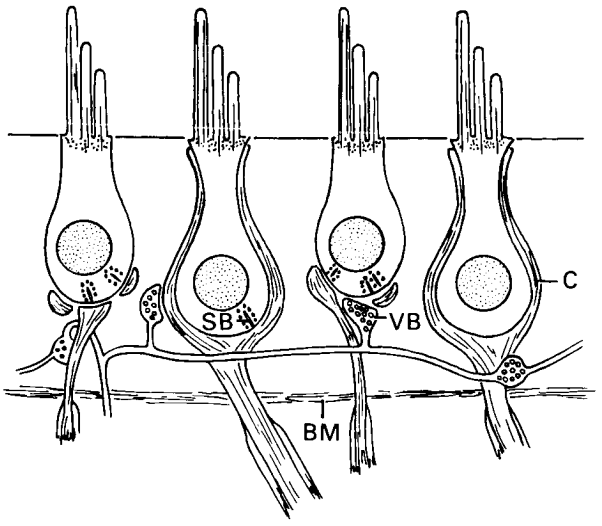



FIGURE 14.—Diagrammatic presentation of four hair cells, their nerve endings, and the relationships of vesiculated boutons (VB) to hair cells, chalice terminals (C), other boutons and nerve fibers in the chinchilla maculae. BM, basement membrane; SB, synaptic bar. It is believed the efferent nerves form a horizontal plexus as drawn.

the brain in company with the cochlear efferent fibers, reach the end organs in company with vestibular dendrites, and terminate on the second-type vesiculated boutons (Fig. 14) of all hair cells in cristae and maculae [123]. Anatomically, these efferent fibers complete a feedback loop, holding out the possibility that central influences of an inhibitory or regulatory nature can be brought to bear on the end organs.

*Primary afferent fibers.* Although primary vestibular fibers have been studied intensively, special mention should be made of the recent findings of Gacek [27], who traced their course from specific end organs to specific central terminations in the vestibular nuclei in the cat. He took into account fiber size at the end organ and, at central termination, cell size in the vestibular nuclei. Figure 13 indicates that primary canalicular neurons, after giving off short collaterals to the interstitial nucleus of the vestibular nerve, enter the brain stem where each axon divides into an ascending and a descending branch. The former terminates in the superior vestibular nucleus (and the cerebellum). The descending branches give off collaterals to the lateral, medial, and descending vestibular nuclei. Gacek was able to trace large and small fibers from the posterior canalicular cristae to large and small cells in the superior nucleus. (In higher vertebrates, the sensory epithelium of the horizontal canal was split off from the superior [vertical] canal; hence, the fibers from both are intermixed and impossible to trace as single bundles.) Primary utricular fibers, after dividing into ascending and descending branches, terminate, respectively, in the lateral and medial nuclei and in the medial and descending nuclei. Primary saccular fibers terminate mainly in the small-group *y* nucleus, with some fibers terminating in the lateral and descending nuclei. In summary, afferents from cristae and maculae are differentially distributed to sites in the vestibular nuclear complex; only canalicular fibers terminate in the interstitial nucleus of the vestibular nerve and in the superior nucleus.

Primary afferents have been traced to the flocculonodular node and ventral part of the uvula comprising the archicerebellum and to the ventral and dorsal paraflocculus and to the lateral dentate nucleus. First-order neurons do not reach the fastigial nucleus, contrary to former belief. Primary vestibular fibers to the cerebellum end as a particular type of mossy fiber, not only in the flocculonodular lobe comprising what Brodal [13] termed the classical vestibulocerebellum, but also the ventral and dorsal paraflocculus. Only a few primary fibers have been traced to the reticular formation, which is shown in Figure 13.

*Interconnections between cerebellum and vestibular nuclear complex.* In general, it is difficult (using classical anatomical procedures) to trace pathways in the vestibular system beyond second-order neurons, but an important exception is in the fastigial nucleus of the cerebellum, which is not a receiving site for primary vestibular fibers. The role of the fastigial nucleus as a relay station for cerebellovestibular fibers would also appear to serve as a major center in the vestibular system. The fastigial nucleus receives fibers from the vermis, paramedian lobule (both sides), dorsal paraflocculus, crus II, and vestibular nuclei. The fastigial nucleus sends fibers to reticular formation, all vestibular nuclei, mainly ipsilaterally, and especially to the lateral vestibular nucleus.

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Vestibulocerebellar fibers, mainly from the descending nucleus but also from the medial nucleus and group $x$, project, chiefly ipsilaterally, to end as mossy fibers in the flocculonodular lobe, ventral part of the uvula, and the fastigial nucleus.

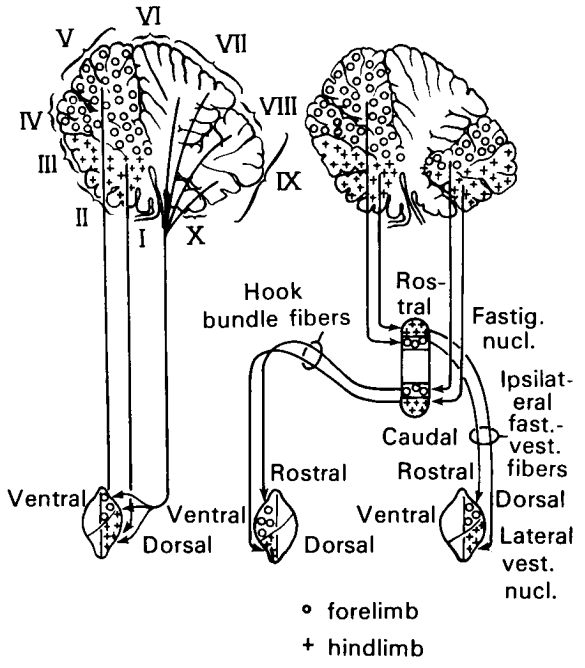



FIGURE 15.—Diagram illustrating major features in the projections from the cerebellar cortex onto the nucleus of Deiters (to the left) and (to the right) in the projections from the cerebellar cortex onto the fastigial nucleus and from this to the lateral vestibular nuclei. Note that the direct cerebellovestibular fibers and projection from the rostral part of the fastigial nucleus end in the dorsal half of the ipsilateral lateral vestibular nucleus, while fibers from the caudal part of the fastigial nucleus via the hook bundle supply the ventral half of the contralateral lateral vestibular nucleus. Within each of these projections there is a somatotopic localization. (From Ref. [13])

With regard to cerebellovestibular fibers, distinction is made between second-order fibers and fibers from the fastigial nucleus. Second-order fibers, which originate in the archicerebellum and in vermal cortices (mainly anterior lobe), project chiefly to the lateral nucleus. Fibers from the fastigial nucleus project to the reticular formation and to all vestibular nuclei; those fibers projecting to the lateral nucleus (which show a somatotopical arrangement) have been intensively studied by Brodal and his group [14]. Figure 15 shows the perseveration of an orderly arrangement from vermal cortices via fastigial nucleus to ipsilateral and contralateral Deiters' nuclei [14]. The ipsilateral system (involving forelimb and hindlimb) projecting to rostral and caudal parts of the lateral nucleus originate, respectively, in rostral and caudal parts of the anterior vermis with their relay stations in the rostral part of the fastigial nucleus. The contralateral system is analogous, except that it projects to the ventral half of the lateral vestibular nucleus and crosses the neuraxis in the hook bundle via a relay station in the caudal part of the fastigial nucleus.

*Ascending projections.* In continuing studies demonstrating a high degree of differential distribution of primary fibers to sites in the vestibular nuclear complex, Gacek [28] traced the ascending pathways from these sites to their termination. Five major pathways are shown in Figure 13; all except the ascending tract of Deiters comprise the (ascending) medial longitudinal fasciculus (MLF). Two pathways in the MLF may be activated by primary canalicular neurons constituting the ascending and descending branches. The former, arising in the superior vestibular nucleus, ascends ipsilaterally, giving off fibers to N IV and N III (some crossing to the nucleus of the medial rectus), and terminating in the interstitial nucleus of Cajal and the nucleus of Darkschewitsch. The continuation of the descending branch (of the primary canalicular fibers) arising in the medial vestibular nucleus after giving off fibers to N VI bilaterally, ascends contralaterally, giving off fibers to N IV and N III (nucleus of the superior rectus and inferior oblique) and terminates in the interstitial nucleus of Cajal and Darkschewitsch's nucleus.

Three pathways may be activated by primary macular fibers constituting the ascending and descending branches. The former splits in the lateral vestibular nucleus, one part coursing outside the MLF (as the ascending tract of Deiters) to terminate in the nucleus of the inferior rectus in N III, the other terminating ipsilaterally in N VI. Continuation of the descending branch (of the primary canalicular fibers) arising chiefly in the medial vestibular nucleus gives off fibers ipsilaterally to N VI, then crosses the midline,

giving off fibers to N VI, N IV, inferior oblique, medial rectus in the oculomotor nuclear complex, and nuclei of the superior rectus.

A direct cerebellar projection to (contralateral) N IV and N III and ipsilateral fibers in the reticular formation are not shown in Figure 13; these might comprise the independent vestibuloocular pathway readily demonstrated in physiologic studies.

*Descending pathways.* The three major descending pathways comprise the lateral and medial vestibulospinal tracts and the reticulospinal tract (Fig. 13). The lateral vestibulospinal tract arises in the somatotopically arranged part of the lateral vestibular nucleus and projects the length of the cord, preserving its somatotopical arrangement. Nyberg-Hansen [104] has described the terminations in great detail, based on Rexed's subdividions of the spinal gray matter, pointing out that fibers in the vestibulospinal tract influence the entire cord by modulating stretch reflexes and muscular tone. The medial tract (Fig. 13), arising chiefly in the medial vestibular nucleus, descends bilaterally in the (descending) MLF and terminates in the upper half of the cord without evidence of a somatotopical arrangement.

In summary, the anatomic organization of the reflex vestibular system, while not extensive (few third-order pathways have been traced outside the cerebellum) is complex. The complexities are evident in the high degree of differentiation from hair cells to sites of terminations of primary and secondary fibers, and in their interconnections both within the vestibular system and between vestibular and nonvestibular systems. Brodal [13] stated, "Much remains to be investigated. The anatomical data available at present indicate functional differentiations between cell groups and parts of nuclei which go beyond what has so far been clarified in physiological studies."

### *Physiological Aspects*

Only a few experiments had been conducted until recently, using electrical stimulation in normal unanesthetized animals. The use of abnormal stimuli, in addition to anesthesia or decerebration, raises the question whether the response is normal or if a vestibular side effect is involved. Depending on the animal used, there is the question of relevance for man. All these considerations are increasingly being taken into account; consider the use of human subjects, techniques that minimize departure from physiologic conditions, and testing at organizational levels characterized by similarity among species. Some examples have in common that vestibular activity involves pathways not yet identified, using classical anatomic procedures. These may involve vestibular projections beyond the reflex system, interactions between vestibular and nonvestibular systems, and the intrinsic organization of the vestibular system.

*Projections beyond the reflex vestibular system.* Razumeyev and Shipov [116] devote Chapter IX of their monograph to "connections of the labyrinth with the cerebral cortex." This excellent summary of the authors' studies (and of other investigators) gives details, as well as representative reports [1, 65, 88, 91, 93, 124, 130].

Studies have been conducted with human and animal subjects; accelerative, thermal, and electrical stimuli; and responses have been measured in electroencephalograms, electrocorticograms, or in recordings of single neuron activity in many parts of the brain.

It was established early that stimulation of the vestibular nerve in the lightly anesthetized cat elicited short-latency responses (around 0.6 ms) in parts of contralateral suprasylvian and ectosylvian gyri, that depended on the functional integrity of the nonacoustic labyrinth. Stimulation of the flocculonodular lobe elicits short-latency responses bilaterally, but removal of the cerebellum does not influence responses from the labyrinth.

Responses in the cat elicited by angular and "alternate" linear accelerations, studied in great detail by Razumeyev and Shipov [116], revealed differences, but, in general, the changes reflected the intensity of stimulation and were characterized by desynchronization in the electrocorticogram over wide (diffuse) areas. Short-latency responses elicited at the onset of acceleration yielded to long-latency response on deceleration and reached the brain by nonspecific pathways in the reticular formation.

Changes in the activity of single neurons in cortical and subcortical regions as the result of linear accelerative stimuli fell into four classes based on impulse frequency: (1) increase, (2) decrease, (3) phasic (with acceleration), and (4) no change. Convergence of vestibular and nonvestibular afferent "signals" were reviewed, categorized, and summarized by Razumeyev and Shipov [116] as:

> Electrophysiological experiments which have been performed to date show that the so-called specific cortical convergence of visual, vestibular, auditory and also, in all probability, somatic afferentation takes place almost exclusively in the anterior portions of the ecto- and suprasylvian gyri; i.e., in the portions of the cortex defined as cortical projection fields of the vestibular analyzer. Therefore, the assumption of Gorgiladze and Smirnov (1967), which states that the "vestibular cortical field is the coordination center which integrates afferent impulses from various sense organs and creates images of spatial relationships between the individual and surrounding objects of the visible world," appears to be correct.

Vestibular connections with the visceral nervous system have been described. Some of the early findings reported by Akert and Gernandt [1] may have to be amended where vestibulovagal connections are inferred. In a subsequent report, Tang and Gernandt [130] demonstrated the vestibular influences above, not below the point where the recurrent laryngeal parts company with the vagus. These authors reported that vestibular stimulation in cats elicited responses in recordings from the phrenic and recurrent laryngeal nerves. The responses were associated with increases in rate and depth of respiration and blood pressure. In a study to be reported, Tang [129] raises the possibility of artifacts vitiating many experiments involving electrical stimulation of the vestibular nerve.

*Vestibular-nonvestibular connections.* Electrophysiological studies have helped greatly in demonstrating connections between vestibular and nonvestibular systems; recent studies contribute much to knowledge of this aspect of vestibular neurology [25, 109, 116, 134]. Pompeiano [109] points out that in deep sleep, activity of second-order neurons in the vestibular nuclei increases phasically due to extralabyrinthine inputs; this results in rapid eye movement (REM) sleep. Wilson [134] has demonstrated that impulses from peripheral nerves ascending the spinal cord facilitate cells in the lateral vestibular nucleus that are sites of origin of the lateral vestibular spinal tract. Frederickson and Schwarz [25] investigated cells in the vestibular nucleus of unanesthetized cats by means of single-unit analysis. Ninety-nine percent of the units were responsive to labyrinthine stimulation and 80% to joint movement. There were no reponses to muscle pressure, optic, or acoustic stimuli, and cerebellectomy did not grossly alter joint influence.

*Intrinsic organization of the vestibular system.* Morphologists are among the first to point out that much work (including the use of electron microscopy) remains before anatomical observations suffice as a basis for functional interpretations. Among many examples, two must suffice for illustration.

The vestibuloocular reflex arc has long been of great interest. Fluur [24] found that selective stimulation of the nerve from individual semicircular canals in cats yielded two types of responses: Type I characterized by spontaneous activity in the extraocular muscles and conjugate deviation of the eyes; and Type II characterized by absence of spontaneous activity and of nonconjugate movements during stimulation. The differences in types of responses were considered in the light of differences in end organ receptors, the functional state either of the extraocular muscles and their proprioceptive mechanisms or the brain stem, and technical factors. Type I responses to selective stimulation of the nerve to the left lateral canal resulted in conjugate eye movement to the right, with activation and reciprocal inhibition of the appropriate muscles; similar stimulation in the left anterior canal caused upward deviation; of the left inferior, down deviation; and of both left vertical canals, counterclockwise rotary deviation. While stimulation of the horizontal canal caused deviation in that plane, stimulation of either vertical canal caused movement in the sagittal plane. In brief, impulses from a single canal must carry messages

not only to the extraocular nuclei, but also to their functional subdivisions. Gacek [28] raised the question—whether one of the two (canalicular) pathways might be inhibitory and the other facilitory, thus making unidirectional eye movements possible.

Cortical, cerebellar, and reticular influences on vestibular activity have generally been regarded as inhibitory, but these broad generalizations must be modified in the light of recent studies [25, 52, 62, 109, 116, 134]. An illustration is a model that Ito [62] has proposed, based on motoneurons in combination with certain receptors and muscles that would form a simple control system with a negative feedback loop (Fig. 16). With the insertion of the cerebellum in this control system, a more complex performance is possible (Fig. 17, part A, left). Ito reported that, insertion of the cerebellar nuclei between the cerebellar cortex and the brain stem may modify the ability of this unit in two respects: (1) integration of excitatory inputs with the inhibitory Purkinje cell signals is performed at the cerebellar nuclei, allowing the brain stem centers to carry out more integration with other signals (Fig. 17, part B, center); (2) a reverberating circuit may be formed between the cerebellar nucleus neurons and those originating in certain cerebellar afferents (Fig. 17, part C, right). Anatomical evidence suggests a reverberating connection between the descending vestibular nucleus and fastigial nucleus, between the paramedian reticular formation and fastigial nucleus, and between the pontine nucleus and the intracerebellar lateral nucleus. According to Ito, these connections would favor maintenance of a certain standard of activity in the cerebellum-brainstem system, which would provide the bias around which the dynamic characteristics of the system may be optimum.

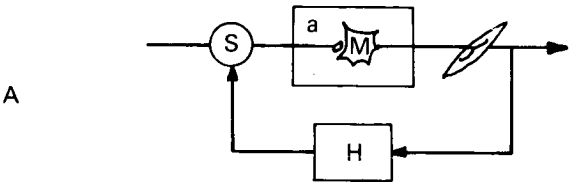



FIGURE 16.—Block diagram illustrating development of the motor control system. M: motoneuron. S: sensory part of the system. H: feedback loop.

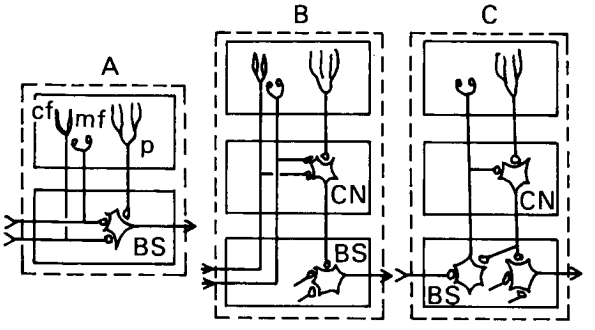



FIGURE 17.—Diagrammatic illustration of variation in cerebellar corticosubcortical connections. BS: brain stem, CN: cerebellar nuclei, cf: climbing fiber, mf: mossy fiber, P:Purkinje cell. See text.

### *Behavioral Aspects*

Vestibular responses (normal and abnormal) elicited in healthy persons have important neurologic implications. Indeed, our point of departure might well have been reversed (discussing behavioral aspects first rather than anatomical) since behavioral phenomena demand explanation while the anatomical do not. Thus, under natural living conditions today, man's nervous system may contain redundant or vestigal elements. Under unnatural conditions (motion environments), we must seek explanations for phenomena that only recently have become part of our lives. In addition to the distinction between behavioral phenomena elicited under natural and unnatural stimulus conditions, the latter (vestibular side effects) may be categorized according to the immediacy and nature of the response. Immediate reflex responses may be divided into those that represent perturbations of normal responses, e.g., nystagmus, and those that do not, e.g., perception of the oculogravic illusion. Delayed responses comprise epiphenomena, best known as motion sickness. Vestibular side effects will be discussed in subsequent sections; it will suffice here to point out a few examples that illustrate neurologic aspects not mentioned in the preceding section.

*Perception of rotation.* Under favorable conditions, when subjects are passively exposed for 10 s to angular acceleration about the vertical axis, thresholds for the perception of rotation

are in range 0.17°/s² [17], and on "sudden stop" after constant rotation at 1 rpm, the perception is "immediate" in behavioral terms. Persons with bilateral loss of vestibular (canalicular) function do not perceive rotation at the highest angular accelerations achieved in the laboratory. These findings suggest that vestibular impulses reach cortical levels when a normal type stimulus many magnitudes lower in intensity is used, rather than that generated during normal head movements.

*Perception of the oculogravic illusion.* This illusion is readily perceived when a person is exposed to change in direction of the gravito-inertial vertical on a certrifuge; it is not readily perceived by subjects with bilateral loss of vestibular function, especially when exposed during water immersion [45]. This influence on perception of the visual upright must involve integration of otolithic and optic neural activity.

*Pseudo-Coriolis illusion.* Recent studies by Brandt and colleagues [12] demonstrated pseudo-Coriolis effects, so termed—a visually induced perception of self-rotation and a pseudo-Coriolis illusion. The Coriolis (or oculogyral) illusion is readily perceived under favorable circumstances in a room rotating at constant velocity if a person rotates his head out of the plane of the room's rotation. The pseudo-oculogyral illusion can be elicited by substituting rotation of the visual environment (a striped drum) for rotation of the "room." Rotation of the head is essential, thus implicating the vestibular organs, although the head movements generate normal accelerative stimuli. After abolition of the visual stimulus, abnormal effects can be elicited for as long as 30 s. The sites of interaction between the normal vestibular inputs and abnormal visual inputs are probably in the medial and lateral vestibular complex and adjacent reticular formation, which was demonstrated by single fiber recordings in rabbits. Some fibers responded not only to accelerative but also to optokinetic stimuli. (In this connection it is important to recall that subjects who have never perceived light may, nevertheless, be highly susceptible to motion sickness when exposed to Coriolis accelerations [38].)

*Motion sickness.* Motion sickness (discussed in the next section) represents a constellation of delayed epiphenomena, precipitated by repetitive vestibular sensory inputs that are either abnormal or (if normal) encounter an abnormal integrative pattern. The immediate origin of cardinal symptoms is in nonvestibular systems; hence, first-order responses (at least) must reach cell groups via preferential pathways (presumably in the brain stem reticular formation) not used under natural stimulus conditions.

In summary, the vestibular system under artificial stimulus conditions readily evokes responses that range from near-normal (the oculogravic illusion) to the absurd (motion sickness). Preferential pathways and unusual interactions with nonvestibular systems deserve study for scientific reasons and for practical benefits.

### Input-Output Relations

The schema in Figure 18 represents an attempt to fit important elements concerned with vestibular input-output relations into a conceptual framework.

In Block I are the types and combinations of natural and artificial accelerative stimuli that reach the semicircular canals and otolith organs. The important contribution to artificial stimulus patterns made by man's motions, especially those involving rotation of the head, deserves emphasis. In the footnotes at the bottom of the schematic are: (1) categories of activation of the vestibular system, some of which are not accelerative in nature, e.g., disease process; and (2) typical activity patterns.

Block II indicates the transducer functions of the end organs. Although a feature common to both end organs is the conversion of the accelerative stimuli to electrical energy, thus altering the temporal and spatial patterning constituting the propogated discharge, the well-known differences between the two end organs must be taken into account.

The cupula-endolymph mechanism in the six semicircular canals responds to impulse and Coriolis accelerations and, for practical purposes, is gravity-independent. Under nearly all natural conditions, the canals are stimulated only by the motions of man that involve rotations of the head. Under artificial conditions, of course, the canals respond to the same types of accelerations generated by a machine. In the absence of head

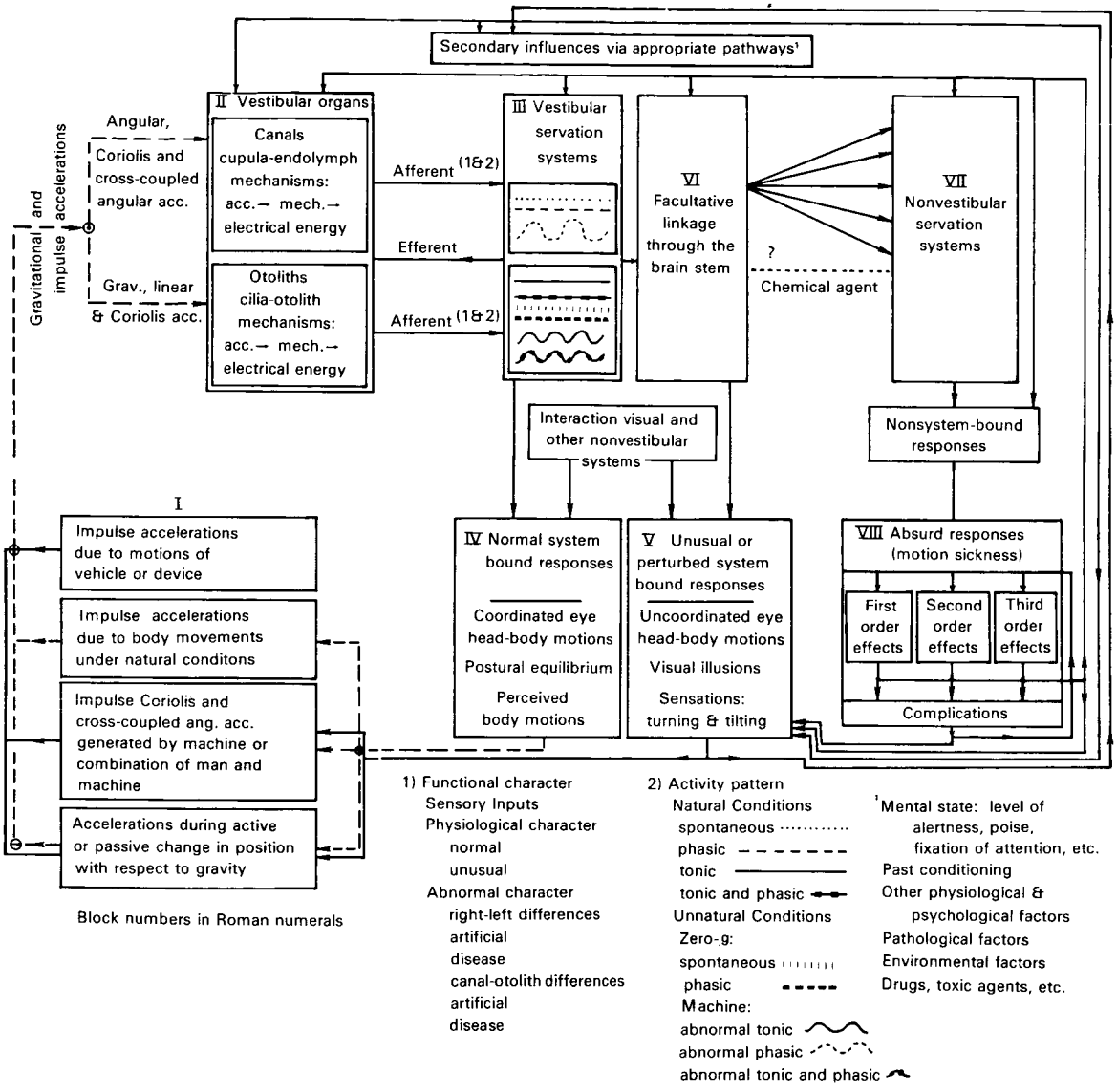



FIGURE 18.—Conceptual framework showing important elements and their interactions underlying system-bound vestibular disturbances and nonsystem-bound disturbances (motion sickness).

motions (active or passive) there is no accelerative canalicular stimulus, but a resting discharge, presumably of chemical origin, is present. Its precise origin and role, however, have not been determined.

The cilia-otolith mechanism in the four otolith organs is activated by gravity and by impulse linear and Coriolis accelerations directed so as to cause a shearing displacement between the otolithic membrane supporting the hair cells. The result is mechanical deformation of the cilia (kinocilia) which, in turn, results in chemical changes affecting the generation of bioelectricity (nerve action potentials).

Block III are the vestibular servation system and its two components (canalicular and otolithic), which have reciprocal modulating influences, and the vestibular efferent fibers ensuring a return flow of impulses to the end organs, thus closing one loop. An effort has been made to indicate typical normal and abnormal canalicular and otolith activity patterns and some opportunities for interaction with nonvestibular systems, notably vision.

### *Natural Terrestrial Stimulus Conditions*

Typical responses to which the vestibular organs contribute under natural terrestrial conditions are shown in Block IV(A) (Fig. 18), and the entire chain of events involves Blocks I through IV(A).

Astonishingly little is known concerning the normal function of the vestibular system in man under natural conditions. The canals and otoliths serve mainly as "participants" in motor functions, and it is exceedingly difficult to elucidate these contributory roles. The reason is that "natural activities" greatly limit the investigator, both in terms of stimulus manipulation and measurement and in the use of specific indications (responses) of canal or otolith stimulation that are available for measurement. Thus, the investigator must resort to unnatural stimulation of canals, otoliths, or both, which elicits abnormal responses that can be measured. In doing this he elicits the same responses experienced by susceptible persons in conveyances of different kinds which generate abnormal patterns of accelerations, with the important difference, however, that in the laboratory, the stimuli are under the experimenter's control.

A classical experimental approach to this question involves the use of human or animal subjects with bilateral loss of canalicular and otolithic functions. Experiments on animals alone, however, will never suffice; the findings are not directly applicable to man. The identification of human subjects with bilateral loss of labyrinthine function (L–D subjects) has been accomplished by screening groups of deaf persons, but experimentation on subjects identified in this way is complicated by the great differences between persons who hear and those who do not. Moreover, in all such subjects there is not only the need to make sure that pathologic changes are quiescent and adaptive changes are complete following any loss of function, but there is also the need to take into account the unmeasurable factor of "compensatory adjustment."

Despite these limitations, the best information has been derived from a comparison in performance of persons with and without vestibular defects. Under present-day ordinary living conditions, severe losses of vestibular function have gone undetected. This is dramatically illustrated by the rare persons with loss of vestibular function early in life but whose hearing had been retained [49]. Two such persons, discovered fortuitously, revealed that neither they, their families, nor their physicians were aware of the loss. Despite the loss of function being readily revealed, this takes little away from the fact that they met not only the ordinary demands of present-day living, but also were above the average in proficiency in a variety of sports. When apprised of their loss, it was brought out that they had experienced difficulties under circumstances where visual cues were inadequate and, possibly, in eye-head-body coordination when visual cues were inadequate.

A comparison between normal and L–D subjects under natural stimulus conditions reveals not only performance decrements but also the important observation that the stimulus to the otolith organs due to gravity generates a tonic discharge over and above the resting discharge. Performance decrement is readily demonstrated using tests for postural equilibrium [26].

When the head is "fixed" in the gravitational field, there is no question that the otolith organs are stimulated, indicated not only by the persistence of ocular counterrolling [98] and the oculogravic illusion [19], but also by these responses varying with hypogravity and supragravity g-loadings [97]. Whether the effective stimulus is due to a constant weight (or pressure) or to slight unavoidable changes in position of the macular plates with respect to gravity (even though the head is presumably fixed) is not clear. In either event the stimulus gives rise to a tonic sensory input over and above the resting discharge, and the significance of this finding for exposures in subgravity and weightlessness is obvious, although Yuganov [140] has brought out evidence that weightlessness may act as a specific stimulus to the maculae.

Nashner [103] has published the results of a sophisticated study on postural sway under near-normal conditions. Based on available information, he developed a general postural control model that, in turn, was used in devising a series of experiments dealing with postural sway resulting from rotation about the ankle. The experimental findings were combined with the general model to develop specific models for the sensory-motor interfaces. Three normal subjects and one L-D subject participated. The latter, age 20, had compensated "to the extent possible" following bilateral transection of the VIIIth nerve, 2 years prior to testing. Four types of tests were conducted:

(1) Reflex response gains. In normal subjects the average gain of the stretch reflex response induced by small rotations was about one-third that necessary for postural stability. In the L-D subject with eyes open the gains were larger than in the normals but below those necessary for postural stability; with eyes closed the average gain increased markedly, and, for extensor muscles, resulted in "rigid" postural stability.

(2) Induced sway: thresholds for perception with eyes open. Threshold values in terms of response time and body angle are shown in Figure 19 [103].

(3) Continuous recording of postural response and body angle motion. With eyes open the "control strategy" is the same for the L-D subject and normal controls, but in making corrections for transient disturbances (higher center commands) performance was better for the normals than the L-D subjects. With eyes closed the strategy remains the same for normal subjects (periods of reflex stability and transient disturbances), but changes for the L-D subject in that reflex stability gives way to continuous oscillation.

(4) Frequency spectra of body angle

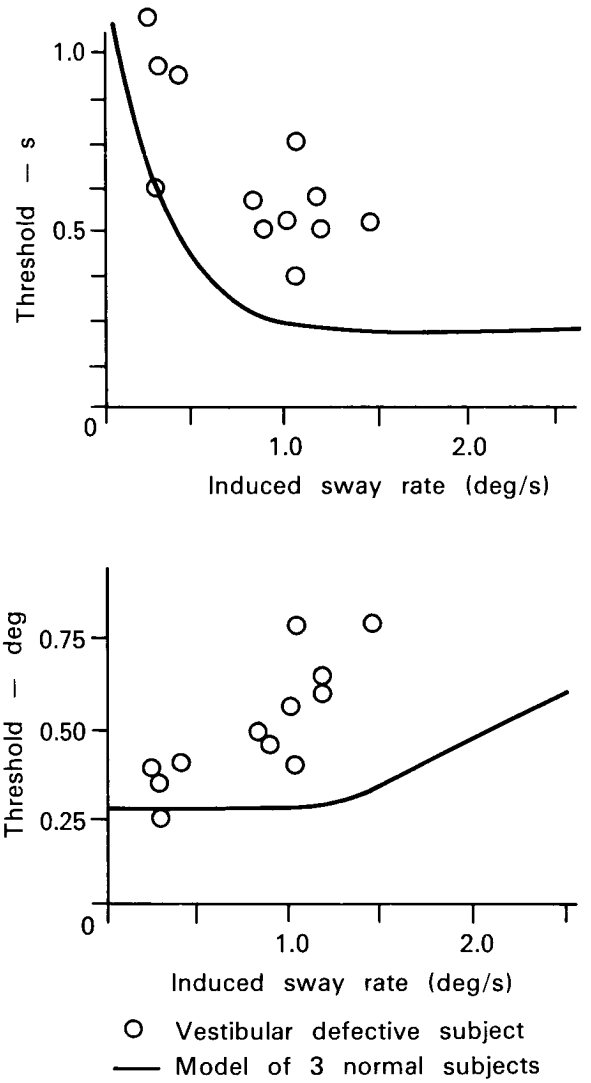



FIGURE 19. – Response threshold to induced sway in terms of response time and body angle; note increase when vestibular cues absent. (From Ref. [103])

motions. Comparative values are shown in Figures 20 A and B [103].

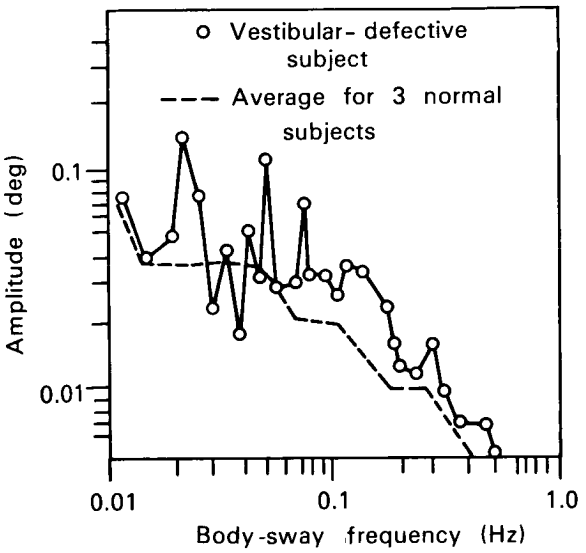



FIGURE 20A.—Body-sway frequency compared in vestibular-defective and normal subjects: Fourier coefficients of body-sway motion for vestibular-defective subject standing on a rigid, flat surface with eyes open. (From Ref. [103])

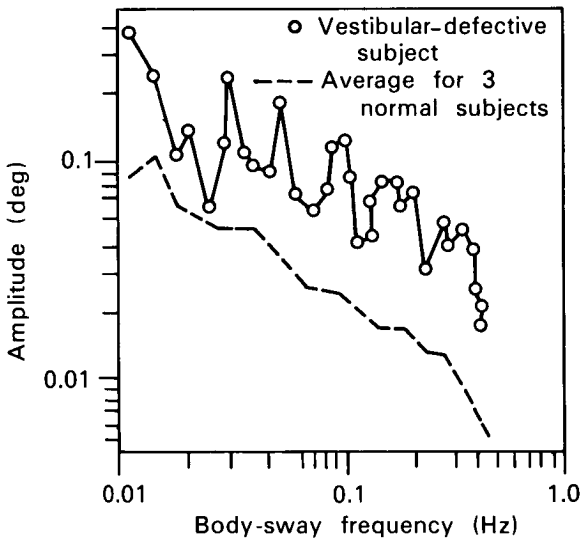



FIGURE 20B.—Body-sway frequency compared in vestibular-defective and normal subjects: Fourier coefficient of body-sway motion for vestibular-defective subject standing on a rigid, flat surface with eyes closed. (From Ref. [103])

In summary, the normal subject regulates posture with a combination of high-frequency (canal and somatosensory receptors) stabilization and low-frequency (otolith and optic receptors) stabilization; with eyes closed he still has otoliths functioning. The L-D subject with eyes closed is without low-frequency stabilization, resulting in a "rigid" stability.

### *Unnatural Stimulus Conditions*

The vestibular responses under abnormal stimulus conditions fall mainly into two categories: system-bound and nonsystem-bound. The main chain of events in system-bound responses involves Blocks I, II, III, IV(B), and V of Figure 18.

*Reflex phenomena.* Some, but not all system-bound responses reflect instability of the vestibular system, which will be referred to as reflex vestibular disturbances (RVD). Typical manifestations in normal persons include nystagmus, the oculogyral illusion, past-pointing, and postural disequilibrium. Systematic studies of reflex manifestations reveal characteristics of the various responses which may be observed or inferred and, in general, have in common: (1) short latencies, (2) maximal response to the initial stimulus, (3) no perseveration of responses unless explicable by continuation of stimulation, and (4) response decline with acquisition of adaptation effects.

A large, important body of information deals with the input-output relations of the semicircular canals, otolith organs, and their interactions. The literature dealing with eye motions [59, 90, 117] has reached the level of a subspecialty with the introduction of nystagmography. Reference may also be made to modeling of the vestibular system [21, 33, 36, 51, 53, 57, 60, 71, 92, 114, 139]; in this chapter, much of a practical nature will be mentioned.

*Delayed epiphenomena.* Nonsystem-bound responses (Blocks I–III and VI–VII of Fig. 18) constitute an epiphenomenon elicited by certain repetitive accelerative stimuli that not only disturb the vestibular system, but allow vestibular influences (by means of a facultative or temporary linkage) to stimulate cells or cell groups outside the system. These responses include the symptoms of motion sickness and are superimposed on any reflex manifestations also present. Inasmuch as they are not elicited in response to physiologic stimuli and serve no useful purpose, they may be properly characterized as absurd manifestations.

Little is known concerning the facultative linkage (Block V). That "irradiating" vestibular activity is demonstrably open to modulating influences points to the use of common pathways in the brain stem reticular formation; mild symptoms of motion sickness have disappeared under the influence of experimenter-directed tasks which may have preempted neural pathways used by irradiating vestibular activity. The vestibular facultative linkage is made unusual but not unique by the readiness with which vestibular activity may get "out of bounds" and elicit the widespread responses which include typical symptoms of motion sickness. The occasional long delay between onset of stimulation and appearance of motion sickness suggests that a chemical linkage may also be involved [37].

Certain secondary etiological influences are categorized in Figure 18 (right lower corner). Some of these influences, e.g., eyes open or closed, are always present, tending to increase or decrease susceptibility to motion sickness. Also, it may be assumed that any factor tending either to evoke or inhibit a response characteristic of motion sickness will affect the susceptibility accordingly.

Although typical symptoms of motion sickness are well-known, a list of first-order responses (let alone the precise sites of origin) has not been compiled (Block VII). At least some first-order responses also act as a stimulus, and so on, until the disturbances involve the organism as a whole.

The cardinal symptoms useful in making a clinical diagnosis include cold sweating, pallor, drowsiness, increased salivation, and the nausea syndrome. Release of the antidiuretic hormone and urinary excretion of 17-hydroxycorticosteroids and catecholamines are among the many biochemical changes that may be manifested [20, 63]. It is apparent that there are great gaps in our knowledge of mechanisms underlying the symptomatology of motion sickness. The starting point in conducting studies, it seems, would not be the full constellation of symptoms and syndromes but rather the first-order responses. Although there is general agreement on what constitutes frank motion sickness, this agreement dwindles with the reduction in number and kinds of responses. It is possible, for example, to elicit either sweating (probably a first-order response) or drowsiness (undoubtedly a second or higher order response) as the only definite overt symptom.

Typical manifestations of motion sickness are: (1) delay in appearance of symptoms (cumulation) after onset of stressful stimuli, (2) gradual or rapid increase in severity of symptoms, (3) modulation by secondary influences, (4) perseveration after sudden cessation of stimuli, and (5) response decline indicating adaptation.

Recovery during continual exposure to stress is complicated. First, the nonvestibular systems (Block VI) must be freed from vestibular influences (Block III) by adaptation taking place in the vestibular system. The point in time at which this occurs is difficult or impossible to determine because it is not immediately reflected by the disappearance of symptoms. Symptoms perseverate until restoration takes place (spontaneously) through homeostatic mechanisms. The time of engagement and disengagement between the vestibular and nonvestibular systems is best determined when a subject is exposed to severe stress for only a short period.

Another important omission in Figure 18 relates to mechanisms underlying adaptation in the vestibular system (with the possible exception of the role of vestibular efferent activity). In general terms, it would appear that individuals differ greatly in the ability to cope with abnormal vestibular inputs. Part of this difference may be attributable to differences in susceptibility and part to differences in the rate at which they can adapt to the abnormal inputs. The need to adjust must be triggered by a recognizable difference between the incoming stimulus pattern and the central patterning into which it must integrate. The fact that motion sickness may be prevented by incremental exposure to otherwise intolerable angular velocities in a slowly rotating room implies that this "recognizable difference" may be smaller than that necessary to elicit symptoms. An additional implication is that adaptation achieved by small increments in stressful accelerations must involve the vestibular system proper (Block III), and not the nonvestibular systems (Block VI) where first-order responses characteristic of motion sickness have their

immediate origin. Money [102] and Reason [118] have reviewed the literature on motion sickness.

## GROUND-BASED STUDIES IN PREPARATION FOR SPACE MISSIONS

In this section of the chapter there are five parts: Functional Tests, Provocative Tests, Adaptive Capacity Tests, Simulation Studies in Parabolic Flight and Rotating Environments, and Antimotion Sickness Drug Therapy. Only simulation studies will be discussed in detail, partly because some of the material is not readily accessible elsewhere, but mainly because they comprise the most important studies in preparation for space missions.

### Functional Tests

The tests described here should be regarded as possibly supplementing, and not taking the place of, a comprehensive clinical otolaryngological examination [7, 59, 68, 90], although there is evidence that function test scores within the normal range have no value in predicting individual differences in susceptibility to reflex vestibular disturbances and motion sickness [68]. They are valuable nevertheless for making comparative measurements, the crewman serving as his own control. The reliability of most vestibular tests is not high compared with vision or hearing tests; hence, there is need or desirability for repeated measurements on the crew serving as experimental subjects. Functional tests should be used not only in the selection of the crew but also in the selection of subjects for vestibular experimentation.

#### *Semicircular Canals*

Nearly all clinical test batteries include tests for spontaneous and positional nystagmus and a modified Hallpike test; hence, those tests will not be described here.

The threshold caloric test [89] is fairly reliable and may be useful; the vestibular disturbance is brief and recovery quick. Irrigating temperatures just below body temperature usually suffice, but if not, stepwise decreases are made until a response is obtained. If irrigating temperatures below 35° C are required to elicit a response, some abnormality should be suspected.

Rotating devices provide not only a physiological type stimulus (albeit abnormal) but also may be instrumented to include preprograming (Fig. 21). The most sensitive indicator is the oculogyral illusion, but "sensations" and nystagmus are used more routinely.

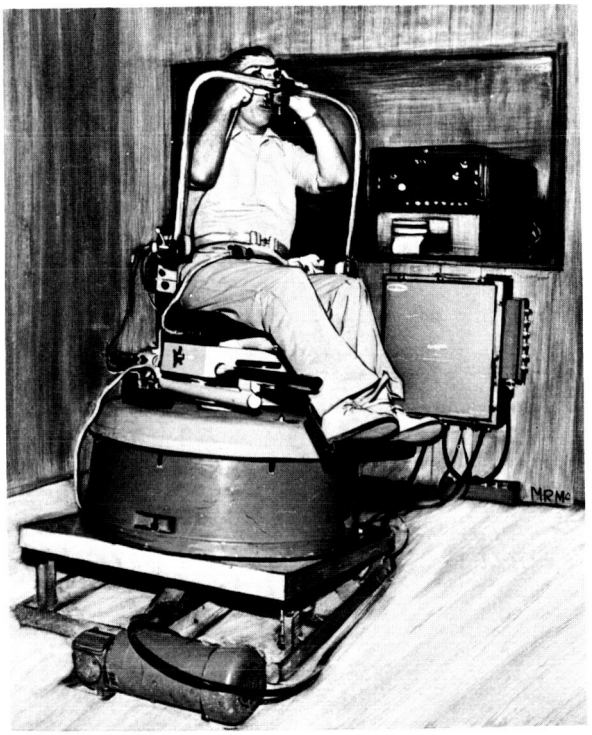


FIGURE 21.—Subject manipulating a dim line of light in darkness, using a goggle device with recording equipment. The target does not furnish a visual cue for space localization but does provide a good indicator. The readout is automatic.

The oculogyral illusion is a form of apparent motion in the direction of angular acceleration; its genesis is in the behavior of the cupula-endolymph mechanism. In measuring "thresholds," favorable conditions include a dimly lighted three-dimensional target viewed in darkness and fixed with respect to the subject, or a goggle device that greatly simplifies the method. The goggle device (Fig. 22), described elsewhere in detail [100], is essentially a collimated line of light in an otherwise dark field. This "line" can be rotated about its center by means of a knurled

knob. A digital readout of "line" position is easily seen and is accurate within ±0.25°. Normal thresholds of perception are in the range of 0.1 to 0.2°/s².

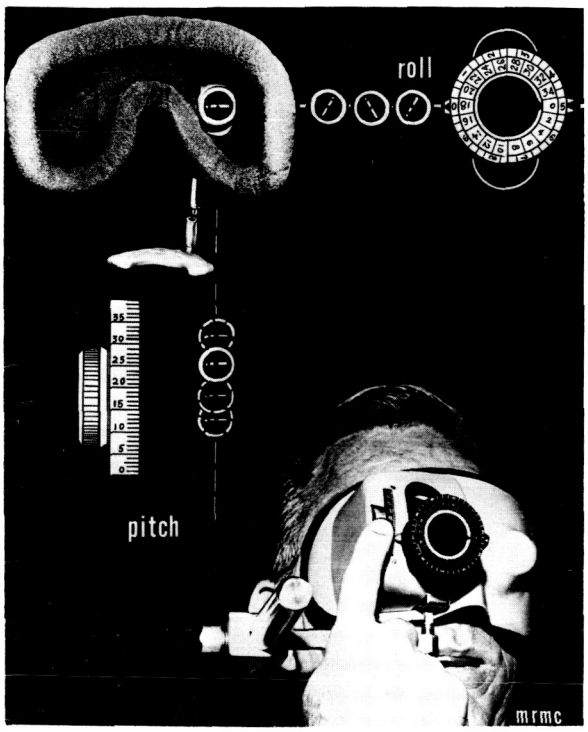



FIGURE 22.—Goggle device without automatic readout; pitch or roll, or both, may be measured. Note the dental appliance (individually fitted) that aids in maintaining the device in a fixed position with reference to the head.

### *Tests of Otolith Function*

Ocular counterrolling has the advantage of not disturbing the vestibular system; hence, it qualifies as a test conducted under near-normal stimulus conditions. Ocular torsion may be defined as the involuntary conjugate rolling movement of the eyes around their lines of sight in the direction opposite the leftward- or rightward-tilted position of the head (and body) with respect to the gravitational upright. The measurements are made by comparing the position of a metal frame, to which the subject is thoroughly secured (head secured by individually fitted dental appliance), and the relative position of the uncovered eye from colored photographs made in the upright and tilt positions. The roll is measured in degrees of arc.

The values obtained with different degrees of rightward and leftward tilt describe curves that can be examined for left-right symmetry. The "index" (one-half the sum of the maximal left and right roll) values obtained in a group of 550 presumably normal persons and 10 L-D subjects are shown in Figure 23 [94]. The rare instances when values fall below 120 seconds of arc are unexplained.

A variety of other tests is available but none is recommended as a substitute for ocular counterrolling. The elicitation of nystagmus in a device that rotates a person about an axis other than the Earth-vertical is receiving attention. Figure 24 shows the type of nystagmus elicited when a normal subject is rotated at constant velocity about an Earth-horizontal axis [44]. During rotation in a clockwise direction, the subject displays a right-beating nystagmus that is diminished as he rotates through the right-ear-down position and is augmented as he rotates through the left-ear-down position. During counterclockwise rotation, he displays a left-beating nystagmus that is diminished as he rotates through the left-ear-down position and enhanced as he rotates through the right-ear-down position. Both a directional bias and a cyclic modulation about the bias level are manifested, indicating that two etiologic factors are operant [126].

### *Test of Combined Canalicular and Otolithic Function*

*The postural equilibrium test battery.* These tests have a limitation in the sense that many systems in addition to the vestibular reflexes are challenged, but a great advantage is that they test natural behavioral mechanisms.

A useful test battery, described elsewhere in detail [26], comprising six individual items, requires the subject to stand or walk in the stringent position of body erect, arms folded against chest. The test items that constitute this battery are:

1. Sharpened Romberg (SR): standing on the floor in strict tandem heel-to-toe position with eyes closed, arms folded against chest, and body erect for 60 s.
2. Walk Eyes Open (Walk E/O): walking heel-to-toe with feet in strictly tandem posi-

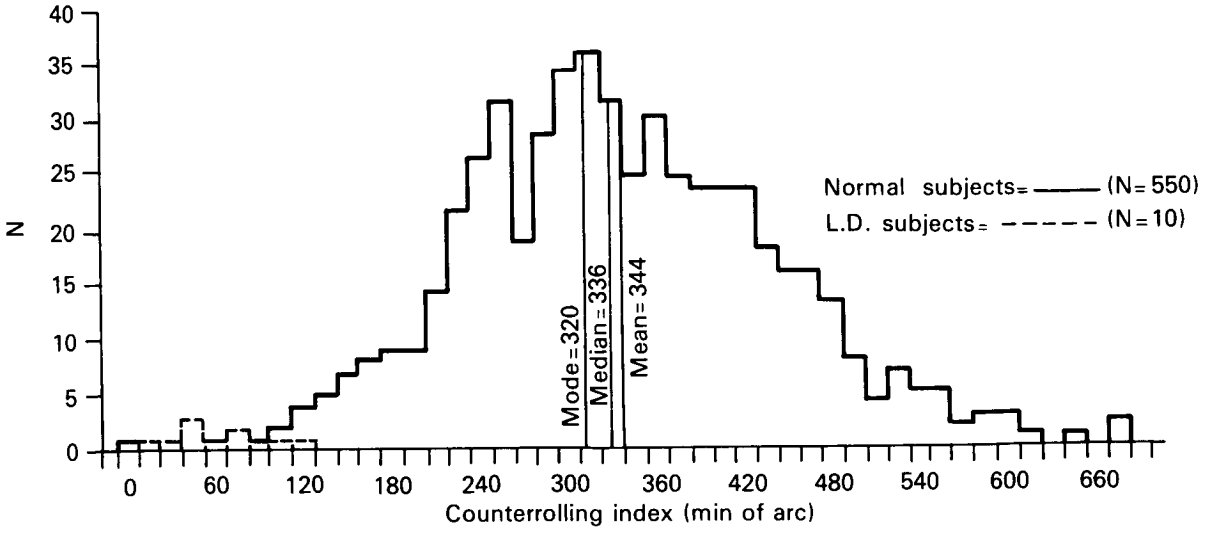



FIGURE 23.—Distribution of counterrolling index among normal and labyrinthine-defective subjects. (From Ref. [94])

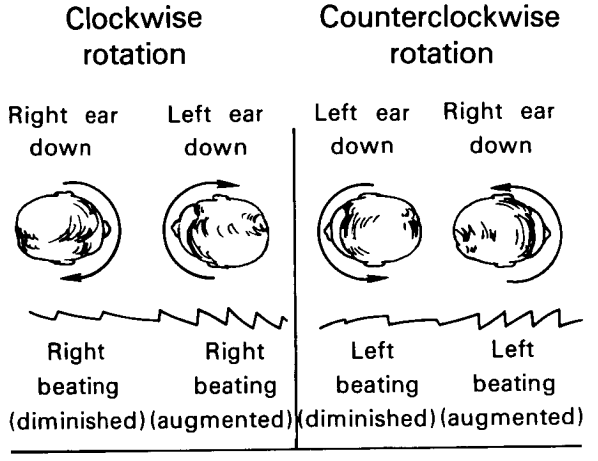



FIGURE 24.—Normal nystagmic responses (drawings) to rotation about an Earth-horizontal axis. Note directional bias and cyclic modulation.

tion and arms folded against chest while in a body-erect position on a 3/4-inch-wide by 8-foot-long rail.

3. Stand Eye Open (Stand E/O): standing heel-to-toe with feet in a strictly tandem position and arms folded against the chest while in a body-erect position on the 3/4-inch-wide rail for a period of 60 s.

4. Stand Eyes Closed (Stand E/C): standing, as for the Stand E/O test, on a 2¼-inch-wide by 30-inch-long rail for a period of 60 s.

5. Stand One Leg Eyes Closed (SOLEC-R and SOLEC-L): standing stationary on the floor on each leg for 30 s while arms are folded against chest and body in erect position.

6. WOFEC: walking on the floor eyes closed in the stringent position of arms folded against chest, body erect, and feet aligned tandemly heel-to-toe.

All subjects are tested while wearing shoes on a hard floor without rugs. The men wear hard-soled shoes and the women wear hard-soled "flats."

The scores and percentile equivalents measured on the normal subjects are shown in Table 1.

Comparative scores (normalized in percentile equivalents) for the other tests are in Table 2. A diagnosis of "frank ataxia" can be made if the walk-on-floor-eyes-closed (WOFEC) score is less than perfect and scores on the other tests are below the 6th percentile. The typical normal range requires a perfect WOFEC and scores about the 40th percentile in the other tests. A test comprising only the "floor" tests is quite satisfactory for screening purposes. In general, improvement in scores suggests normality, and its absence, abnormality.

TABLE 1.—*The WOFEC Test Scores of Normal Men and Women; Means, Standard Deviations, and Percentile Equivalents* [26]

| N = 287 Normal men Ages 17–61 (Mean age = 24.5; S. D. = 8.73) | | N = 100 Normal women Ages 18–65 (Mean age = 33.2; S. D. = 11.72) | |
|---|---|---|---|
| WOFEC score | Percentile equivalent | WOFEC score | Percentile equivalent |
| 30 | 100th–5th | 30 | 100th–12th |
| 29 | 4th | 29 | 11th |
| 27–28 | 3rd | 28 | 10th |
| 23–26 | 2nd | 27 | 9th |
| ≤ 22 | 1st | 26 | 8th |
| | | 24 | 6th |
| | | 23 | 5th |
| | | 22 | 3rd |
| | | 19–21 | 2nd |
| | | ≤ 18 | 1st |
| Mean: 29.7 S. D.: 1.65 | | Mean: 29.3 S. D.: 2.60 | |

## Provocative Tests

Provocative tests of many types are widely used [5, 30, 31, 40, 64, 83, 86, 95, 101, 106, 108, 128], serve the important purpose of evaluating susceptibility to reflex vestibular disturbances and to motion sickness, and measure ability to cope with such disturbances either with or without the aid of countermeasures, including the use of drugs. Factors of etiologic significance in addition to the motion environment may be introduced, to simulate more completely the anticipated operational conditions or to explore their role in affecting an individual's susceptibility to novel circumstances. The distinctions between provocative and simulation tests involve primarily duration and, secondarily, specificity in terms of the global exposure conditions; thus, the predictive value of provocative tests is less than that of simulation tests. The validity of the findings, similar to functional tests, is compromised if the person tested is either suffering from active disease involving the vestibular systems, or, indeed, has not compensated completely following permanent injury that is no longer active.

In conducting and interpreting the results of provocative tests, difficulties are encountered and precautions must be taken, which are not unrelated. The origins of the difficulties are: (1) the individual differences in susceptibility with regard to a given test; (2) intra-individual differences in susceptibility, when exposed in different gravitoinertial force environments; (3) preternaturally high susceptibility if insufficient time has not elapsed between exposures; (4) that adaptation occurs as an inevitable consequence of every test, with much individual variation in the rate of acquisition and loss of adaptation; and (5) the difficulty in expressing the results in absolute values. The use of normalized scores and standardization of techniques would provide great advantages.

Advantages of provocative tests include: (1) the low "cost" in terms of time and equipment that

TABLE 2.—*A Comparison of Scores of Normal Men with the L–D Men on the Ataxia Test Battery; Means, Standard Deviations, Mean Differences, and Validity Coefficients* [26]

| Chron. age and ataxia tests | Normal men (N = 287) | | L–D men (N = 22) | | Mean differences | Validity coefficients (r pt. bis) |
|---|---|---|---|---|---|---|
| | mean | S.D. | mean | S.D. | | |
| Age. | 24.5 | 8.73 | 27.5 | 8.11 | 3.0 | .089 |
| WOFEC | 29.7 | 1.65 | 14.1 | 7.82 | 15.6 | .838 |
| SR | 224.8 | 35.65 | 19.3 | 14.13 | 205.5 | .837 |
| SOLEC-R | 125.9 | 35.75 | 15.9 | 5.57 | 110.5 | .647 |
| SOLEC-L | 126.2 | 35.03 | 14.2 | 5.85 | 112.0 | .634 |
| Walk E/O | 12.6 | 2.48 | 7.0 | 2.51 | 5.6 | .502 |
| Stand E/C | 88.8 | 55.08 | 9.2 | 3.21 | 79.6 | .360 |
| Stand E/O | 35.3 | 29.64 | 8.6 | 2.17 | 26.7 | .234 |

makes a "test battery" feasible; (2) individual testing; and (3) their use in studying vestibular mechanisms and in evaluating countermeasures.

A number of provocative tests are in use, but brevity dictates limiting descriptions to a few representative tests relevant to spaceflight operations.

Standardized tests have been devised for determining susceptibility to vestibular side effects (usually motion sickness) in the Naval Aerospace Medical Research Laboratory, Pensacola, Fla., slow rotation room (SRR), with eyes open or in a rotating chair device with eyes closed. The stressful accelerations are generated by having the subject actively rotate his head (and body) out of the plane of the room's rotation. The head movements (front, back, left, and right) are limited by "stops," usually through arcs of 90°. Eight head movements, "over" and "return," in the four quadrants are randomized, and a taped recording sets the cadence.

Figure 25 shows the stress profile (Provocative Incremental Test Schedule) used in a slow rotation room in comparing susceptibility to motion sickness with eyes open and eyes closed [106]. The end point was a motion sickness score of

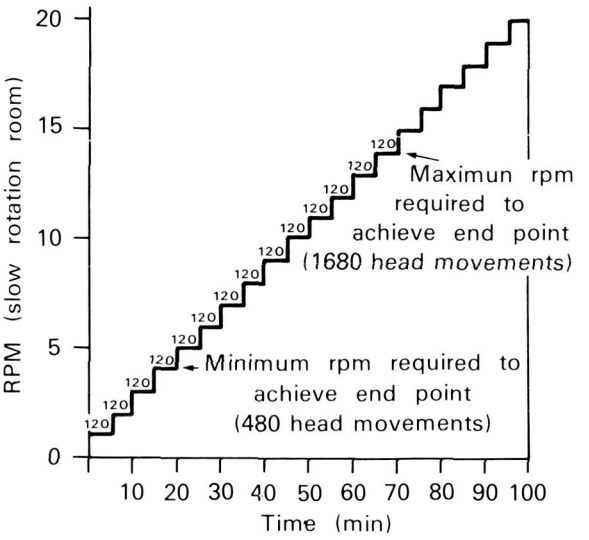



FIGURE 25.—Stress profile used in testing motion sickness susceptibility of 24 subjects. 120= number of head movements made in 4 quadrants at each step increase in velocity of the room. End point was 12 units on a scale used in grading severity of motion sickness.

FIGURE 26.—Off-vertical rotating chair device with recording equipment. Chair may be used upright for stimulating semicircular canals and in off-vertical (at constant angular velocity) for stimulating otolith organs. Instrumentation permits fairly complete programing.

C-4

TABLE 3.—*Diagnostic Categorization of Different Levels of Severity of Acute Motion Sickness* [96]

| Category | Pathognomonic 16 points | Major 8 points | Minor 4 points | Minimal 2 points | AQS [1] 1 point |
|---|---|---|---|---|---|
| Nausea syndrome | Nausea III [2] retching or vomiting | Nausea II | Nausea I | Epigastric discomfort | Epigastric awareness |
| Skin | | Pallor III | Pallor II | Pallor I | Flushing/subjective warmth ≥ II |
| Cold sweating | | III | II | I | |
| Increased salivation | | III | II | I | |
| Drowsiness | | III | II | I | |
| Pain | | | | | Persistent headache ≥ II |
| Central nervous system | | | | | Persistent dizziness<br>Eyes closed ≥ II<br>Eyes open III |

Levels of severity identified by total points scored

| Frank sickness | Severe malaise | Moderate malaise A | Moderate malaise B | Slight malaise |
|---|---|---|---|---|
| (FS<br>≥ 16 points | (M III)<br>8–15 points | (M IIA)<br>5–7 points | (M IIB)<br>3–4 points | (M I)<br>1–2 points |

[1] AQS—Additional qualifying symptoms.
[2] III—severe or marked, II—moderate, I—slight.

approximately 12 points (Table 3) [96]. Using the (terminal) rpm reached as the "normalized" score has the advantage of comparing susceptibility within and between subjects.

*The Coriolis Sickness Susceptibility Index.* This test represents a modification of the test just described, using a rotating chair instead of a room, and the subject is rotated with eyes closed [95]. A noteworthy feature of this test is the method of scoring, which yields a single value, the "index," enabling the investigator to make comparisons within and among subjects.

*Off-Vertical Rotation Test.* In contrast with the two tests just described, which initially "disturb" the canalicular system, exposure to rotating linear acceleration vectors or to rotation (at constant velocity) other than in the gravitational or gravitoinertial upright, initially disturbs the otolithic system. The device, shown in Figure 26, consists of a rotating chair [40] mounted on a platform that can be tilted either by a hand crank or by an electric motor, and the degree of tilt read from a large protractor. With each revolution of the off-vertical rotation (OVR) device the subject continually changes position with regard to the gravitational upright. Thus, receptors in the paired maculae of the utricles and saccules and nonvestibular proprioceptors are continually exposed to an unusual stimulus pattern. (This chair-device serves different purposes, including use in the upright mode.) In provocative testing, both the angle of tilt and the rpm are manipulated in different ways. At a predetermined angle of tilt, the rotation, programed on a time axis, involves periods of acceleration at 0.5°/s² for 30 s, followed by periods of constant velocity for 6 min, until either the end point is reached or 6 min completed at 25 rpm, the cut-off point. In effect, this program represents unit increases of 2.5 rpm every 6.5 min after the initial step. The end point can be expressed in terms of elapsed time at terminal velocity, as total elapsed time at terminal velocity, or as total elapsed time, which serves as an index of susceptibility to motion sickness. The findings in a group of healthy men, the great majority attached to a naval air station, are shown in Figure 27 [40]. All but 12 men reached the predetermined end point (M IIA) (Table 3) at a 10° tilt

[40]; all but five of the remainder reached it only when the angle of tilt was increased to 20°. Thus, the scores ranked 95 subjects in terms of their susceptibility to this unusual gravitoinertial force environment and demonstrated that five were highly insusceptible.

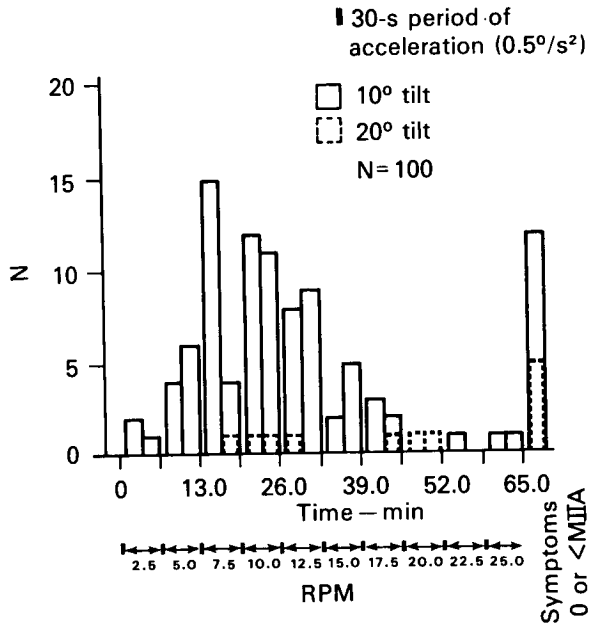



FIGURE 27.—Motion sickness susceptibility index in subjects exposed to off-vertical rotation according to programed stress indicated on abscissa. (From Ref. [40])

Figures 28 and 29 are plots comparing susceptibility to motion sickness with scores obtained in testing, respectively, the function of the semicircular canals and otolith organs [40]. Although it appears that significant relationships were not found between functional test scores and susceptibility to motion sickness, it is worth adding that, when extreme values are compared, susceptibility was lower in subjects with high rather than low values for the counterrolling index.

### Adaptive Capacity Tests

The relevant Soviet literature should be consulted for a detailed knowledge of their tests and procedures along with validating studies dealing with the important subject of vestibular training and adaptation [10, 58, 66, 107, 110, 142].

Khilov [66], in reviewing this material, began

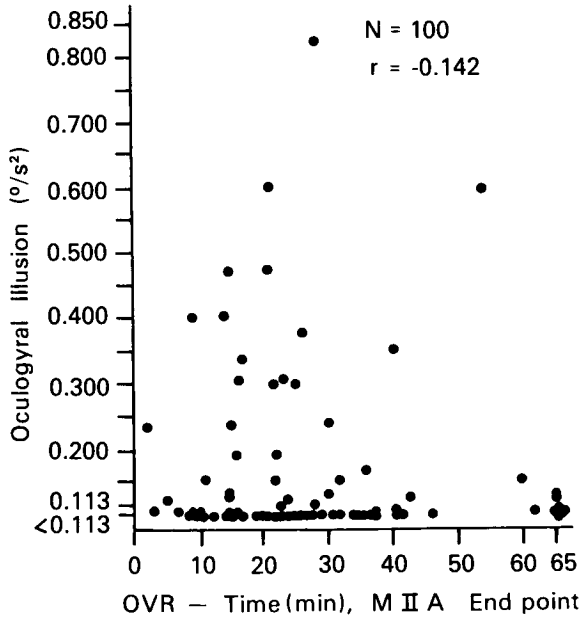



FIGURE 28.—Comparison of motion sickness susceptibility with scores on test of semicircular canal function (the oculogyral illusion). (From Ref. [40])

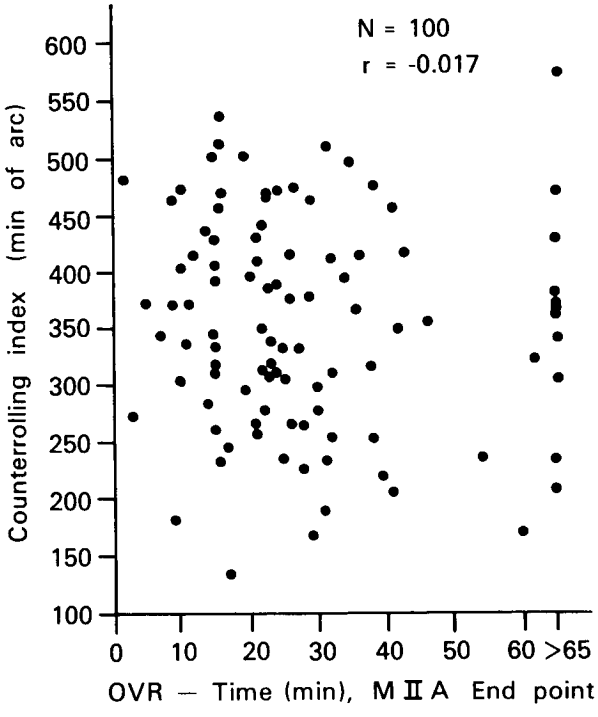



FIGURE 29.—Comparison of motion sickness susceptibility with scores on test of otolith function (counterrolling index). (From Ref. [40])

with the well-known tests used in the early days of aviation in the USSR, then described successive additions to the test battery. These additions were required to meet the need of not only selecting flyers with normal vestibular function but also to discriminate among those with normal function regarding their "sensitivity" to sensory inputs, the genesis of which are in successive increases in magnitude and complexity of the force environments in aircraft and spacecraft, including weightlessness. No less than seven such additions have been made, and validation of these additional test items has been carried out.

Khilov referred to one of his earlier articles expressing the opinion that otologists were ignoring the function of the otolith apparatus which, in flight, is subjected to greater stimulation than the semicircular canals.

With regard to otolith function, Khilov described a test based on the interaction between canals and otoliths (proposed by Voyachek in 1914). With trunk "inclined downward" the subject is rotated in a Bárány chair. After cessation of rotation "when primarily the frontal canals are responding" to the acceleration, the subject returns to the upright, "which is an adequate stimulus to the utricle altering the response of the canals. Persons in whom the duration of rotary nystagmus is reduced and in whom the reaction of falling is intensified with the simultaneous manifestation of autonomic reflexes, are not admitted to flight school."

Khilov rightly emphasized the point that persons with normal function of the canals and otoliths manifest great differences in sensitivity (motion sickness) and other innate or behavioral responses when exposed in different gravitoinertial force environments. This sensitivity may be overt or latent, and if so, it may be brought out by decreasing cortical inhibition through the use of chloral hydrate. The force environments associated with space were separately considered, although there is overlap with flights in aircraft.

In connection with space flight, he discussed the problems of repeated exposure to sustained high-level linear accelerations which can be simulated on a human centrifuge, the transition into weightlessness which can be simulated in parabolic flight, Coriolis accelerations which also are a possibility and readily simulated, and made recommendations on conducting a training program.

The importance of distinguishing between basic susceptibility to motion sickness and the role of adaptation has been explored at the Naval Aerospace Medical Research Laboratory in Pensacola, Fla. On a given occasion, a person's susceptibility to motion sickness is determined by: (1) the ease with which the vestibular system is disturbed in a particular motion environment, thus providing the opportunity for the escape of neuronal activity beyond its normal bounds; and (2) thresholds (presumably in the brain stem reticular formation) permitting the escape of this neuronal activity along certain preferential pathways (not normally used) to sites where first-order symptoms of motion sickness originate. If this person is exposed to an incremental adaptation schedule, additional information is gained regarding adaptation to the motion environment, but the level of susceptibility measured now comprises the two factors determining susceptibility just mentioned, minus the amount of adaptation acquired. These factors can be separated to some extent by using a modification of the stress profile mentioned previously, termed an incremental adaptation schedule (IAS).

Two "standard" stress profiles have been used. One required the execution of 120 head movements at each 1-rpm increase in rotation (clockwise or counterclockwise) between 0 and 6 rpm, and, after a single-step gradual return to zero velocity, the execution of 120 head movements either immediately after the return ("no delay") or after delay periods varying from 1 to 24 hours. The other standard stress profile differed by the addition of a second incremental adaptation schedule (IAS) in which the direction of rotation was reversed either immediately after return to zero velocity or after delay periods measured in hours. The terms "initial IAS" and "reverse IAS" are used because the initial direction of rotation was semirandom.

After each discrete head movement the subject signalled ("yes" or "no") whether he detected a "sensation" of movement, an apparent movement (visual illusion), or a tendency to be deflected from the plane in which the movement was carried out. The severity of motion sickness symptoms was given numerical scores according to the diagnostic criteria described in Table 3.

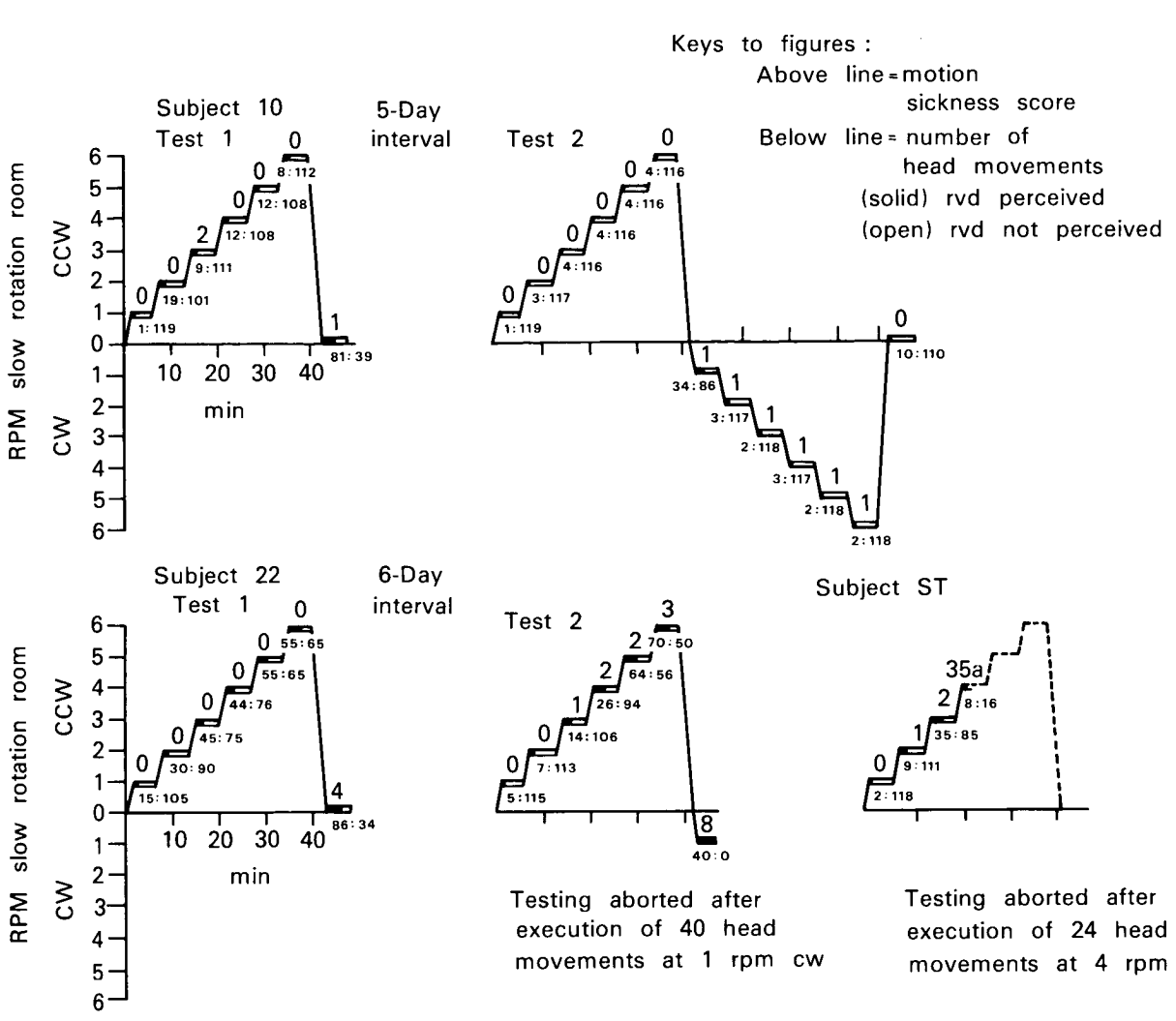



FIGURE 30.—Stress profiles, motion sickness scores, and occurrence of reflex vestibular disturbances in three normal young subjects: first and second numbers below line indicate, respectively, number of head movements when (one or more) reflex disturbances "perceived" and "not perceived." Subject 10 was virtually symptom-free (of motion sickness) despite a high occurrence of RVDs after return to zero velocity (Test 1) and during step 1 after reversal of direction in Test 2. Subject 22 experienced a high occurrence of RVDs but relatively low susceptibility to motion sickness in both tests. Subject ST demonstrated a relatively low incidence of RVDs but high susceptibility to motion sickness. For implications see text.

Figure 30 shows the measurements obtained in three young healthy subjects. On the two occasions Subject 10 was tested he was virtually free from symptoms of motion sickness. During the initial IAS in Test 1, the low susceptibility might be attributed mainly to the low level of instability in the vestibular system indicated by the low incidence of reflex vestibular disturbances (RVD). During the execution of head movements after return to zero velocity, however, a high level of vestibular instability is shown without elicitation of significant symptoms. In other words, insusceptibility involved the maintenance of both high stability in the vestibular system, and, even when the system was disturbed (after return to zero velocity), a high threshold preventing vestibular activity reaching sites of origin of motion sickness symptoms.

In the second test the incidence of RVD during the initial IAS was lower than in the first test, probably reflecting some retention of adaptation effects. On reversing the direction of rotation, the incidence of RVD rose sharply (at 1 rpm), but the subject again remained virtually immune to motion sickness.

Subject 22 was tested on two occasions. In Test 1 he did not manifest symptoms of motion sickness during the IAS, although the incidence of RVD's was high. During the challenge after return to zero velocity, very mild symptoms of motion sickness were experienced along with a substantial increase in the incidence of RVDs. The high incidence of positive responses (implying loss of stability in the vestibular system) was associated with a high threshold for spread of vestibular activity. In Test 2, during the initial IAS very mild symptoms of motion sickness were experienced, and rapid incremental increase in the incidence of RVD. On reversal of rotation, after 40 head movements testing was aborted due to nausea, and the RVD incidence was 100%. Despite the abort during reversal (indicating the acquisition of direction-specific adaptation effects during the IAS) the findings are in accord with those in Test 1.

The measurements obtained in exposing subject ST (Figure 30) to the stressful accelerations indicate typically normal findings during the execution of 120 head movements during the first three incremental steps, then an abort after 24 head movements at 4 rpm. The number of positive responses denoting instability of the vestibular system gave little or no clue to the impending abort. The time for acquisition of adaptation was brief, hence the high susceptibility, presumably, was the consequence of a low threshold permitting the escape of vestibular activity beyond its normal bounds.

In summary, a single test may reveal a great range of individual differences in adaptive capacity, and a succession of exposures can be used to reveal both the acquisition and the retention of adaptation, as described next in this section.

## Simulation Studies

Some problems posed in attempting to predict susceptibility to vestibular side effects under the novel conditions in a rotating space base are pointed out in Figure 31. A slow rotation room (SRR), which can be used to simulate the angular velocity, is a completely enclosed space and provides for prolonged exposures and sudden transitions between the rotating and nonrotating states. The SRR fails to simulate space-base conditions in such notable aspects as weightlessness, subgravity levels, man's orientation when upright with regard to the axis of rotation, and the Coriolis forces while walking and handling objects. Stated differently, the SRR provides a useful simulation device for the important study of effects of Coriolis accelerations[6], except for the fractional subgravity levels and man's orientation with respect to the axis of rotation. The SRR is useful in demonstrating the qualitative aspects of the vestibular organs' role in postural equilibrium and in walking, but here, nonvestibular factors also play an important role. The necessary use of small rotating devices poses limitations in terms of visual references, length of exposure, and postural equilibrium.

Parabolic flight offers the opportunity to study the effects of weightlessness and fractional subgravity levels for brief periods. Orbital flights prior to the establishment of a space base offer not only the opportunity to use small or even fairly large rotating devices for validation of ground-based experimental findings, but they also offer the advantages of prolonged exposure to study adaptation effects.

### *Parabolic Flight*

Studies involving parabolic flight have been conducted in the USSR and the US [68, 69, 72, 78, 80, 96, 141]. Insofar as the studies have used similar methods, the findings are not only concordant but also agree with findings on astronauts [9] and cosmonauts [66] in orbital flight.

Studies dealing with susceptibility to motion sickness in the weightless phase of parabolic

[6] There is general agreement in using the term Coriolis acceleration to define the "added acceleration" generated by one angular and one linear velocity. When the "added acceleration" is generated by two angular velocities acting on a mass simultaneously, some investigators (for clarity) have substituted for Coriolis acceleration such terms as "cross-coupled angular accelerations" or "angular Coriolis accelerations."

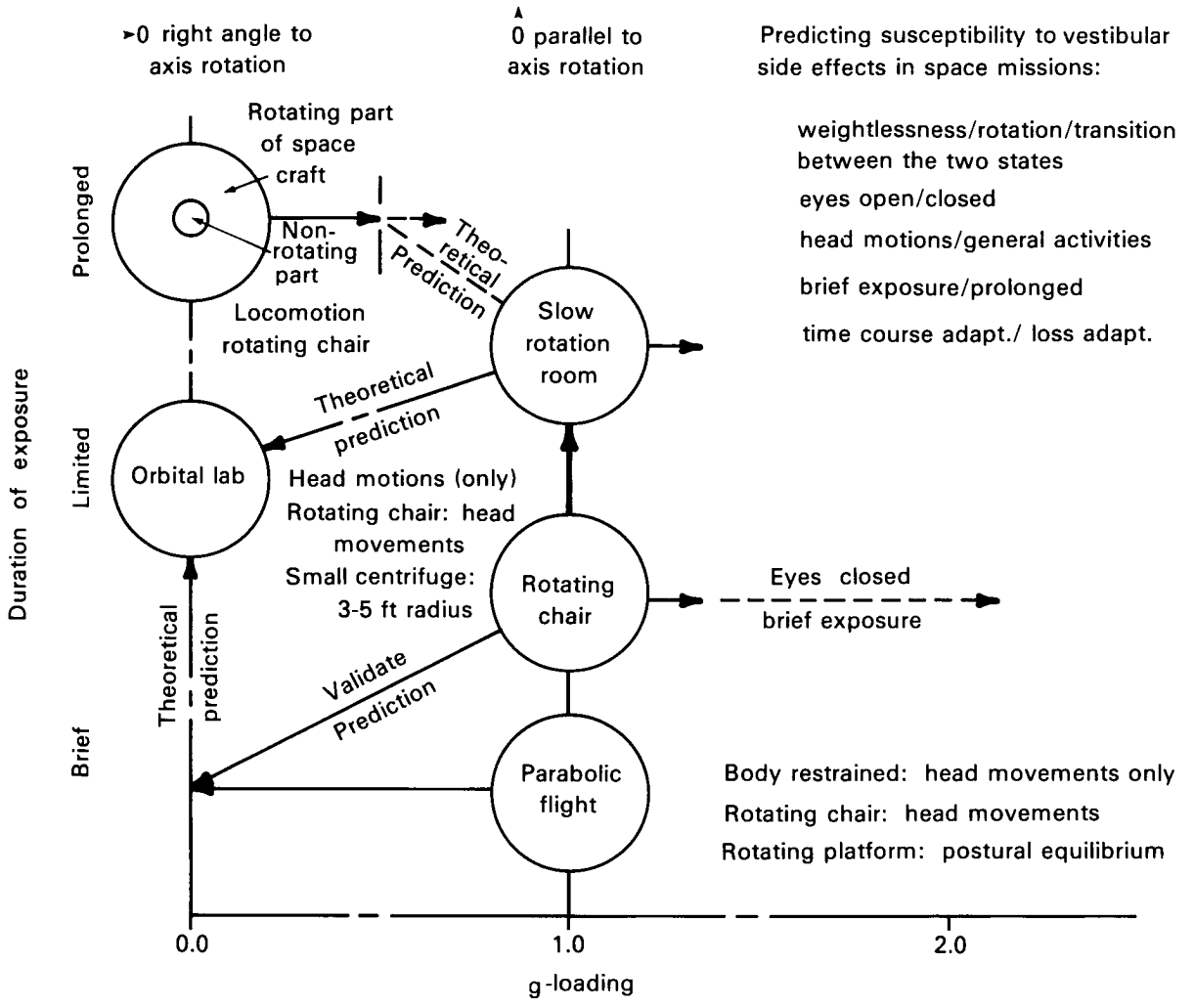



FIGURE 31.—Problems in predicting vestibular side effects in rotating space base.

flight have been mainly of two types. In one, the subjects were restrained in their seats and required to make standardized head motions during the weightless phase only. The findings in one experiment [96] are summarized in Figure 32 and demonstrate that, among the 12 subjects tested in this manner, six were asymptomatic. Five of the remaining six experienced symptoms only when making head motions; the last subject demonstrated increased susceptibility when making head motions compared to the head restraint (control) condition.

The second kind of experiment involved a rotating chair device, and the subjects were required to make standardized head motions similar to those used in the SRR but with eyes blindfolded. Each subject served as his own control; comparisons were made between susceptibility under terrestrial conditions and during parabolic flight, using similar periods of rotation and nonrotation. The findings on 74 subjects are shown in Figure 33 [96]. Susceptibility in weightlessness compared with ground-based conditions is ranked on the Y-axis, the topmost subject experiencing the greatest increase in susceptibility in weightlessness compared with terrestrial conditions. This ranking was made possible by the use of "equivalent head movements" (EHM), a universal scoring procedure described elsewhere in detail [95]. Scores on subjects tested on

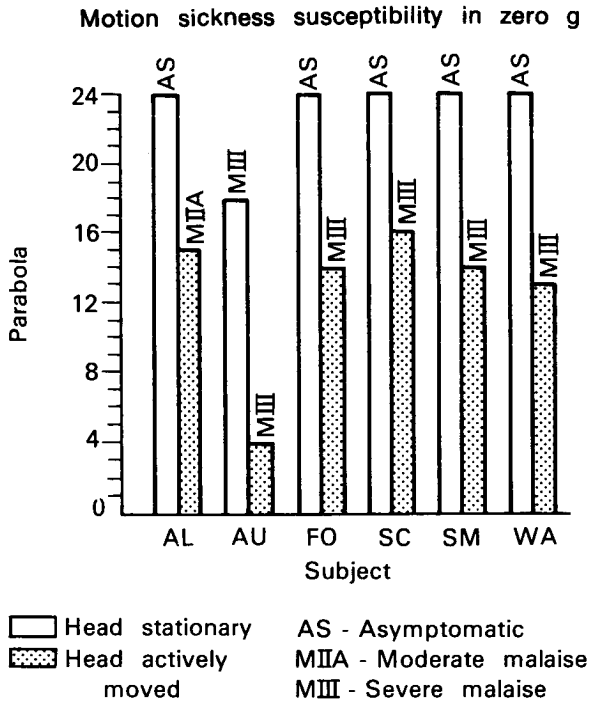



FIGURE 32. — Among six susceptible subjects, effects of active head movements relative to the restrained condition upon sickness susceptibility measured in terms of the number of parabolas required to provoke severe malaise. (From Ref. [96])

more than one occasion are given in chronological order, and the open circles indicate that the moderate malaise IIA (Table 3) end point was not reached [46]. The data indicate that more subjects have decreased than increased susceptibility in weightlessness and that in these subjects, the end point frequently was not reached. Susceptibility under ground-based conditions proved to be a poor indicator of susceptibility aloft.

The effects of preadaptation to the stressful accelerations generated by standardized head movements during rotation have been evaluated in 10 subjects.

Preadaptation to terminal velocities of either 7 or 10 rpm was accomplished by the use of so-called incremental adaptation schedules in a slow rotation room, the subject's eyes remaining open. In every instance the preadaptation was beneficial, often to a striking degree. Thus, in one subject, prior to adaptation, susceptibility was far greater in weightlessness than under terrestrial conditions, but after adaptation the subject was symptom-free in weightlessness. Moreover, whereas prior to adaptation the subject was susceptible to motion sickness in parabolic flight even when not rotating, after the adaptation he was symptom-free. In varying degrees, this transfer effect (from rotating room to nonrotating conditions in weightlessness) has been demonstrated in other subjects.

### *Rotating Environment*

A great number of experiments has been carried out with normal subjects exposed to continual rotation at varying angular velocities and for periods up to 25 days. Many of these investigations were concerned with the overall response pattern [3, 18, 22, 29, 42, 50, 56, 64, 70, 74, 76, 79, 84, 132], while others were directed toward more specific goals: response thresholds [34], effects of varying body position [55, 82, 85] and of concomitant accelerations [77], transfer effects (between horizontal and vertical positions) [47], effects on hearing [137] and on sleep [6, 35, 105], cardiovascular effects [131], and the release of stress hormones [20, 63].

A person is not subjected to stressful stimuli in a rotating environment unless he rotates his head outside the plane of the room's rotation; hence, the situation differs from that in ships and planes where a person cannot avoid stressful accelerations generated by motions of the vehicle. On the other hand, it is difficult to carry out tasks without generating stressful accelerations. Moreover, if stressful head movements are avoided, adaptation effects are not acquired. Generally speaking, it is found that, for a given level of bodily activity, the two important factors governing the appearance of symptoms are susceptibility of the unprotected subject and the angular velocity of the room. With regard to angular velocity, even subjects highly susceptible to vestibular side effects are not handicapped on sudden exposure to 1 rpm, but above this level, countermeasures must be taken to prevent elicitation of symptoms.

The prevention of side effects, especially motion sickness, involves avoiding or at least minimizing nonvestibular etiological factors, thus reducing the problem to the prevention of that

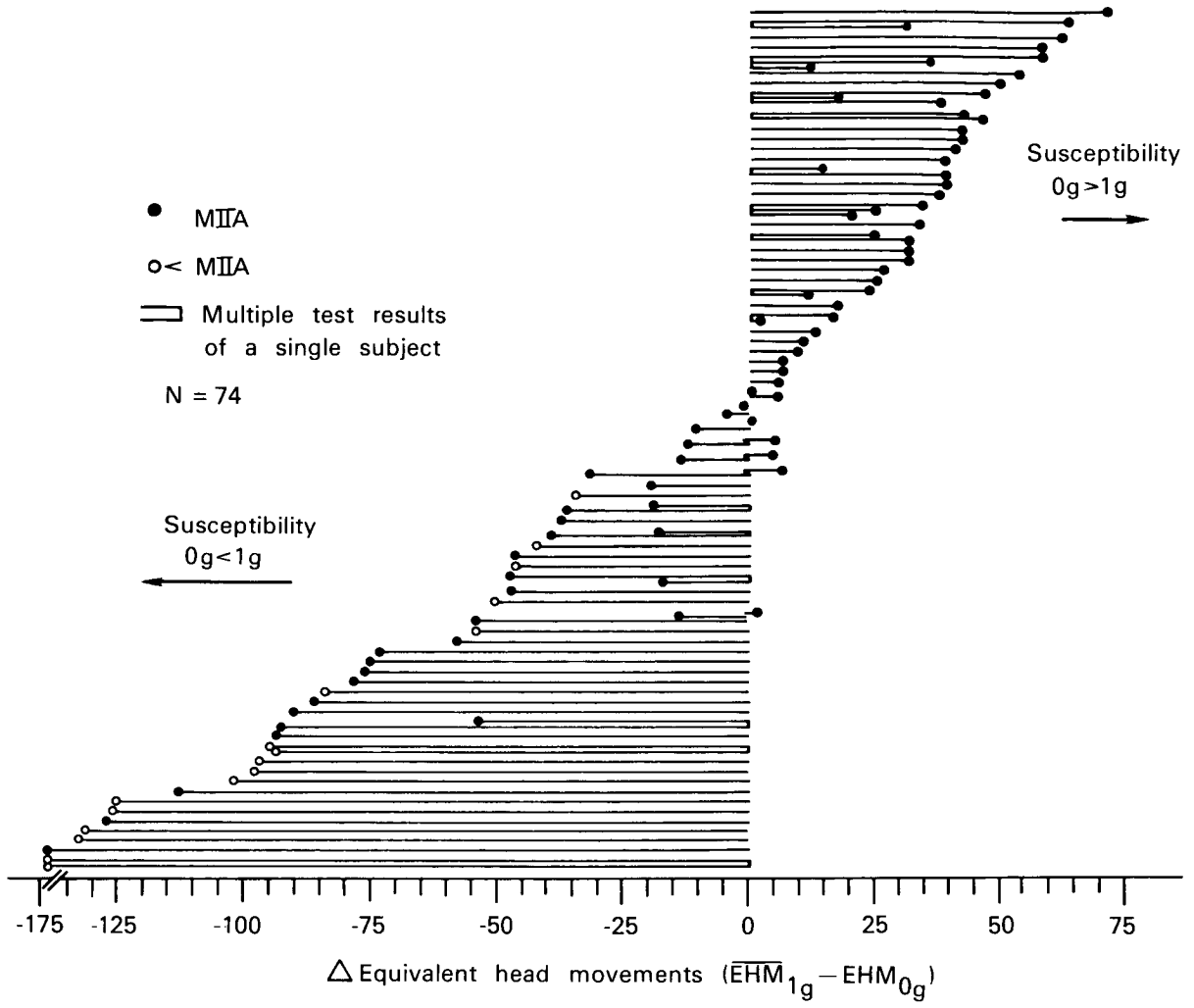



FIGURE 33.—Changes in susceptibility to motion sickness in 74 subjects on transition into the weightless phase of parabolic flight compared with similar stimulus conditions (standardized head movements during rotation) on Earth. The same end point was used but the stress was varied in intensity (rpm) and duration (indicated by number of head movements). "Equivalent head movements" implies these two stress factors were equaled (see text). Only a few subjects (scores near 0) experienced no change in susceptibility aloft, a majority were less susceptible (− scores), and a minority (+ scores) were more susceptible aloft than on the ground.

which might be termed vestibular sickness. Minimizing nonvestibular factors involves selection of typically normal spacemen (i.e. those whose responses to provocative tests are physiologic in character) and avoiding intrinsic and extrinsic factors known to lower susceptibility to motion sickness. Dealing with vestibular sickness eventually gets down to the selective process (discussed under **Adaptive Capacity Tests**), the use of incremental adaptation schedules and, possibly, antimotion sickness drugs.

With regard to adaptation schedules, three attempts to prevent motion sickness by step increases to a terminal velocity of 10 rpm at the Pensacola SRR [8] were unsuccessful; two involved three incremental steps during approximately 3 days, and the third a series of 40 incremental steps during 40 hours. In the next attempt [43], overt symptoms (with the probable exception of drowsiness) of motion sickness on exposure to otherwise intolerable stressful accelerations were prevented solely by means of

nine stepwise increases in rotational speeds during 25 days to a terminal velocity of 10 rpm. This experiment demonstrated the possibility of achieving (virtually) symptom-free adaptation to a rotation velocity of 10 rpm, but the time required was too "costly" for operational use, even for a terminal velocity of 4 rpm.

An attempt was then made to effect asymptomatic incremental adaptation in an experiment with three subjects required to execute experimenter-paced head-body movements [41]. The actual time spent making 1000 head movements was slightly more than half an hour. In Figure 34 [41] is shown the stress profile, the number of head movements made at each step (each up-down counting as one movement), and the level of symptoms experienced by the subjects. One subject, TA, was quite susceptible, becoming very drowsy at 2 rpm, experiencing epigastric discomfort at 5 rpm, and minimizing or refraining from making head motions at the higher rpm. The two remaining subjects experienced mild symptoms at terminal velocity, which became more severe on cessation of rotation. TA resorted to the use of an antimotion sickness drug. Noteworthy features were: (1) inability of TA to keep up with the schedule, (2) appearance of symptoms resulting from inadequate adaptation in the remaining two subjects, and (3) increase in symptoms experienced by all subjects on cessation of rotation.

The findings shown in Figure 35 [41] are from a similar test, except that more head movements were made at the higher angular velocities. Symptoms of motion sickness were trivial except in subject RO who experienced very mild symptoms at 8 and 9 rpm and on cessation of rotation. Except for ataxia, which was aggravated by head movements, complaints were minimal on cessation of rotation.

These findings confirmed the inferences drawn from the earlier studies and demonstrated that the time required to effect adaptation can be greatly shortened through control over head movements as well as over angular velocity and by setting up an adaptation schedule. The prob-

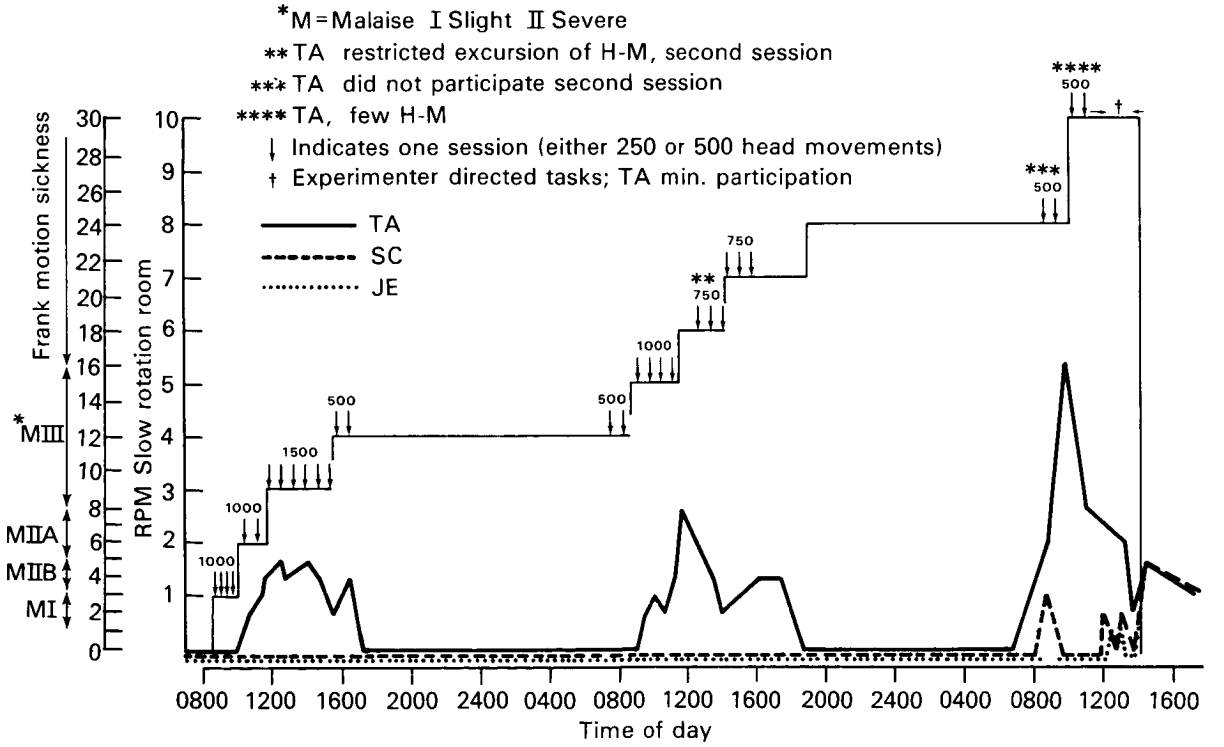



FIGURE 34.—Stress profile in the slow rotation room and manifestations of motion sickness in three healthy subjects exposed to rotation for more than 2 days. (From Ref. [41])

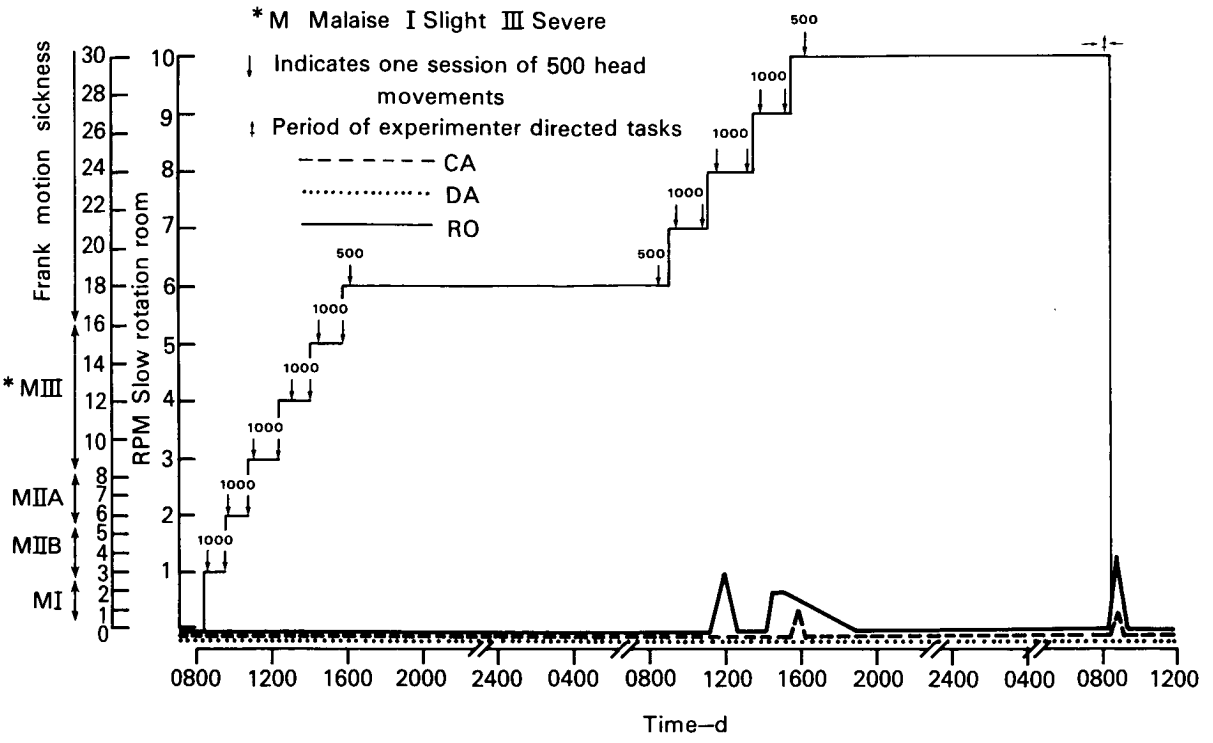



FIGURE 35.—Stress profile in the SRR and manifestations of motion sickness in three healthy subjects exposed to rotation for about 2 days. The large number of head motions accounts for the rapid adaptation. (From Ref. [41])

lems encountered were greater at relatively high compared with relatively low velocities, and that, except in one instance, problems were not experienced if the unit increase was 1 rpm.

A series of experiments was carried out in the SRR to determine if there were differences in susceptibility to vestibular side effects dependent upon man's orientation to the axis of rotation and if the acquisition of adaptation effects acquired in one orientation mode transferred to the other. A unique feature of this experiment was the provision for subjects to walk on the "wall" of the circular SRR and carry out their tasks while horizontal with respect to the Earth-vertical [47]. This was made possible by the use of air-bearing supports and custom-fitted articulated fiber glass molds. Four subjects participated in two different experiments involving adaptation to the stimulus conditions with the room rotating at 4 rpm for a period of either 4 or 5 days. One pair of subjects, initially in the horizontal mode, was changed to the vertical mode near the middle of the perrotation period when symptoms of motion sickness had disappeared; in the second experiment they began in the vertical mode. The order was reversed for the second pair. When in the horizontal mode, the subjects spent approximately 6 h/d in the airbearing device, 6 to 10 min upright, and the remainder of the time recumbent on a bunk. The findings, summarized in Figure 36, indicate no significant difference in susceptibility in the two modes and that transfer of adaptation is excellent. On cessation of rotation only mild symptoms of motion sickness were manifested. A byproduct of the experiment was the demonstration of important differences between motion sickness and postural disequilibrium during adaptation to the rotating environment and subsequent return to the stationary one. In the start-horizontal mode, adaptation ensuring freedom from symptoms of motion sickness on change to the vertical mode did not prevent ataxia. In the start-vertical mode, the adaptation resulted in greatly decreased ataxia; this adaptation perseverated throughout the finish-horizontal mode and as long as 36 h

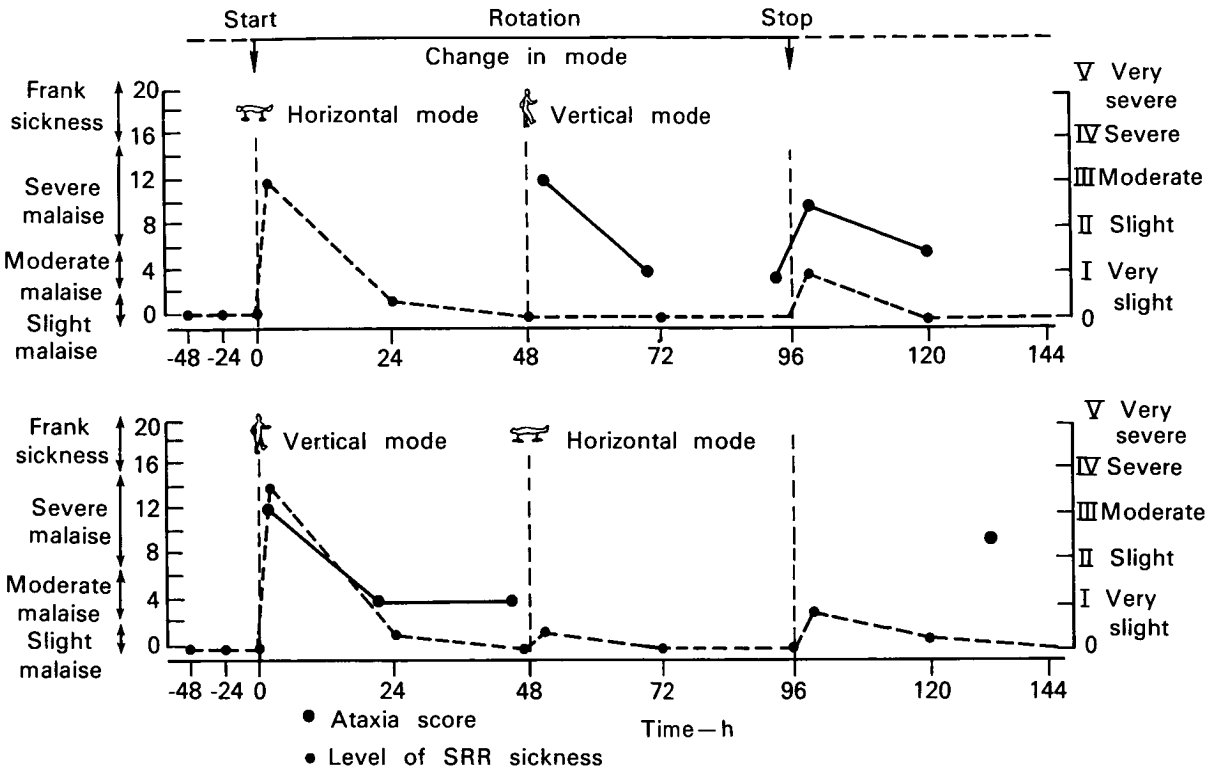



FIGURE 36.—Approximate mean changes in level of symptoms of motion sickness and in postural disequilibrium in four young healthy subjects exposed to continual rotation at 4 rpm.

after. This implied that the dynamic processes underlying postural homeostasis involved muscular activities largely rendered static when subjects were in the horizontal mode.

In the light of the experiment just described, earlier studies involving prolonged exposure in the SRR were reviewed, particularly from the standpoint of manifestations of motion sickness on cessation of rotation. An experiment in which four subjects were exposed at 10 rpm over a period of 12 d was notable in this regard [50]. Despite the severe symptoms that were experienced, especially in the first half of the perrotation period, manifestations of motion sickness on cessation of rotation were trivial or absent.

*Rapid transitions between a rotating and a nonrotating environment.* It has long been observed that normal persons with mild symptoms resulting from exposure to stressful accelerations in an SRR might experience an aggravation of motion sickness on cessation of rotation. In consequence, it appeared that sudden transitions between the weightless (nonrotating) and rotating parts of a space station posed the most serious aspect of generating artificial gravity. A series of studies demonstrated that: (1) persons remaining symptom-free during exposure to an incremental adaptation schedule (counterclockwise rotation) experienced motion sickness when the direction of rotation was reversed [37]; (2) head movements executed on return to zero velocity, after achieving symptom-free adaptation in an incremental fashion, would elicit symptoms of motion sickness [119]; and (3) adaptation to rotation in one direction transferred to rotation in the other direction [120].

The findings in an experiment to be reported [120] can be briefly summarized with the aid of Figure 37. Three subjects participated, and the adaptation schedule was the same for all subjects; the procedure was essentially the same as that described above in connection with Figure 30. On Day 1, while rotating counterclockwise, subjects executed 40 head movement sequences at 2 rpm, 50 at 3 rpm, 70 at 4 rpm, 90 at 5 rpm, and 110 at 6 rpm. The subjects, while rotating, were

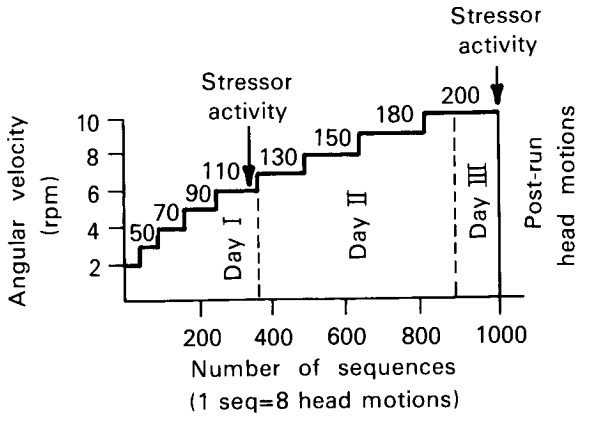



FIGURE 37.—Stimulus profile for a 3-day adaptaion schedule on the slow rotation room.

then transferred to carrying out highly stressful generalized activities in an attempt to evoke motion sickness, and their performance indicated that the head motions had produced a substantial degree of protection with respect to both reflex vestibular disturbances and motion sickness. On Day 2 the subjects executed 130 head movement sequences at 7 rpm, 150 at 8 rpm, 180 at 9 rpm, and 80 at 10 rpm. The subjects were again transferred to generalized activities, and their performance was similar to that on Day 1. On the morning of Day 3 after 120 head movement sequences at 10 rpm, the room was brought to a stop, and the subjects executed the same head motions as during rotation. There were no symptoms of motion sickness, and all reflex effects quickly disappeared.

The findings shown in Figure 38 were obtained on the same three subjects when they executed an incremental adaptation test before and after

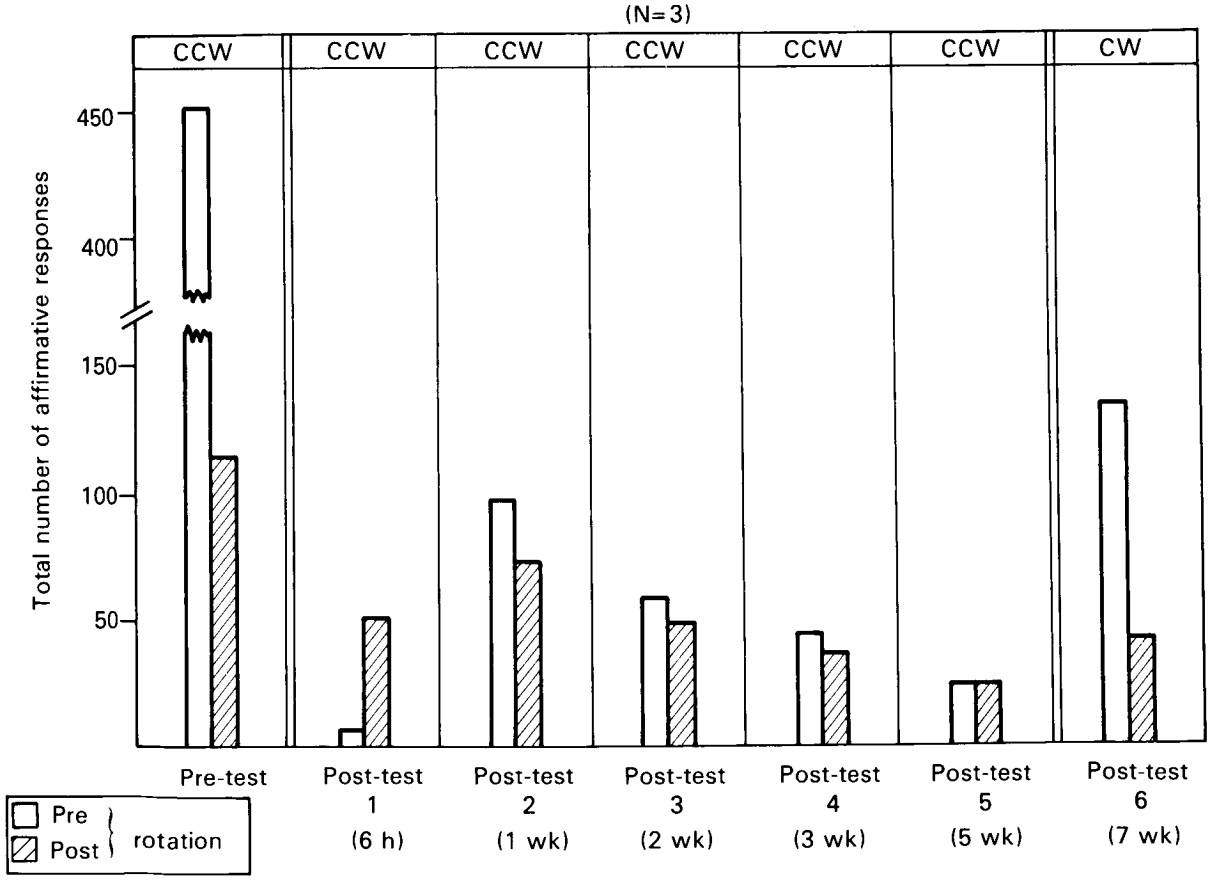



FIGURE 38.—Pre- and post-tests associated with a 3-day adaptation schedule for three subjects in the slow rotation room.

participating in the 3-day experiment just described. This test is also identical with the incremental adaptation test described in connection with Figure 30. The noteworthy findings are: (1) the small number of affirmative responses 6 hours after the 3-day experiment ended, (2) weekly exposures led to increasingly better performance, and (3) when the subjects were rotated in the opposite direction (clockwise), their performance was far better than on the first preexperimental test, indicating transfer of adaptation effects acquired during counterclockwise rotation. The findings support the conclusion that sudden transfers between the rotating and nonrotating environments are not only feasible in the SRR, but also the adaptation effects may not decay rapidly and with weekly practice may not only be retained but improved.

In a recent series of experiments [39], it was demonstrated that during exposure to an incremental adaptation schedule, normal persons simultaneously acquired both short-term (direction-specific) adaptation effects and long-term nondirection specific effects. The short-term effects disappeared spontaneously. This was demonstrated by requiring the subjects to remain seated or recumbent with head fixed for delay periods (measured in hours) after exposure to standardized incremental adaptation schedules. Figure 39 shows that the rate of decay (22 subjects) is exponential, revealed by the decline in susceptibility to motion sickness when head movements were executed at zero velocity.

With the spontaneous disappearance of direction-specific (short-term) adaptation effects, long-term adaptation effects are revealed that are nondirection-specific or nearly so. This important aspect was not investigated systematically but is illustrated by the findings in four subjects (Fig. 40). Measurements on subject 11 during his first four tests are shown in Figure 40, Part a. Prior to the one-step reversal in Test 4, the only other exposure in a reverse direction had been 20 days earlier in Test 2. The absence of symptoms during the execution of head movements at 6 rpm (CW) and especially after return to zero velocity suggests that subject 11 was completely insusceptible, with small likelihood that this insusceptibility was due solely to adaptation acquired during the reverse incremental adaptation schedule (IAS) in Test 2.

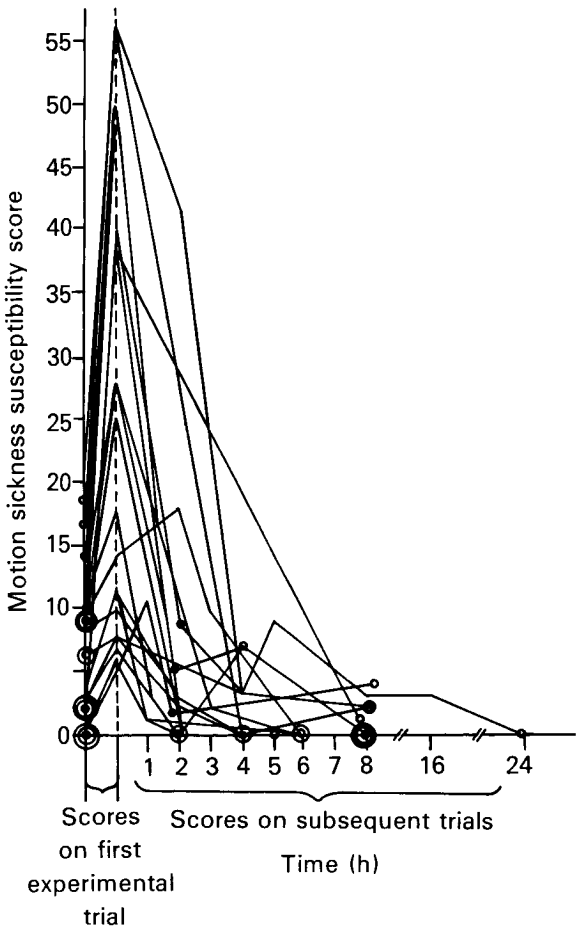



FIGURE 39.—Decay in direction-specific adaptation effects as a function of the time elapsed between completion of a standard incremental adaptation schedule (and return to zero velocity) and execution of head movements at zero velocity. The first two points on the graph represent susceptibility scores obtained in Test 1 of the series, at 6 rpm and after executing head movements at zero velocity, respectively. Thereafter, each point on the graph (or circles in lieu of points) represents susceptibility scores obtained in subsequent tests. An exponential curve characterizes the decay trend. (From Ref. [39])

Figure 40, Part b, shows the measurements obtained on subject 25 during his first four tests. In the second test he was completely symptom-free except for a score of 1 point during the reverse IAS after a 2-hour delay. In Test 1 the score of 6 points on return to zero velocity (a relatively weak challenge) suggests that testing would have been aborted during exposure to a

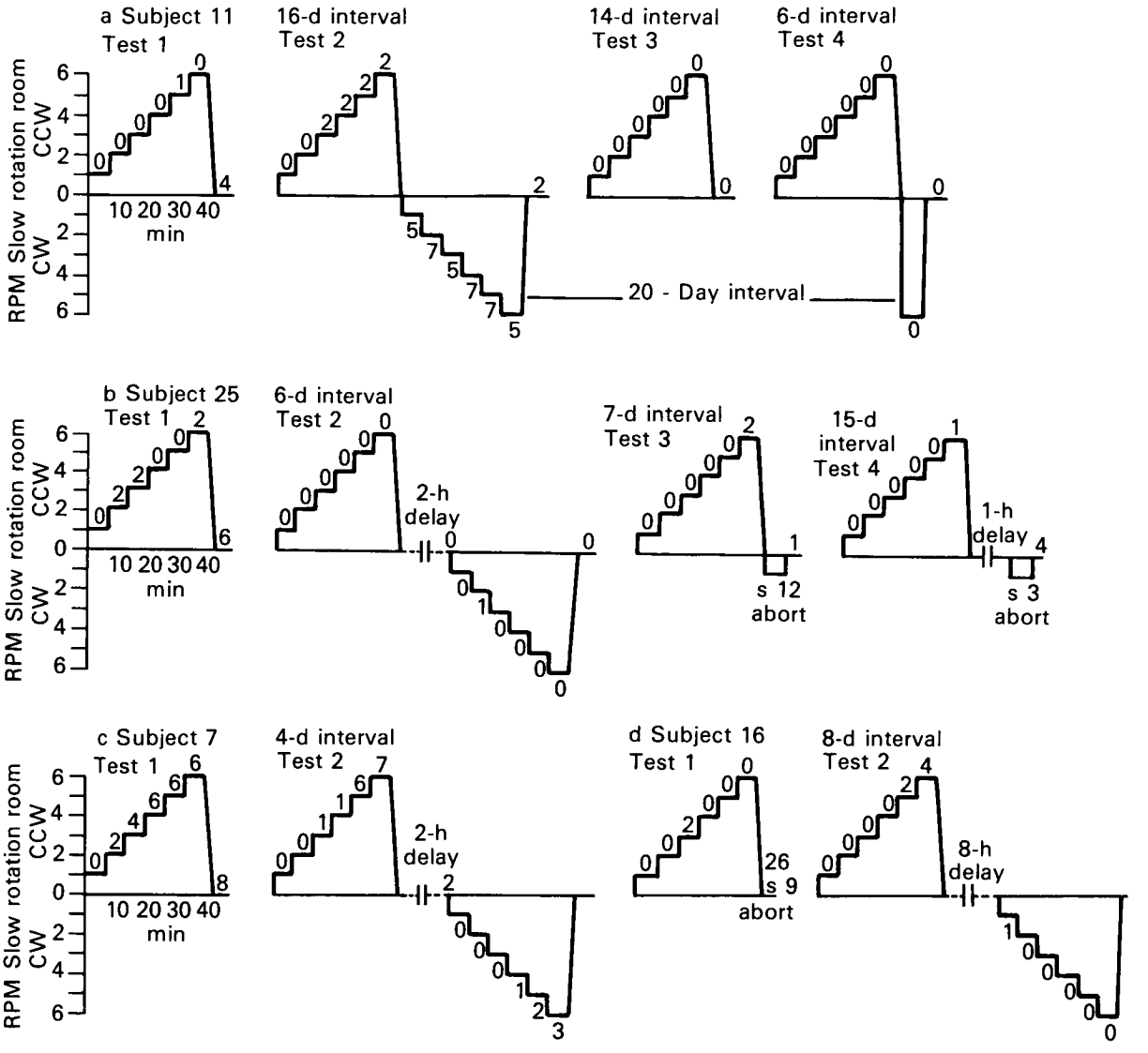



FIGURE 40.—Stress profiles and motion-sickness scores in four subjects, suggesting simultaneous acquisition of direction-specific and nondirection-specific effects. (a) Freedom from symptoms during the execution of 120 head movements after a one-step change from 6 rpm CCW to 6 rpm CW rotation, more likely to be the result of adaptation to CCW rotation (4 exposures) than one exposure to CW rotation, Test 2. (b) Freedom from symptoms during CW rotation and return to zero velocity (Test 2) only explicable by the 2-h delay (see Tests 3 and 4) and the previous adaptation to CCW rotation. (c) The mild symptoms during the reverse IAS and especially the score of 1 point on return to zero velocity, likely explained by adaptation to CCW rotation in Tests 1 and 2. (d) Explanation similar to that in (c). (From Ref. [39])

reverse IAS if it had been done at that time, which was definitely confirmed in Tests 3 and 4. The only reasonable explanation for the virtual absence of symptoms during the reverse IAS in Test 2 and during the challenge after return to zero velocity is that the 2-hour delay was sufficient for the direction-specific effects acquired during the initial IAS to disappear, revealing the non-direction-specific adaptation acquired during that initial IAS and, probably, during the IAS in Test 1.

Subject 7 (Fig. 40, Part c) demonstrated sus-

ceptibility to motion sickness during the initial IAS in Tests 1 and 2 but demonstrated only very mild symptoms during his first exposure, after a delay of 2 hours, to a reverse IAS (Test 2). The score of 1 point on final return to zero velocity suggests that less central vestibular repatterning occurred than in Test 1 (8 points). Only the delay of 2 hours prevented an abort in subject 7, and only prior acquisition of nondirection-specific adaptation could account for the less severe symptoms during the first reverse IAS (and return to zero of Test 2) compared with the initial IAS of Tests 1 and 2.

Figure 40, Part d, shows that for subject 16 in Test 1, testing was aborted during the challenge after return to zero velocity, implying that testing would have been aborted during a reverse IAS if this had been measured at that time. Symptoms during the initial IAS in Test 2 were more prominent than in Test 1, implying that central vestibular repatterning was taking place and that, in the light of Test 1 results, testing would have been aborted during a no-delay reverse IAS had this been done. The 8-hour delay allowed nearly all of the direction-specific effects to disappear, thereby revealing prior acquisition of nondirection-specific adaptation that accounted for the virtual absence of symptoms during the reverse IAS and, more importantly, for the 1-point score on final return to zero velocity.

The findings just described imply that with the disappearance of direction-specific adaptation, with a short time constant, nondirection-specific adaptation with a long time constant is revealed. The practical significance of these findings is twofold. On making transitions from a rotating to a nonrotating portion of a space station, it is necessary to ensure that short-term direction-specific adaptation effects are not present and that long-term adaptation to weightlessness has not been lost. On the transition from the weightlessness to the rotating part, it is only necessary to ensure that adaptation to rotation has not been lost.

It is interesting to speculate on the problem of ataxia and past pointing in making transitions between rotating and nonrotating parts of a space station. Based on the incidental findings [47] that adaptation to rotation with respect to walking in the SRR was preserved during exposure to walking under simulated fractional G-loads and during a subsequent period upwards of 24 hours, it would seem that exposure to weightlessness would not result in loss of adaptation to walking in the rotating environment. With respect to past pointing, visual cues would serve to minimize any tendency to past point in making transitions between the weightless and rotating environments.

### Antimotion Sickness Drugs

Numerous reports have been published recommending many drugs, chemicals, and other agents in the prevention of motion sickness, but competent reviewers, pointing to faulty experimental design, have made sweeping criticisms of the validity of many findings. The major criticisms center around: (1) lack of understanding of the complex etiological factors; (2) lack of control over stimulus conditions in the field; and (3) inadequate design, making statistical treatment of the findings of doubtful value. Inasmuch as antimotion sickness drugs are effective in any "motion environment," bioassay under laboratory conditions is recommended. Important aspects in experimental design involve: (1) selection of typically normal subjects; (2) control and measurement of the stressful accelerations; (3) standardization of the laboratory environment; (4) measurement of responses elicited; (5) use of an end point short of vomiting; and (6) use of a double-blind technique in the administration of the pharmacologic agents.

Prior to the introduction of the antihistamines, hyoscine (scopolamine) was generally regarded as the most effective antimotion sickness remedy, although the side effects from moderate doses were annoying and from large doses, unacceptable. The antihistamines ushered in a new and better era in the use of antimotion sickness drugs, although most of the preparations reaching the public have not been thoroughly evaluated for effectiveness.

Recent studies have confirmed the effectiveness of hyoscine [11] and the effectiveness of some drug combinations [4, 15, 81, 135, 136]. Figure 41 summarizes bioassays conducted in a

slow rotation room using mild motion sickness (malaise III) as an end point [136]. A constant (predetermined) level of stress was used, and differences in susceptibility were measured in terms of the number of head movements required to reach the end point. A 10-unit Latin-square design was used (seven drugs and three placebos regarded as drugs). A "placebo" baseline was drawn and findings on the 10 subjects were treated as a group. When these drugs were ranked in order of their effectiveness in reducing susceptibility to acute motion sickness, it was found that they also were arranged in terms of their pharmacologic actions. Drugs with central sympathomimetic or parasympatholytic action were effective not only individually, but also their effects tended to sum. It is not shown in Figure 41 that the relatively high effectiveness of ephedrine 50 mg in combination with promethazine 25 mg have minimal side effects [135].

The procedure just described does not permit valid analysis of the bioassay findings in terms of the individual and has two other undesirable features. The velocity (rpm) of the SRR at which a subject experienced the motion sickness end point after executing about 50 head movements was used as his "baseline." When some subjects were tested after administration of an effective drug, it was impossible to achieve the end point even after 300 head movements. Another handicap was the difficulty in ranking subjects with regard to susceptibility, using both rpm and the number of head movements as a combined score. An experiment just completed on another project (comparing susceptibility with eyes open and eyes closed) used an incremental provocative test schedule (Fig. 25) that yielded a single score (rpm). This permitted measuring and ranking all intraindividual and interindividual differences in susceptibility. An effort is being made at present to reevaluate all drugs and drug combinations that will yield reliable information on individuals as well as groups.

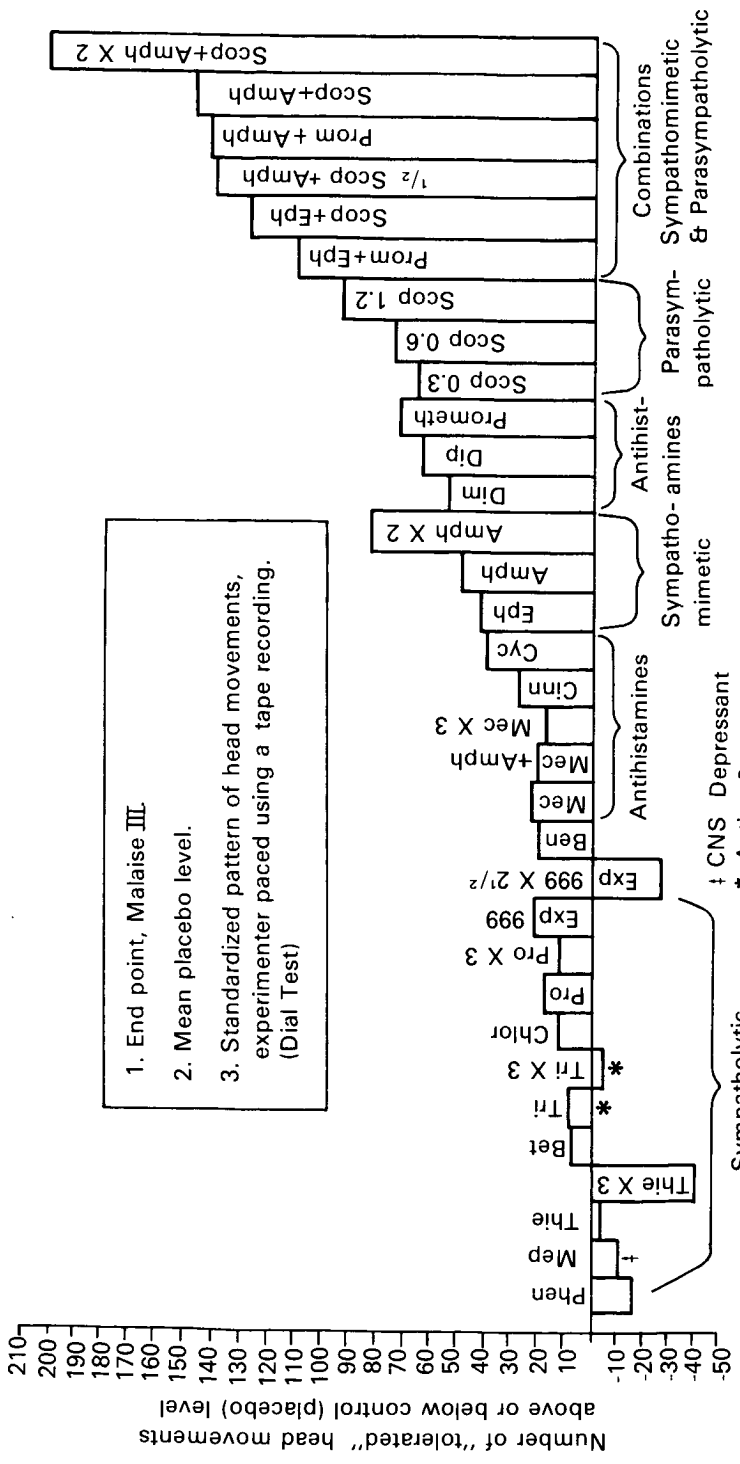
## SPACE MISSIONS

The basic difficulty in coping with vestibular problems in space exploration is that our knowledge of vestibular side effects, especially motion sickness, is mainly empirical. Consequently, it is not possible to predict accurately susceptibility to motion under the novel conditions that may be encountered in space. Aside from the use of drugs, we must rely on such countermeasures as the selection process and preflight adaptation in preventing vestibular disturbances.

There are some encouraging aspects. First, the motion environments encountered under nominal conditions are not highly stressful, and vestibular side effects, if experienced, are limited through the acquisition of adaptation. The exception to this generalization regarding adaptation to weightlessness or to a rotating environment arises if the crewman makes rapid transitions between rotating and nonrotating parts of a space station. A second favorable aspect is that the symptomatology of motion sickness is similar even when the eliciting causes are different. In other words, ground-based studies dealing with symptomatology per se have validity in space, which, it appears, also applies to drug therapy. A third favorable aspect is the feasibility (small number involved) to evaluate and test each crewman on an individual basis.

An extensive testing program is needed in view of great individual differences that pervade nearly every mechanism underlying vestibular side effects. Thus, there are differences between susceptibility to reflex phenomena and motion sickness, differences in the rate of acquisition and decay of side effects in different motion environments, and differences in the effectiveness of antimotion-sickness drugs.

A full understanding of the relation between reflex vestibular phenomena and delayed epiphenomena (motion sickness) is needed. Although susceptibility to reflex vestibular disturbances (loss of stability) has some value in predicting susceptibility to motion sickness, there are many exceptions to this generality. Thus, improving the stability of the reflex vestibular system (through training and conditioning) [37, 39, 66, 116] may be quite independent of the ease with which vestibular influences, as soon as they have escaped from their normal confines, may travel along preferential (though common) pathways to sites where first-order symptoms originate. Indeed, it is likely that some nonvestibular etiological factors also exert their influence in this way.




| | | | |
|---|---|---|---|
| Phenoxybenzamine 20 mg | Benactyzine 3 mg | Dimenhydrinate 50 mg | Scopolamine 0.3 mg + |
| Meprobamate 400 mg | Meclizine 50 mg | Diphenidol 50 mg | Amphetamine 5 mg |
| Thiethylperazine 10 mg | Meclizine 50 mg + | Promethazine 25 mg | Promethazine 25 mg + |
| Thiethylperazine 30 mg | Amphetamine 10 mg | Scopolamine 0.3 mg | Amphetamine 10 mg |
| Betahistidine 4 mg | | Scopolamine 0.6 mg | Scopolamine 0.6 mg + |
| Trimethobenzamide 250 mg | Meclizine 150 mg | Scopolamine 1.2 mg | Amphetamine 10 mg |
| Trimethobenzamide 750 mg | Cinnarazine 50 mg | Promethazine 25 mg + | Scopolomine 1.2 mg + |
| Chlorpromazine 25 mg | Cyclizine 50 mg | Ephedrine 50 mg | Amphetamine 20 mg |
| Prochlorperizine 5 mg | Ephedrine 50 mg | Scopolamine 0.6 mg + | |
| Prochlorperizine 15 mg | Amphetamine 10 mg | Ephedrine 50 mg | |
| EXP 999 10 mg | Amphetamine 20 mg | | |
| EXP 999 25 mg | | | |

FIGURE 41.—Effectiveness of antimotion-sickness drugs in preventing SRR sickness in 70 subjects exposed on 500 occasions in a rotating environment, using the Dial Test. See item 3 (box) in the chart for explanation of Dial Test. (From Ref. [136])

Although the immediate concern at present is the prevention of, or coping with, vestibular side effects in weightlessness, attention is being given to validating aloft the ground-based measurements made in the laboratory and in parabolic flight. Attention is also being given to experimental studies, looking ahead to the possibility that, tomorrow, artificial gravity will be generated by rotation of part of a space station. Thus, tackling the problems aloft will be carried out on an incremental basis.

The crewman necessarily plays a key role in this integrated effort: as subject, he can serve as his own control in validating studies; as onboard experimenter, he is essential in conducting experiments and making observations aloft; as astronaut or cosmonaut, he has responsibilities in connection with the prevention of vestibular side effects during the mission. Prevention involves taking charge, rather than responding to, events which requires close cooperation between the crew aloft and the biomedical representatives in the ground-based control center. Preflight preparations include such major elements as selection (or secondary selection), instruction, and, if needed, preflight adaptation. The inflight period involves making observations and measurements aloft and ground-based monitoring during the mission. Postflight, the debriefing and initial assessment should be followed by measurements at repeated intervals until stable values are obtained.

## Weightlessness

The literature dealing with orbital flights is discussed elsewhere in this volume; only a few comments on prevention of vestibular side effects will be mentioned here.

On transition into weightlessness, the crewman's head movements (rotations) generate normal angular accelerative stimuli, but the resulting sensory input encounters unusual central vestibular patterning due to absence of the constant stimulus of gravity. The susceptible astronaut is immediately confronted with the maximum stressful effect possible; the analogous situation in a rotating environment would be immediate transition from zero velocity to terminal velocity. To achieve adaptation, however, head movements must be made. A program based on present knowledge should be helpful. As soon as symptoms are experienced, especially the nausea syndrome, time must elapse before dynamic changes in the vestibular system disappear and restoration through homeostatic mechanisms has taken place in nonvestibular systems. If feasible, it is highly desirable for the crewman to restrict head movements for a period well beyond the time during which symptoms have disappeared. This will allow time for antimotion-sickness drugs taken by mouth to be absorbed (90 min), after which head movements may be cautiously resumed. If it is not feasible to restrict head movements, drugs may be injected—a collar device fixing the head relative to the thorax is beneficial, providing circumstances permit.

## Rotating Environment

Although the rotating environment represents a novel experience, the principal unknown element concerns the effect of the fractional G-load. This information will be available, however, long before rotating space stations are a reality. With rare exceptions, the initial transition will be made from the weightless part to the rotating part, spinning at terminal velocity. Thus, the crew making normal head movements out of the plane of passive rotation would be exposed to maximal accelerative stimuli. In the event that symptoms are elicited, head movements (out of the plane of rotation) should be restricted if possible; otherwise, countermeasures described above should be taken. If there is opportunity to restrict head movements until 1 or 2 h have elapsed after symptoms have disappeared, an incremental adaptation schedule is feasible under antimotion-sickness drug therapy. This would be accomplished in a Bárány-type chair rotating opposite the room's rotation, permitting manipulation of the angular velocity between zero velocity and the terminal velocity of the space station. After a demonstration that symptoms were not elicited at (resultant) zero velocity while executing standardized head movements, a cautious stepwise increase (0.5 rpm) in angular velocity should be scheduled.

Sudden transitions between rotating and nonrotating parts of a space station involve novel stimulus conditions. Experience in the rotating room has demonstrated the need to distinguish between direction-specific and nondirection-specific adaptation effects. If this is kept in mind, the prevention of vestibular side effects on making rapid transitions between rotating and nonrotating portions of a spacecraft should not pose a problem [39, 120].

Postural disequilibrium, on making the transitions, is a separate problem because receptor systems serving touch, pressure, and kinesthesis are involved mainly, and because direction-specific effects, it would seem [47], do not disappear spontaneously but require active whole-body motions.

The suggested preventive measures indicated today will almost surely be different tomorrow. The need for full understanding of vestibular side effects, including self-diagnosis, prevention, and treatment may not change.

## VESTIBULAR INVESTIGATIONS IN SPACE MISSIONS

### Operational Aspects

The character of prolonged space missions will inevitably change with advances in spacecraft technology, and the emphasis will gradually change from exploration to exploitation of extraterrestrial opportunities. Travelers at present must be able to withstand the severe stresses incidental to launch and reentry which demands a state of fitness that seems to dwarf the subtle effects of weightlessness. Even among travelers in superb health, maintenance of fitness during prolonged exposure in a weightless spacecraft will make great, continuous demands on the spaceman's time; illness aloft, precluding exercise, could pose a hazard. With tomorrow's advent of a space shuttle or its equivalent, stresses incidental to launch and reentry will be greatly reduced; the limiting factor with regard to fitness for travel will be conditions aloft, and the most important of these factors is weightlessness. The generation of artificial gravity by causing the spacecraft to rotate represents a "technological fix," and this alleviates an otherwise potentially continuous hazard, weightlessness.

The major need (it was indicated earlier) is to be able to predict, on the basis of ground-based tests, the responses (mainly or partly of vestibular origin) of space flyers in the novel environments of the weightless space vehicle and the rotating space station. The limitations in simulating such novel stimulus conditions force an extension of ground-based studies to include validating observations and experiments conducted aloft. Identifying motion sickness as the chief vestibular problem still leaves the problem to be solved, and this must be accomplished in ground-based laboratories.

### Scientific Aspects

It is doubtful if the precise role of gravity under terrestrial conditions (distinguishing between gravitoinertial forces and agravitoinertial mechanical forces) can be made without the opportunity to conduct studies in space. Transition into weightlessness abolishes the stimulus to the otolith organs due to gravity in an elegant and harmless manner, and also abolishes the stimulus to receptors serving touch, pressure, and kinesthesis due to weight. Psychophysical experiments using mechanical pressure can be conducted in weightlessness that should add to our knowledge of these perceptual systems. For example, experiments conducted in Gemini 5 and 7 [48] dealing with egocentric visual localization suggested that elicitation of the A and E phenomena depends not only on otolithic inputs (already known) but also on nonotolithic mechanoreceptor systems. The vestibular experiments to be conducted in Skylab 2 and 3 flights will not only extend the measurements made in Gemini 5 and 7 but will also include measuring changes in canalicular susceptibility, susceptibility to motion sickness, and two-dimensional perception of nonvisual extrapersonal space [99]. Devices designed for these purposes (see Figs. 21, 26, and 42) have many uses in ground-based laboratories.

It is not an overstatement to say that the opportunities to study the vestibular system aloft constitute a major historical landmark in the advancement of knowledge not only in the vestibular but also in related areas. Resting dis-

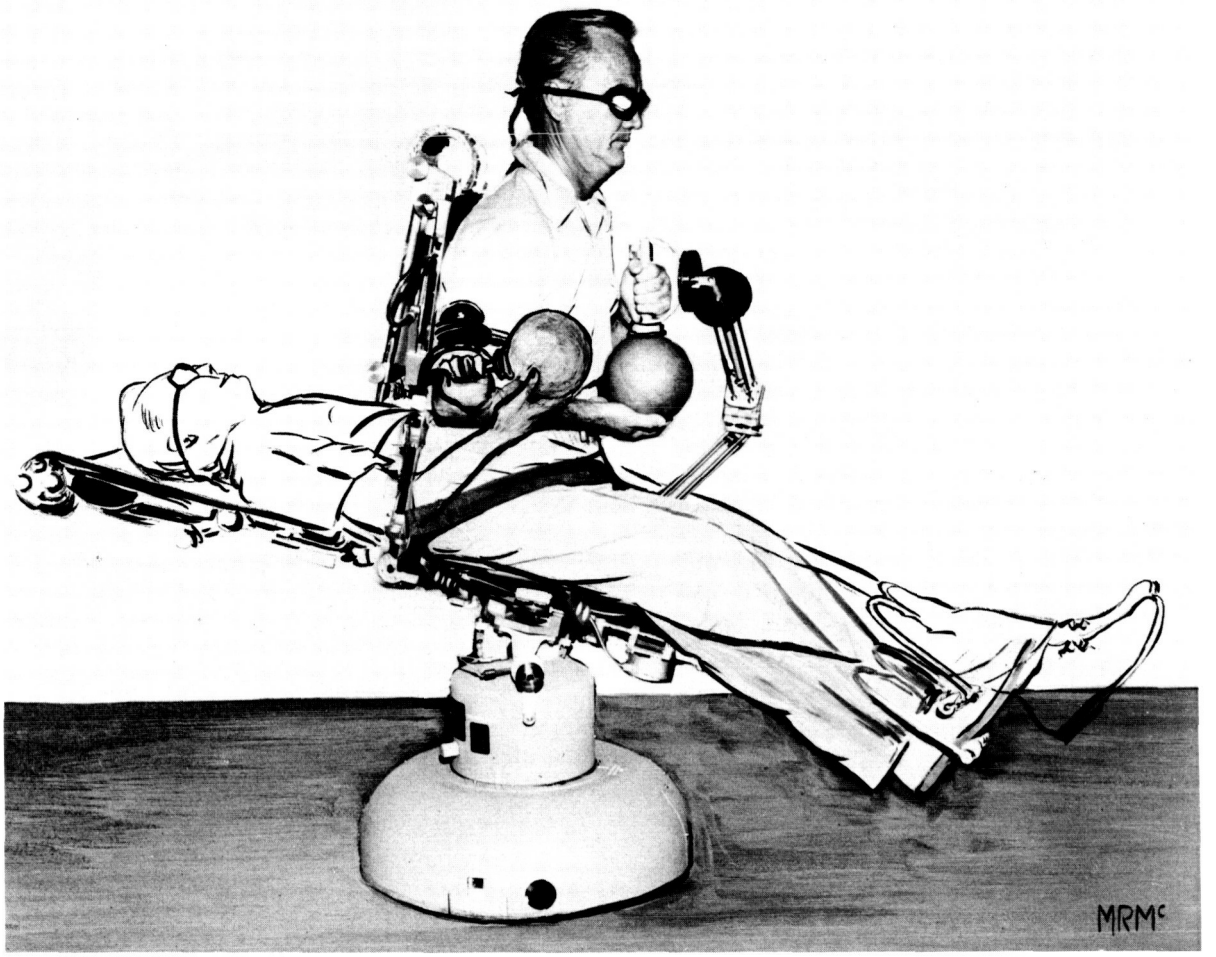



FIGURE 42.—Rod and sphere device for measuring nonvisually perceived personal and extrapersonal space. The device has a pointer held by magnetic attraction to a hollow steel ball. With eyes covered, the subject slides the rod over the ball until spatial localization is achieved; note absence of constraints that are usually provided to maintain the indicator in one plane. The readout is semiautomatic.

charges [54] and the contribution of tonic otolithic sensory inputs modulating stretch reflexes and muscle tone in man [16, 73, 75, 84, 104, 115] can be investigated. Distinctions between otolithic and canalicular influences that are so closely intertwined might be unraveled.

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