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# THE ROLE OF THE VESTIBULAR ORGANS IN THE EXPLORATION OF SPACE

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Pensacola, Fla.

JAN. 20-22, 1965



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Symposium On  
**THE ROLE OF THE  
VESTIBULAR ORGANS IN THE  
EXPLORATION OF SPACE**

Held under the auspices of the Office of Advanced Research and Technology  
National Aeronautics and Space Administration

*U.S. Naval School of Aviation Medicine*

*Pensacola, Fla.*

*Jan. 20-22, 1965*

*General Chairman: ASHTON GRAYBIEL*  
U.S. NAVAL SCHOOL OF AVIATION MEDICINE



*Scientific and Technical Information Division*  
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## Foreword

By sponsoring research, the aerospace agencies have given a great impetus to investigations dealing with the vestibular organs, the semicircular canals, and otolith apparatus. The reasons for their interest are no mystery. Man, in breaking away from the Earth, is transported in vehicles which move in three-dimensional space and generate inertial forces that create environmental factors to which he is not accustomed either by inheritance or experience. The vestibular organs not only are uniquely structured to respond to angular and linear accelerative forces but also they contain the only sensory cells whose sole, and indeed obligatory, purpose is to act as transducers signaling forces acting at a distance: the cupula-endolymph system of the canals responding to angular accelerations and the cilio-macular system of otolith organs responding to linear accelerations. The vestibular organs may be damaged by overload, respond in curious fashion to stimulation outside their physiological response range, or, within this range, signal appropriately for the force environment but inappropriately in terms of the object environment and visual environment. This may result in spatial disorientation, postural and visual "illusions," and a wide variety of symptoms best known under the term motion-sickness. Persons who have lost the function of the vestibular organs do not become motion-sick, illustrating the significance of these organs in causing disturbing effects when man is exposed to unusual force environments.

There are particularly good reasons why NASA should be interested in the labyrinthine organs inasmuch as the otolith apparatus is deafferented (suppressed) in weightlessness. Exposure in a rotating environment results in unusual patterns of canalicular stimulation when a person rotates his head out of the axis of rotation of the vehicle. It is quite appropriate, therefore, that NASA from time to time sponsor a meeting that serves a dual purpose in bringing together the investigators under the eyes of the administrators.

The striking feature of this symposium was not the presentation of facts, important though they were, but the enthusiasm of the participants. This spirit, unfortunately, cannot be captured from a reading of the proceedings. Nor can the proceedings reflect adequately either the great scope of investigations now underway or the numerous problems still awaiting investigation. Rather, the proceedings might be regarded as a series of vignettes, usually reportorial but sometimes polemical in nature, contributing only a limited number of restricted views of the entire picture.

Inasmuch as all presentations were by invitation, the manuscripts remain as submitted except for minor changes that leave unaltered the meaning of the author. Any editorial temptation to pluck motes from the eye of a

colleague has been resisted. This also applies to the discussions which, with one or two exceptions, were edited by the participants.

The initial stimulus for this meeting was provided by Eugene Konecci at the time of the dedication of the vestibular laboratory at the U.S. Naval School of Aviation Medicine. He suggested the date, place, and the emphasis on participation by those sponsored by aerospace agencies or industry. After Dr. Konecci moved to the Space Council, NASA implementation fell into the willing hands of Walton Jones who joins me in acknowledging the help of many persons of good will and good capabilities.

ASHTON GRAYBIEL

*U.S. Naval School of Aviation Medicine*

# Welcome

JAMES L. HOLLAND

*U. S. Naval Aviation Medical Center*

It is appropriate, I believe, to use this forum for making two additional comments: first, in recognition of the splendid support we have received from NASA, and second, to acknowledge the farflung congenial relationships we have with other investigators and institutions they represent.

Although we have been interested in vestibular problems as far back as I can remember, it was a relatively small effort carried on as best we could under our budgetary restrictions. The support we have received from NASA has made a great impact and accounts for the increased activity and scientific output from the vestibular laboratory. I should like to take this opportunity

to acknowledge this aid and to thank the NASA representatives here in attendance.

If a star were placed beside the names of those here who have collaborated with us in furthering our vestibular program, it would be impressive although incomplete. Some of the pioneers are here: Dr. Clark, Dr. Mann, and Dr. Johnson. I will not attempt to list the present collaborators by name, but they constitute a wide representation from the Air Force, Army, Canadian Defence Medical Laboratories, the British RAF and Navy, a number of European and Stateside universities, and industry. Speaking selfishly, we gained a great deal from this cross-fertilization, and, speaking unselfishly, we have shown a willingness to share our ideas, dollars, and facilities.

# Opening Remarks

WALTON L. JONES

*Headquarters, National Aeronautics and Space Administration*

Dr. Graybiel and the naval personnel at Pensacola have been studying vestibular physiology and disorientation problems for over 20 years. Much of this work was directed toward the aviator's problems in military aircraft. With this background, the availability of a highly skilled staff, and unique facilities with on-going related work, it was logical for NASA to turn to Dr. Graybiel and ask for his assistance in the study of vestibular problems posed by manned space flight.

This meeting represents the fulfillment of a need recognized by many to bring together investigators, especially those supported by NASA and DOD, working on the solution of vestibular problems posed by manned space flights. These problems appear to fall into two categories; namely, the prevention of vestibular disturbances in weightlessness, and the possible need to generate artificial gravity.

The disturbing symptoms experienced in weightlessness require much detailed study. We are fortunate that the present age has made available many new disciplines and new techniques which may be brought to bear on this problem. Most experts, I believe, are convinced that we will solve these problems; but, we will not be absolutely sure until we have conducted some experiments in orbit under the weightless condition for considerable time.

The second problem arises out of the possibility that artificial gravity will be needed. This is not the place to debate the pros and cons of this possibility but for you to tell us what provisions should be made if the need eventuates. In orbiting spacecraft, a constant level of artificial gravity can be provided only by the generation of centripetal force either by rotation of the entire spacecraft or by what amounts to a human centrifuge. It would seem as if we must make provision for either or both if we keep in mind the requirement for fitness aloft and the greater requirement for fitness on a reentry. The two mutually dependent problems requiring solution are how much artificial gravity is needed and how does one prevent any undesirable side effects.

From the standpoint of those charged with the responsibility of insuring that the problems are solved, the question arises as to how best this can be accomplished, keeping in mind time-stress and, contrary to what people think, dollar-stress. I am hopeful that additional guidelines will appear out of the deliberations of this symposium. Indeed, it will be a disappointment if this is not the case. Although we have a fine appreciation of the desire and need for basic background information, we request that the same appreciation is generated for our need to furnish specific engineering data at specific points in time.

**SESSION I**

**Chairman: WALTON L. JONES**

**Headquarters, National Aeronautics and Space Administration**

**Cochairman: ROBERT MAYNE**

**Goodyear Aerospace Corporation**



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# Ultrastructural Studies of the Labyrinth in Squirrel Monkeys

HEINRICH H. SPOENDLIN

*University of Zurich, Switzerland*

All sensory epithelia of the vestibular labyrinth show basically the same structure. They consist of the sensory cells imbedded in supporting cells, the nerve fibers and nerve endings, and finally of a specific suprastructure such as the cupula and the otolithic membrane which can be considered as a mechanical device to transform a particular form of energy into the stimulating mechanism of the sensory elements. This stimulating mechanism most probably is a sheering motion between suprastructure and sensory epithelia producing a deviation of the sensory hairs (Békésy 1952, Loewenstein 1955).

The ultrastructure of the vestibular sensory epithelia has been studied by many authors (Wersäll 1956, Engström and Wersäll 1958, Bairati 1960, Friedmann 1963, Engström, Ades, and Hawkins 1962, Flock and Wersäll 1962, Spoendlin 1964, Flock 1965). Two types of hair cells can clearly be distinguished: The type I hair cell has the shape of a bottle and with the exception of the apical parts is surrounded by a nerve chalice (fig. 1). The type II hair cell shows, however, an irregular cylindrical shape and is in contact with several small nerve endings (fig. 2). Both cell types are found over the entire surface of the maculae and cristae. The type I hair cells are, however, particularly concentrated in the central part of the maculae and on the vertex of the cristae. The zone of concentrated type I hair cells in the maculae seems to correspond with the "striola" which was described earlier by Werner as a band going

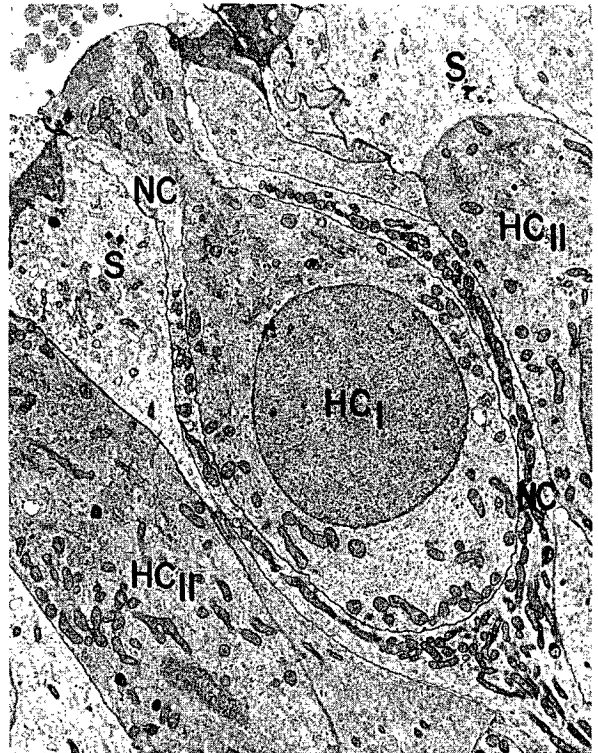


Figure 1.—Hair cell type I (HC<sub>I</sub>) surrounded completely by a nerve chalice (NC). On either side is a hair cell type II (HC<sub>II</sub>). On the right-hand side the nerve chalice is in direct contact with the hair cell type I and the hair cell type II which is not frequently observed. Supporting cells (S) fill out the spaces between the sensory cells.

slightly curved through the middle of the maculae, where the sensory cells are especially large, and all supporting cells close to the bottom of the epithelium.

In this report I would like to concentrate on a few questions of the ultrastructural or-

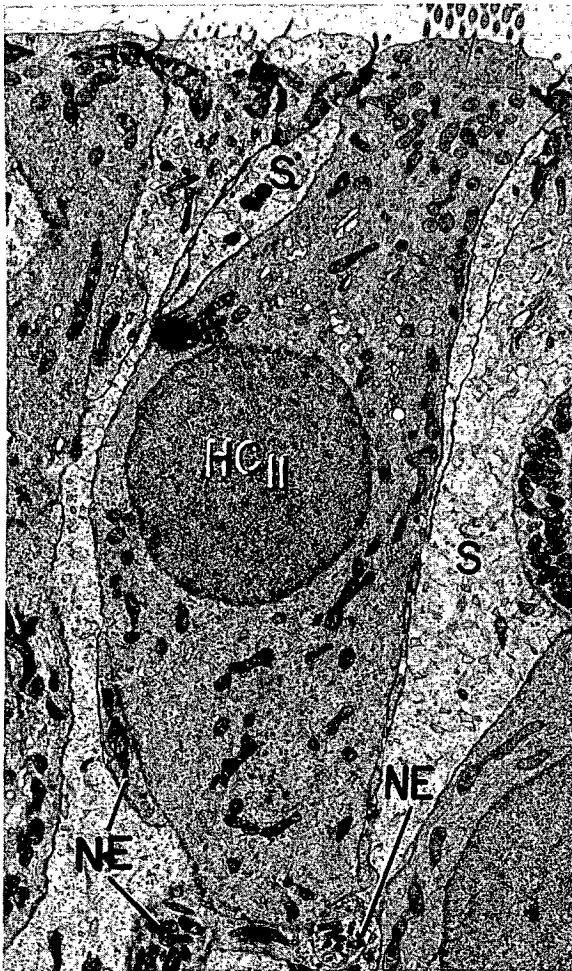


Figure 2.—Irregular cylindrical hair cell type II ( $HC_{II}$ ) in contact with several individual nerve endings (NE). Supporting cells (S). The very dense cytoplasm of the hair cell is obvious.

ganization of the vestibular sensory epithelia which might be brought into a certain relation to the functional behaviour of those sensory receptors. In the last century Ewald (1892) established, on the basis of careful physiological experiments in pigeons, certain rules according to which the semicircular canals are functioning. Since then those rules have been known as the second law of Ewald which states that, in the lateral semicircular canal, an ampullopetal stimulation has a much stronger effect than an ampulofugal cupula deviation (fig. 3). In the vertical canals this condition is reversed.

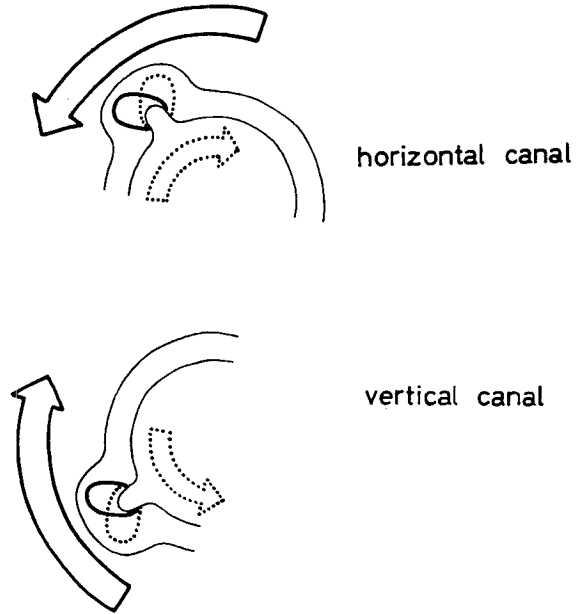


Figure 3.—Schematic representation of Ewald's finding in pigeons: Utriculopetal deviations of the cupula in the horizontal canal have a greater effect than equal utriculofugal deviations. Reversed pattern in the vertical canals where utriculofugal cupula deviations have a greater effect.

Two basic features of the cristae are expressed in this law:

1. The receptors of the semicircular canals are direction specific.
2. There is a quantitative difference in the response to equal ampullopetal and ampulofugal stimulation.

Such a directivity and an asymmetric mode of action of the vestibular sensory epithelia should be expected to express itself somehow in their structural organization.

The direction specificity as a basic functional feature is probably based on a structural polarization of those sensory epithelia, which is clearly visible at the level of the sensory hairs. At the apex of each sensory cell there is a great number of sensory hairs with increasing length from one side of the cell surface to the other, comparable to organ pipes measuring from 1 micron to approximately 12 microns in the maculae and much more in the cristae (fig. 4). Wersäll (1956) was the first to show that the sensory cells

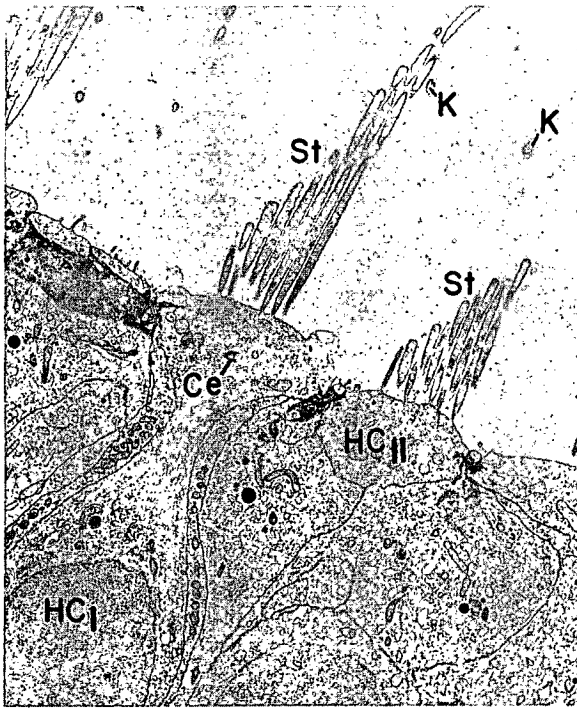


Figure 4.—Sensory hairs of a hair cell type I ( $HC_I$ ) and a hair cell type II ( $HC_{II}$ ). The stereocilia (St) are arranged like organ pipes on the surface of the sensory cell. Kinocilium in cross section (K). Additional centriole in hair cell type I (Ce).

of the crista contain two types of sensory hairs, the stereocilia and kinocilium-like processes very similar to motile cilia of the respiratory tract.

In horizontal sections through the sensory hairs we see that each vestibular sensory cell of a macula or crista indeed carries from 60 to 100 stereocilia and one kinocilium in a strict geometrical arrangement (fig. 5).

The stereocilia are morphologically defined as homogeneous, clublike, and as rather stiff rods emerging from the cuticular plate where they are anchored with small roots which penetrate usually through the entire thickness of the cuticula (fig. 6).

The kinocilium is the longest of the sensory hairs. It seems to be much more flexible than the stereocilia and it is always next to the longest of stereocilia. It originates from a specific basal body in a cuticula-free area of the cell surface (fig. 7). In transverse sections the typical pattern with nine pe-

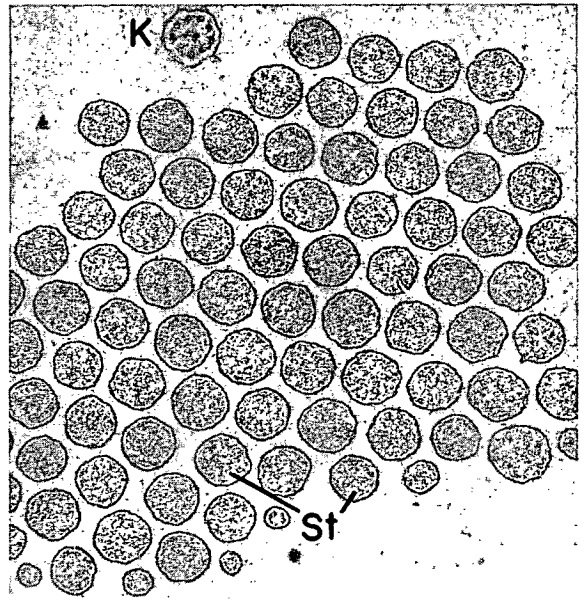


Figure 5.—Horizontal section through the sensory hair bundle of one sensory cell. The stereocilia (St) are geometrically arranged in form of a hexagonal packing. On one side of the stereocilia bundle is the kinocilium (K) with its typical nine peripheral and two central filaments.

ripherally arranged double tubular filaments and two centrally located single tubular filaments is evident (fig. 5). This typical nine plus two pattern is found in all kinocilia wherever they exist in all kinds of tissues and animals, such as, for instance, in the respiratory epithelia, in the oviduct, or in unicellular flagellates. The filaments of the kinocilium extend into the basal body where they assume a triple tubular shape and appear in a spiral arrangement (fig. 7). Such basal bodies reveal an almost identical structure with the centrioles. The great importance of the centrioles in the organization of every cell and the concept that the basal bodies of kinocilia and centrioles are homologous structures certainly justify the assumption that the kinociliar basal body in the vestibular sensory cell represents important centers, which might be related to a functional polarization of the cell.

The kinocilium is, as mentioned above, a very elementary structure found in almost identical forms in many different tissues of

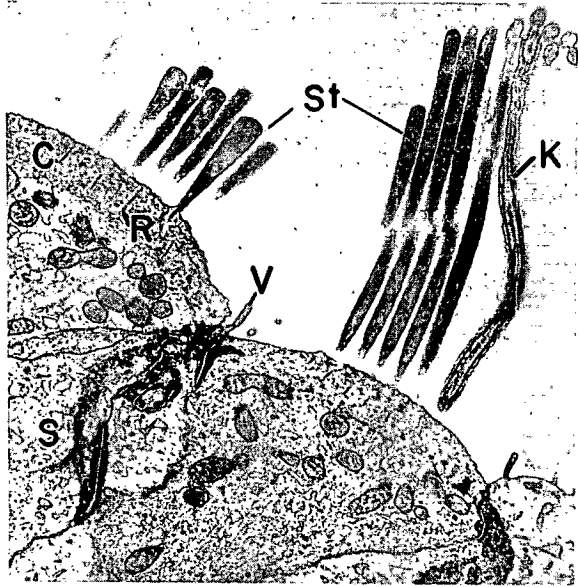


Figure 6.—Sensory hairs of two sensory cells. Stereocilia (St) appear as stiff club-like, rather homogeneous rods which are anchored in the cuticular plate (C) with small rootlets (R) which usually penetrate through entire thickness of cuticular plate. Kinocilium (K) seems to be a much more flexible structure. Between the sensory cells are supporting cells (S) which have microvilli (V) at surface of sensory epithelia.

all animals. Its primary role seems to consist of motility as is expressed in its name. In certain situations, however, its motility is very unlikely and it may have quite a different functional significance. Thus kinocilia are found not only in many types of parenchymatous tissues (Barnes 1961, Dahl 1963) but also in many types of sensory cells such as the rod cells of the retina, the olfactory sensory epithelium, or sensory organelles in primitive unicellular animals (Wolken 1956). This certainly suggests that the kinocilia might play an active role in sensory systems.

The assumption that the kinocilium with its basal bodies indicates a functional polarization of the sensory cells gets further support from the spatial arrangement of the kinocilia in relation to the stereocilia over the entire sensory epithelium. In horizontal sections through sensory epithelia just above the surface of the cell all the bundles of sen-

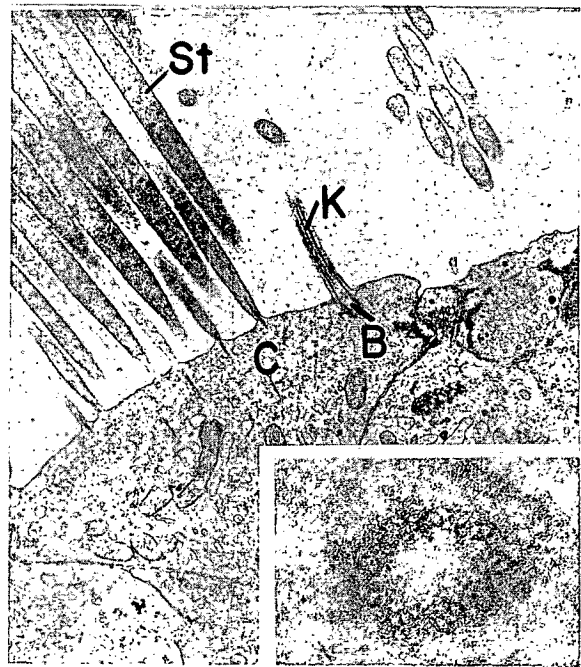


Figure 7.—Sensory hairs of a macular sensory cell with stereocilia (St) and one kinocilium (K) which originates from a basal body (B) from a cuticula-free area of the cell surface. Cuticula with roots of stereocilia (C). Inset at lower right shows a horizontal section through a basal body at high magnification where spiral arrangement of triple tubular shaped filaments is clearly visible.

sory hairs are transversely cut, and their spatial arrangement can be studied (fig. 8). In most of the sensory cells in one area the kinocilia are always found on one and the same side of the stereociliar bundles. They originate from the same pole of the cellular surface. Such a uniform polarization was shown by Loewenstein and Wersäll (1959) in the cristae of fish and a bidirectional polarization in the lateral line organ of fish by Flock and Wersäll (1962) and Flock (1965).

We investigated the polarization of the vestibular sensory hairs of the entire surface of the cristae and maculae in mammals such as guinea pigs, cats, and monkeys with phase contrast microscopy which allows us to study large surfaces in one section. Since the morphology of the sensory hairs is well



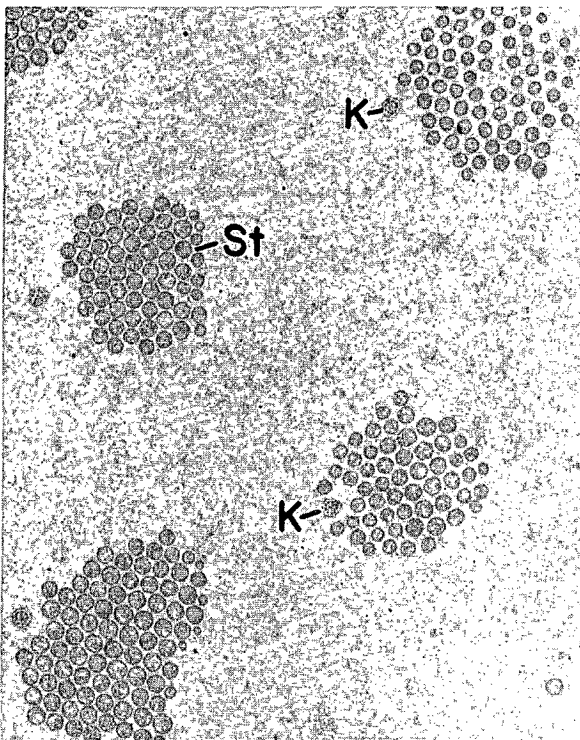


Figure 8.—Horizontal section through a number of sensory hair bundles of a crista of a monkey. Kinocilia (K) are always found on same side of stereociliar bundles (St) indicating a morphological polarization of sensory cells.

known from electronmicroscopic investigations, the kinocilia can be recognized as dark spots and differentiated from the stereocilia in about 1-micron-thick sections in phase contrast microscopy (fig. 9). This, however, is only possible in horizontal sections at the level of the cell surface or slightly above it where either the basal body of the kinocilium is visible or where the kinocilium as such is conspicuous by being much thicker at this level than the neighbouring stereocilia. More distally, the cilia are no longer discernible.

In the cristae the kinocilia of all sensory cells are found on one and the same side of the hair bundles or the cell surface. The sensory hairs are therefore uniformly polarized. In the cristae of the horizontal canal the kinocilia always are at the utricular pole of the surface of the sensory cell. In the

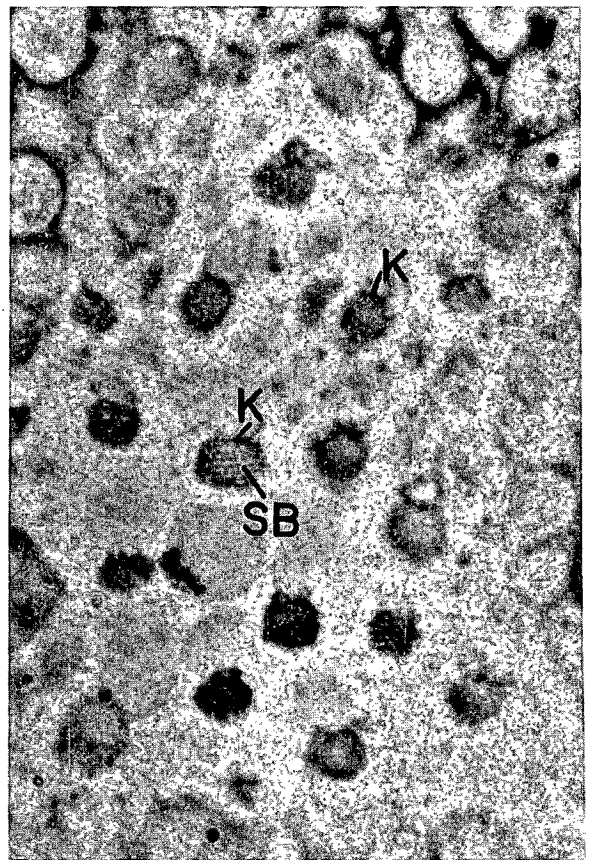


Figure 9.—Phase contrast picture of a horizontal section through surface of a macula of a monkey showing stereociliar bundles (SB) each with one kinocilium (K) as a black spot on one and the same side of stereociliar bundle.

crista of both vertical canals they are, however, always at the distal pole of the cell surface (fig. 10). The reversed functional pattern of horizontal and vertical crista as it is shown in Ewald's law is therefore related to a reversed morphological polarization of the sensory hairs.

In electrophysiological experiments Loewenstein and colleagues (1940, 1955), were able to confirm and elucidate Ewald's law a little further. In the majority of single sensory units they found a spontaneous activity. In the lateral ampulla this activity was increased with ampullopetal and decreased to a lesser extent with ampullofugal stimulation. In the vertical ampullae the

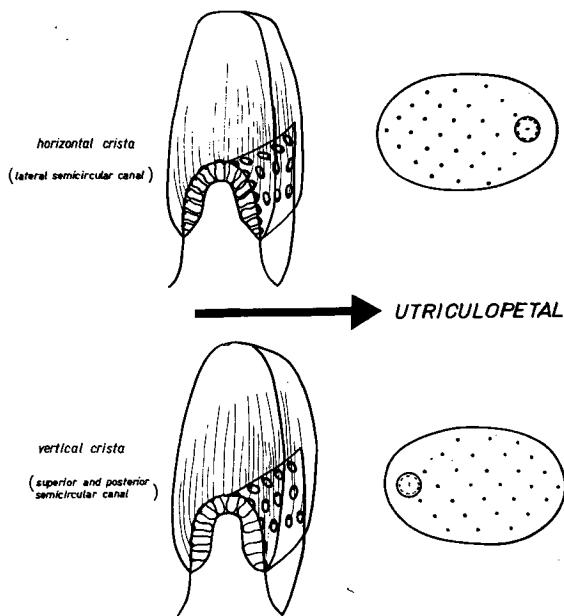


Figure 10.—Schematic representation of orientation of kinocilia in cristae of semicircular canal. In horizontal cristae all kinocilia are always oriented toward utricle whereas in vertical canals they are oriented away from utricle.

pattern is reversed in accordance with the second law of Ewald.

A similar directional behaviour of the cristae was found by Trincker who studied the resting potential in the ampullae of guinea pigs. He found that a utriculopetal cupula deviation in the horizontal ampulla always results in a depression of the resting potential, whereas a utriculofugal deviation increases this potential. Here again the pattern was reversed in the vertical ampullae.

Such a direct relation between the arrangement of the kinocilia and the function of the sensory cell shows that the kinocilium as such is an important structure for the sensory cell stimulation. A deviation of the sensory hairs in the direction of the kinociliar pole would, according to Trincker's interpretation of his own findings, produce a depolarization of the sensory epithelium and, therefore, an increased nervous activity as found by Loewenstein and Sand (1940). A deviation of the sensory hairs away from the kinociliar pole, on the other hand, would

create a hyperpolarization of the sensory epithelium and a decreased nervous activity.

In the maculae the situation appears to be more complex. The sensory hairs are somewhat shorter than in the cristae but otherwise have an identical structure. The kinocilia are also uniformly polarized over wide areas. However, the direction of polarization differs for different parts of the maculae (fig. 11). In reconstruction of the macular surface by means of horizontal serial sections we find that the directions of polarization spread fanlike from the medial and anterior part of the macula up to a curved boundary line beyond which the polarization of the sensory hairs is reversed. The kinocilia on either side of this dividing line are facing each other (fig. 12).

Although the great majority of the sensory hairs in one area of the maculae are polarized in the same direction, there is always a certain number of sensory cells with different polarization of their sensory hairs, as illustrated in the following table:

Direction of polarization	Section		
	I	II	III
Anterior -----	1	1	14
Lateral -----	6	15	5
Posterior -----	35	2	1
Medial -----	3	0	1

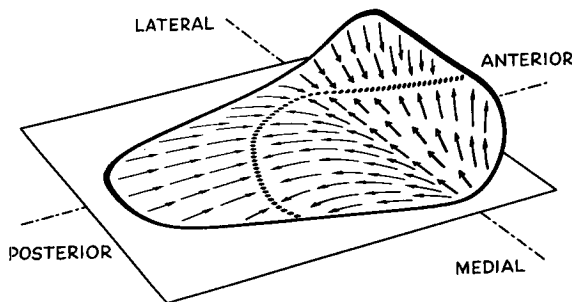


Figure 11.—Schematic representation of polarization pattern of sensory cells in macula utriculi of guinea pig. Arrows indicate the direction of polarization showing how it spreads fanlike from one side of macula up to a certain line beyond which polarization is reversed. Kinocilia on either side of this dividing line are facing each other.

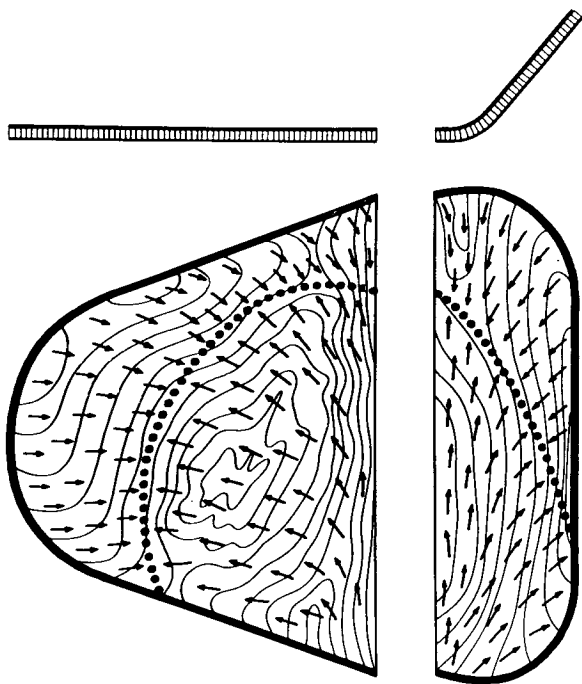


Figure 12.—Schematic drawing showing how reconstruction of surface of macula utriculi was done with serial sections of horizontal and inclined part of macula. Since surface of the macula utriculi is not entirely flat, each section of the serial sections hits actual surface of the sensory epithelium along a certain line which is indicated in this drawing by the fine curved lines. Arrows indicate direction of polarization of kinocilium as evaluated in each section with aid of phase contrast microscopy.

Each column represents the number of sensory cells in one section which have been counted and sampled according to the direction of their kinocilia. Each section shows a different direction of polarization. On some occasions we even observe sensory cells where the kinocilium is not polarized, all being in the middle of the stereociliar bundle (fig. 13). All possible directions of polarization are represented in one macula utriculi.

A different pattern of polarization is observed in the macula sacculi (fig. 14). Here too we find a curved dividing line going through the entire sensory epithelia on either side of which the polarization of the sensory hairs is opposite. Here, however, the kinocilia are not facing each other as in the macula utriculi but are facing away from

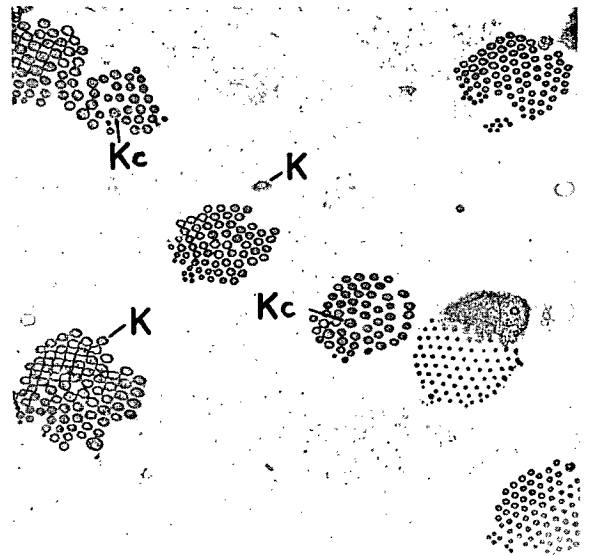


Figure 13.—Horizontal section through some sensory hair bundles of a macula utriculi showing two examples of an unusual position of kinocilium more or less in center of a stereociliar bundle (Kc). All other kinocilia are oriented in same direction (K).

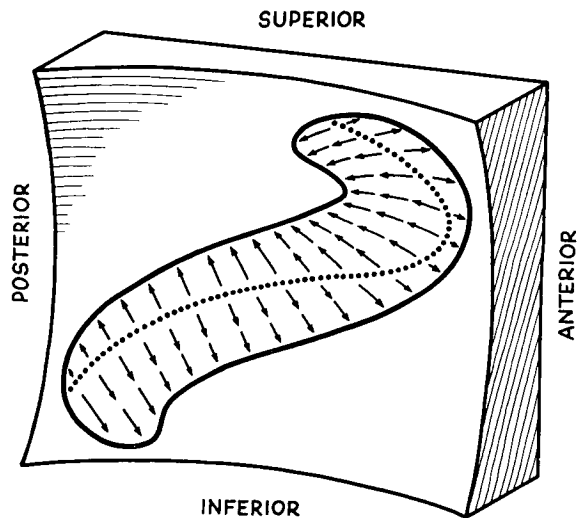


Figure 14.—Schematic representation of polarization pattern of sensory cells in macula sacculi of guinea pig. Here polarization is again reversed along a certain line going through entire macular surface (indicated as dotted line). In contrast to macula utriculi kinocilia on either side of this boundary line are not facing each other but facing away from each other.

each other. Not all directions of polarization are represented in the macula sacculi. The sensory cells are mainly polarized to approximately equal parts in antero-inferior and postero-superior direction.

It is very striking that the site of the dividing line of sensory hair polarization in the macula utriculi and sacculi seems to correspond fairly well with the site of the striola, that special zone in the center of the macula described by Werner. It might well be that here we are dealing with a particularly sensitive and well-developed zone in the sensory epithelium. Furthermore, we find a similar structural pattern within the otolithic membrane insofar as large otoliths in the distal part of the macula utriculi can be distinguished from smaller ones in the proximal and medial part of the macula. The boundary line between the two kinds of otoliths again corresponds roughly with the curved line of a reversed polarization of the striola of Werner. Similar differences in the otoliths have been reported by Lorento de N6 (1926) and Werner (1940).

If we assume that, in the macula, the same mechanism of hair cell stimulation is taking place as in the cristae, a positive stimulation with increased nervous activity will always arise when the sensory hairs are deviated toward the kinociliar pole of the sensory cell. Since all four directions of polarization are represented in one macula utriculi, we would expect that one macula would be able to respond to rectilinear accelerations in all directions. This is in perfect agreement with the electrophysiological findings of Loewenstein and Roberts (1951). According to their results, one single macula utriculi responds to tilting from normal position around all horizontal axes. A single functional unit, however, will have its maximum positive and negative response to tilting around one specific horizontal axis, exactly as we would expect it from its morphological polarization.

There is certainly much morphological evidence that the kinocilium plays an important role in the receptor mechanism of

the vestibular sensory cell. The basic structural identity with motile kinocilia, as for instance in the respiratory epithelium, has to be kept in mind. Active movements of motile kinocilia are strictly directional, beating always in the same direction. Those movements are evoked by a stimulation which spreads throughout the cilium. It is possible that in the case of the sensory epithelia, we are dealing with a reversed mechanism. The passive movement of the kinocilium in the correct direction would cause a stimulation, as already suggested by Loewenstein and Wersäll (1959). Even the asymmetric intrinsic structure of the kinocilium denotes a directional function. The odd number of nine peripheral tubular filaments in the kinocilium might be the basis and the condition for direction-specific function.

However, we have to be careful not to go too far in our interpretation. On one hand,

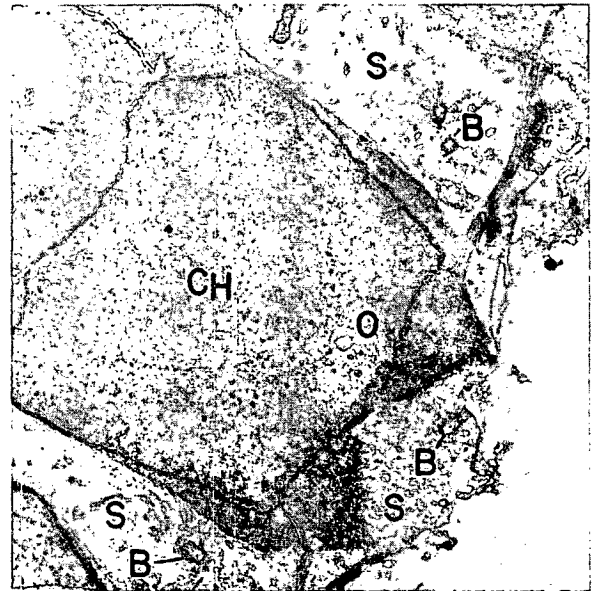


Figure 1. Horizontal section through the cuticular plate (CH) of an outer hair cell of the organ of Corti of a adult cat with its typical opening in the cuticular plate on one side of the cellular surface (O) where we would expect a kinociliar basal body in vestibular sensory cells. In this cochlear hair cell kinociliar basal body is lacking however. In each supporting cell (S), however, one or two basal bodies (B) are clearly visible.

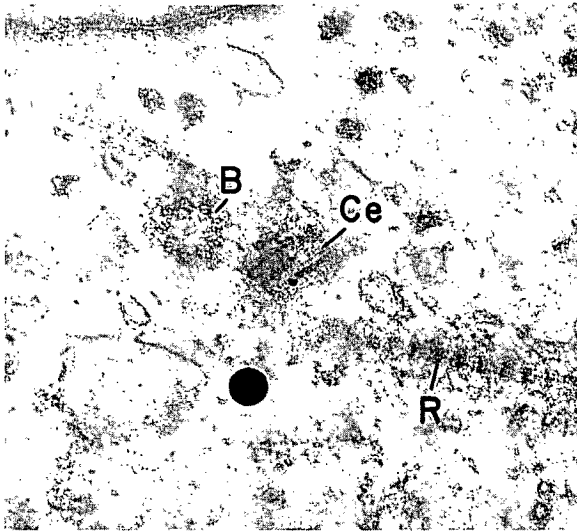


Figure 16.—Horizontal section through a typical kinociliar basal body (B) with its associated centriole (Ce) of a supporting cell in macula. Typical striated rootlets (R) which are usually associated with those basal bodies in supporting cells.

the cochlear sensory cells which are stimulated by the same type of sheering motion have no kinocilia and in some species, as for instance the cats, not even isolated kinociliar basal bodies (fig. 15). On the other hand, each supporting cell contains also a modified kinocilium with a typical basal body and usually an associated centriole (fig. 16). Such basal bodies appear to be very elementary structures and are not necessarily and directly involved in the receptor mechanism (compare Engström et al., 1962). We believe, however, on the basis of the correlation of functional and morphological polarization of the sensory cells, that the fully developed kinocilia of the vestibular sensory cells are important for their excitation. The stimulating sheering motion is very slow and monophasic in the vestibular receptors as compared with the cochlea. This might be the reason why the vestibular sensory cells are provided with a kinocilium whereas in the cochlear receptors it is not needed.

It is more difficult to find a structural basis for the second feature in Ewald's law, that is, the quantitative difference between

equal ampullofugal and ampullopetal cupula deviations. Again it was Loewenstein and his colleagues (1956) who found an explanation for this phenomenon on an electrophysiological basis. They found essentially two types of sensory units, ones with and others without spontaneous activity at rest. The spontaneously active receptors represent the majority, but there is also a considerable number of spontaneously silent units. Utriculopetal stimulation in the lateral ampulla increases not only the activity of the spontaneously firing units but also activates the spontaneously silent units which appear to have a higher threshold. Utriculofugal stimulation, however, reduces the activity of spontaneously silent receptors. Thus, the total deviation from the resting activity is stronger with utriculopetal stimulation than with utriculofugal stimulation in the area of the lateral ampulla and vice versa in the case of the vertical ampullae. Between those two types of sensory receptors transitional types are also found (fig. 17).

At first thought it would be tempting to correlate those two types of sensory units, the spontaneously active and the spontaneously silent units, to the two types of sensory cells. This, however, was shown to be incorrect since there is only one type of sensory cell in the labyrinth of fishes, although electrophysiologically both types of sensory units are represented (Wersäll 1958). There must, therefore, be another distinctive feature between those two sensory units, and we have to consider the innervation of the sensory cells.

If the sensory cells are responsible for the first step in the transformation of mechanical into electrical energy, it is the connection between the sensory cells and the nerve endings where the second step, the initiation of nerve impulses or of generating potentials, is taking place.

It is generally believed that, in most cases, the impulse transmission from the sensory cell to the nerve ending occurs only at certain places with specific synaptic structures. The morphological multitude of such synaptic

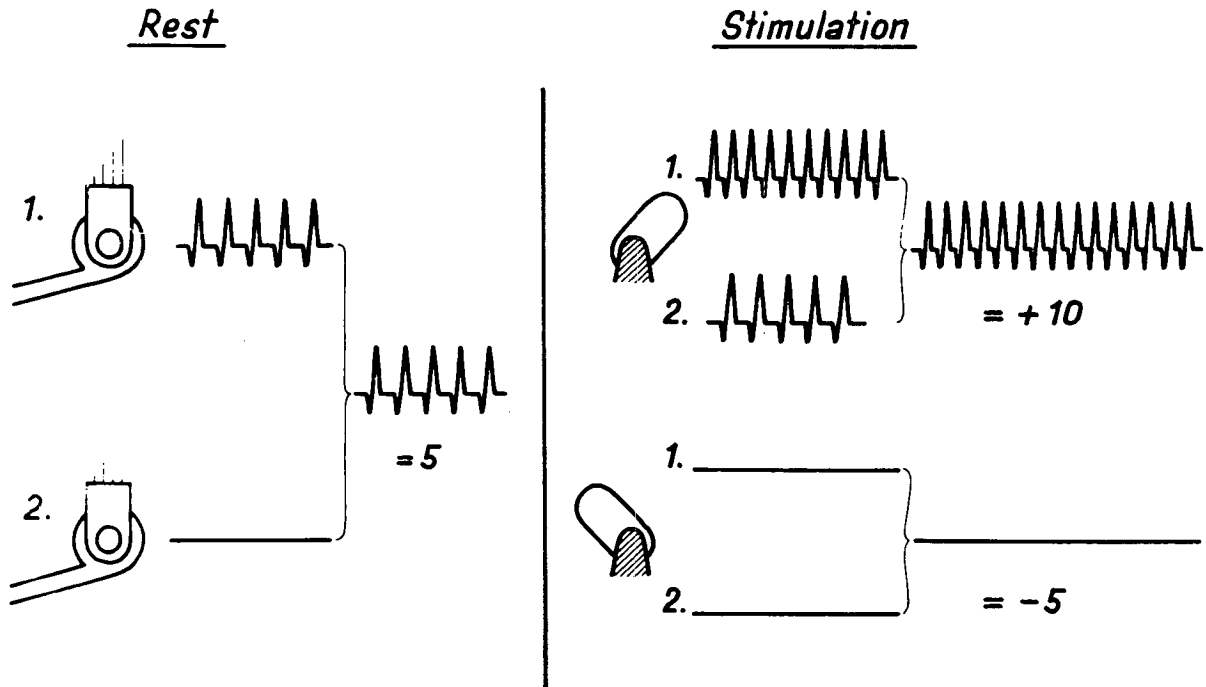


Figure 17.—Schematic representation of spontaneously active (1) and spontaneously silent (2) units as described by Loewenstein et al. The number of impulses are chosen arbitrarily to demonstrate how this arrangement can be used to explain the difference between utriculopetal and utriculofugal cupula deviations. The total deviation from activity at rest is greater with cupula deviation in one direction (+10) than in opposite direction (−5).

structures within the macula of monkeys is striking. The most basic and regularly observed features of possible synaptic structures is a narrowing of the intercellular space between axon and cell membrane with a thickened axon membrane. Next to such "synaptic" areas, the intercellular space is often considerably widened. Frequently such areas are invaginated into the sensory cell (fig. 18).

In addition, however, there is a variety of accessory synaptic structures in the cytoplasm of sensory cells (figs. 19, 20). They consist usually of a very dense osmiophilic structure frequently surrounded by small vesicles of the dimensions of synaptic vesicles (De Robertis 1959). The most common of these formations are synaptic bars similar to what has been described by Smith (1961) in the organ of Corti. Instead of bars there are also found small round masses, spherical structures which, in section, appear as rings

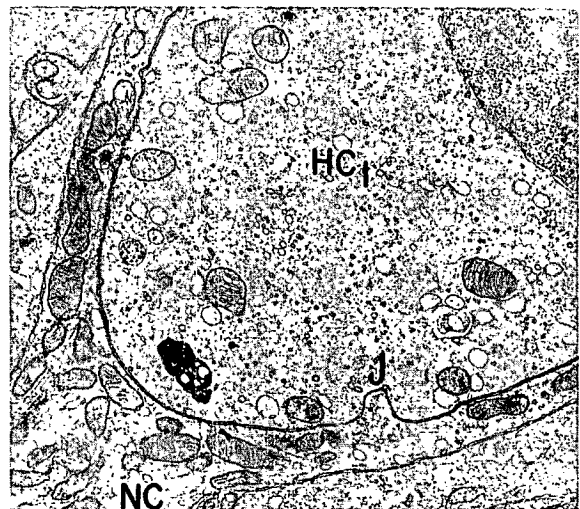


Figure 18.—Relation of a nerve chalice (NC) to a hair cell of type I ( $HC_1$ ) with a typical invagination of the nerve chalice (J) where the intercellular space is markedly reduced, which probably can be regarded as a synapse. Note also abundance of ribosomes within hair cell whereas no ribosomes are visible in nerve chalice.

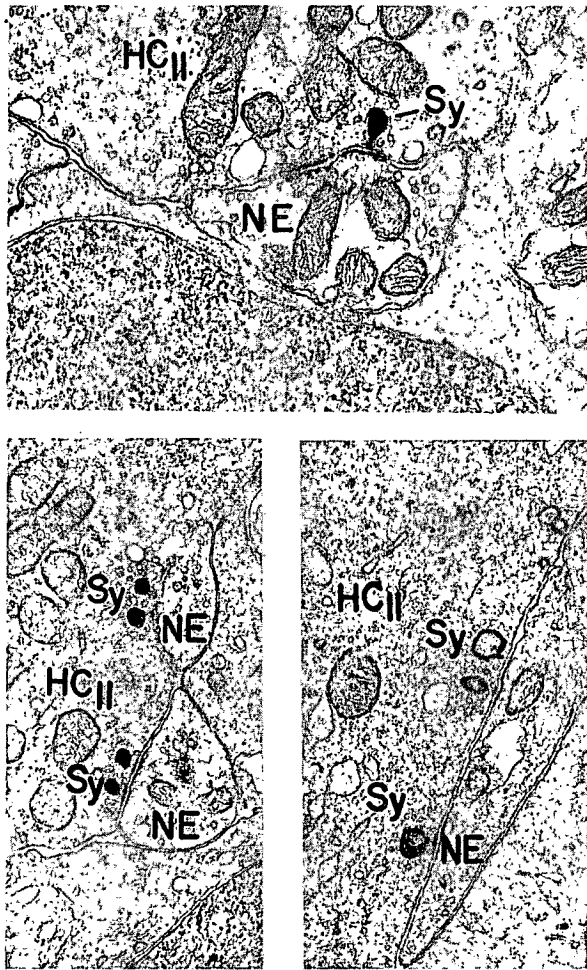


Figure 19.—Some different accessory synaptic structures (Sy) between individual nerve endings (NE) and hair cells of type II (HC<sub>II</sub>).

or long straight or bent laminae extending far into the cytoplasm of the sensory cell. The latter structures could only be found in the hair cells of type I (fig. 20). All the others appear in both types of sensory cells. Not only the appearance but also the number of synapses per sensory cell and nerve endings vary greatly.

As revealed in serial sections there are sensory cells with very few or no synaptic structures while others have a large number. This suggests that not all sensory cells show the same functional behaviour at a given time, and the question remains whether morphologically different types of synapses

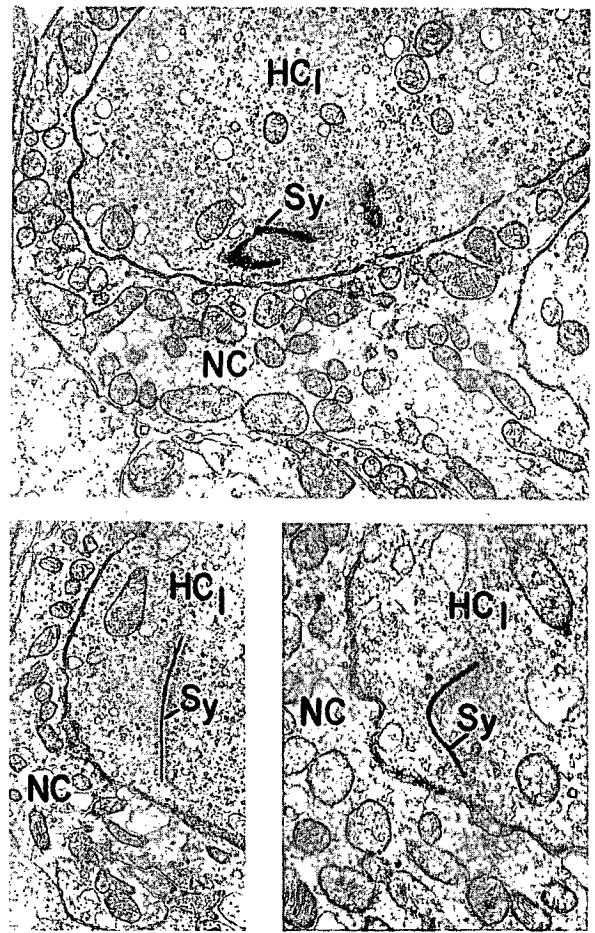


Figure 20.—Different forms of accessory synaptic structures (Sy) in hair cells of type I (HC<sub>I</sub>) usually surrounded by a great number of small vesicles. Nerve chalice (NC).

represent qualitatively or quantitatively different functions or whether they correspond to different functional states of a synapse. The difference in functional behaviour between the spontaneously silent and active sensory units as revealed in electrophysiological investigations might be in relation to the number and state of the synapses in the different sensory cells. The spontaneously silent receptors which are considered to be less sensitive would correspond to sensory cells with fewer functional synapses.

A very primitive schemata might illustrate my thoughts. As already suggested by Loewenstein (1955), we might compare the

sensory cell nerve-ending unit with a triode. The anode current comes basically from the grid voltage and from the number and size of the grid meshes. That the behaviour of the sensory units can be changed by polarizing DC-currents (grid voltage) has been demonstrated by Loewenstein. We wonder, however, if the second factor, the size and number of grid meshes, could influence the behaviour of sensory units as well. In such a model the synapses would correspond to the grid meshes. More or larger meshes are present in the grid at a given grid voltage, the current flow from cathode to anode being freer. For the sensory cell it would mean that a great number of synapses facilitate the transmission from the sensory cell to the nerve ending and would therefore lower the threshold for the initiation of nerve impulses (fig. 21).

The enormous variety and large number of synaptic structures in the vestibular sensory epithelia of the squirrel monkey have not yet been described in other animals. They might be related to the great functional

importance of the gravity receptors in those squirrel monkeys where acrobatic skill is important for survival. Uninterrupted synaptic transmission must be provided in order to guarantee a steady function.

The fact that, in some sensory cells, no synaptic structures at all are found certainly suggests that synaptic structures are not of a permanent nature but that they wear out and are built up in time. It could hardly be conceivable that a sensory cell would remain throughout lifetime without any synaptic relations to nerve endings.

How much and in what way the efferent innervation interferes with the function of the vestibular receptors still remains an open question.

There are undoubtedly myelinated efferent fibers from the lateral vestibular nucleus reaching the sensory epithelia, as shown by Gacek and Rasmussen (1961) and in histochemical acetylcholinesterase demonstrations by Ireland and Farkashidy (1961) as well as by Schuknecht and Nomura.

This efferent system most probably has its endings at the sensory cells. Some of the bud-like endings at the hair cells of type II are indeed filled with vesicles comparable to the cochlear efferent endings; and they could therefore be considered as efferent endings from the morphological point of view (fig. 22). However, the morphological distinction between afferent and efferent fibers in the vestibular sensory epithelia is not so clear as in the cochlea where the efferent innervation is much more abundant. Nerve endings with synaptic vesicles are frequently found associated with synaptic bars and agglomerations of synaptic vesicles on the sensory cell side of the synapse as well. Only on rare occasions could we observe a synapse between a vesiculated nerve ending and another nerve fiber or nerve chalice. We do not believe that places where vesiculated nerve structures are just adjacent to other nerve fibers can be considered as synaptic areas in which impulse transmission is taking place.

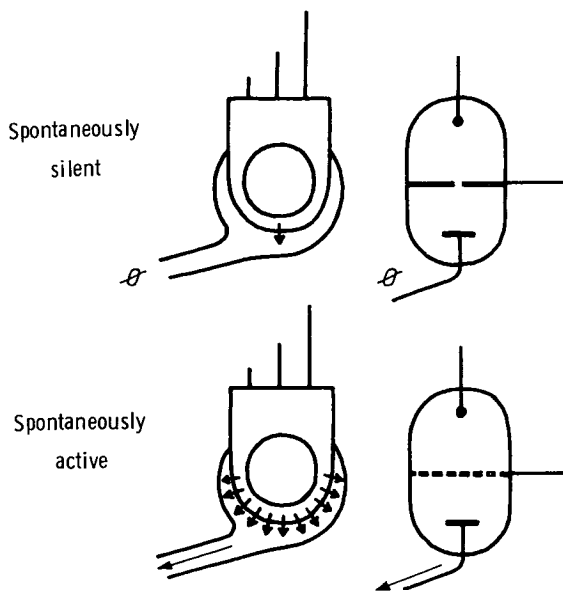


Figure 21.—Possible model of spontaneously silent and spontaneously active sensory units where number and quality of synapses between sensory cell and nerve ending are compared with different size and number of grid meshes in a triode.



Generally speaking, there are unquestionably many more vesiculated nerve endings in synaptic contact with hair cells of type II than with the nerve chalices of the type I hair cells. In accordance with those findings, Gacek, Schuknecht and Nomura (personal communication) found, with histochemical methods, much less acetylcholinesterase activity in the area of the striola, where we have a particular concentration of type I

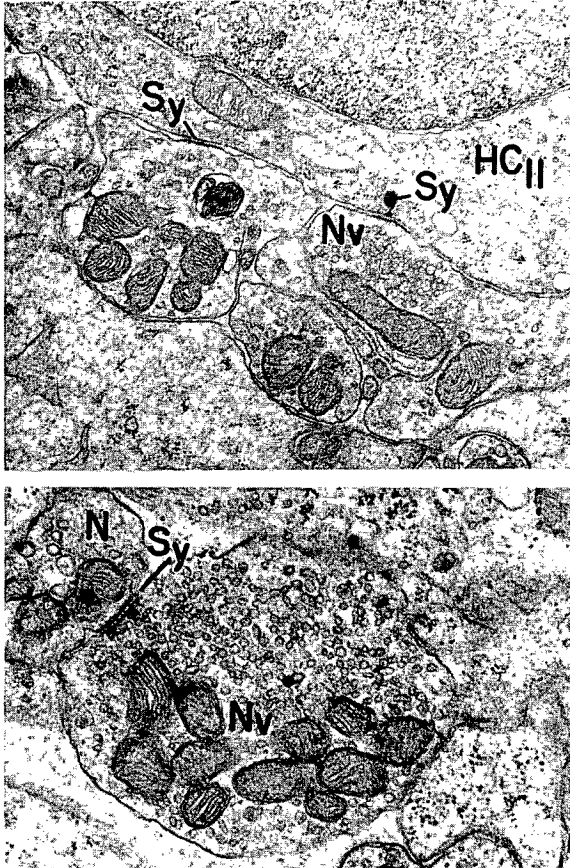


Figure 22.—Upper: vesicle-containing nerve ending (NV) in synaptic contact with a hair cell of type II (HC<sub>II</sub>). A synaptic bar is present in the sensory cell adjacent to the vesiculated nerve ending (Sy at right). The example of a probably basic synaptic pattern with narrowing of intercellular space associated with thickening of axon and cell membrane is visible at left (Sy). Lower: synaptic contact (Sy) between a vesiculated nervous structure (NV) which from morphological point of view could be of efferent nature with another nervous structure (N).

hair cells. It is most likely that the vesiculated endings correspond to acetylcholinesterase active structures and that they are the terminals of the efferent vestibular fibers as described by Gacek (1961), although the direct proof is still lacking.

Acetylcholinesterase activity, however, does not prove the presence of a cholinergic innervation as recently demonstrated by several authors (Desmedt, personal communication). It is, therefore, not yet clear what type of system those efferent fibers represent.

With a new histochemical method which was introduced by Falk (1962), adrenergic fibers can be demonstrated in the fluorescence-microscope. In this way the perivascular adrenergic network around the labyrinthine artery can easily be shown (fig. 23). This perivascular network, however, is no longer present in the vessels of the membranous labyrinth.

On the other hand, we were able to demonstrate a very fine network of adrenergic fibers in the macula utriculi. They did not seem to be in a direct relation to blood vessels which, at this level, usually are in the order of capillaries. The precise endings of those adrenergic fibers, however, could not yet be detected (fig. 24).

In electronmicroscopic sections through the nerve fibers underneath the macular epithelium we see that the great majority of nerve fibers are myelinated and have a diameter of 3 to 9 microns. They lose their myelin sheath just before they penetrate into the sensory epithelium. At closer examination one finds, however, a certain number of very fine unmyelinated fibers from 0.2 to 1.0 micron thickness (fig. 25). We have always wondered about the significance of those fibers and now we think that they might correspond to adrenergic fibers. We have not yet been able, however, to follow them to the actual endings. In any case, they never were found adjacent to blood vessels. It seems that some of them do penetrate into the sensory epithelium where they can

be recognized above the basement membrane between supporting cells (fig. 26).

All we can say now is that there is an adrenergic nerve plexus in the macula and that this plexus is independent from the

blood vessels. Further experiments will give more information about the origin and possible role of this adrenergic system in the receptor mechanism. It might have an opposite effect on the receptor than the cholinesterase active fibers, which seem to be inhibitory. Thus, sensory receptors would be regulated by two different types of innervation, myelinated cholinesterase active fibers (Gacek) and an adrenergic unmyelinated system.

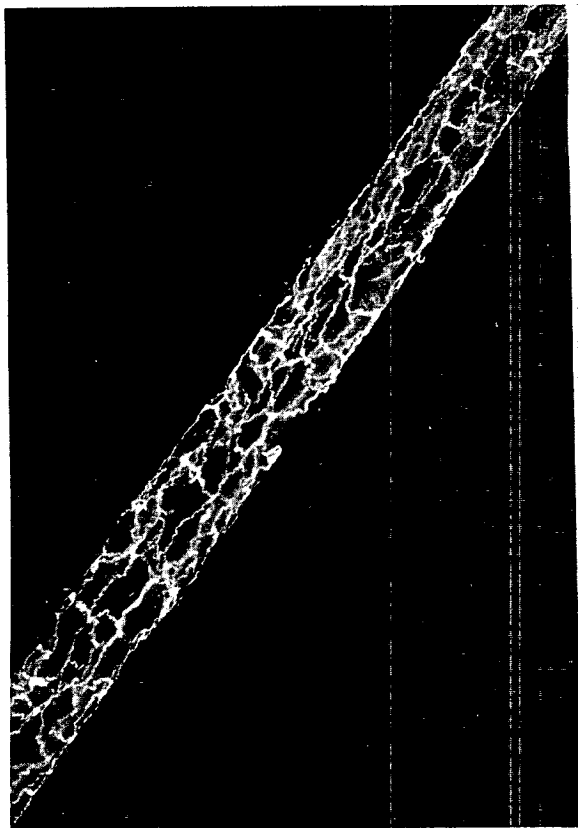


Figure 23.—Specific demonstration of adrenergic nerve plexus around labyrinthine artery in a cat in fluorescence microscope, according to technique of Falk and Hillarp. With this technique noradrenalin and associated compounds appear in the fluorescence microscope with a bright green fluorescence. (Study in collaboration with Dr. Lichtensteiger, Department of Pharmacology, University of Zürich.)

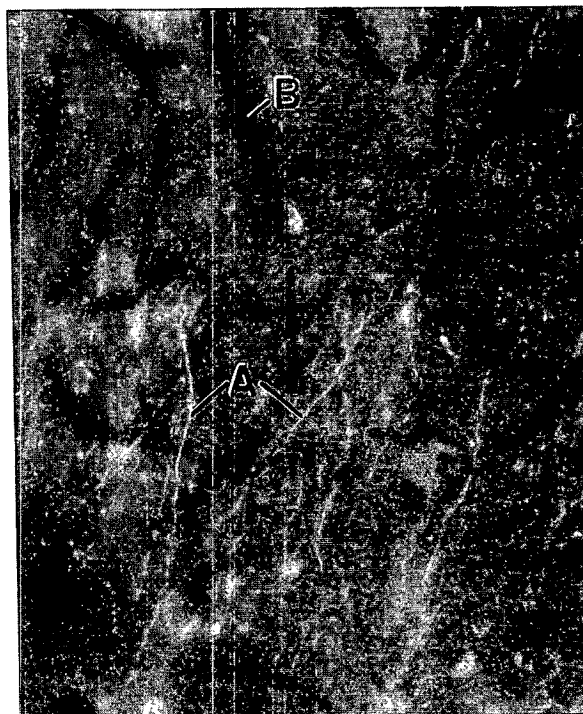


Figure 24.—Specific demonstration of noradrenergic nervous plexus within macula utriculi of a guinea pig (A) in a fresh tease-preparation of a macula of a guinea pig. Those adrenergic fibers are not associated with blood vessels (B).

#### REFERENCES

- BAIRATI, A., JR.: *Acta Oto-Laryngol.*, Suppl. 163, (1960).
- BARNES, B. S.: *J. Ultrastruct. Res.* 5, (1961), pp. 453-467.
- BÉKÉSY, G. V.: *J. Acoust. Soc. Amer.* 24, (1952), pp. 72-76.
- DAHL, H.: *Z. f. Zellforschung* 60, (1963), pp. 369-386.
- DE ROBERTIS, E.: *Triangel, Sandoz-Zeitschrift* 5, (1961), pp. 76-88.
- DE ROBERTIS, E.: *Int. Rev. Cytol.* 2, (1959), p. 503.
- ENGSTRÖM, H.: *Acta Oto-Laryngol.*, 49, (1958).
- ENGSTRÖM, H.; WERSÄLL, J.: *Intern. Review of Cytology* 7, (1958), pp. 535-585.
- ENGSTRÖM, H.; WERSÄLL, J.: *Exp. Cell Res. Suppl* 5, (1958), p. 460.
- ENGSTRÖM, H.; ADES, H.; HAWKINS, J.: *J. Acoust. Soc. Amer.* 34, (1962), pp. 1356-1363.
- EWALD: *Endorgan des Nervus Acusticus* (1892).

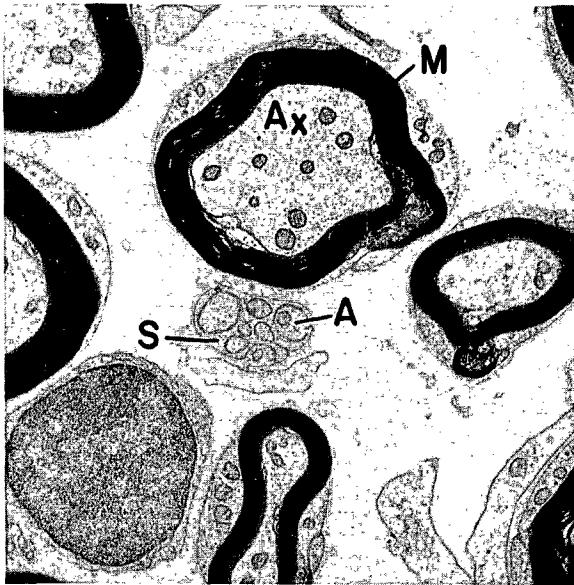


Figure 25.—Cross section through a number of nerve fibers underneath the macula utriculi with large myelinated nerve fibers of an average diameter of 5 microns with their axons (Ax) and myelin sheaths (M). Among those fibers there are found regularly unmyelinated small nerve fibers of a diameter from 0.2 micron (A). Several of those unmyelinated small nerve fibers are usually surrounded by one single supporting cell (Schwann's cell) (S).

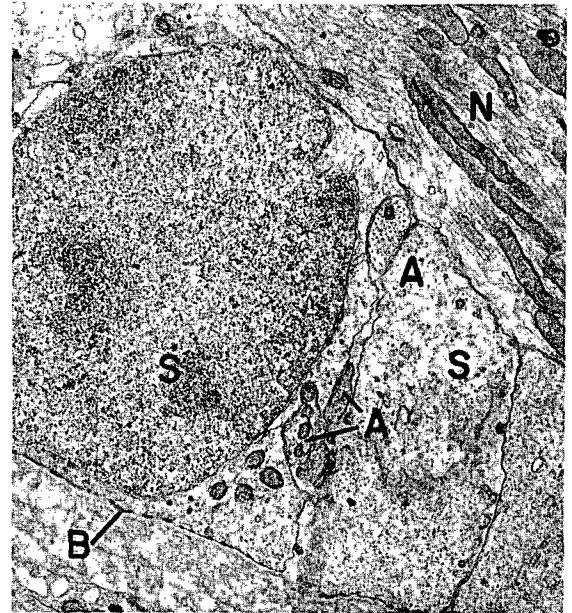


Figure 26.—Small unmyelinated nerve fibers can be followed into the sensory epithelium. Here those small fibers (A) can be found between basal portions of supporting cells (S) above basement membrane (B) of sensory epithelium. A large nerve fiber can be seen at upper right (N). It has lost its myelin sheath before entering the sensory epithelium. Small nerve fibers might eventually be associated with the histochemically demonstrated adrenergic nerve fibers.

- FALK, B.: *Acta Physiol. Scandinav.* 56, Suppl 197 (1962).
- FAWCETT, D. W.: *The Cell* 2, (1961), pp. 217-297.
- FLOCK, A.: *J. Cell Biol.* 22, (1964), pp. 413-431.
- FLOCK, A.: *Acta ORL Suppl* 199, (1965).
- FLOCK, A.; WERSÄLL, J.: *J. Cell Biol.* 15, (1962), pp. 19-27.
- FRIEDMANN, I.: *Triangel, Sandoz-Zeitschrift* 6, (1963), p. 74.
- GACEK, R. R.; RASMUSSEN, GRANT L.: *Anat. Rec.* 139, (1961), pp. 455-463.
- GERNANDT, B.: *J. Neurobiology* 12, (1949), pp. 173-184.
- IRELAND, P.; FARKASHIDY: *Ann. of ORL* 70, (1961), pp. 420-563.
- LORENTO DE NÓ, R.: *Trav. recherches viol. Univ. Madrid* 24, (1926), p. 53.
- LOEWENSTEIN, O.: *J. Physiol.* 127, (1955), pp. 104-117.
- LOEWENSTEIN, O.; ROBERTS, T. D. M.: *J. Physiol.* 110, 392 (1949) 114, (1951), p. 471.
- LOEWENSTEIN, O.; SAND, A.: *Proc. of the Royal Soc. Series B Biol. Sciences* 129, (1940).
- LOEWENSTEIN, O.; WERSÄLL, J.: *Nature* 184, (1959), pp. 1807-1808.
- LOEWENSTEIN, O.; WERSÄLL, J.: *Nature* 184, (1959), pp. 4701, and 1807.
- ROBERTSON, J. D.: *J. Biophys. Cytol.* 2, (1956), p. 381.
- SMITH, C.: *Ann. of ORL* 70, (1961), pp. 504-527.
- SMITH, C.; SJÖSTRAND, F. S.: *J. Ultrastruct. Res.* 5, (1961), p. 184.
- SPOENDLIN, H.: *Z. f. Zellforschung* 62, (1964), pp. 701-716.
- TRINCKER, D.: *Pflüg. Arch. ges. Physiol.* 264, (1957), pp. 351-382.
- WERNER, C. F.: *Georg Thieme, Leipzig*, 1940.
- WERSÄLL, J.: *Neural Mechanisms of the Auditory and Vestibular Systems.* Charles C Thomas, Springfield, Ill., 1960.
- WERSÄLL, J.: *Acta ORL* 126 (1956).
- WERSÄLL, J.; HAWKINS, J.: *Acta ORL* 54, (1962), p. 1.
- WOLKEN, J. J.: *J. Protozool* 3, (1956), pp. 211-225.

## DISCUSSION

M. JONES: It had occurred to me that perhaps the kinocilium might act here in its more usual mode, for instance as a motile organ. Do you think that it might be involved in modifying the cupular elastic restoring coefficient? Recent theoretical considerations of R. Mayne and myself have shown that there might be a great advantage in being able to do so.

SPOENDLIN: Such a functional significance of the kinocilia is certainly conceivable. As a matter of fact, Ewald himself had a similar concept about the function of the sensory hairs. It does, however, not explain the close relationship between the electrophysiological behavior of the sensory units and the polarization of the kinocilia on the surface of the sensory cells.

MONEY: I would like to comment about the second law of Ewald: When Ewald did his famous experiment, the ear which was giving the responses was opened in two places and was exposed to a hydraulic hammer during the experiment. You would expect lots of artifacts from that. Dohlmann did a later experiment in which he deformed the ampulla and thereby mobilized the cupula in pigeons. He found a postrotatory nystagmus from single horizontal canals equal in both directions. We repeated the same thing in pigeons by plugging the canal duct and found that the single horizontal canal remaining gave postrotatory nystagmus equal in both directions. We have done the same thing in cats, dogs, and monkeys. Although the electrophysiological evidence shows that the increases in the resting dis-

charges are greater than the decreases, this can be explained by the fact that now the central mechanism handles this information; just how is not known. If it receives, on a logarithmic scale, a smaller decrease, it could give a bigger response than with a larger increase in the resting discharges. I don't think Ewald's law is true in the intact animal for the horizontal canal.

SPOENDLIN: It was actually not Ewald who established his law. He just reported the findings from his physiological experiments on pigeons. No matter what artifacts may have influenced his results, they pointed to the basic pattern of a direction specific and asymmetric response of the semicircular canal receptors which was later electrophysiologically confirmed for the first order neuron by different authors. Therefore, I think the second law of Ewald has to be considered first of all at the level of the first order neurons. If we consider the entire vestibular system with its numerous central connections, we are dealing with too many unknown factors. On the other hand, the difference in response to utriculopetal and utriculofugal deviations probably becomes evident only with strong stimuli because the spontaneously silent units have a relatively high threshold.

BERGSTEDT: Ewald's second law holds for the first neuron but not for the entire vestibular system. It does not show in caloric tests. As discussed at a symposium in Switzerland, it is probably a misconception of Ewald's law if it is considered for the entire animal.

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# Form and Innervation of the Vestibular Epithelia

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## SUMMARY

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The present study has shown that the vestibular sensory cells are of two basic types; however, cells are also found which are intermediate in form. These presumably represent stages of partial differentiation from the more primitive type II cell toward the type I.

It has been clearly established that sensory cells of both types may be innervated by the same nerve fiber. It is apparent also that the pattern of innervation of vestibular sensory cells is much more complicated than has been believed hitherto.

Finally, this study has shown that well defined "synaptic regions" can be found between the nerve calyx and the base of the type I sensory cell, and that these are very much like the supposed synaptic regions which are seen between the sparsely granulated type of nerve ending and the type II sensory cells.

Author

## INTRODUCTION

In mammals the vestibular sensory cells are localized in five sensory areas in each ear. These areas are the three cristae ampullares, one in each ampulla of the three semicircular canals and the two maculae, one in the utricle and one in the saccule. Although there is a close resemblance in the arrangement of the sensory cells and supporting elements among cristae and maculae, there is a considerable difference in the covering structure over the epithelia between cristae and maculae. The jelly-like cupula over the crista has little in common with the membrane of statoconiae covering the macular surface.

The basic study of the ampullar cristae of the guinea pig by Wersäll (1956) added greatly to our knowledge of the vestibular epithelia. Further studies by Smith (1956), Engström and Wersäll (1958), Bairati

(1961), Engström (1958, 1961), Wersäll (1961), and Flock (1964) and others demonstrated that there is a general principle recognizable in all vestibular sensory epithelia in higher mammals as well as in fishes. It is also seen in the lateral line organ of fish.

It is now generally accepted that there are two types of hair cells in vestibular sensory epithelia. The general features of these cells are recognized from figures 1 to 5. During the last few years, however, it has become evident that the description of the vestibular sensory cells has been oversimplified and that important features in their structure have been overlooked. This is especially so with respect to the arrangement of the sensory hairs and the innervation of the hair cells; we have, therefore, considered it of interest to give a new description of these sensory cells and their innervation.

As we have found that some of the basic

principles in the form and structure of the maculae have been discussed very little in recent years, and as we believe that some basic functional vestibular properties have thereby been overlooked, we thought it appropriate to take up some of these problems also for brief discussion.

#### MATERIAL AND METHODS

Our studies are based upon a very large number of specimens collected during three decades and including inner ears from many different animals and man. In the years up to 1950 this material was studied only by conventional sectioning and light-microscopy methods. From 1950 on, an increasing part of the material has been studied by electron-microscopy after embedding in media such as acrylate or epoxy resins. In recent years we have devoted much time to a direct study of the unfixed or fixed vestibular sensory regions as seen, unsectioned, under the preparation microscope. Much of the work has utilized surface specimens, by which we mean that the sensory epithelia as a whole have been freed from the rest of the labyrinth, and observed under phase contrast microscopy or after nerve staining. On many occasions we have then microdissected the individual sensory regions to study physical or structural properties of individual cells or cell organelles.

During part of our work we have used X-ray crystallography to study the statoconiae. This was done together with D. Carlström who has since continued the work and recently has published a comprehensive study of the crystals forming the otoliths or statoconiae.

#### OBSERVATIONS

There are two distinct kinds of sensory cells in all the vestibular sensory epithelia we have studied. Their characteristic features can be seen in figures 1 to 5. There are also several cells of intermediate type and groups of cells which are enclosed in one nerve ending or nerve calyx (fig. 6). It also becomes evident if we compare figures 1 and 2, which are taken from earlier publications,

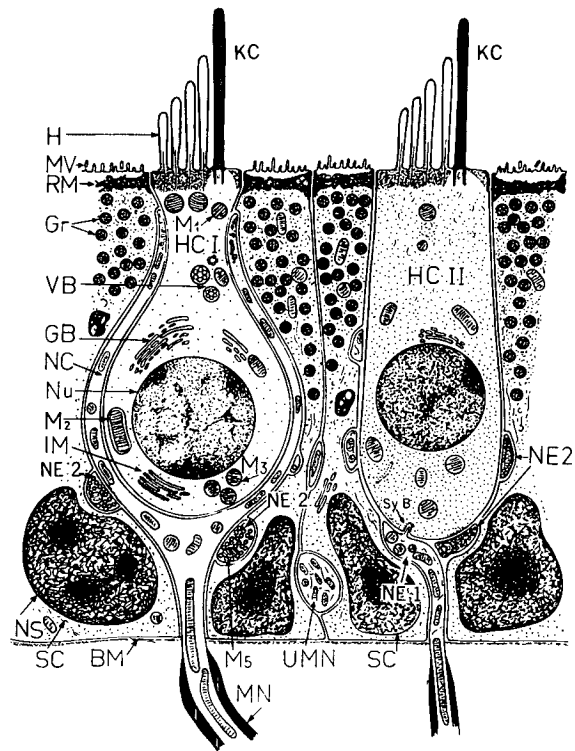


Figure 1.—Schematic drawing of two types of vestibular sensory cells. The flask-shaped type I cell (HC I) is surrounded by a nerve calyx (NC), which makes contact on its outer surface with granulated (presumably efferent) nerve endings (NE 2). Unmyelinated nerve fibers (UMN) are extensions of myelinated fibers (MN) which lose their myelin sheath as they pass through basement membrane. Type II sensory cell (HC II) is roughly cylindrical and is supplied by two types of nerve endings (NE 1 and NE 2) which can be seen at its basal end. Several groups of mitochondria ( $M_1$ – $M_5$ ) are found in the sensory cells and neural elements. Two kinds of hairs project from the surfaces of sensory cells, stereocilia (H) and kinocilium (KC), single kinocilium always being the longest on each cell. Supporting cells are easily distinguished from sensory cells by virtue of their numerous population of rather uniformly distributed granules (Gr).

that considerable modifications can be recognized. The major differences from earlier descriptions are found in the arrangement of the sensory hairs and in the innervation of the cells.

In a recent publication, Engström, Ades, and Hawkins (1962) described the two dis-

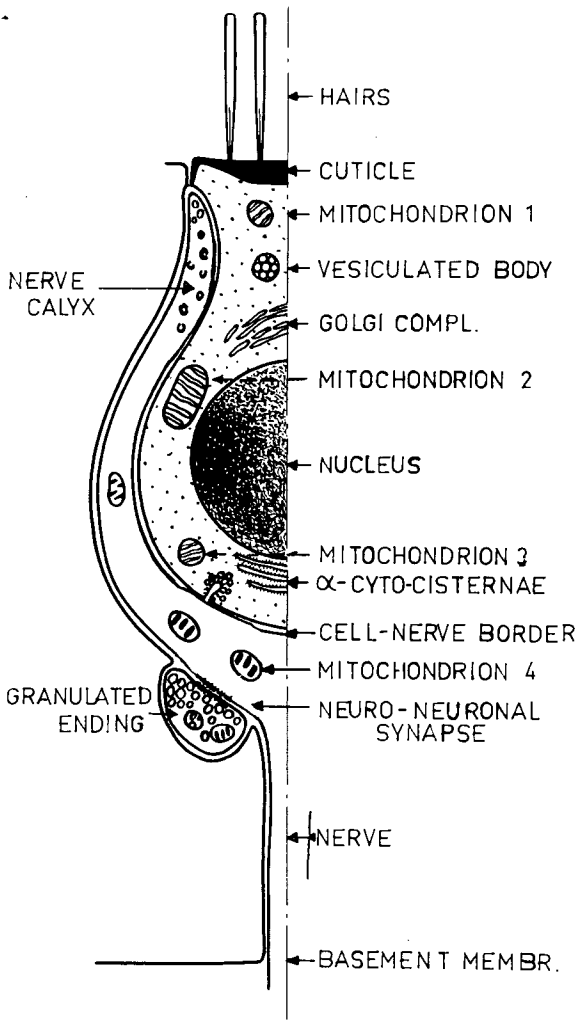


Figure 2.—Schematic drawing of a type I sensory cell, showing its inner structure and its neural connections.

tinct kinds of sensory hairs seen on vestibular sensory cells. Each cell characteristically displays a single kinocilium and about seventy stereocilia. This general situation had been known before; however, the fact that the stereocilia vary in length in a systematic way had been overlooked. The pattern is such that the stereocilia situated close to the kinocilium are the longest, and the length of the rest decreases progressively with increasing distance from the kinocilium (fig. 5). The gradient of diminishing length is sharp and pronounced, easily discernible by either phase or electronmicroscopy. Ac-

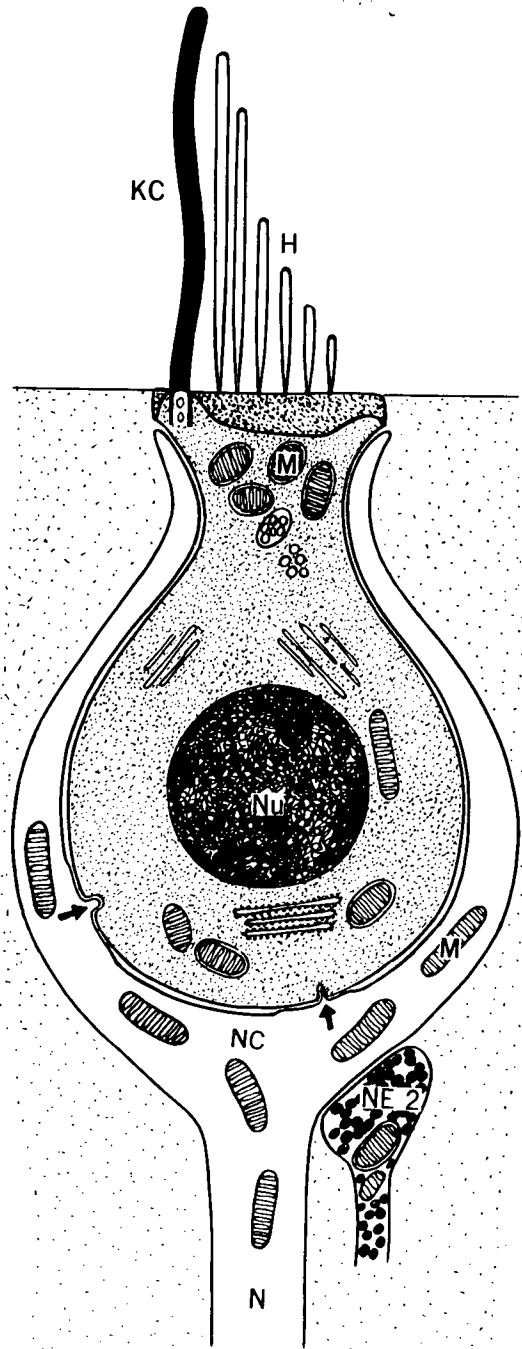


Figure 3.—Schematic drawing of type I sensory cell showing its nucleus (Nu), mitochondria (M), and two types of hairs (H and KC). Surrounding nerve calyx (NC) shows two synaptic areas (arrows). Applied to outer surface of calyx can be seen a granulated, presumably efferent, nerve ending (NE 2).

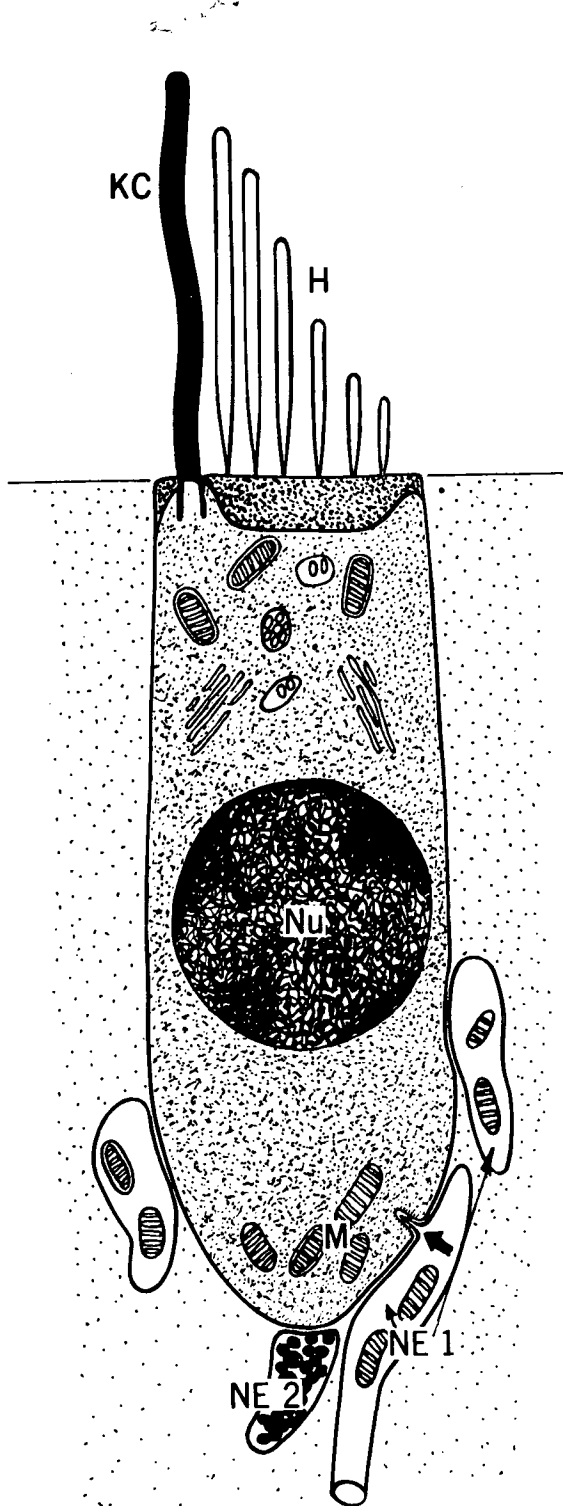


Figure 4.—Type II sensory cell showing two kinds of nerve endings (NE 1 and NE 2), and two types of hairs (H and KC).

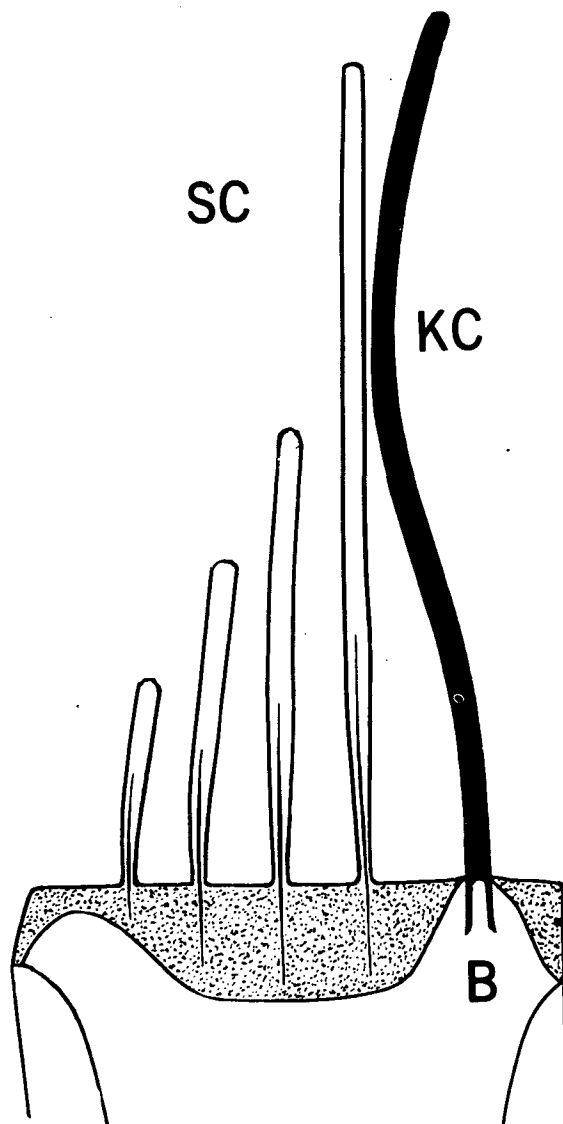


Figure 5.—Schematic drawing of a section through cuticular region of a vestibular sensory cell showing arrangement of stereocilia (SC) and kinocilium (KC) and origin of latter from basal body (B).

tually, Retzius hinted at this systematic variation of stereocilia length, but this has never been taken account of in discussions of the physiology of vestibular sensory cells. The gradient of increasing length of stereocilia toward the kinocilium indicates a polarity which is quite characteristic; moreover, the polarity of all cells in a particular region is uniform (Engström, Ades, and Hawkins,



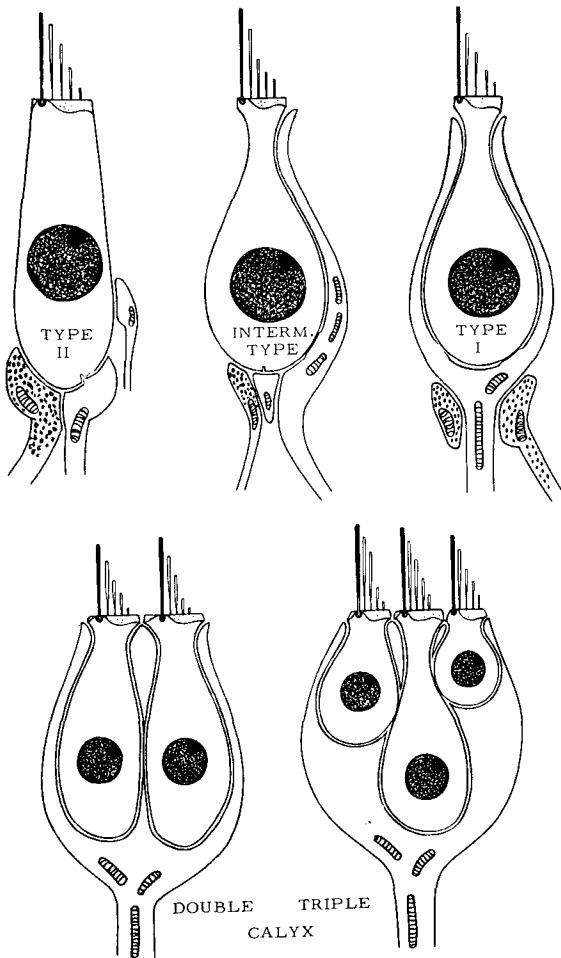


Figure 6.—Schematic drawing showing further complexities of sensory cell arrangement and nerve endings which may be found in same region of vestibular sensory epithelium.

1962), and characteristic of that region from one ear to another. Similar observations have been made by Flock (1964), and by Spoendlin (personal communication). Polarization of the kinocilium in a uniform direction is apparently characteristic of the neuromasts of fish, of the lateral line in *Xenopus* (*Kalmijn*: Dijkgraaf, 1963), and of the lateral line of fish (Flock et al., 1962); however, these authors did not indicate whether or not the stereocilia are of different lengths. Flock (1964) did describe different lengths of stereocilia in *Lota*.

It has been clearly established that the polarization of the cells has a distinct phys-

iological importance, and that a bending of the hairs toward the kinocilium corresponds to a rise in activity. Dijkgraaf (1963) has discussed the importance of this polarization. Interest in the kinocilium has been further increased since it has been shown that the primitive otolith of the ascidian tadpole is enclosed in what seems to be a modified kinocilium, and that no stereocilia are present on the cell (Dilly 1962). In this connection it is of great interest that a modified kinocilium is also present on every cochlear hair cell as shown by Flock et al. (1962) and by Engström, Ades, and Hawkins (1962). These observations and several others have prompted us to consider the kinocilium as a most important sensory hair. It could even be that the bending of the stereocilia is not directly responsible for excitation of the hair cell, but only indirectly through its mechanical effect on the kinocilium and the basal body. It is significant to note that modified kinocilia are now being found on most sensory cells and even inside the central nervous system. In the vestibular sensory epithelia with the kinocilium leading in length and with the rather stiff stereocilia decreasing in length, it must be assumed that a shearing movement of the statoconia as well as an increased pressure upon the hair-bearing surface must be met by a gradual increase in resistance. This, in turn, could result in a graded form of response and/or serve as an important safeguard to prevent damage to the sensitive organelles of the cell. It will be of great interest to see if the staircase pattern in the arrangement of stereocilia is present also in the side-line organ of, for instance, *Xenopus*, where every other row of sensory cells is oppositely polarized.

In hair cells of type I (fig. 3) the infracuticular region is densely populated by mitochondria (fig. 7) indicating a high enzymatic and metabolic activity. It is of great importance to elucidate what function these mitochondria have. In the outer hair cells of the cochlea of the guinea pig and the squirrel monkey, there is a marked accumulation of

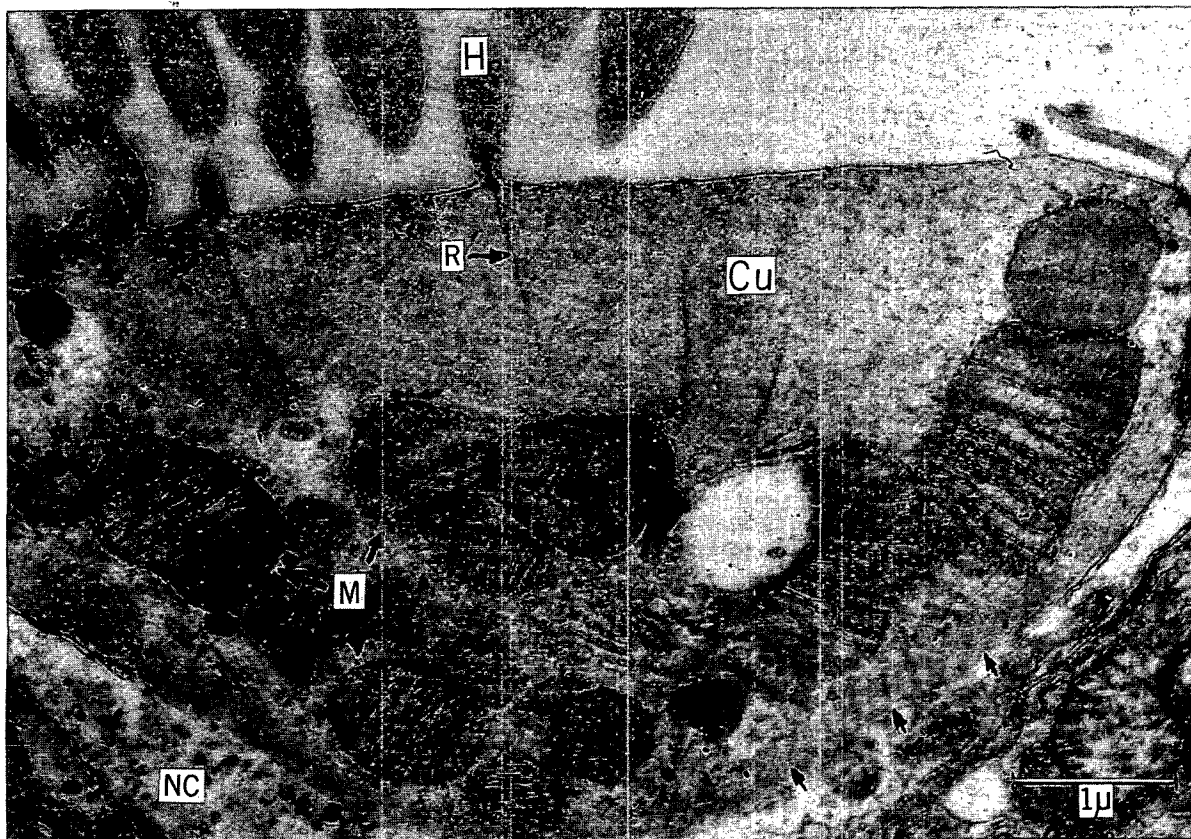


Figure 7.—Electron micrograph through cuticle of a type I sensory cell from macula utriculi of a guinea pig, showing detail of rootlets of sensory hairs. From base of hairs (H) rootlets (R) extend down through cuticle (Cu) to region close to large rounded mitochondria (M). Three black arrows toward lower right indicate a region of regularly arranged fibrous structures. NC marks nerve calyx.

mitochondria in the region under the basal body, the structure corresponding to the kinocilium of the vestibular cells. It seems very plausible that the modified kinocilium is in close functional relationship with the nearby mitochondria, as these often closely surround the basal body. It would be of great interest to determine definitely whether the rootlets of the vestibular stereocilia may not penetrate the cuticle and make contact with the large mitochondria. It seems likely from our micrographs that the rootlets at least extend very nearly through the cuticle (fig. 7).

The hair cells of type I usually have a characteristic arrangement of cytoplasmic organelles which can be seen in figures 1 to 3, but as very little of importance can be

added at this time, we refer to earlier publications. The hair cells of type II have a form which varies considerably. In the utricular and saccular maculae the cells may be short and bulky, but the majority have a form corresponding to figures 1 and 4. In the cristae the cells often are taller and thinner. The endoplasmic reticulum and the location of the cytoplasmic organelles differ considerably from the arrangement in the cells of type I (cf. fig. 3 with fig. 4). It is of considerable interest to note that the sides or slopes of the cristae appear to be more densely populated with sensory cells than the crest and that many of these are of type I (figs. 8 and 9). It was originally supposed by Wersäll (1956) and by Engström and Wersäll (1958) that the two types of sensory



Figure 8.—Light photomicrograph. Section of crista ampullaris of guinea pig with nerve stain showing nerve fibers of different caliber leaving epithelium. Dense population of type I sensory cells is easily seen, especially on side of crista.

cells could be innervated by nerve fibers of different caliber. This has proven true to a certain extent; however, the innervation of the sensory cells is more complicated than we earlier believed. A clear overlapping of the innervation takes place so that type I and type II cells may be innervated by the same nerve fiber. As some of the fibers also branch below the epithelium and a reconstruction of their course is rather difficult, our knowledge is as yet far from complete.

A type II cell, in all probability, can be innervated from a nerve calyx that surrounds a type I cell (cf. figs. 10 to 13). There is reason to believe that the so-called type II cell actually is ontogenetically the more primitive cell. For this reason, and because the innervation of the type II cell makes an easier starting point, we shall begin by discussing its innervation.

The hair cell of type II is innervated by at



Figure 9.—Light photomicrograph of a section through one-half of a crista ampullaris of guinea pig, showing fibers of thicker caliber going to nerve calyces and thinner fibers forming a basal network in lower part of sensory epithelium.

least two different kinds of nerve endings. One of these, known as type 1 ending, is sparsely granulated and forms a synaptic contact with the hair cell plasma membrane. The synaptic region often shows a small indentation into the hair cell and in some instances the nerve ending may be partially enclosed by the sensory cell. The synaptic indentation varies in form and size as described by Engström (1961). Sometimes it is smooth and rounded (as in fig. 14); sometimes it is oblique, with a few vesicles inside the sensory cells. In other cases it is very similar to what Smith and Sjöstrand have called a "synaptic bar," but as far as we have found, there is always an invagination or indentation of the sensory cell plasma mem-

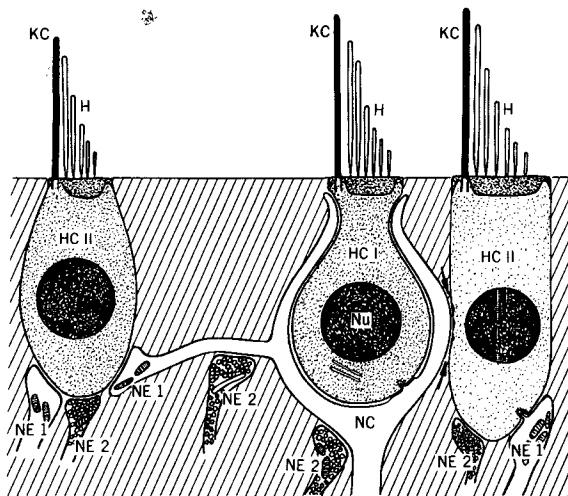


Figure 10.—Schematic drawing of vestibular sensory cells of type I (HC I) and type II (HC II), showing complexity of neural interconnections which may be found in vestibular sensory epithelia. Hair cells of both types may have contact with the same nerve calyx (NC). A side branch from a nerve calyx may course for some distance to make contact with a type II cell. Various ways in which both type 1 (NE 1) and type 2 (NE 2) nerve endings may terminate are shown.

brane. In some cases there is a denser region on both sides of the invagination. As just mentioned, there are sometimes vesicles on the same side as the sensory cell; but in other cases, it looks more as if there were short spines or radiating denser structures, about 200 Å in length. Synaptic vesicles are occasionally found in the hair cell close to the invagination. Further studies are necessary to clarify these structures. The general characteristics of these endings are such as to support the belief that they are post-synaptic, which should mean that they have a centripetal conduction. Along one cell surface there are found several endings of this type varying in both size and form. Some have the appearance of boutons, while others are long and slender, making extended contact with the sensory cells, so extensive, in some cases, that they resemble a half of a nerve calyx of the type described below for the type II cells.

The other kind of nerve ending, called type 2, is less numerous. They are often bouton-

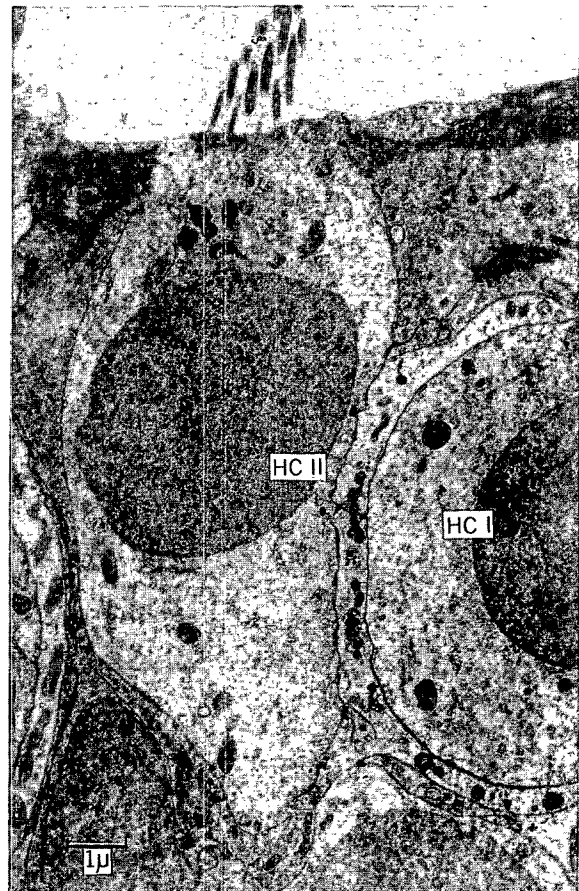


Figure 11.—Electron micrograph, showing how both type I (HC I) and type II (HC II) cells may be in direct contact with same nerve calyx.

shaped and very densely granulated in the guinea pig (fig. 14). For the squirrel monkey these granulated endings are also present but less prominent. They differ considerably in structure from the sparsely granulated type 1 ending. The granulated endings are in direct contact with the sensory cell surface. The contact area often shows a distinct thickening of the plasma membrane of both sensory cell and nerve endings. There is clear evidence that granulated nerve endings of this type can also contact nerve fibers belonging to type 1, forming synaptic thickenings with them. Exactly the same kind of granulated ending is found in contact with the nerve calyces of vestibular sensory cells of type 1. These fibers evidently originate from



Figure 12.—Higher magnification, showing detail, of a part of preceding figure (fig. 11). Nuclei (Nu) of both sensory cells may be seen with their double membranes. Type II hair cell (left) shows at its lower end a thickened region of contact with the calyx. See also following figure.

fibers of very small caliber which form a rich plexus in the lower half of the sensory epithelium.

The vestibular sensory cell of type I is almost completely surrounded by a sparsely granulated nerve calyx (figs. 3, 15, 16), the two being separated by a distance of 250–300 Å. It has been supposed that this distance was uniform at all points except at the neck of the hair cell, making plausible the assumption that only the neck region should be regarded as the area of synaptic contact. This assumption received support from the fact that the nerve calyx in the neck region contains vesicles similar to synaptic vesicles. Our studies have clearly shown, however,

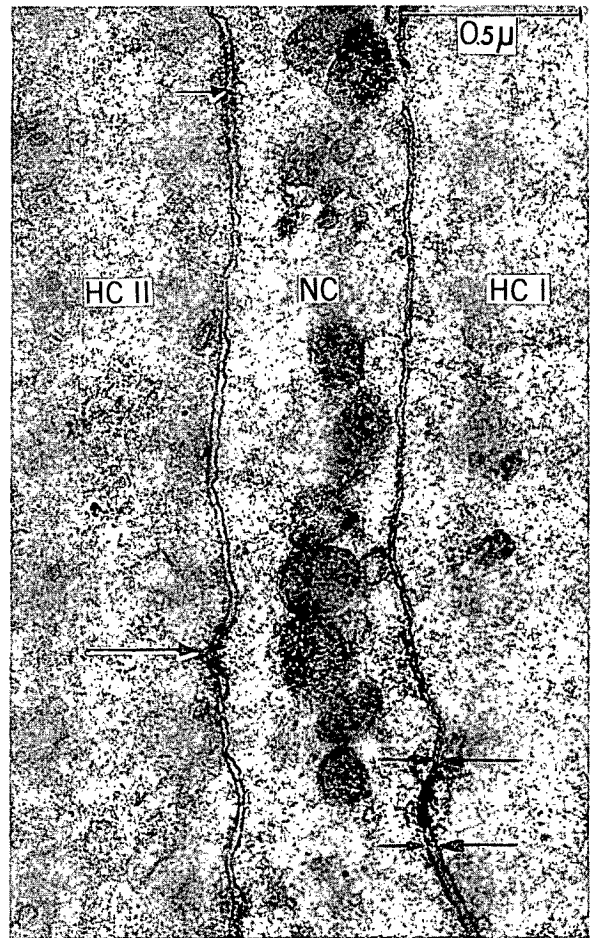


Figure 13.—Still higher magnification of detail from preceding figure (fig. 12), showing how hair cells of both type I (HC I) and type II (HC II) may border same nerve calyx (NC). Black arrows indicate regions where space between calyx and sensory cells is reduced. Black and white arrows on left indicate regions which have been interpreted as synaptic areas.

that the distance between sensory cell and nerve calyx in the guinea pig is *not*, in fact, constant all the way around the cell. Rather, there are distinct areas where the space between calyx and cell is reduced. In the center of these areas an invagination can often be clearly discerned (fig. 16), and occasionally a thin invagination similar to “synaptic bar” can be found. Small vesicles with a diameter of 200–400 Å are often found inside the thin regions and especially in contact with the invaginations. The invagina-



Figure 14.—*Electron micrograph, type II sensory cell (HC II) with nerve endings of two varieties. A sparsely granulated ending (NE 1) forms a synaptic invagination into hair cell. A richly granulated ending (NE 2) forms a synaptic contact (arrows) with hair cell.*

tions are sometimes rather large and a densely granulated nerve ending of type 2 can often be found outside the nerve calyx in the same region. One or more of such densely granulated endings are almost always found outside the nerve calyx, as previously described by Engström (1958, 1961).

In our earlier publications we assumed that sensory cells of type I and II were innervated from different types of nerve fibers. The present study has clearly shown that the outer surface of a nerve calyx often makes contact with a sensory cell of type II, distinct osmiophilic "synaptic regions" being seen along the contact surface. On several occasions we have seen that a nerve calyx can send a branch to a nearby hair cell of type II (fig. 10). Such collateral branches, as we have seen them, show a sparsely granulated terminal (i.e., type 1 ending), and, in the course of such a branch, a densely granulated, type 2 ending is often seen.

Finally, another interesting feature seen

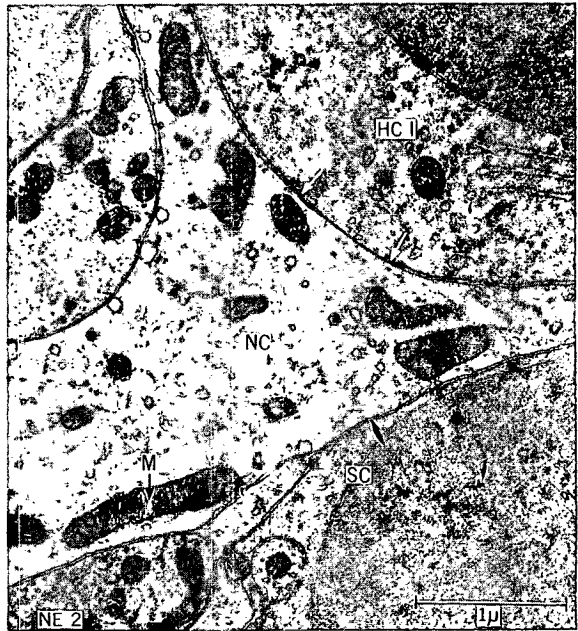


Figure 15.—*Electron micrograph, guinea pig, showing base of a type I sensory cell (HC I), surrounded by a nerve calyx (NC), containing mitochondria (M). White arrows indicate a thin, presumably synaptic, area. Such an area often shows an invagination in its center. A richly granulated nerve ending (NE 2) is seen at lower left, in contact with outer surface of calyx. A supporting cell (SC) is seen at lower right.*

both in the maculae and in the cristae is the occurrence of calyces which enclose two, three, four, or even five sensory cells (fig. 6). These are often found in the maculae of the squirrel monkey. The lack of space often leads to oblique positioning of the enclosed sensory cells and, oftentimes, to distortion of the necks of these.

In the course of a study of large numbers of specimens such as this, in which the inner ear sensory epithelia are regularly dissected free and examined under low magnification as well as by more detailed methods, many features are observed which are of considerable interest, but which do not as yet constitute a systematic body of information. Several of these are briefly discussed in the following paragraphs and some are illustrated in figures 17 to 30. Some of the ob-



Figure 16.—*Electron micrograph of guinea pig showing base of a type I sensory cell with its nerve calyx. Arrows indicate two (synaptic) invaginations. Within these folds, space between calyx and sensory cell is noticeably diminished as compared with rest of calyx-hair cell border.*

servations point up the need for additional systematic study.

One of the most interesting problems in need of elucidation is that of vascular supply to the vestibular sensory areas. The constant activity of the cristae and maculae requires a rich blood supply. As can be seen in several of the micrographs, an abundant subepithelial vascular network is found under the vestibular sensory epithelia. An extensive systematic study of the circulatory pattern of the entire labyrinth is one of the projects now going on in our laboratory.

Previous studies of vestibular physiology have discussed the form and size of the statoconial membrane to a very limited extent, which is amazing, considering the regularity of arrangement of the statoconia in many species, and the significance this character-



Figure 17.—*Electron micrograph showing a myelinated nerve fiber (beginning in lower left corner) as it loses its myelin and then divides into three branches which run, unmyelinated, for a considerable distance (500  $\mu$ ) before entering the epithelium. The region of the termination of the sheath shows the characteristic infolding of myelin and the increase in mitochondrial concentration in the Schwann cell cytoplasm.*

istic structure must have for macular function. In particular, the statoconia of the macula sacculi show a very orderly pattern which is faithfully repeated from one animal and one species to another (fig. 31). The statoconia at the edges form a thin layer, while, along a curved central line, they are heaped into the appearance of a "snowdrift" which is several times thicker than at the edges. A similar variation in thickness is seen also in the macula utriculi. In each case, the line of heaped statoconia corresponds to a central, curving line on the macula which marks its division into two large areas of oppositely polarized hair cells, implying a clearly functional significance of the statoconial pattern. This view is strengthened by evidence that the statoconia form a coherent mass rather than being free to move individually without relation to each other. Actually, it is usually possible to lift



Figure 18.—Ampulla of the semicircular canal of a guinea pig showing the canal widening slightly at the entrance into the ampulla. The blood vessels form a rich network in the crest of the ampulla among the nerve fibers and, as seen here, along the wall.



Figure 20.—Phase contrast micrograph of section of crista ampullaris of squirrel monkey. The difference in form from that of the guinea pig is apparent when figures 2 and 3 are compared. Note that the epithelium of the crest is thinner than that of the sides. Correspondingly, the density of sensory cells is higher along the lower margin of the sides than at the crest.

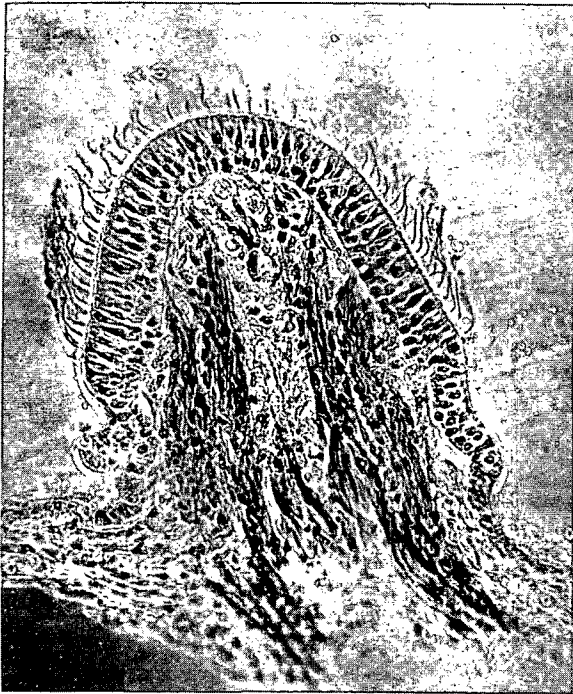


Figure 19.—Phase contrast micrograph of section of crista ampullaris of guinea pig from acrylate-embedded specimen. The long tufts of hairs from the sensory cells can be seen at the surface. Note that the innervation of the sides of the crista is at least as rich, and possibly more so, than that of the crest. Likewise, the sides as well as the crest are densely packed with sensory cells.



Figure 21.—View of exposed labyrinth of guinea pig. The white areas show the macula of the utricle (MU) and of the saccule (MS). The utricle (U) is seen as a dark cylinder with pigmented cells in the wall. At the extreme left is a part of the ampulla of one of the semicircular canals. The fine, dark fibers seen on the macula utriculi are nerve fibers.



off the statoconial layer in a single piece. This implies, in turn, movement as a single mass, meaning that the cells, being of opposite polarity on opposite sides of the central line, must be subject to stimulation along different vectors for any given direction of movement. While this consideration apparently suggests an increasing complexity in

correlation with function, it also offers more degrees of freedom in accounting for the subtleties of behavioral reactions to vestibular stimulation. It also suggests possible patterns to be sought in the projection of the macula on brain-stem nuclei, and a basis for searching out neural coding patterns of vestibular input to central pathways.

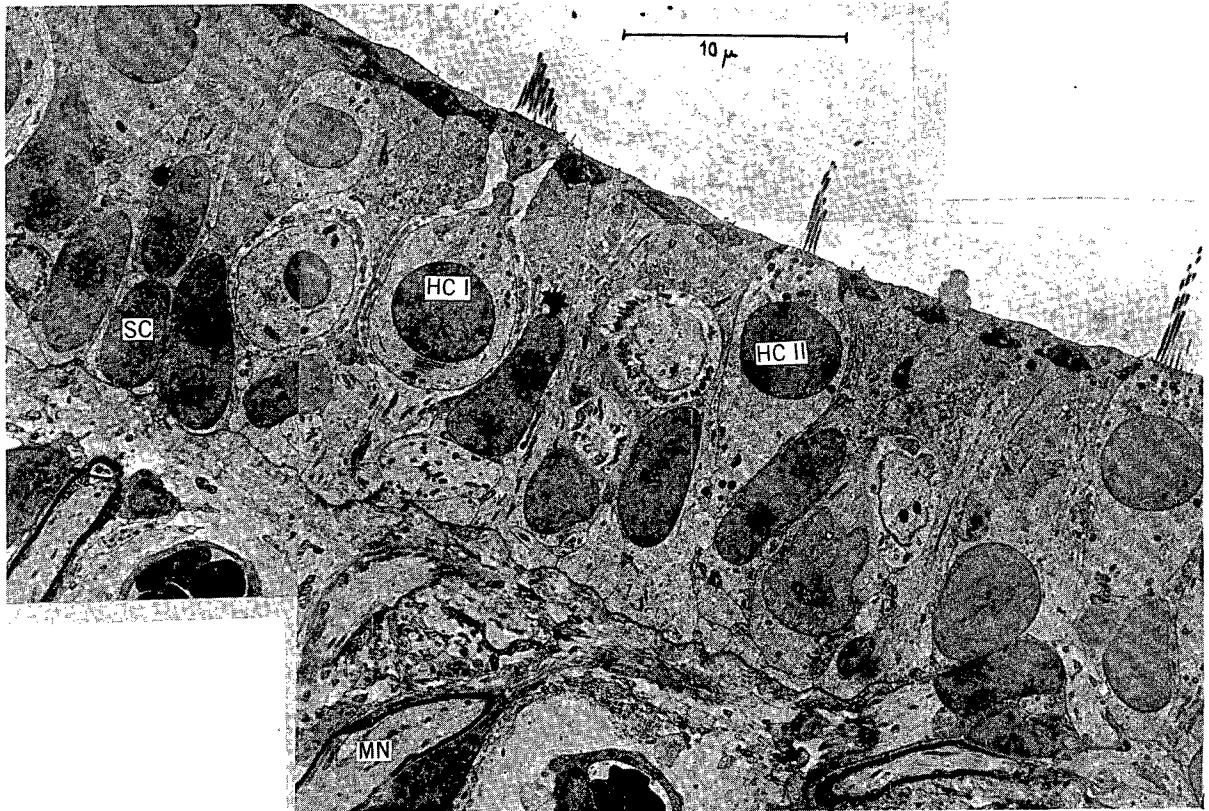


Figure 22.—*Electron photomicrograph of an extensive area of sensory epithelium from the macula utriculi of the guinea pig. Several sensory cells of type I (HC I) and several of type II (HC II) can be seen. Note the difference between sensory cell and supporting cell (SC) nuclei. At the surface of the sensory cells, the sensory hairs show the typical graduation of size, in this case from shortest hairs on the right to longest on the left. Myelinated nerve fibers (MN) can be seen entering the base of the epithelium, losing the myelin sheath just before passing through the basement membrane. Within the epithelium, the now unmyelinated fibers pass to their terminations among the sensory cells.*

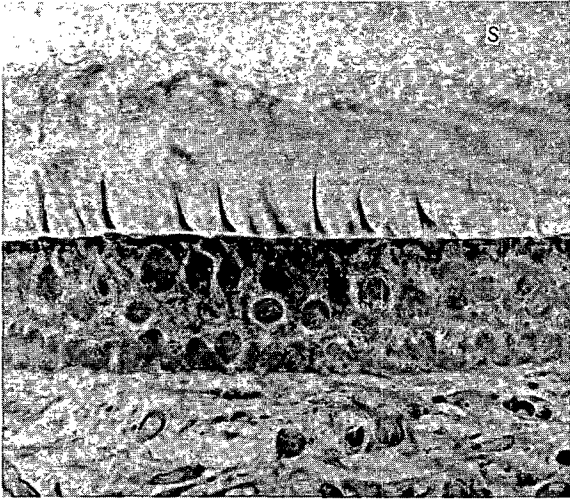


Figure 23.—Section of macula utricle of squirrel monkey showing the hair tufts at the surface. The orderly progression of length of the hairs from low to high on each cell can be seen clearly. The longest hairs are always found close to the kinocilia, which, over large areas of the macula, are oriented in the same direction, denoting a structural polarization of the cell surface. The light colored mass (S) above the surface is the layer of statoconia, formed by hexagonal crystals of calcite whose specific gravity is about 2.74.

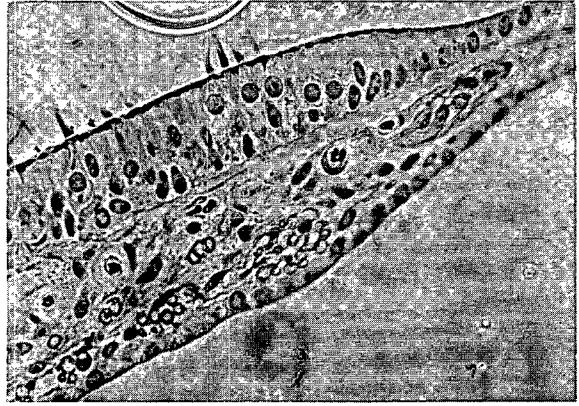


Figure 24.—Section of border region macula utricle of guinea pig showing the transition between sensory epithelium and columnar epithelium which rapidly changes to cuboidal. Below the epithelium can be seen the connective tissue layer with its blood vessels and nerve fibers.

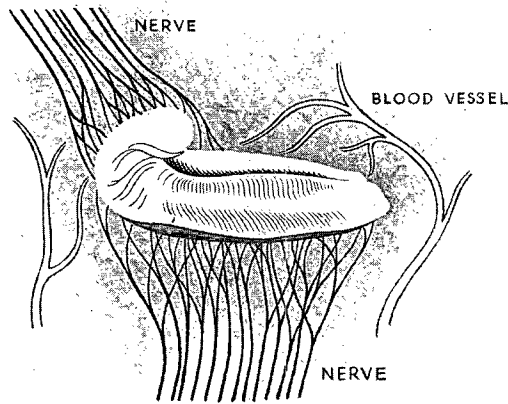


Figure 25.—Schematic drawing of guinea pig macula utricle showing the separate innervation of its two portions, one branch from the upper and the other from the lower division of the vestibular nerve. The macula is also supplied by a third bundle of thin caliber nerve fibers (not shown here) which presumably belong to the efferent system.

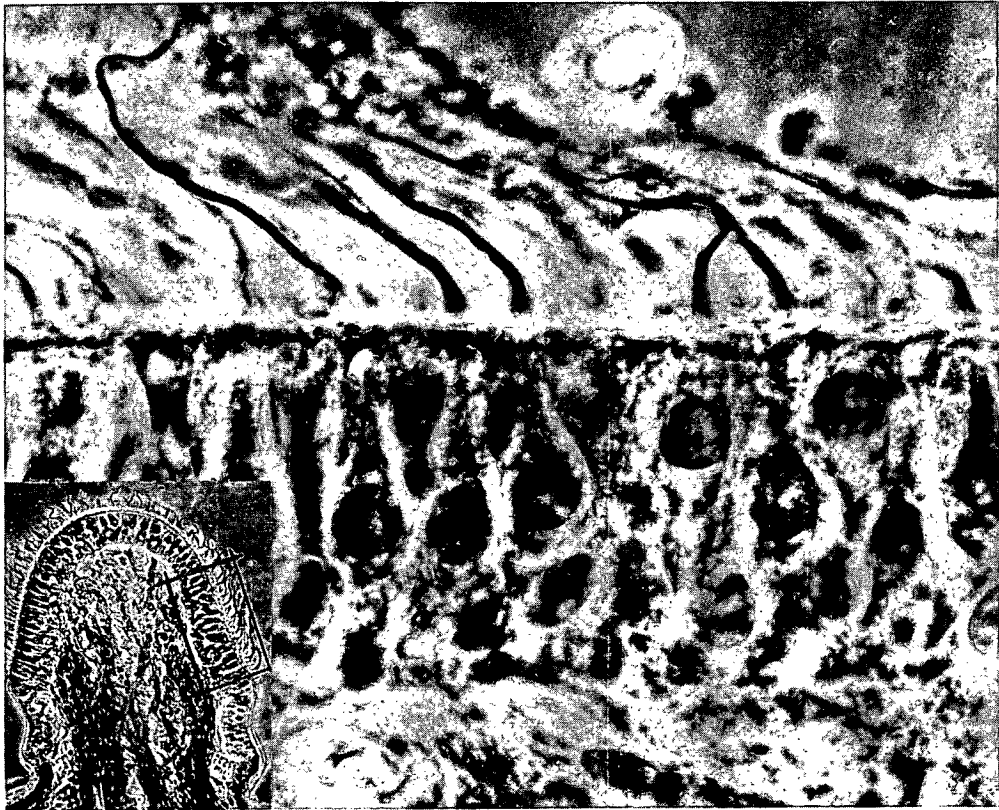


Figure 26.—Crista ampullaris of guinea pig, phase contrast photomicrograph showing sensory hair tufts from cells on the side of the crista (main figure). Inset shows lower magnification of whole section of crista with portion shown in main figure outlined.

#### REFERENCES

- BAIRATI, A.: Recentes connaissances sur la structure submicroscopique des organes du vestibule. *Acta Oto-Laryngol. Suppl.* 163, 1961, p. 9.
- DIJKGRAAF, S.: The Functioning and Significance of the Lateral Line Organs. *Biol. Rev.*, 38, 1963, pp. 51-105.
- DILLY, P. N.: Studies on the Receptors in the Cerebral Vesicle of the Ascidian Tadpole: 1. The Otolith. *Quart. J. Microscopical Science*, 103, Part 3, 1962, pp. 393-399.
- ENGSTRÖM, H.; AND WERSÄLL, J.: The Ultrastructural Organization of the Organ of Corti and of the Vestibular Sensory Epithelia. *Exp. Cell Res., Suppl.* 5, 1958, p. 460.
- ENGSTRÖM, H.: On the Double Innervation of the Sensory Epithelia of the Inner Ear. *Acta Oto-Laryngol.*, 49, 1958, p. 109.
- ENGSTRÖM, H.: The Innervation of the Vestibular Sensory Cells. *Acta Oto-Laryngol., Suppl.* 163, 1961, p. 30.
- ENGSTRÖM, H.; ADES, H. W.; AND HAWKINS, J. E., JR.: Structure and Functions of the Sensory Hairs of the Inner Ear. *J. Acoust. Soc. Am.*, 34, 1962, pp. 1356-1362.
- FLOCK, Å.: Structure of the Macula Utriculi With Special Reference to Directional Interplay of Sensory Responses as Revealed by Morphological Polarization. *J. Cell Biol.*, 22, 1964, pp. 413-431.
- FLOCK, Å.: Electron Microscopic and Electrophysiological Studies on the Lateral Line Canal Organ. *Acta Oto-Laryngol., Suppl.* 199, 1965, pp. 1-90.
- FLOCK, Å.; KIMURA, R.; LUNDQUIST, P. G.; AND WERSÄLL, J.: Morphological Basis of Directional Sensitivity of the Outer Hair Cells in the Organ of Corti. *J. Acoust. Soc. Am.*, 34, 1962, p. 1351.
- FLOCK, Å.; AND WERSÄLL, J.: A Study of the Orientation of the Sensory Hairs of the Receptor Cells of the Lateral Line Organ of Fish. *J. Cell Biol.*, 15, 1962, pp. 19-27.
- SMITH, C. A.: Microscopic Structure of the Utricle. *Ann. Otol.*, 65, 1956, p. 450.
- SMITH, C. A.; AND SJÖSTRAND, F. S.: A Synaptic Structure in the Hair Cells of the Guinea Pig Cochlea. *J. Ultrastruct. Res.*, 5, 1961, pp. 184-192.
- WERSÄLL, J.: Studies on the Structure and Innervation of the Sensory Epithelium of the Cristae Ampullares in the Guinea Pig. *Acta Oto-Laryngol., Suppl.* 126, 1956, pp. 1-185.
- WERSÄLL, J.: Vestibular Receptor Cells in Fish and Mammals. *Acta Oto-Laryngol., Suppl.* 163, 1961, p. 25.



Figure 27.—*Electron micrograph, guinea pig, showing two areas of presumptively synaptic significance. On the wall of a type I sensory cell (HC 1) where it borders on a nerve calyx is seen a thinned region featuring a fold involving the walls of both calyx and hair cell. A richly granulated nerve ending (NE 2), probably of efferent nature, which also borders the nerve calyx shows a thickened "synaptic" area (Sy).*



Figure 28.—*Electron micrograph showing a myelinated nerve fiber as it loses its sheath (MN) a short distance below the basement membrane of the epithelium. The myelin sheath terminates by folding inward around the nerve fiber, enclosing mitochondria as it does so. Note the slight expansion of the nerve fiber as it emerges from the sheath.*



Figure 29.—Electron micrograph; myelinated nerve fiber showing region of termination of the sheath. Several large mitochondria (M) are regularly found in this region, forming, in conjunction with the folding of the myelin sheath, a kind of terminal collar. At the lower left corner a Schmidt-Lanterman incisure can be seen.



Figure 30.—Electron micrograph showing the complicated infolding of myelin sheath terminations as often seen just below the macular epithelium.

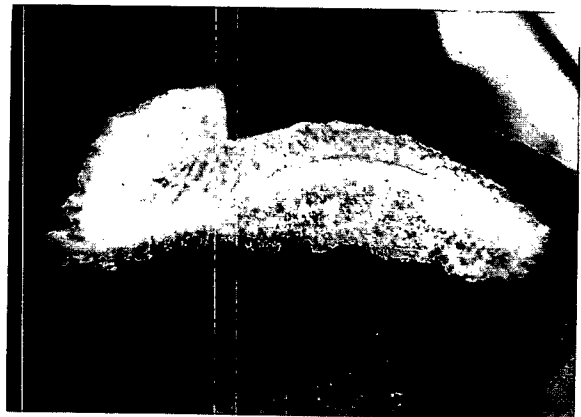


Figure 31.—Surface view of macula sacculi of guinea pig. White surface is composed of large numbers of statoconia showing typical pattern of their distribution, most prominent feature of which is "snowdrift" of crystals heaped along a curving line. This "snowdrift" line is found regularly in this and other species which have been studied. It has also been found that this line marks a corresponding line in sensory epithelium, on two sides of which the structural polarization of cells is reversed.

## DISCUSSION

MAYNE: Apparently the sensory cells themselves are capable of a high degree of organization. The question arises as to the nature of this organization. Could it be that adaptation takes place at the low level of these cells rather than in higher neural centers? Could it be that the organization is to the end of providing optimum coding of sensory signals to provide the highest possible rate of information over existing channels?

ENGSTRÖM: The problem of cellular and neuronal interaction in the inner ear is one of the most interesting problems in our present work. We know from the cochlea that there is a very geometrical or mathematical arrangement of the cells and that one nerve fiber innervates several sensory cells. It is therefore probable that the fixed system of sensory cells corresponds to a fixed system of neuronal interplay. Some of these problems have been discussed by Flock and Wersäll and by Dijkgraaf for the side line organ. We have to elucidate cellular pattern and neuronal interconnection in the vestibular apparatus to fully understand how economical the information-transmission is. Nature has solved very many problems in a very elegant way and still using very minute dimensions.

QUESTION: Is there any difference in the structure or innervation of sensory cells in a normal human subject and an albino?

ENGSTRÖM: I have never looked with the electron microscope at the inner ear of an albino animal except guinea pigs and I have never seen any literature dealing with that problem although I know that it is a very interesting one. We have on several occasions talked about looking upon the sequence of sensory-cell—nerve-termination loss in some of the albino animals but it has not yet been done.

MCDONALD: I would like to comment on Dr. Engström's micrograph and ask about some of his technical procedure, particularly whether glutinaldehyde fixation was used for some of the nerve fibers we saw.

The second thing I would like to ask or comment on is that perhaps we are underrating the stereocilia in all this. It seems like they are more limited to the ear whereas kinocilia are found in many places, as mentioned, and in addition in the tail of spermatozoa. It seems to me that maybe some of this bidirectional phenomena of Ewald might be explained in the stereocilia being of varied length so that from the equilibrium position the cupula may be pushed more easily in one direction than in the opposite direction.

Third, I might comment on the question of various types cells. The earlier forms of fish particularly

only have the type II cell, and this may be an evolutionary phenomena of a more specialized structure developing rather than one of nature planning the engineering needs beforehand for the particular types of cells we see.

ENGSTRÖM: About the method we are using. We work with 1½% veronal buffered osmic acid as fixative and for high resolution with epoxy resin embedding. For light microscopy we often use acrylate embedding instead of celloidin. Our specimens are stained with lead hydroxide or uranyl-acetate. Most of it is standard technique now universally applied.

Regarding the importance of the cilia at the cellular surface, I fully agree that the stereocilia presumably are very important from a functional point of view. We only wanted to point out that vestibular, and also cochlear sensory cells are provided with two different systems of interacting cilia. In all older literature only the stereocilia have been discussed, and as it is now known that modified kinocilia play an important role in olfaction, in vision, and trigger activity in primitive animals, etc., we have to consider the function of the kinocilia on inner ear sensory cells also.

The arrangement of the stereocilia on the cochlear hair cells indicates a functional difference between the base of the cochlea and the top. The angle inside the W-formation of the approximately 120 hairs is at the base around 120° and at the top only 60°.

As to the function of the kinocilium it could be that it only forms a cytocentrum from which the stereocilia are formed. It is known that basal bodies can act as centers for new formation of regularly arranged organelles as can be seen in many primitive ciliated animals. It took around 60 years for people to understand the important function of the modified kinocilia of the eye. It is now around 60 years since the kinocilia of vestibular sensory cells were first observed so it is high time that we solve the problem of the functional significance of the kinocilia and the stereocilia in the inner ear.

SPOENDLIN: The basal body of the kinocilium doesn't seem to be necessary for the receptor action of the sensory cells. There are many cochlear hair cells without a kinociliar basal body, as for instance in the cat. The basal body is probably very important in the evolution of the sensory cells. Once the cell is fully differentiated it is, however, not any more essential for the sensory cell function.

As far as the polarization of the macular sensory cells is concerned, it seems to me that the findings of Flock on the macula of fish correspond fairly well with our findings on the maculae of guinea pigs and monkeys.

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# An Experimental Approach to the Dynamics of the Vestibular Mechanisms

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A problem of potential significance to manned space vehicle operations is that of adverse effects associated with the exposure of on-board personnel to stress level linear and angular accelerations of both static and dynamic form. These accelerations range from high-level linear G loads during lift-off and re-entry; sustained near threshold angular motions arising from vehicle drift or routine movements of the crew members; the weightless state itself; and Coriolis accelerations produced by head, torso, and limb motions when the vehicle or station is rotated so as to create a gravitational-like force field. The biological effects of this environment may be manifested by the appearance of disorientation, of interference with visual monitoring processes, of inability to perform operational tasks efficiently, or of disabling motion sickness. More specifically, the crewmen may be unable to read critical instrument displays because of nystagmus-induced blurring of his visual field or he may be rendered ineffective by severe nausea or even vomiting. Any of these factors can limit the operational efficiency of the human component of the spacecraft system, and reflect on the degree of mission success.

Man's keen ability to sense and control his relationship to a force environment and his motions within that environment originate in the inner ear anatomy of the vestibular system. The semicircular canals serve as the primary biological transducer for the detection of angular motions while the oto-

lith mechanisms are considered to be the equivalent sensor for linear acceleration stimuli. As with the analysis of the over-all performance of a physical system, the prediction of the over-all response of the vestibular system to the force parameters of the space environment must be initiated by studying the nature of the input sensing elements. Thus a knowledge of the individual performance characteristics of the canal and otolith mechanisms to their driving accelerations serves as the starting point for system analysis with the collection of quantified response data under precisely controlled stimuli conditions being a primary objective.

The development of the vestibular field has been such that a great deal more data are available to describe the response behavior of the semicircular canals than those available to quantify the stimulus response characteristics of the otolith mechanism. As early as 1931, Steinhausen pointed out that the cupula-endolymph system of the individual canals can be considered to act as a simple torsion pendulum with a high degree of viscous damping, and as such, could be described by a simple linear differential equation of second order. With such a mathematical representation of the canals, the response characteristics could be related in analog form to a simple physical system. About 20 years later, Groen and his colleagues experimentally established approximate numerical values for the coefficients of Steinhausen's equation based on subjective



sensations of angular rotation on a rotating chair and a torsion swing. The techniques, as these experimenters pointed out, are limited by the sensitivity of the subjects and their ability to make the required subjective judgments.

With the work of these investigators serving as a primary reference, the authors have developed and are implementing a research program which offers quantified insight into the prediction of vestibular response to the nonphysiological static and dynamic acceleration stimuli of the space environment. The approach is based on the use of sinusoidal angular acceleration stimuli of variable frequency and magnitude which can be objectively quantified in such a form that the overall response of the oculovestibular system can be quantified on either an intra or inter individual basis.

A mathematical formulation of the basic stimulus-response relationships relating steady-state nystagmic eye velocity to periodic angular accelerations based on the system transfer function concept as well as objective experimental data has been described in detail in recent reports. The formulation begins with Groen's representation of Steinhausen's equation of motion for the canals with the substitution of the damping ratio and undamped characteristic angular frequency performance parameters of the control and servomechanism areas for the original coefficients of the differential equation. The basic form of the equation is

$$\ddot{\xi} + 2\zeta\omega_n\dot{\xi} + \omega_n^2\xi = -\alpha(t)$$

$$\xi \equiv \xi(t)$$

where

$\xi, \dot{\xi}, \ddot{\xi}$  = equivalent angular displacement, velocity, and acceleration of the cupula-endolymph system

$\zeta$  = equivalent damping ratio of the cupula-endolymph system

$\omega_n$  = undamped characteristic angular frequency of the cupula-endolymph system

$\alpha(t)$  = angular acceleration of the skull representing the driving torque stimulus

By making  $\alpha(t)$  of sinusoidal form and noting the time lag characteristics of a given response measure, say the oculogyral illusion, body sensation, or nystagmus, behind the driving stimulus, a highly attractive experimental technique may be developed. In essence, the magnitude and phase relationships of the resultant data define the frequency response characteristics of the system.

The device used to generate the sinusoidal angular motion stimuli for our early studies was the Human Disorientation Device (HDD) shown in figure 1. This device, having two degrees of rotational freedom, permits simultaneous and independent rotation of the subject about Earth-referenced vertical and horizontal axes. The cab and yoke of the device revolve about the vertical axis; the cab alone revolves about the horizontal axis. The HDD is designed for operation with the subject's head centered at the intersection of the two axes of rotation. The head can be centered and fixed with respect to the device by means of an adjustable chair and head-restraining device. The sinusoidal angular acceleration stimuli to the semicircular canals are then generated by applying the output of a low frequency sine wave generator as a command signal to the HDD drive system.

The advantages resulting from the use of such periodic stimuli become most pro-



Figure 1.—Human disorientation device (HDD).

nounced in terms of collecting quantified objective data when they are used in conjunction with a nystagmus transition technique that has been developed to describe the time-characteristics of the response.

In figure 2, there are presented recordings of an ocular-nystagmus response to a high-level sinusoidal angular acceleration stimulus. Each tracing represents the observed response to a single stimulus frequency. The stimulus frequencies shown range from 0.02 to 0.20 cycle per second. The response curves reflect alterations in corneo-retinal potentials recorded from electrodes placed at the outer canthi of the eyes and represent nystagmic eye movements in the horizontal plane produced by oscillation about the Earth-ver-

tical axis with the subject seated upright. The peak angular acceleration was held constant at 40 degrees per second squared for each test frequency.

It may be noted that the recordings show characteristic transitions in the direction of nystagmus related to changes in direction of the stimulus. Figure 3 presents two such nystagmus transitions in larger scale. When a man is accelerated to the left, his eyes move slowly to the right and return quickly to the left to begin a new slow movement to the right. We define a left transition as one which is followed by such a pattern as shown in the upper tracing. On the other hand, acceleration to the right will result in a slow movement to the left followed by

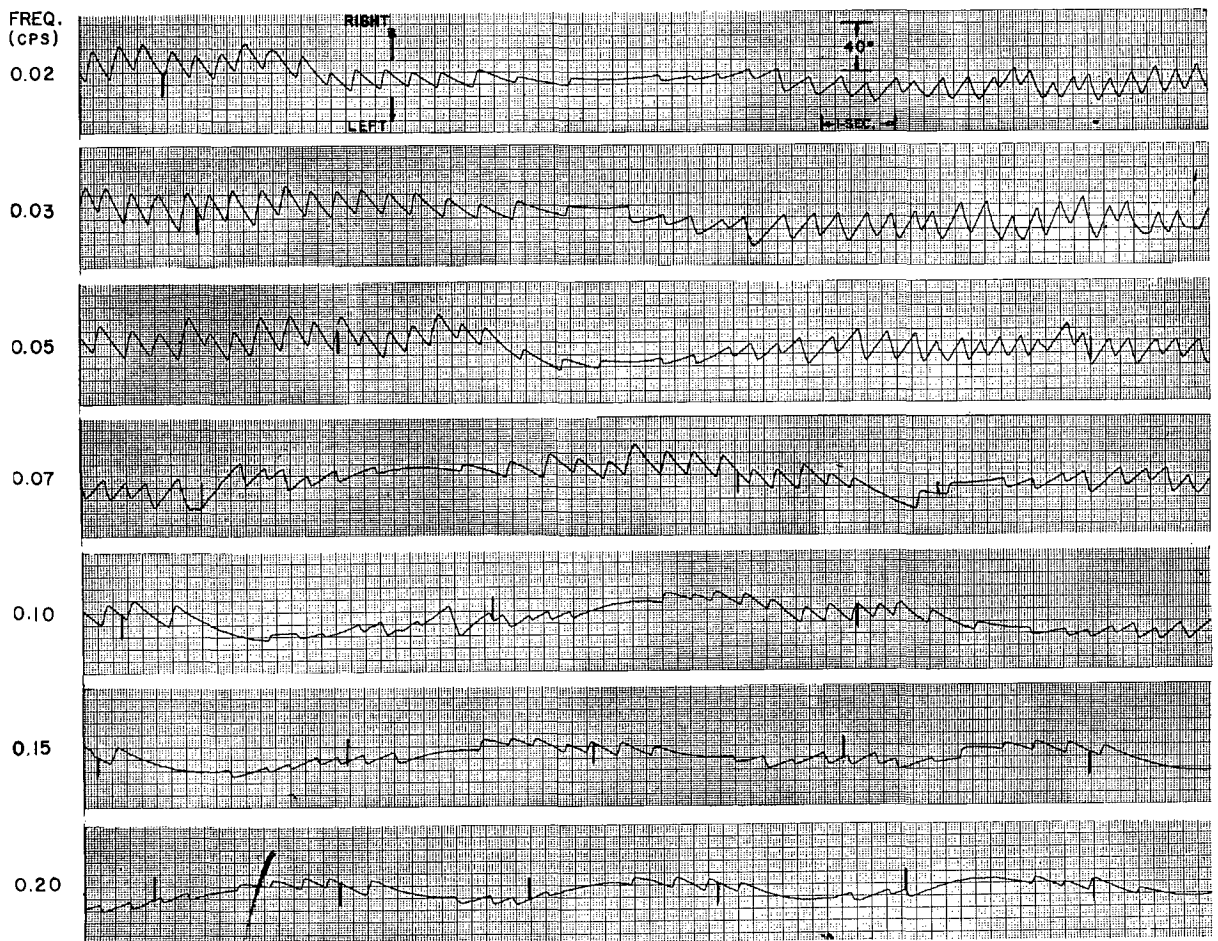


Figure 2.—Recordings of horizontal eye nystagmus arising from rotational stimulation of horizontal semi-circular canals with sinusoidal angular accelerations of variable frequency and fixed magnitude.

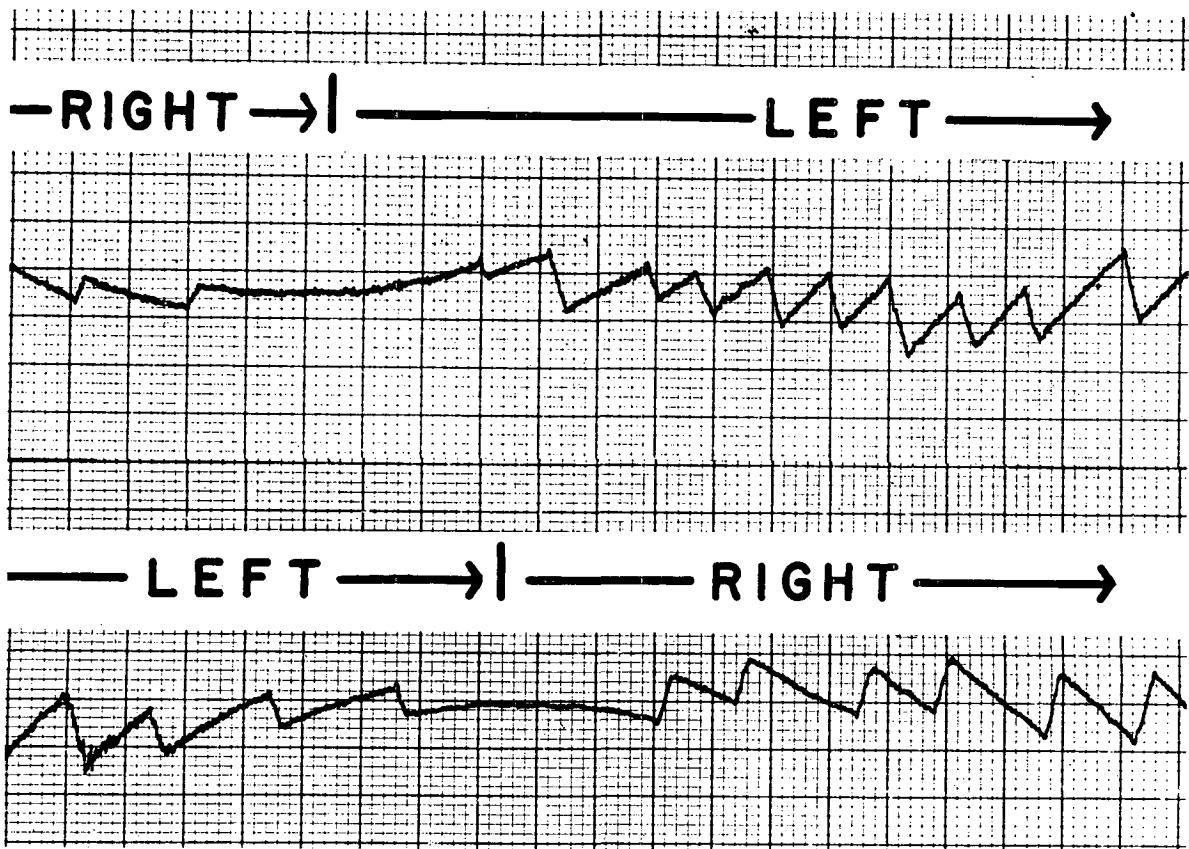


Figure 3.—Typical nystagmus transitions in response to a sinusoidal angular acceleration.  $F = 0.10$  cps;  
 $a_{\text{peak}} = 60$  deg/sec<sup>2</sup>.

quick return to the right. We identify the point at which such a pattern is initiated a right transition as in the lower tracing. It is clear that the slow phase velocity of the eye in a given direction systematically decreases following a change in the direction of acceleration. It finally has zero velocity for an instant, and then reversing, gradually increases in velocity again. As compared to duration or magnitude measures of nystagmic response, the transition technique offers a readily measurable data point, which is not heavily dependent on the exact amplitude of the nystagmic eye motions.

Since the angular acceleration of the skull represents the driving force producing the torque stimulus to the cupula-endolymph system, the phase lag of each nystagmus transition is referenced to the corresponding acceleration transition. This procedure is

illustrated in figure 4. The schematic nystagmus represents an eye which has been responding with slow movements to the right in response to acceleration to the left. It then undergoes a right transition through zero eye velocity in response to an acceleration to the right, producing eye movements in the opposite direction. The phase shift or lag is measured as the distance from the point at which the stimulus acceleration is zero to the transition point for eye motion direction. It can also be estimated as the midpoint between the end of the last pre-transition slow component and the beginning of the first post-transition component.

By using variable frequency angular acceleration stimuli and expressing the phase lag of the nystagmus transition response in electrical degrees, a numerical expression of the frequency characteristics of the overall

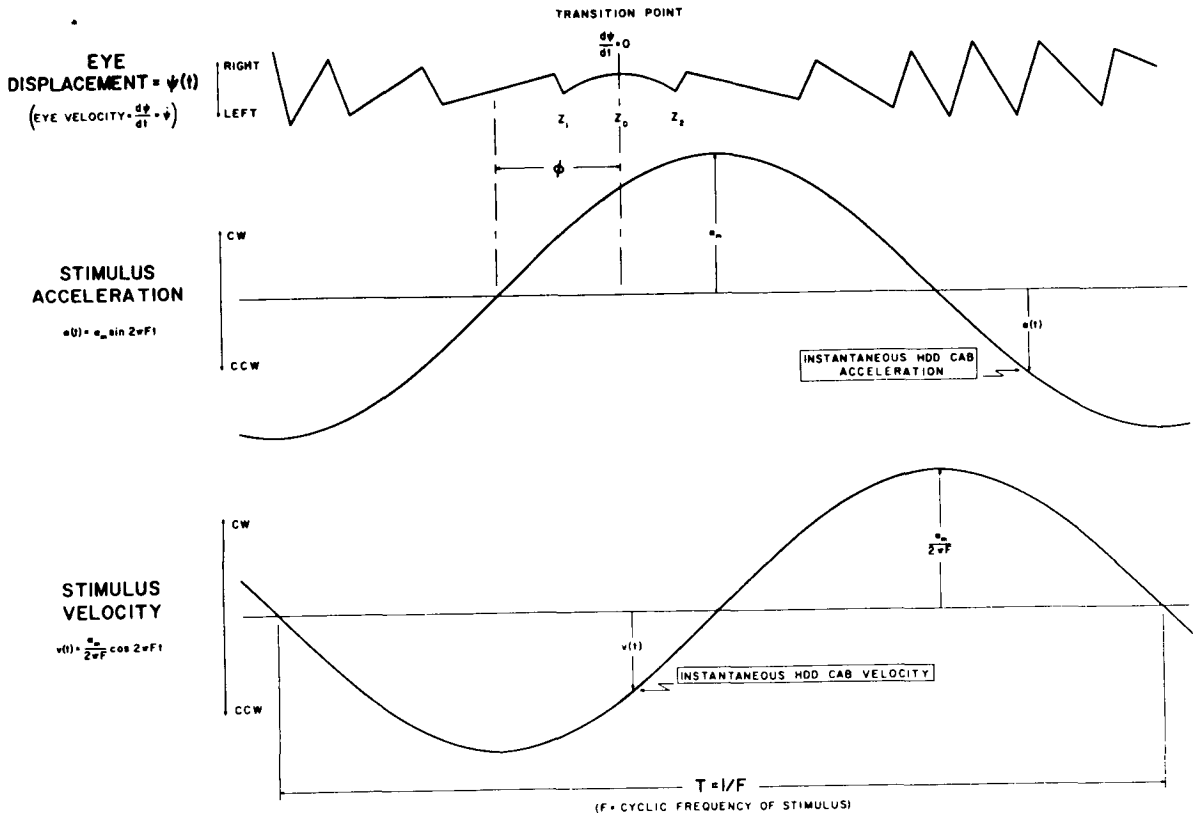


Figure 4.—Relationship of an idealized nystagmus response to sinusoidal stimulus variables.

system results. Specifically, by experimentally determining the frequency which produces a 90 degree phase lag, the undamped characteristic angular frequency  $\omega_n$  can be established; by using a stimulus frequency much lower than  $\omega_n$ , a quantified determination of the ratio  $2\zeta/\omega_n$  (theoretically equivalent to the  $\Pi/\Delta$  constant of conventional cupulometry practices) can be measured, and by combining the above data, a quantified description of  $\zeta$  results. Thus the damping ratio and natural frequency of the over-all oculovestibular mechanism for a specific individual can be quantitatively defined with the periodicity of the stimulus permitting ready replication of data.

Figure 5 presents a demonstration of the application of frequency response analysis to the experimental evaluation of the phase shift angle. Sinusoidal angular accelerations have been applied about the vertical axis of a subject. The resultant nystagmic eye dis-

placements in the horizontal direction are shown for five different cyclic frequencies of the stimulus source. It should be noted that the real-time scale-factor has been normalized by increasing recording speed with frequency so that equal increments on the horizontal axis represent equal increments of electrical phase degrees. The actual direction of the acceleration stimulus is shown in the upper trace and the onset of a new stimulus direction is represented by markers on the nystagmus records. The recordings show the expected directional response, i.e., that acceleration to the right produces a right-beating nystagmus with the slow component to the left and conversely for acceleration to the left.

Such data have been summarized graphically for six subjects in figure 6. In each case, the experimentally determined phase shift is plotted as solid circles. To these points there have been fitted theoretical rep-

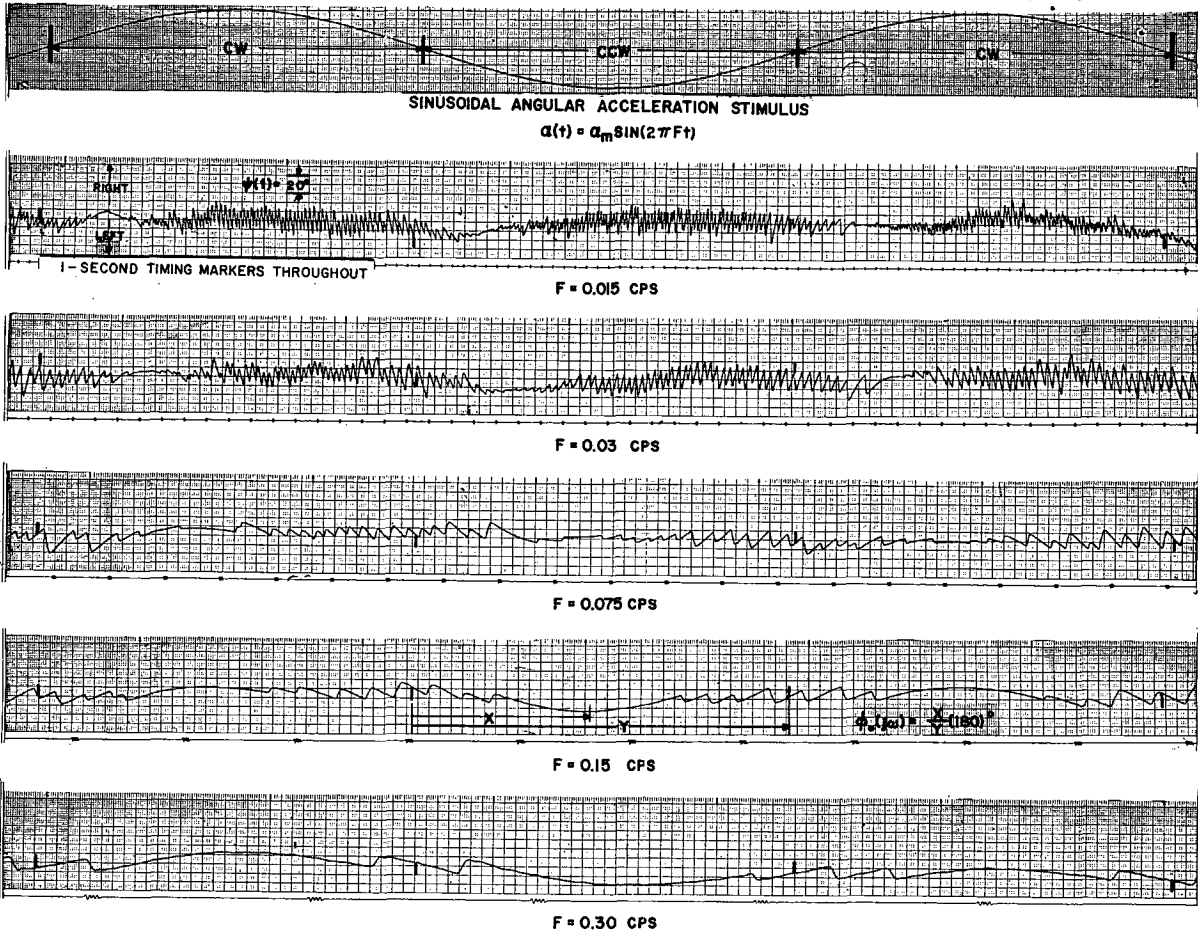


Figure 5.—Steady state horizontal nystagmus responses as produced by sinusoidal angular accelerations of variable frequency applied about vertical axis.

representations of the observed frequency dependence; the curves were based on values of the natural frequency derived graphically as the stimulus frequency at which the phase shift was 90 degrees, and of the damping ratio calculated from the observed phase shift at 0.02 cycle per second. Note that the natural frequency is in the range of 0.2–0.4 cycle per second for three of the six subjects.

The nystagmus transition technique can be applied equally effectively to an evaluation of the linearity of the system. If the ocular nystagmus response were a linear function of the stimulus acceleration, then it would be possible to demonstrate that the waveform of the output response would not be affected by variations in the amplitude of the stimu-

lus. Such variations should be reflected only in the amplitude of the output response, that is, the velocity of the slow component. On the other hand the phase difference between the stimulus acceleration and the nystagmic eye motions should remain constant; that is, there should be no change of phase even when the magnitude of the torque is changed.

The effectiveness of this technique in revealing nonlinearities is demonstrated in figure 7. Test persons were exposed to each of four frequencies of sinusoidal angular acceleration stimuli. At each stimulus frequency they were subjected to several different magnitudes of peak acceleration. It is evident from the trends of the data that little change in phase occurs for the 0.08 and 0.20 cycle per second range of stimulus magni-

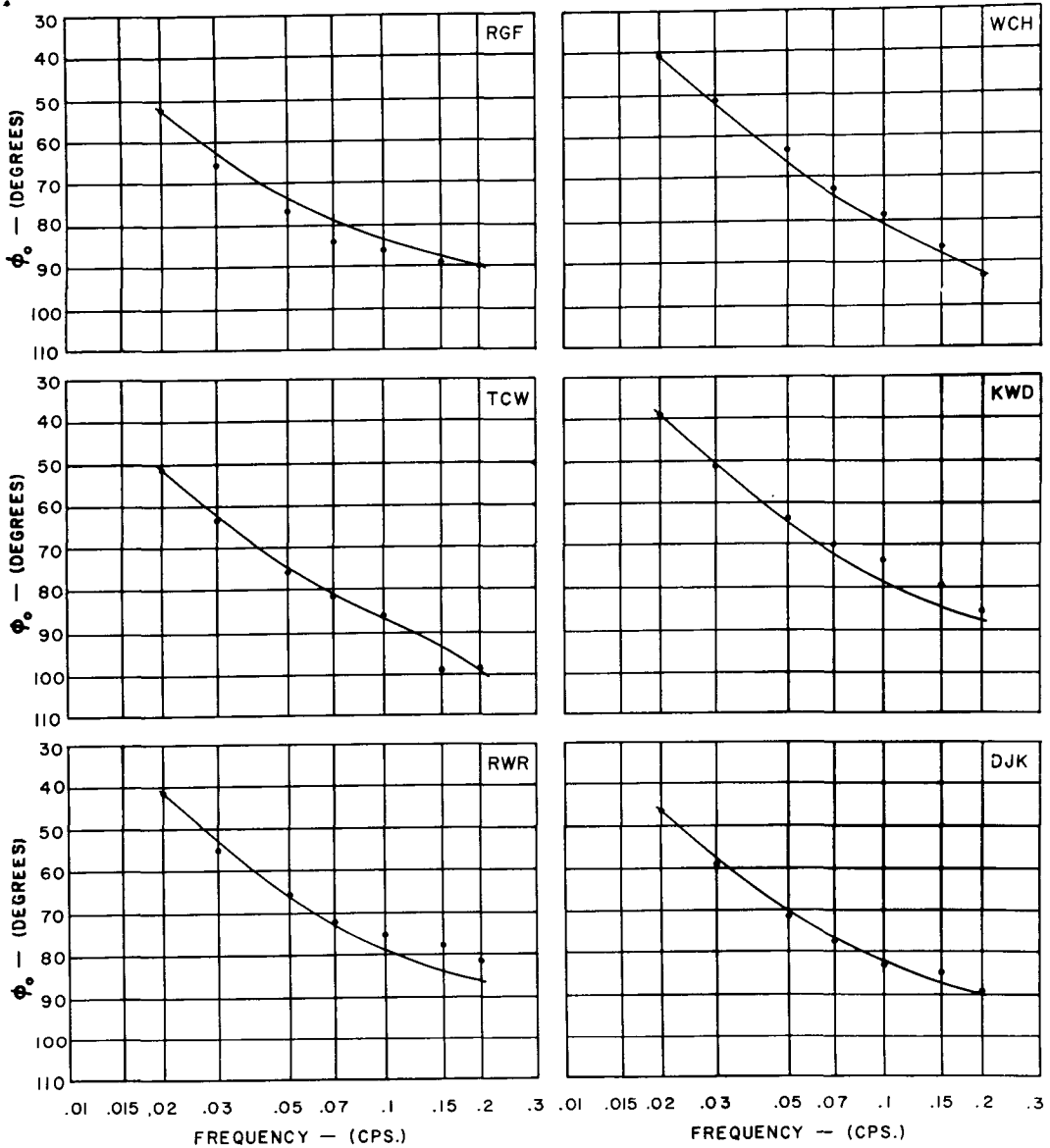


Figure 6.—Actual and theoretical nystagmus phase shift as a function of frequency.

tudes. The constancy of the phase shift indicates that here the cupula-endolymph system is responding in a linear range. As the stimulus frequency is decreased to 0.04 and further to 0.02 cycle per second, the phase shift increases more and more for a given increase in the magnitude of the stimulus; i.e., the slope increases noticeably.

If these same data are replotted in figure 8 in the frequency response form, a rough estimate of the damping present in the sys-

tem can be made by observing the relative slope of the phase curve. A system with little damping has a greater slope; that is, the observed phase shift will increase more rapidly with an increase in the frequency of the stimulus. It can be readily seen that the estimate of system damping would vary as a function of the stimulus magnitude selected by the experimenter.

Data describing the damping/stiffness ratio of the canals are shown in figure 9 as

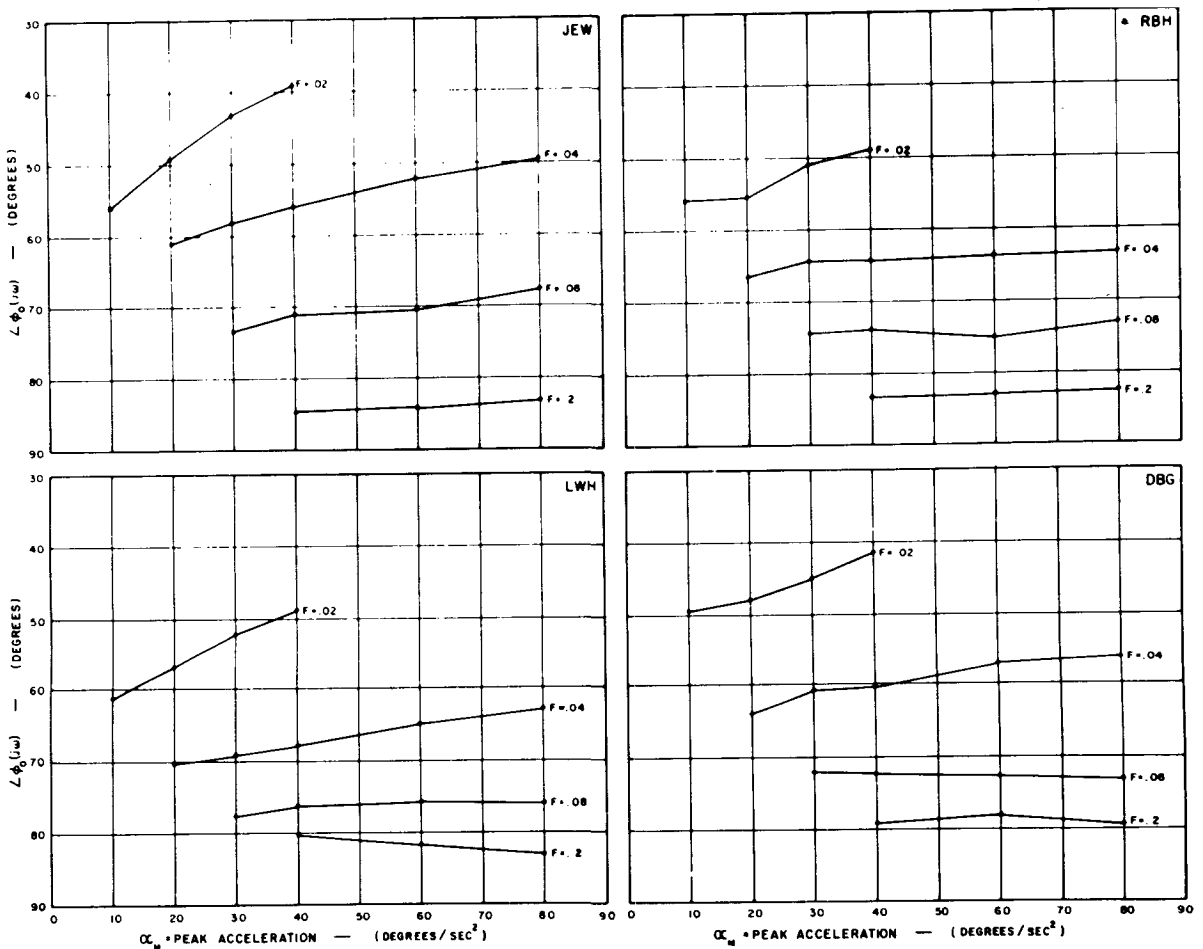


Figure 7.—Nystagmus phase shift data from four subjects plotted versus peak acceleration level of sinusoidal angular acceleration stimulus for various cyclic frequencies.

a function of the magnitude of the acceleration stimulus. The decrease with increase of stimulus strength probably reflects saturation or overloading effects which may arise with single excessive stimuli or as a result of habituation associated with repeated exposure to aerobatics or similar acceleration exposures. We have found also in pilot experiments with individuals confirmation of Aschan's findings that the damping/stiffness ratio based on nystagmus will reflect differences between degree of habituation. If calculated separately for right and left accelerations, it often reveals directional differences between the two ears, where the caloric test shows no difference. High values of  $2\xi/\omega_n$  seem to be associated with sensitivity to

motion sickness. It is also of interest that we have observed high values of natural frequency in subjects resistant to motion sickness.

In effect, these magnitude data offer a quantified approach to the identification of the nonlinearities long associated with the response of the oculovestibular system. In fact, they demonstrate that the deviation of nystagmus magnitude responses to ramp or impulse type stimuli from the idealized logarithmic form are readily covered by Steinhilber's equation of motion by application of nonlinear mathematics. The data in general demonstrate the integrative action of the system as the stimulus frequency is raised and approaches the frequency spec-

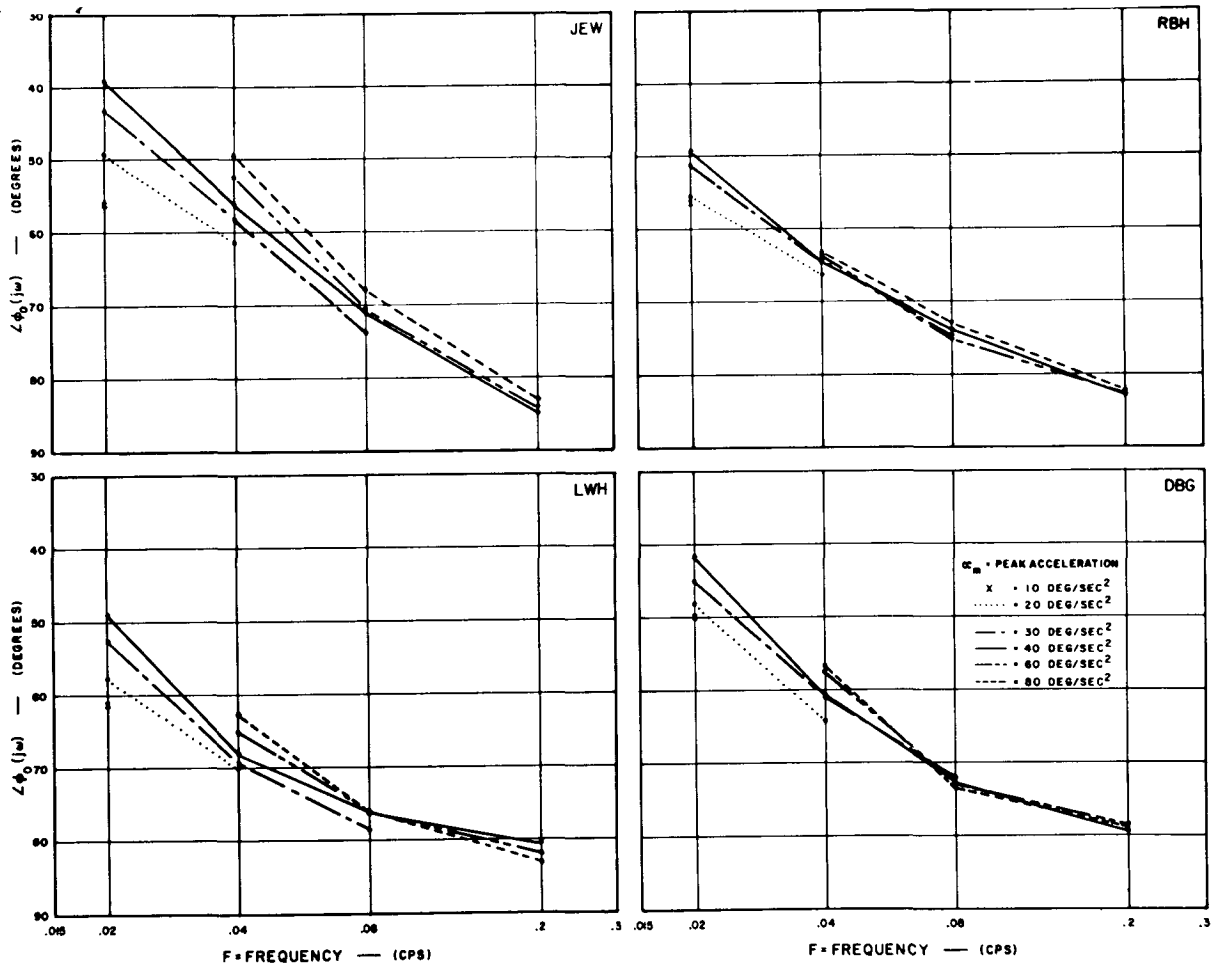


Figure 8.—Nystagmus phase shift data from four subjects plotted versus the cyclic frequency of sinusoidal angular acceleration stimulus for various peak acceleration levels.

trum of normal physiological head motions which is predominately above the natural frequency  $\omega_n$ .

Up to this point, the discussion has been concerned primarily with the development lines of an experimental technique for the determination of the performance characteristics of the semicircular canals in their transduction of angular accelerations and with a brief survey of potential applications. Linear accelerations and the otolith organs are obviously of equal interest. The School has recently placed into operation a new device, the Coriolis Acceleration Platform, shown in figure 10, which can produce the sinusoidal linear accelerations necessary for a frequency response analysis. A 40 foot track extends

through the center of the capsule. On this track is a platform to which various seating devices can be attached. The platform can reach a peak linear velocity of 16 feet per second and a peak linear acceleration of 3 g's with a payload of 500 pounds.

Our initial efforts with this device have been directed toward observing the response to periodic linear acceleration stimuli of sinusoidal form. A preliminary study has been completed in which frequencies of 0.2, 0.4, 0.8 cycle per second were used, all with peak linear accelerations of approximately 0.6 g in the Earth-horizontal direction. The results are of real interest to the vestibular field.

Figure 11 shows DC corneo-retinal poten-



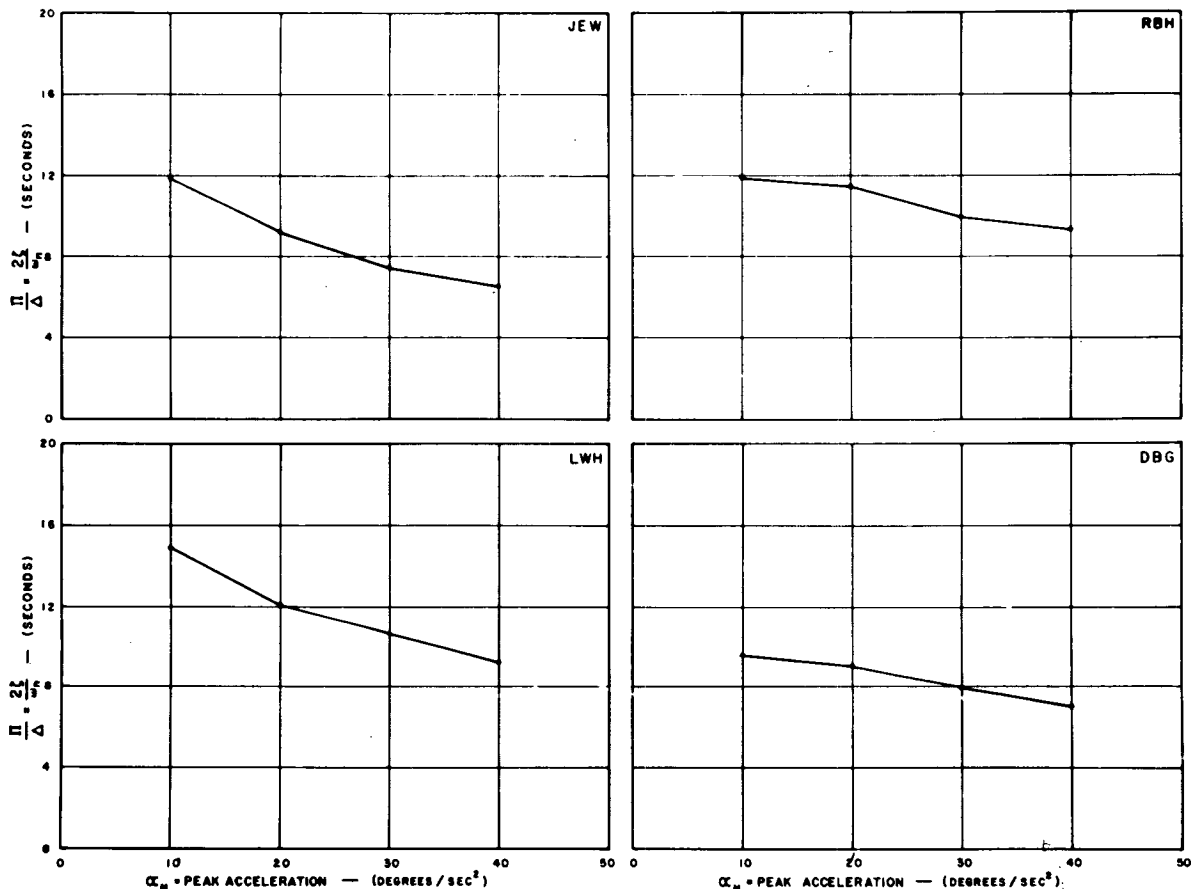


Figure 9.—Calculated values for theoretical ratio of damping to the stiffness of the cupula endolymph mechanism as derived from nystagmus phase shift data produced by a sinusoidal angular acceleration stimulus of cyclic frequency 0.02 cps for various peak acceleration levels.

tial recordings of eye displacements recorded at these test frequencies. The orientation of the subject with respect to the track movement was such that the stimulus was acting in his horizontal plane with his head rigidly

fixed by a custom-molded, plaster head cast. The resulting sinusoidally varying horizontal nystagmus shown in the figure was quite unexpected, as we had instead anticipated, at most, only sinusoidal eye motions without nystagmic beats.

The subject was then reoriented to bring the stimulus acceleration into the frontal plane (fig. 12). Again there resulted a sinusoidal horizontal nystagmus, which, both in this figure and the previous one, has the general appearance of the nystagmus produced by angular acceleration stimulation of the canals. However, there are basic differences; measurement of the phase lag indicates quite different orders of magnitude. For example the phase angle was about 25

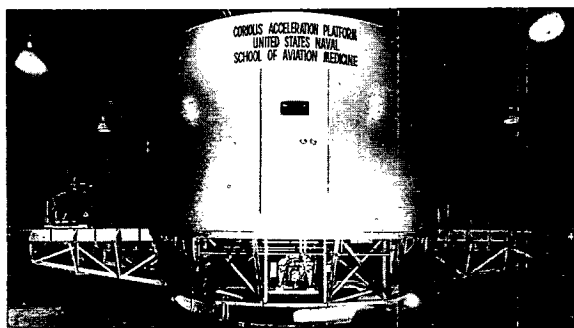


Figure 10.—Coriolis acceleration platform (CAP).

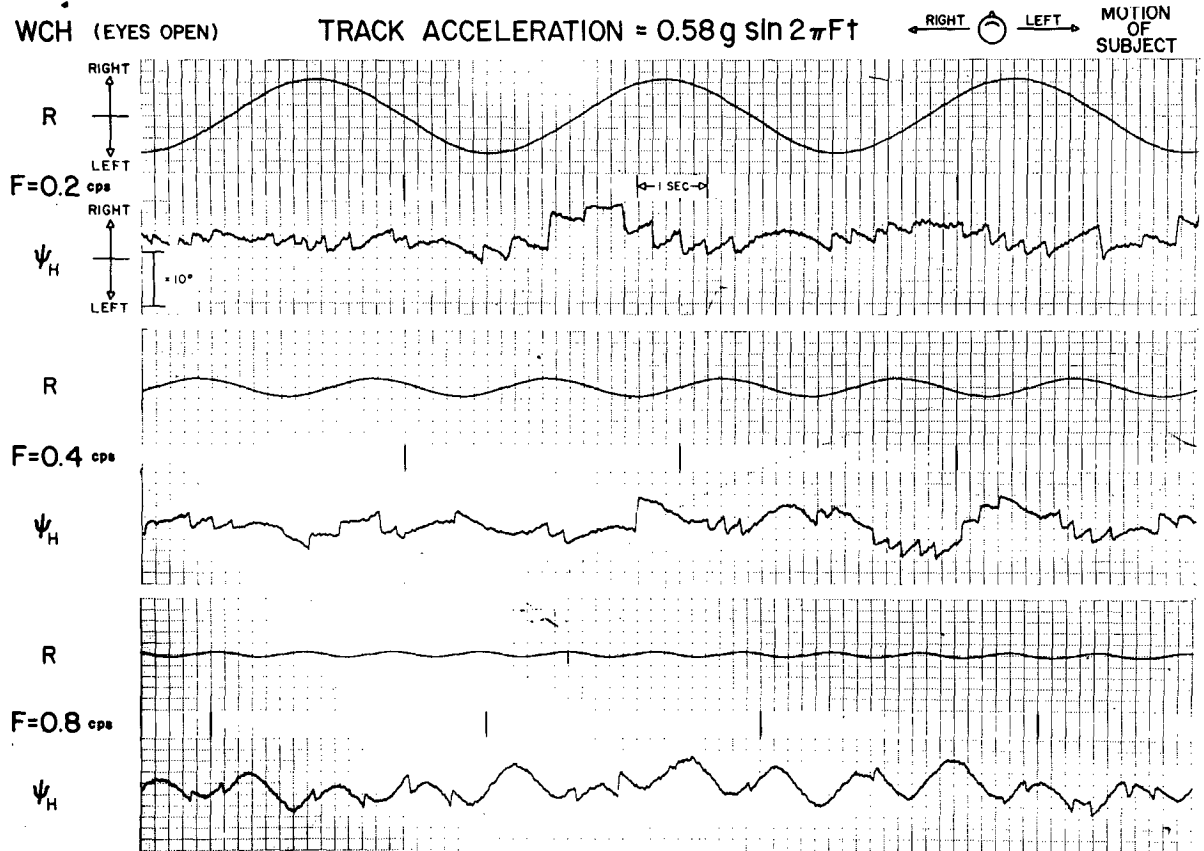


Figure 11.—Horizontal nystagmus response to a periodic linear acceleration applied in the frontal plane.

degrees at 0.2 cycle per second for the linear stimulus as compared to 85 degrees for an angular stimulus for the same subject.

It was not found possible, however, to obtain similar vertical nystagmus patterns when the subject was oriented in the head-foot direction (fig. 13) or the fore-aft direction (fig. 14). Eye movements were non-existent, random, or marked by blinks or similar artifacts.

These results show for the first time strong highly systematic, stimulus bound, horizontal nystagmus produced by periodic linear acceleration stimuli whether the subject's head was lightly constrained or rigidly fixed during his linear oscillations. The authors take care to note that these data (discussed in a paper by J. I. Niven, W. C. Hixson, M. J. Correia entitled "The Elicitation of Horizontal Nystagmus in Man by Periodic Linear

Accelerations" in preparation) do not allow the precise responding mechanisms, i.e., otoliths or canals, to be identified.

All the previously described efforts are based on using precisely defined periodic as well as transient linear and angular acceleration stimuli, making quantified measures of response data, and the assimilation of the resultant input-output response relationships in a form that will allow the dynamic characteristics of the various elements of the vestibular system to be separately defined. With this program, it is hoped to establish objective measures which will facilitate a quantified approach to the evaluation of potential deleterious effects of continued, bizarre acceleration environments on the vestibular mechanisms, as well as afford objective functional indices of vestibular status for selection purposes.

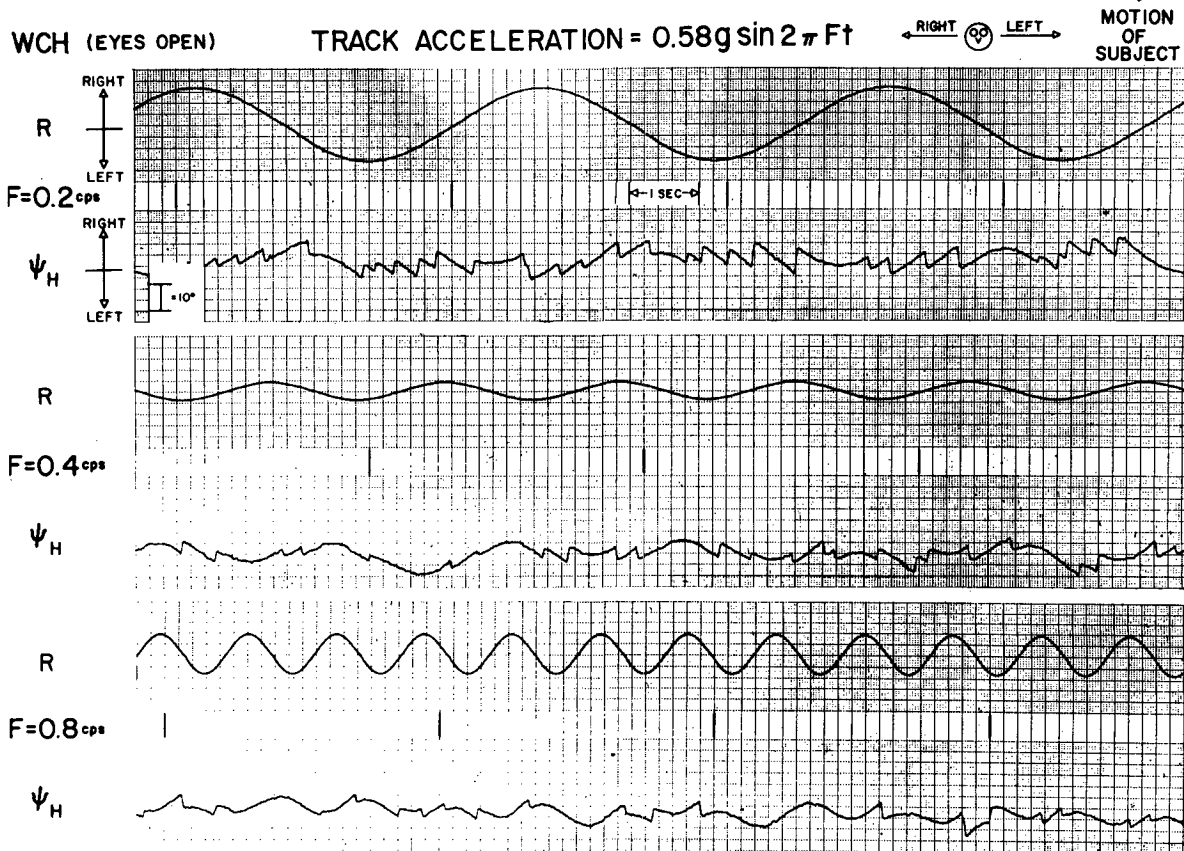


Figure 12.—Horizontal nystagmus response to periodic linear acceleration applied in horizontal plane.

#### REFERENCES

- HIXSON, W. C.; AND NIVEN, J. I.: Application of the System Transfer Function Concept to a Mathematical Description of the Labyrinth: I. Steady-state Nystagmus Response to Semicircular Canal Stimulation by Angular Acceleration. BuMed Project MR005.13-6001 Subtask 1, Report No. 57 and NASA Order No. R-1, Naval School of Aviation Medicine, 1961.
- HIXSON, W. C.; AND NIVEN, J. I.: Frequency Response of the Human Semicircular Canals II. Nystagmus Phase Shift as a Measure of Non-linearities. BuMed Project MR005.13-6001 Subtask 1, Report No. 73, NASA Order R-37, Naval School of Aviation Medicine, 1962.
- NIVEN, J. I.; AND HIXSON, W. C.: Frequency Response of the Human Semicircular Canals: I. Steady-state Ocular Nystagmus Response to High-level Sinusoidal Angular Rotations. BuMed Project MR005.13-6001 Subtask 1, Report No. 58 and NASA Order No. R-1, Naval School of Aviation Medicine, 1961.

#### DISCUSSION

BERGSTEDT: I found especially the last part of the paper very interesting. It could point to the otolith organ as really producing nystagmus. I would suggest that there is real reason to be careful in interpretation. Many subjects, so-called not normal subjects, show positional nystagmus about 20 to 30 percent as a result of hangover from alcohol, or of other causes, too, and I wonder whether you have found results of different intensity in different individuals, stronger in some than in others.

NIVEN: There were individual differences in subjects' responses. It is one reason why these tests show promise in being able to differentiate among individuals. However, the horizontal nystagmus was quite clear cut for each subject.

BERGSTEDT: I agree with you that the different time lags for the linear and angular oscillation stimulus could point to the otolith organ.

NIVEN: Well, we are not willing to go that far yet. We have presented these data simply as evidence

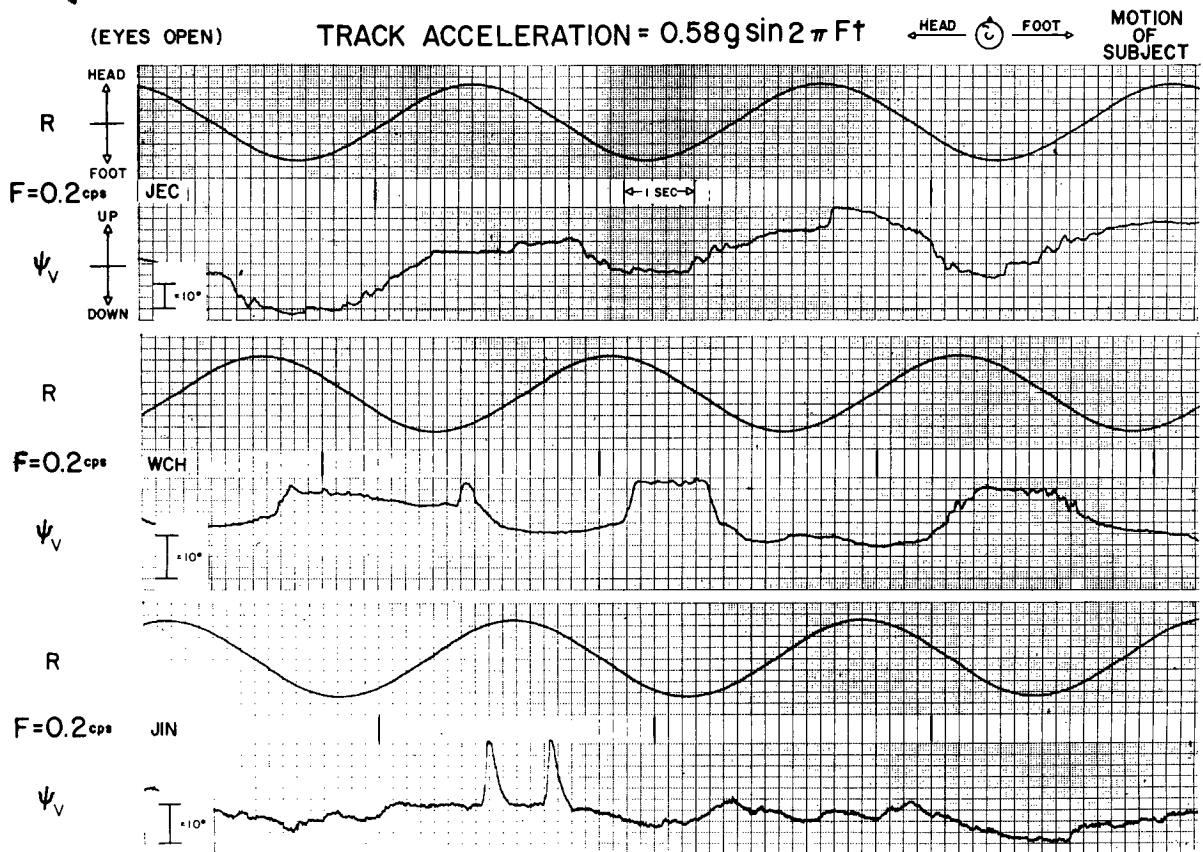


Figure 13.—Vertical nystagmus response to periodic linear acceleration applied in head-foot direction.

that highly systematic ocular nystagmus can be produced by sinusoidal linear acceleration stimuli. I might add one interesting observation. In one of Dr. Money's squirrel monkey preparations all six semicircular canals had been plugged. Caloric and rotational tests indicated that there was no remaining canal function. When we exposed them to similar linear oscillations, there was no observable nystagmus. A control monkey with functioning canals and normal responses gave us the same type of tracing that we have shown. It is a little too early to take a definite position.

M. JONES: Your data seem to suggest that at about 3-4 cps angular head movement there would be a poor dynamic response of the vestibulo-ocular reflex system. Yet if I shake my head about a vertical axis at these frequencies, I seem to have a beautifully fixed retinal image, as evidenced by a strikingly clear visual impression of the scene I am looking at. Do you think it possible that the responses elicited by you at low frequencies might be attributable to non-adapting neural units in the primary vestibular afferent nerve fibers, of the kind demonstrated by Groen, Löwenstein, and Vendrik? Perhaps at higher

frequencies additional adaptive neurons are brought into play, thereby improving the frequency response of the system as the frequency of head movement increases.

NIVEN: Certainly at the lower frequencies I indicated the existence of nonlinearities although at the higher frequencies the system seemed to be operating in a linear fashion. We have no direct experimental data on phase shift above 0.2 cycle per second, because of apparatus limitations.

WENDT: I collected data of this kind back in 1930 and analyzed the turns successively. It has been a long time since I published the paper, but, as I recall the data, there are progressive changes in phase relationships as you go along from the first turn to the second, third, fourth, etc. There were also very considerable individual differences especially based on any pre-existing imbalance of the vestibular response of the subject. I wonder whether you analyzed these data from this point of view.

NIVEN: We found small but inconsistent alterations in phase during short-term exposures for a given subject, although there were individual differences and directional differences in some subjects.

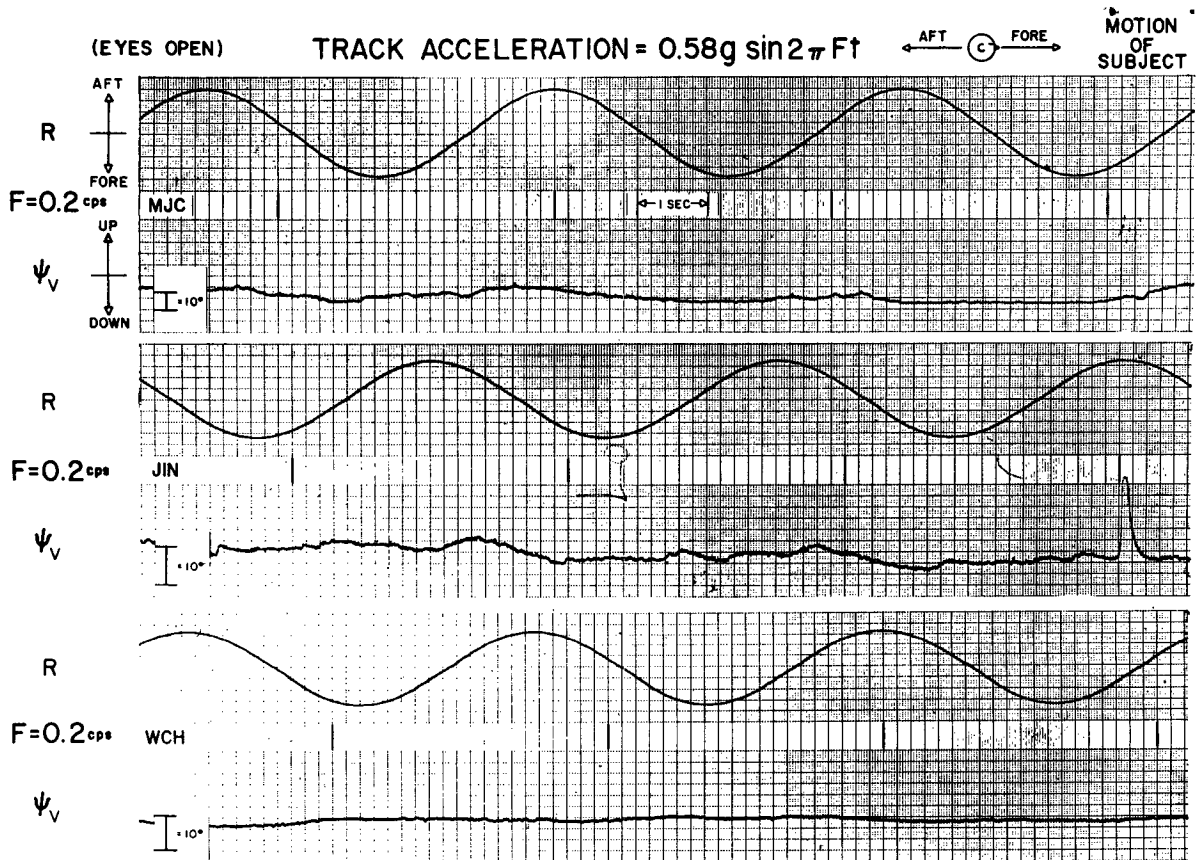


Figure 14.—Vertical nystagmus response to periodic linear acceleration applied in fore-aft direction.

STEELE: The eye movement is not to compensate for acceleration but for motion relative to a stable inertial-reference system. For phase shift, you should calculate zero eye movements against zero movement of the subject. This would be an integration of the acceleration and when you are calculating the frequency of the system, it does matter which phase relationship is regarded as zero. It would have been more to the point to have used the actual velocity rather than the acceleration because if the system is working perfectly, you will find zero phase shift between eye velocity and head velocity. At maximum acceleration (in sinusoidal movement), the eye velocity should be zero.

NIVEN: That's one way of looking at it, but you can use either, and acceleration is the stimulus that results in the eye movement rather than velocity per se. Since angular acceleration serves as the torque driving stimulus for the cupula-endolymph system, the authors have always referenced nystagmus phase lag response measures accordingly. These data may be readily plotted against angular velocity, or even angular displacement, by noting the constant 90 degree phase difference which exists between sinusoidal displacement and velocity as well as be-

tween velocity and acceleration. In interpreting the functional performance of the system when exposed to rapid head motions one can certainly realize elucidative advantages by considering the nystagmic response to the instantaneous angular velocity of the head. Our data serve to reinforce Steinhausen's highly damped torsion pendulum concept by clearly outlining the integrative nature and thus the velocity nature of the system at the higher stimulation frequencies. Further these data do not imply that this integrative facility will be lost in the frequency spectrum of rapid physiological head motions.

QUESTION: If you have the same subject and determine the phase shift from subjective judgments and from your method, how big is the difference in the natural frequency?

NIVEN: There is quite a bit of difference. The natural frequency from nystagmus data apparently runs much higher than from subjective sensations of body rotation or from reports of the oculogyral illusion. Our problem was, however, especially at the higher stimulus frequencies, such a great variability of the subjective measures that we could not place much reliability on them.

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# The "Match" of the Semicircular Canals to the Dynamic Requirements of Various Species

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## SUMMARY

The concepts of frequency response and elementary information theory are utilized in this paper to extend an investigation by Jones and Spells (1963) into the adaptation or "match" of the semicircular canals to the dynamic requirements of various species.

Author

## INTRODUCTION

Jones and Spells (1963) used dimensional analysis to investigate the effect of critical dimensions of the semicircular canals on their dynamic characteristics and to determine the likely relation between the weight of species and the speed of their movements. They obtained, then, measurements of the canals of a large number of species and showed that the dimensions of the canals could be generally predicted as a function of weight with the assumption that these sense organs are adapted to the dynamic requirement of the animals. They showed that very small changes in the internal radius and radius of curvature of the canals were sufficient to provide suitable match for very large weight variations.

The application of frequency response and elementary information theory to the analysis was discussed in private correspondence with Jones and is presented here for whatever additional insight it may give into the problem.

The history of the evolution of the theory of the semicircular canals is too well known to bear repetition here. Suffice it to say that Steinhausen (1931) expressed the behavior of the canals by an equation of the form:

$$\ddot{\theta} + L\dot{\theta} + P\theta = 0 \quad (1)$$

With oscillatory motion impressed on the head, the equation becomes:

$$\ddot{\theta} + L\dot{\theta} + P = A\omega^2 \sin \omega t \quad (2)$$

where

$\theta$  = angular displacement of the endolymph fluid

$$L = \frac{\Phi}{I}$$

$$P = \frac{\Delta}{I}$$

and

$\Phi$  = the moment of viscous friction exerted on the endolymph per unit angular velocity

$I$  = moment of inertia of the endolymph

$\Delta$  = elastic moment exerted on the endolymph per unit angular displacement

$A$  = maximum angular displacement of the head

$\omega$  = circular frequency of the oscillation

The system is greatly overdamped, and the theory expressed by equation (1) has been called that of the "overdamped pendulum." Van Egmond, et al. (1948-1949); Groen, et al. (1948, 1952), explored numerous aspects of the behavior of the canals of man and of the ray on the basis of the above formulation and calculated the constants of the equation. Mayne (1950) computed the frequency

response of the canals for ranges of frequencies said to represent normal body movements.

The response of a system defined by the equation (1) can be expressed as a ratio of output to input or by a so-called "transfer function." The transfer function may be in terms of endolymph displacement output to acceleration, velocity, or displacement input. It can be assumed that cupular displacement and, therefore, semicircular canal output signals are proportional to endolymph displacement. Figure 1 shows a plot of the three transfer functions for the canals of man on the basis of constants computed by Mayne (1950). It will be noted that there are ranges

of frequency where the response is flat for each of the three forms of input. Obviously, a highly desirable characteristic of a measuring instrument is a flat response to the quantity to be measured. This permits a direct reading of the quantity without complicated corrections. A flat response is particularly important in the case of the measurement of transient phenomena since without it a different correction would be required for every frequency component of the transient. The semicircular canals could then be used as accelerometers, velocity meters, or seismographs in different ranges of frequencies.

We note, however, that the range of fre-

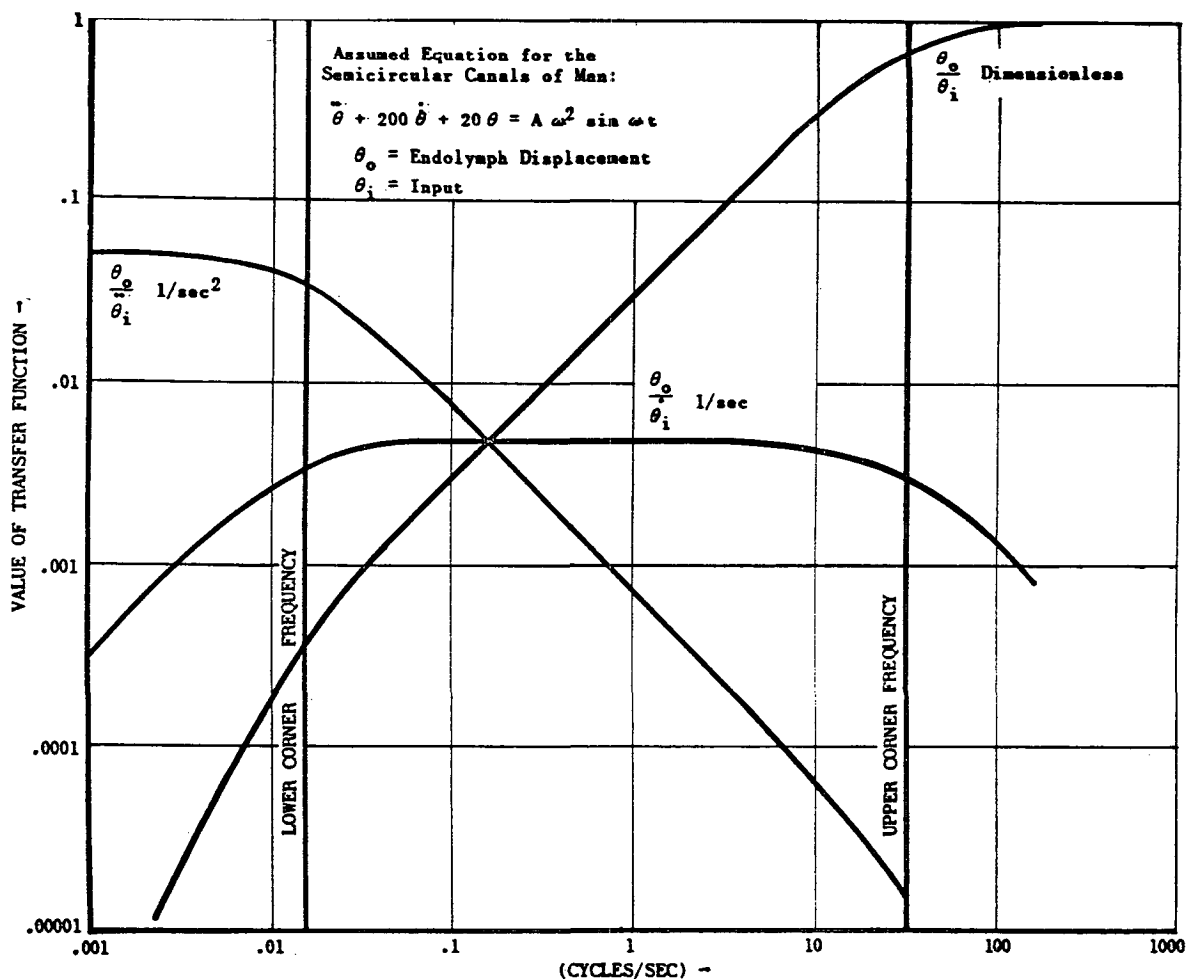


Figure 1.--Transfer functions of the semicircular canals of man for acceleration, velocity, and displacement inputs.

quencies or “bandwidth” where the response to velocity input is reasonably flat, is roughly from 0.04 cps to 4 cps and this appears to cover the range of body movements. In contrast, flat responses to acceleration and displacement lie in ranges below 0.002 cps, and above 100 cps, respectively. Obviously, oscillations having a period of more than eight minutes, or less than ten milliseconds, are not within the range of normal body movements. We say, then, that the semicircular canals are adapted to measure the velocity of the body’s transient movements within a range of frequencies, and to the accuracy defined by the frequency response curve of figure 1. Van Egmond, et al. (1949), stated that “. . . the function of the semicircular canals is to control body movements by indicating the attained velocity and the angle of rotation executed.” The statement agrees with the present view although obviously “the angle of rotation executed” is not given directly by the canals, being the result of further neural computations, but is ambiguous unless the limits of frequencies for which it is true to a given accuracy are indicated.

It is well known that any transient phenomenon, such as the sound of music, or a body movement, can be expressed as a summation of pure sinusoids of various frequencies, amplitudes and phases by a so-called Fourier transform. The characteristics of body movements of various animals vary with their size and it is the purpose of this investigation to extend that of Jones and Spells in determining how the semicircular canals are matched to these characteristics. Four numbers are significant in defining the behavior of the semicircular canals—the upper range of the frequency band, the lower range of the frequency band, the resolution, and the limit of linearity. We shall be concerned here mainly with the first three. It must be emphasized that equation (1) represents only a first approximation of the canal behavior. So-called “adaptation” is known to modify considerably the response of the canals (Guedry and Ceran, 1959), but

perhaps mostly outside the normal bandwidth of the system. We shall neglect this phenomenon in this paper.

*UPPER RANGE OF THE FREQUENCY BAND*

The upper and lower limits of the frequency bandwidth may be defined in terms of the so-called corner frequencies. These frequencies define a point in the spectrum where the response is 0.707 of the maximum and the phase shift 45 degrees. The corner frequencies do not correspond in any way to the usable bandwidth as the distortion is considerable at those values, but they are convenient points of reference. The upper corner frequency ( $f_u$ ) of a highly overdamped system such as the semicircular canals is expressed to a close approximation by the formula:

$$f_u = \frac{L}{2\pi} \text{ cps} \tag{3}$$

$L$  can be computed from hydrodynamic considerations and the Hagen-Poiseuille law of laminar flow in small circular pipes.

Schmaltz derived the formula

$$L = \frac{8\eta}{\rho r^2} \tag{4}$$

where

$\eta$  = coefficient of viscosity of the endolymph

$\rho$  = density of the endolymph

$r$  = internal radius of canals

Van Egmond, et al. (1949), altered the formula to:

$$L = \frac{4\eta}{\rho r^2} \tag{5}$$

on the ground that the canals include only half a circumference, the remaining portion consisting of the very much enlarged utricle. It appeared to them that viscosity in the utricle could be neglected. There are reasons to believe that the alteration of the formula cannot be justified on this ground. It is valid, however, in showing that the top corner frequency is inversely proportional to  $r^2$ . We then turn to an investigation of the likely maximum frequency of body movements of various species. We first consider mammals alone for reasons that will be discussed later.



Jones and Spells used dimensional analysis to relate canal dimensions to animal weight. They expressed equation (1) in dimensional parameters and derived a relation for the speed of head movement of various animals as a function of their mass. They argued that higher "sensitivity" of canals should correspond to the more sluggish head movement of heavier animals. Dimensional analysis then led to the conclusion that both  $r^2$  and  $R$  should vary as the same function of the mass  $m$  of the animals. The concern of this paper is for frequency, more specifically the upper and lower frequencies in a flat bandwidth of response, but the results of both methods of analysis are generally similar. Frequency has the same dimension as angular velocity and the two are numerically equal for unit angular amplitude of oscillation.

While Jones and Spells are undoubtedly correct in assuming that the semicircular canals should be matched to the head angular movement with respect to the body, it would seem that they should also be matched to gross body movements. Mayne (1950-1951) suggested that the main function of the vestibule is to act as proprioceptive sensor for gross movements of the body when no other sensors can supply the information. It is possible to derive expressions for over-all body motions as a function of mass similar to those obtained by Jones and Spells for head movements with respect to the body.

The body-muscle system of a mammal may be considered as an oscillatory system with moment of inertia, damping and restoring moment. The restoring moment is that due to the muscle system operating under feedback control. The stiffness of the system depends on gain and is adjustable, depending on the desired speed of movement.

There is, however, a maximum effort or a maximum power which a muscle system can exercise or develop as limited by its size and weight. If we assume that the maximum angular amplitude of movement is the same for all animals, the maximum moment developed by the muscles of various animals

corresponds then to the same displacement, and we can write:

$$\Delta_{\max} \propto \frac{M_{\max}}{A} \quad (6)$$

where

$\Delta_{\max}$  = the maximum stiffness of the system

$M_{\max}$  = maximum moment

$A$  = maximum amplitude of motion of the body within the limits of linearity of the system

Since  $A$  is a non-dimensional constant, we have:

$$\Delta_{\max} \propto M_{\max}$$

and therefore,

$$\omega_{\max} \propto \sqrt{\frac{M_{\max}}{I}} \quad (7)$$

where:

$\omega_{\max}$  = maximum frequency of oscillation

$I$  = the moment of inertia

We now attempt to determine the likely relation of  $M_{\max}$  to the mass ( $m$ ) of animals. We assume first geometrical similarity between animals corresponding to a similar allocation of weight to muscles. Jones and Spells assumed further that the maximum moment which can be exerted by the muscles is proportional to the weight of the animal. It would be more realistic to assume that the power or energy rate ( $\dot{E}$ ) is so proportioned.

We can write:

$$\dot{E}_{\max} = [\dot{\theta}_i M]_{\max} \quad (8)$$

where:

$$\theta_i = A \sin \omega t$$

and

$$\begin{aligned} \dot{\theta}_i &= A \omega \cos \omega t \\ M &= -A I \omega^2 \sin \omega t \end{aligned}$$

then:

$$\dot{E} = -A^2 \omega^3 I \frac{1}{2} \sin 2 \omega t$$

giving

$$\dot{E}_{\max} \propto I \omega^3_{\max} \quad (9)$$

According to previous assumption:

$$\dot{E}_{\max} \propto m$$

If we take

$$I \propto m^{5/3}$$

we have:

$$\omega_{\max} \propto \sqrt[3]{\frac{m}{m^{5/3}}} \propto m^{-0.223} \quad (10)$$

Under the same assumptions of geometrical similarity, Jones and Spells obtained an angular velocity proportional to  $\frac{1}{m^{1/3}}$ . The difference between the two results, outside of the formulation in terms of velocity instead of frequency, is based on the different assumptions as indicated above.

It can be shown that the damping of some body control (Mayne, 1951) for high gain is nearly critical. The maximum frequency of body oscillation as calculated above should represent, therefore, the upper limit of linear operation of the body movements.

Jones and Spells argue that the relation of:

$$\omega \propto \frac{1}{m^{1/3}}$$

represents too rapid a decrease of angular speed of movement as the mass of animals is increased. If true, this relation would mean that the linear speed of movements would remain constant regardless of animal size, and the paw of a tiger would then move linearly no faster than that of a cat. Relation (10) provides a somewhat slower decrease of frequency with weight but appears still too fast. The difficulty is apparently with the assumption of dimensional similarity between animals. In the first place, it is probably not possible to maintain the same ratio of muscle weight to body weight as the animals become smaller. There must be limits in the way the skeleton, and other non-power producing parts of the body can be miniaturized. Jones and Spells point out that in order to keep gravitational stresses constant, more muscle must be provided than determined by dimensional similarity, and therefore more muscle is available for turning the head. On the basis of this assumption, they obtain the relation:

$$\omega \propto \frac{1}{m^{1/12}}$$

where  $\omega$  is again the speed of movement. They argued that the true exponent of  $m$  should be between  $1/12$  and  $1/6$ . We may use still another criterion; namely, that acceleration stresses on the body should be constant. We could then write for both centrifugal and reversal stresses:

$$\omega_{\max}^2 \ell = C \quad (11)$$

We would then have:

$$\omega_{\max} \propto \sqrt{\frac{1}{\ell}} = \frac{1}{m^{1/6}} \quad (12)$$

Jones and Spells find that the dimensions of the internal radius of the semicircular canals of mammals can be related to the mass of mammals by the expression:

$$\log_{10} 100r^2 = (0.1433 \pm 0.0421) \log_{10} m + 0.0406 \quad (13)$$

The equation can be modified to express  $f_u$  directly. The constant of proportionality can be determined, approximately, on the basis of data on the semicircular canals of man, giving an upper corner frequency of 32 cps for a mass of 70 kg. With the average exponent of  $m$  the equation becomes:

$$f_u \approx \frac{58.5}{m^{0.143}} \quad (14)$$

It will be noted that the exponent of  $m$  is 0.143 or  $1/7$  instead of  $1/6$  as in equation (12). This figure agrees also with Jones' and Spells' statement that the exponent should lie between  $1/3$  and  $1/12$ . We can well agree with them, also, that the next step in the investigation should be one of measurement of the spectrum of the body movements of specific animals. The allocation of weight to muscles, organs, and skeleton could then be determined together with critical dimensions of canals. Some of the differences in canal dimensions of different species of the same weight can only be reconciled on the basis of such specific investigation. Weight is obviously not the only consideration determining the upper frequency response of an animal.

#### LOWER RANGE OF THE FREQUENCY BAND

The upper corner frequency of the canals is a measure of the maximum frequency at

which the canals give reliable velocity information within amplitude limits of linearity. Similarly, the lower corner frequency is a measure of the lowest utilizable frequency. As mentioned above, the corner frequencies are convenient points of reference but do not represent the limits of the usable bandwidth which is much narrower. Some body motions may utilize the higher portion and others the lower part of the bandwidth. A diver, for instance, may utilize the upper range in the control of a  $1\frac{1}{2}$  turn from a low board and depend on the lower limit in a swan dive from a high board. The canals of an airplane pilot in a turn are operating below their normal range of frequencies and may cause abnormal sensations.

The lower corner frequency is given to a close approximation by:

$$f_l = \frac{1}{2\pi\tau} \quad (15)$$

where  $\tau$  is the time constant represented by

$$\tau = \frac{L}{P} = \frac{\Phi}{\Delta} \quad (16)$$

Van Egmond, et al. (1949), showed that from elementary hydrodynamic considerations:

$$\Phi = 8\pi^2\eta R^3 \quad (17)$$

where  $\eta$  is a constant as defined in equation (4).

Jones and Spells give

$$\Delta = \pi r^2 \mu R \quad (18)$$

where  $\mu$  is the coefficient of elasticity assumed to be constant.

From these two relations they derived:

$$\tau \propto \frac{R^2}{r^2} \quad (19)$$

If the ratio of the high frequency in the bandwidth to the low frequency is to remain the same for all species,  $\tau$  should then be expressed as a function of the same power of animal mass as  $r^2$ . This should be nearly realized by expression (19), for if  $r^2$  and  $R$  are similar functions of ( $m$ ) as predicted by Jones and Spells, we should have:

$$\tau \propto \frac{R^2}{r^2} \propto R \propto r^2 \text{ approx.} \quad (20)$$

However, Jones' and Spells' measurements show that  $r^2$  and  $R$  are not exact functions of the same power of  $m$ . Furthermore, the relations between canal dimensions and mass represent only averages for a large number of animals and there are wide variations for species of the same weight. While equation (20) may be nearly true as an average, it does not necessarily apply even approximately to a specific specie. It gives, for instance, values at wide variance from the experimentally derived time constants for man and for a ray. At the same time a plot of these values versus the mass of animals does not indicate a statistically significant relation. It would seem better to express the probable value of the time constant directly as:

$$\tau \propto r^2 \quad (21)$$

Equation (19) is derived by Jones and Spells under the assumption that  $\mu$  is constant in equation (18). We next derive the value of  $\tau$  without this assumption so that we may observe possible variations of cupula stiffness.

We assume that ampullae and cupulae of different sizes are geometrically similar. The total pressure on the cupula is equal to the pressure times the cupula area which may be taken as  $V^{2/3}$  where  $V$  is the volume of the ampulla. The moment  $M$  on the hinge is then:

$$M \propto \epsilon^{1/2} P V^{2/3} V^{1/3} \propto P V \quad (22)$$

where:

$M$  = elastic moment per unit deflection of cupula

$\epsilon$  = angular deflection of cupula

But we can also write from equality between the volume of endolymph displaced in the canal and that swept by the cupula:

$$\theta r^2 R \propto \epsilon V^{1/3} V^{2/3} \propto \epsilon V \quad (23)$$

Combining equations (22) and (23):

$$P = \frac{\theta M r^2 R}{V^2} \quad (24)$$

But  $\Delta$  is the moment on the endolymph per unit displacement so that:

$$\Delta \propto \frac{P r^2 R}{\theta}$$

or, from equation (24)

$$\Delta \propto \frac{M\gamma^4 R^2}{V^2} \quad (25)$$

From

$\tau = \Phi/\Delta$  and equation (17) we derive:

$$\tau \propto \frac{RV^2}{M\gamma^4} \quad (26)$$

and from equation (21):

$$M \propto \frac{RV^2}{\gamma^6} \quad (27)$$

Expression (27) indicates how  $M$  should be adjusted to other dimensions of the canals to make  $\tau$  proportional to  $r^2$ .

From equation (25) it can be shown that Jones' and Spells' assumption that  $\mu$  is constant in equation (18) corresponds also to a variable  $M$  as expressed by:

$$M \propto \frac{V^2}{r^2 R} \quad (28)$$

It appears, therefore, that cupula stiffness must be introduced as one of the adjustable parameters determining canal characteristics. Nor can we assume that elasticity is always a rigid function of canal dimensions. Experimental data by Van Egmond, et al. (1948), indicate, for instance, that the stiffness of the cupula for clockwise is different from that for counter-clockwise rotation as judged by cupulograms of subjects having suffered unilateral loss of a labyrinth. Equation (27) is based on the assumption of the same bandwidth for all animals. It is more likely that larger animals would have a wider bandwidth so that  $\tau$  may be proportional to a function of  $r$  greater than two.

From equation (13) and a time constant of 10 for man having a mass of 70 kg, we derive the following expression using the average coefficient of  $m$ :

$$f_t = \frac{0.03}{m^{0.143}} \text{ cps} \quad (29)$$

Table I gives some computations of  $\tau$  on various bases in comparison with experimentally derived values.

#### RESOLUTION

Resolution of a measuring instrument is usually defined as the reciprocal of the size

Table I.—*Computations of  $\tau$  on Various Bases*

[The constants in the expression for  $\tau$  are computed to give  $\tau = 10$  for man.]

Species	Mass	Experimental	$\tau = \frac{0.015 R^2}{r^2}$	$\tau = 500 r^2$
Man -----	70	<sup>a</sup> 10	10	10
Ray -----	10	<sup>b</sup> 35	8.6	55
Mammals c_	10	--	5.5	7.2
Fish c_-----	10	--	10.4	26.5

<sup>a</sup> Van Egmond, et al. (1948-1949).

<sup>b</sup> Groen, et al. (1932).

<sup>c</sup> Computed from data taken from Jones and Spells (1962).

of a resolvable element which may be, in turn, taken as a measure of the limit of accuracy of the instrument. A navigational system, for instance, which measures velocity to an accuracy of plus or minus three feet/second may be said to have a resolvable element of six feet. Another characteristic of a measuring instrument is the ratio of the maximum amplitude of the quantity it measures to the size of the resolvable element. This ratio is usually expressed in db and is designated as the dynamic range of the instrument.

Physiological sensors can be assumed similarly to possess characteristics such as resolution and dynamic range. Fechner is generally credited with the pioneering work in the determination of the size of the resolvable elements of various sensors or "just noticeable differences" (jnd). His studies indicated that the jnd must be specified in statistical terms. More recently his work has come under considerable scrutiny, with an application of statistical theory of signal recognition (Swets, 1964).

The experimental determination of jnd generally involves the statement by a subject that he does or does not perceive a change in a sensation, be it the pitch of a tone, the weight of an object, or the intensity of a light when the stimulus is increased gradually. The experimental situation, there-

fore, involves higher neural or mental functions in the way of perceptions and their verbalization. The work of Swets has been directed in part toward the isolation of the physiological reaction from the "noise" created by these higher functions.

The semicircular canals are designed to operate autonomically in the control of body movements. It would seem that their resolution should be determined on the basis of the analysis of thoroughly habituated motions in which higher functions have no part (Mayne, 1951). Any professional golfer needs no demonstration that higher mental functions can introduce severe "noise" in the control of body movements and will work hard to acquire a mechanical, so-called "grooved swing" as independent as possible from any conscious interference.

For the purpose of this study we will assume there exists a semicircular canal resolvable element. We recognize this element should be expressed statistically. We associate this element with the autonomic control of body movement, free from the interference of higher functions. Within these limitations we let:

$n$  = number of resolvable elements for maximum amplitude of velocity input within the limit of linearity.  
 $n$  may be considered as a measure of resolution.

The maximum number of resolvable elements processed per second is then:

$$\dot{n}_{\max} \propto f_u n \quad (30)$$

There is a similar expression in communication engineering—the so-called "Hartley Law"—differing mainly from the above in its logarithmic formulation. The physical justification for the formulation of the Hartley Law is that it expresses a measure of the required complexity of a channel needed to handle a given rate of yes and no pieces of data assuming optimum coding. The nature of the physiological coding of sensor data is not known in the way of what "trade-offs" may have been made between simplicity of coding and simplicity of chan-

nels. The formulation of (30) is left purposely loose so as to be free from any implications in this regard. It should correspond, nevertheless, to an adequate physical concept for the purpose of this paper.

We investigate now the relation between canals, ampulla size, and cupula deflection for unit change in angular velocity input. The solution of equation (1) for a region within the bandwidth gives very nearly:

$$\Delta\theta = \frac{\Delta\dot{\theta}_i}{L} \quad (31)$$

where

$\Delta\theta$  = an increment deflection of endolymph corresponding to:

$\Delta\dot{\theta}_i$  = an increment of velocity input

Since  $L \propto \frac{1}{r^2}$  we have:

$$\Delta\theta \propto \Delta\dot{\theta}_i r^2 \quad (32)$$

The volume of endolymph delivered to ampulla is then:

$$\Delta V \propto \Delta\theta r^2 R \propto \Delta\dot{\theta}_i R r^4$$

The cupula deflection becomes:

$$\Delta\epsilon \propto \frac{\Delta V}{V} \propto \frac{\Delta\dot{\theta}_i R r^4}{V} \quad (33)$$

and:

$$\frac{\Delta\epsilon}{\Delta\dot{\theta}_i} \propto \frac{R r^4}{V} \quad (34)$$

If we assume that a resolvable element corresponds to a constant increment of deflection  $\delta$  of the cupula, expression (34) is then proportional to the number of resolvable elements per unit change of angular velocity of the head and to  $n$ . On the other hand, we can assume that the number of resolvable elements is some function of the number of cells in the cristae. Let us assume, or guess, "pour fixer les idées," that the number of sensory cells in the cristae is proportional to the volume of the ampulla and that because of the random stimulation the number of resolvable elements is proportional to the square root of this number. We then have from equation (34):

$$n \propto \frac{R r^4}{V^{1/2}}$$

and from (30) :

$$\dot{n}_{\max} \propto n \frac{Rr^2}{V^{1/2}} \quad (35)$$

It may be noted that if  $V \propto r^2R$  as indicated by Jones and Spells, this equation would become :

$$n_{\max} \propto \frac{1}{R^2r} \quad (36)$$

It may be well to retain equation (35) because of the variability of the relation between  $V$  and  $r^2R$ .

THE SEMICIRCULAR CANALS OF FISH

Jones and Spells point out that the dimensions of  $r$  and  $R$  for fish are relatively large compared to those of mammals. The contrast of these dimensions is shown in table II taken from their paper.

Table II.—Dimensions of  $r$  and  $R$  for a Mammal and a Fish Each of 10 Kg Mass

Animal class	$r$ (mm)	$R$ (mm)
Mammal -----	0.12	2.30
Fish -----	0.23	5.99

Table I also indicates values of experimentally determined time constants for man and for the ray. The dimensions of the canals correspond to a very much lower bandwidth of frequency response for fish than for mammals. It is easy to visualize why this should be so.

The head of the fish is fixed to the body without a connecting neck; the rotation of the head can only take place with a rotation of the body. The analysis of Jones and Spells regarding head movements is, therefore, not applicable. But neither is our own analysis. There is nothing in the turning of a fish which corresponds to that of the torso, or to a jump. The fish must have forward motion in order to turn, and in this characteristic it resembles a ship, an airplane, or even an automobile. There may be small restoring rolling moments when the fish is stationary, but these appear rather inconsequential. The fish applies the bulk of its power to obtain forward motion and then

needs to exercise only small control forces to effect a turn. The angular speed of the turn depends on the linear velocity of the fish as he enters the turn, the amount of control forces, and is limited by fluid dynamic considerations such as the stalling of control surfaces under excessive lift, and probably by centrifugal force stresses. There is no correspondence between this situation and that of a mammal where estimated muscle power and moment of inertia determine a frequency of oscillation. It is easy to see why Jones and Spells could not obtain statistically significant relations between canal dimensions and the mass of fish. The turning radius of a fish must be relatively large and must correspond to relatively low angular speed, related in turn to a lower bandwidth of frequency.

The vestibule of a fish is no doubt one of the key sensors in the computation and control of the beautiful pursuit course executed by a trout in catching a minnow, or the evasive maneuvering of a fish under attack. The relative size of the canals for mammals and fish is one of the best illustrations of the match of these sensors to biological systems. It may be noted that the above discussion applies also in some degree to birds and their canals have somewhat larger dimensions than mammals of the same weight. The difference is less, however, than for the fish because birds must be adapted for movements on the ground as well as for flight.

DISCUSSION AND CONCLUSIONS

The present investigation, as far as it goes, confirms the broad findings of Jones and Spells (1963) regarding the matching of the semicircular canals to the requirements of various species. However, while Jones and Spells indicate that this match can be accomplished by adjustments of two independent parameters—the internal radius and the radius of curvature of the canals—the present paper suggests that five adjustable parameters are needed to provide an appropriate degree of flexibility in the design. These parameters are said to include: the

internal diameter of the canal, the radius of its curvature, the volume of the ampulla, the stiffness of the cupula, and possibly the number of sensory cells in the cristae. The next step in the investigation should be one of experimental measurement of body movement, canal characteristics, canal dimensions, and attempted correlation of the data.

If an engineer were to be given the task of designing a semicircular canal having specified to him by the system group an upper and lower limit of frequency bandwidth and a maximum processing rate of resolvable elements, he might proceed as follows. He would write down the three equations relating these quantities to canal parameters.

$$f_u = c_1 \frac{1}{r^2} \quad (a)$$

$$f_l = c_2 \frac{Mr^4}{RV^2} \quad (b)$$

$$\dot{n} = c_3 \frac{r^2 R}{V^{1/2}} \quad (c)$$

where the constant of proportionality would have been established. He would first select  $r^2$  from equation (a) to give the desired  $f_u$ , and would substitute this value in (b) and (c), giving:

$$f_l = c_4 \frac{M}{RV^2} \quad (d)$$

$$\dot{n} = c_5 \frac{R}{V^{1/2}} \quad (e)$$

From (e) he would obtain:

$$V^2 = c_6 R^4 \quad (f)$$

since  $\dot{n}$  is known.

Substituting in (d) would give:

$$f_l = c_7 \frac{M}{R^5} \quad (g)$$

Reasonable values for  $M$  and  $R$  would then be selected from (g) on the basis of good de-

sign practice and space available. The value of  $V$  would then be determined from (e).

But our engineer would really have performed only a routine function in the design of the canals. The more difficult job would have been done by the system group in arriving at specifications for  $f_u$ ,  $f_l$ , and  $\dot{n}$  to provide suitable match of the system in all possible respects such as power available in muscles, information handling capacity of neural channels and processing centers, resolution of time, resolution of force and position in body proprioceptive sensors, and, of course, the desired function of the system. An error analysis would have been made allocating an appropriate portion of the maximum overall permissible error to every component, including the semicircular canals.

The notion that components of a biological system are matched is at the root of the basic motivation to undertake investigations such as conducted by Jones and Spells. It originates from an assumption of efficient design and economy in the configurations of nature. An oversized component in a system is wasteful in that its performance is limited to that of other components. An undersized component is also wasteful as it limits the performance of all other components. But the most remarkable match of all is between the expectation and the findings of reason that biological systems are matched.

In the history of human thought and experience, three agencies have been either proposed or observed to produce matched designs—God, evolution, and an engineering organization. The same characteristic of reason which leads man to look for matched or economical design in nature will lead him to try to compress these three agencies into one.

#### REFERENCES

- CLARK, BRANT; AND GRAYBIEL, ASHTON: *J. Psychology*, 28, 1949, pp. 235-44.  
 GROEN, J. J.; AND JONGKEES, L. B. W.: *J. Laryng.*, 1948.  
 GROEN, J. J.; LÖWENSTEIN, O.; AND VENDRIK, A. J. H.: *J. Physiology*, 1952, 117, pp. 329-346.  
 GUEDRY, F. E., JR.; AND CERAN: U.S. Army Med. Res. Lab., Report No. 376, 1959.

- HIXSON, W. C.; AND NIVEN, J. I.: NSAM-830, 1962.
- JOHNSON, WALTER H.; MEEK, JOSEPH C.; AND GRAYBIEL, ASHTON: *Annals of Otology, Rhinology and Laryngology*, vol. 71, no. 2, June 1962, pp. 289-298.
- JONES, G. M.; AND SPELLS, K. E.: *Proc. Roy. Soc. B*, vol. 157, 1963, pp. 403-419.
- MAYNE, ROBERT: *J. Comparative Psychology*, vol. 43, no. 4, Aug. 1950.
- MAYNE, ROBERT: *J. Am. Inst. Elec. Eng.*, 1951.
- MAYNE, ROBERT: *Bull. Math. Biophys.*, vol. 14, 1952.
- NIVEN, J. I.; AND HIXSON, W. C.: NSAM-459, 1961.
- ROSENBLITH, WALTER A.: *Rev. Mod. Phys.*, vol. 31, no. 2, 1959.
- SCHMALTZ, G.: *Proc. Roy. Soc. Med.* 25, 1932, pp. 359-381.
- SCHMITT, OTTO H.: *Rev. Mod. Phys.*, vol. 31, no. 2, 1959.
- STEINHAUSEN, W.: *Pflüg. Arch. Ges. Physiol.*, 1931, pp. 229 and 439.
- SWETS, JOHN H. (ED.): *Signal Detection and Recognition by Human Observers*, John Wiley & Sons, Inc., 1964.
- VAN EGMOND, H. A. J.; GROEN, J. J.; AND JONGKEES, L. B. W.: *J. Laryng.*, 1948, pp. 62-63.
- VAN EGMOND, H. A. J.; GROEN, J. J.; AND JONGKEES, L. B. W.: *J. Physiology*, 1949, 110, pp. 1-17.



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# The Modulating Influence of the Otolith Organs on Semicircular Canal Functions

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When I was pondering on the subject of this symposium, which is the role of the vestibular organs in the exploration of space, it occurred to me that nobody has considered the possibility that this role could ever be rated along the positive side of the scale. Indeed, some people have even gone so far as to suggest that the astronauts' labyrinths could better be eliminated either temporarily or permanently as they could only bring mishap to their owners. We have all been so busily engaged in counting the undoings of the vestibular organs under unphysiologic or rather uncommon conditions that we are bound to forget that these organs might well serve a positive function.

Indeed, I think they may be as useful to the space-traveler as they are to the dweller on earth if only we take care that the situation the astronaut will meet will be adapted to him as well as vice versa. It is therefore a fortunate coincidence that so many studies have recently been concerned with problems of habituation, response decline and adaptation as we will want to know to what extent we can deviate from normal circumstances without compromising the astronauts' well-being and proficiency in handling and reasoning.

Consequently I feel that the questions to be answered at this symposium might be formulated as follows:

- (1) What dynamic situation will the space-traveler have to face according to current technical concepts?
- (2) Does our present knowledge of vestibular mechanisms suffice to judge in how far these situations envisaged in space flight may be provocative of untoward reactions? How should these reactions be classified and what will be their character?
- (3) What means do we have to circumvent or prevent any such reactions? I think in terms of selection procedures, indoctrination, training, drugs.
- (4) The fourth question is rather a reciprocal one; it does not concern the role of the vestibular organ in the exploration of space but the reverse, i.e., the role of space flight in the exploration of the vestibular organ. Speaking more generally, this may well be the most important and fascinating side of space flight; not what it will reveal to us of distant worlds but what it will unveil to us about ourselves. Does not the physiologist engaged in the study of vestibular organs eagerly await observations under weightless conditions just like the psychologist is anxious to learn more about man from his conduct when distantly separated from his

fellow man and deprived of his customary surroundings and like the metaphysicist is longing to substantiate the transcendental nature of our knowledge of ourselves and our world by exposing us to dimensions of distance and time that reach beyond the system of co-ordinates in which we visualize our world?

Returning to question (1), I think we will agree that the dynamic situations to which the astronaut will be exposed will be weightlessness and artificial, centrifugal weight.

The second question has had considerable attention both in the laboratory and in the satellites but we still do not feel quite confident as to the innocuousness of weightlessness and even less as to the harmlessness of centrifugal weight.

In considering where the possible harm should reside we once again hit upon the old controversy about the cause of motion-sickness. Leaving aside all other factors—visual cues, central coordination, psychic constitution—it is the vestibular input that upsets the minds of the investigators as much as the homeostasis of the traveler, and a vivid controversy still reigns among the students of motion-sickness. There are ardent protagonists of the semicircular canals as eliciting agents of the disturbing symptoms. Others remain faithful—though in a negative sense—to the otoliths as being the culprits of the troubles. A third group feels that neither the otoliths nor the semicircular canals suffer undue unphysiologic stimuli as a result of which motion-sickness ensues. They hold that it is a conflict in information that causes the symptoms and they gleefully welcome all recent data on learning processes and habituations as this fits very well in with their theory of conflicting sensorial cues. Not overstimulation but discordant stimulation is in their opinion the item to be looked for. Among the many studies of vestibular adaptation those of Guedry and Graybiel deserve our special attention as they were directly aimed at pro-

viding evidence on the behavioral problems in a rotating space station.

It will be remembered that the subjects had to move their heads in a frontal plane which engenders a "false" impulse in the semicircular canals causing a tilting reaction and a sensation of tilt. After a few days habituation occurred and when the rotation of the room was stopped, similar false responses—nystagmus included—were again elicited by these head movements but now in the opposite direction.

Let me take these experiments as a framework to bring into the discussion a few more points.

First, are we justified in considering the rotating room situation to be equivalent to the situation in a rotating spaceship? I do not think so. In the rotating room an inclination of the head in the frontal plane will elicit always the same cupula deflection whatever direction the man is standing. Thus habituation will be possible. Conversely, on the rotating spaceship the impulse will depend upon the subject's position. Even worse is the fact that the most normal movements we know of, i.e., the rotations of the head or the body around the longitu-

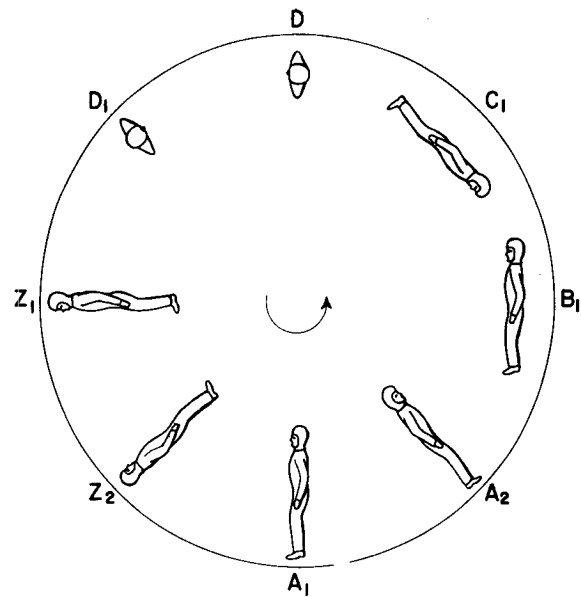


Figure 1.—Body orientation in the experiment.  
(From *J. Aerospace Med.*, ref. 1.)

dinal axis, will not cause any false response in the occupant of the rotating room but they will do so in the occupant of the spaceship and again in a treacherous and—for the subject unpredictable—unhabituable way. Consequently, I feel that on giving our advice to the engineers of rotating spaceships we should be conservative in our estimates of what angular velocity will be acceptable.

A second point I would like to make is that the impulses to the cupulae that we are discussing are from a point of mechanical sense organ physiology quite innocent. Indeed after stopping the room the head movements will occasion only the normal momentaneous cupula deflection and yet the effects are quite different. I believe that these facts argue

in favor of the conflicting-clue theory and that the conflict resides in the otolithic and canal signals.

Past are the good old days where semicircular canals and otoliths were separate organs with separate functions and separate effects. Although it is true that the canals will respond to angular impulses and the otoliths basically to linear accelerations, they are linked together for better and worse. We have tried to find some principles governing this interaction. To this end 315 runs were performed on the Pensacola centrifuge (ref. 1); the subjects, 26 in all, were placed in different seated and recumbent positions. The angular impulse was kept pretty well constant. (See fig. 1.) The magnitude of

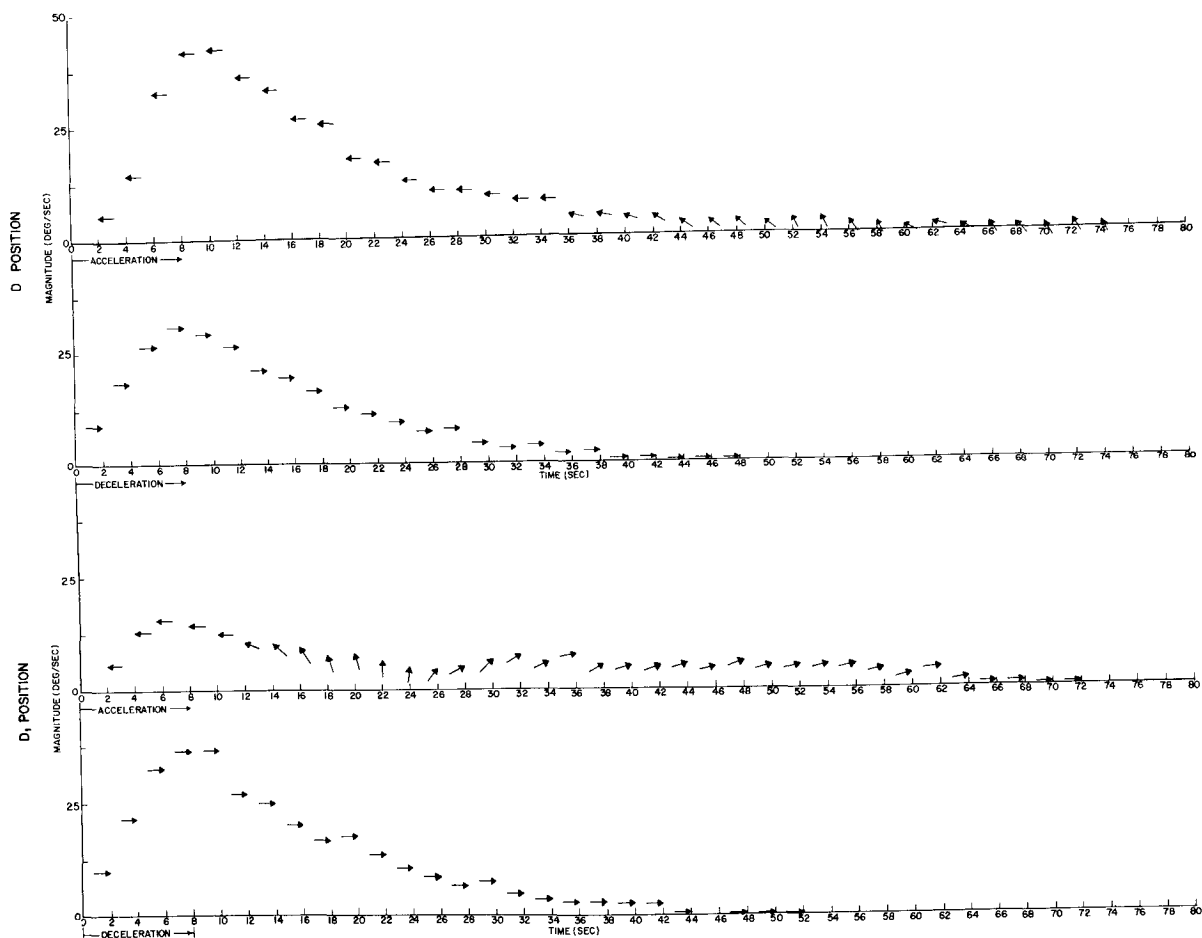


Figure 2.—Vectorial presentation of nystagmus. Response magnitude is indicated by height above the x-axis. (From J. Aerospace Med., ref. 1.)

the response should accordingly be constant. The response, as measured by electronystagmography, should as desired by classic vestibular dogmas obey Ewald's first law stating that the response will be in the plane of stimulation.

The results of our experiments contradicted such attractive simplicity of response patterns (figs. 2 and 3). In several instances, where the orientation of the canal system

relative to the plane of rotation remained unchanged, the magnitude and direction of the nystagmus proved to vary as a function of the subject's heading. For instance heading forward the nystagmus was strong and of long duration. Conversely, when heading backward the nystagmus did not reach its anticipated magnitude; moreover, it lasted shorter than expected and then gradually changed *plane* and direction. The implication

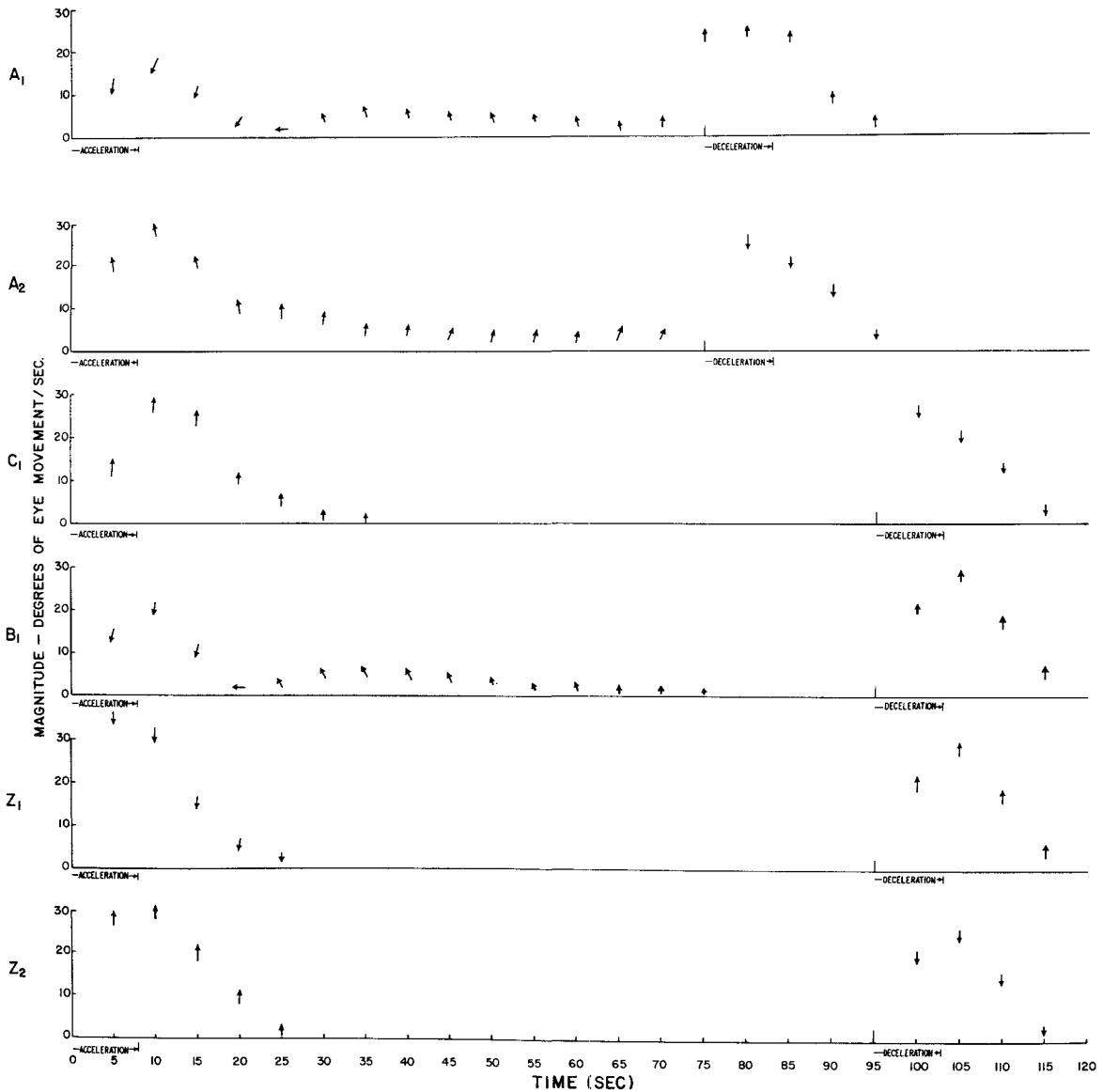


Figure 3.—Vectorial presentation of nystagmus obtained by method of selected beats. (From *J. Aerospace Med.*, ref. 1.)

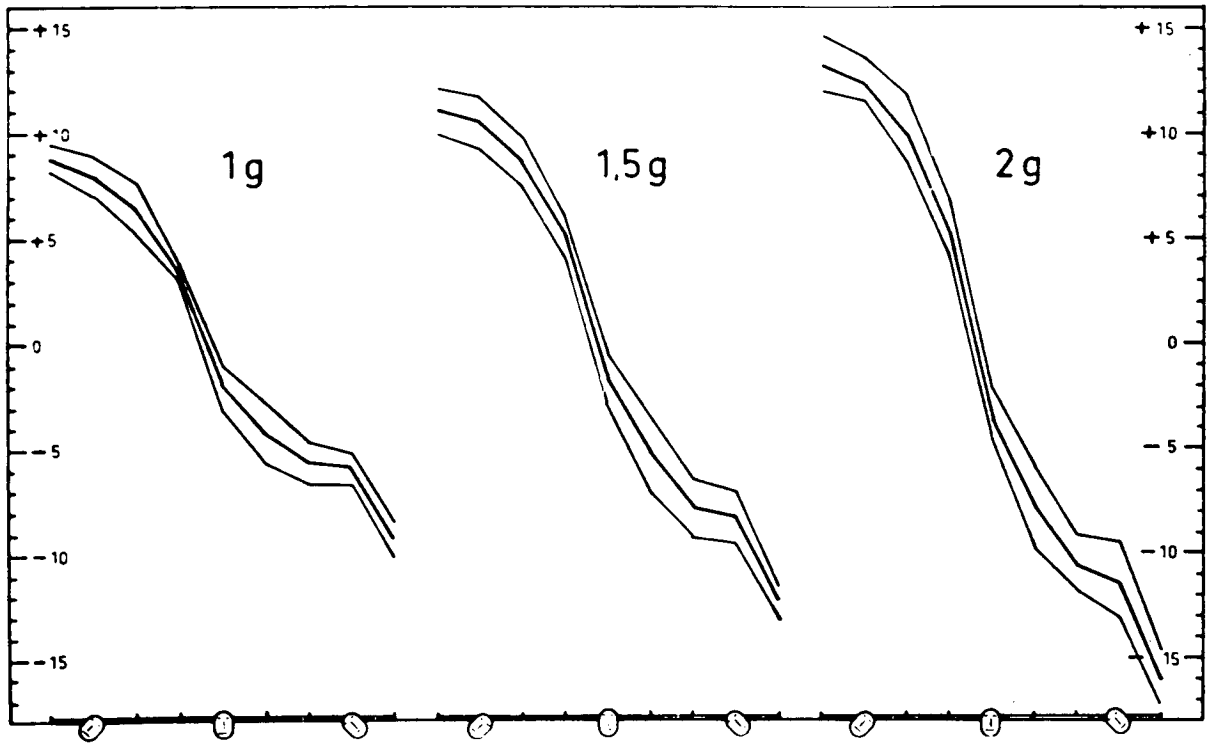


Figure 4.—Counterrolling, the curves obtained at 1G, at 1.5G, and at 2G. (From *Aeromed. Acta*, ref. 2.)

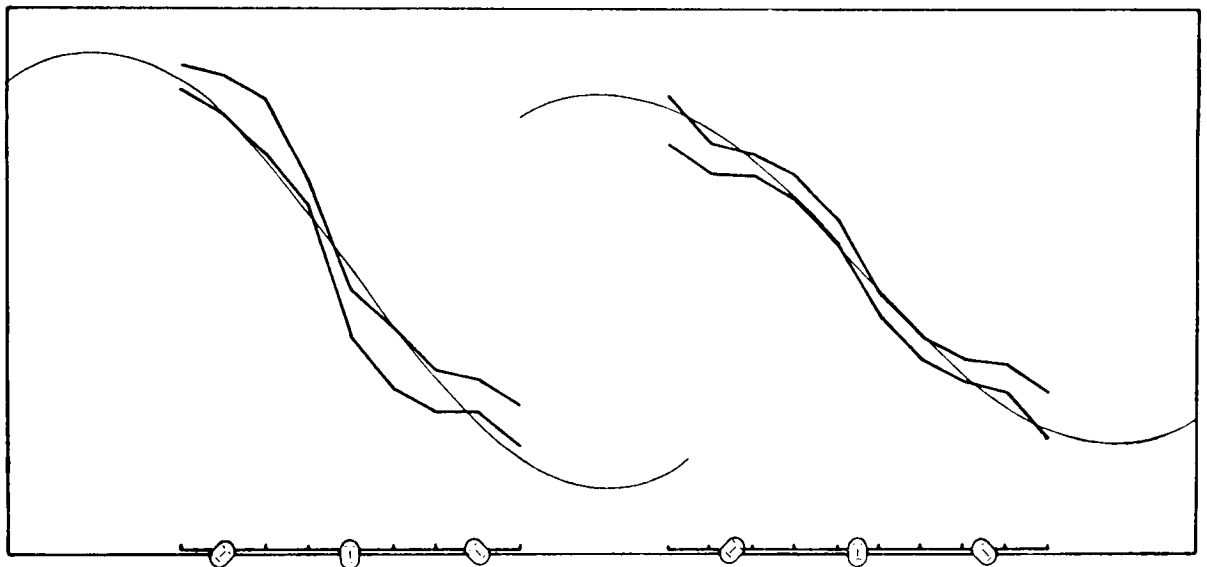


Figure 5.—Counterrolling, line curves fitted to 1G data curve (left) and to curve of estimated otolith effect (right). (From *Aeromed. Acta*, ref. 2.)

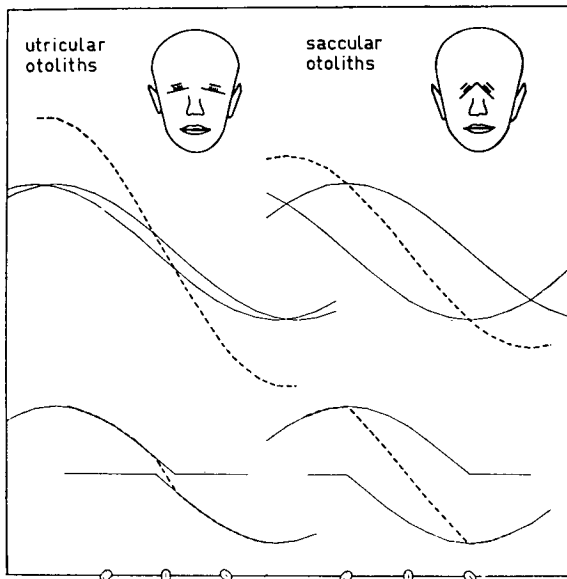


Figure 6.—Otolith signal, predicted signal from each pair of maculae, on assumption that shearing force is active in both directions (upper curves) or in a lateral direction only (lower curves). (From *Aeromed. Acta*, ref. 2.)

of these experiments seems to be that the otoliths can modulate an ampullar signal or, even stronger, have a nystagmus-modifying and a nystagmus-generating power.

As for the otoliths, some recent experiments, performed on our centrifuge by Colenbrander (ref. 2), may be of interest, experiments which to my mind reflect originality and thoroughness. In the free-swinging gondola 7200 observations of counterrolling of the eyes and of the subjective horizontal were made at 1G, 1.5G, and 2G levels in order to establish which component of the force acting upon the otoliths determines its stimulus (fig. 4). Breuer's concept (1874) of the sliding movements furnishing the adequate stimulus enjoyed little popularity mainly because the displacement of the otoliths was hard to demonstrate. Magnus and Quix, both working in Utrecht, proposed in 1924

rival theories of, respectively, pulling and pressure as the active agent.

It was not until 1949 that Loewenstein lent new support to the shearing force theory which gained further support from Van Holst's experiments in 1950. Alas, in 1962 Miller's technically refined measurements could not be fitted to the simple sine function which the shearing force seemed to demand. However, these measurements were taken at 1G, whereas Colenbrander, measuring at different G levels, found a simple linear proportionality between the outward shearing force of the otoliths on the utricular maculae and the counterrolling of the eyes (fig. 5).

The question may be raised why then the 1G counterrolling data do not follow the sine curve.

Further experiments led Colenbrander to believe that it is the impression one has formed about one's spatial position that is superimposed upon the otolithic influence. (See fig. 6.) This impression, stemming from neck and body receptors, would in his centrifuge studies be independent of G. The 1 G counterrolling curve should consequently be a combined curve. The pure otolithic effect showed a good linearity in the range between 1 G and 2 G and it is tempting to extrapolate to 0 G. Further theoretical implications of the 0 G curve, interesting as they are, call for confirmation from a laboratory in space. So here we are back at the reverse of our starting point which is the role of space flight in the exploration of the vestibular organ.

#### REFERENCES

1. LANSBERG, M. P.; GUEDRY, F. E., JR.; AND GRAYBIEL, A.: Effect of Changing the Resultant Linear Acceleration Relative to the Subject on Nystagmus Generated by Angular Acceleration. *Aerospace Med.*, 36, 1965, pp. 456-460.
2. COLENBRANDER, A.: Eye and Otoliths. A Centrifuge Study on the Ocular Response to Otolith Stimulation. *Aeromed. Acta*, 9, 1963-1964, pp. 45-91.

## DISCUSSION

PATTERSON: The steps in Dr. Lansberg's argument that results on a slow rotation platform could not be applied to a rotating space vehicle are not clear to me at all. This is a rather serious broadside attack. I could not follow the argument. If this is true, a lot of money has been wasted and a lot of time spent. Is this an airtight conclusion?

LANSBERG: No, on the contrary. I definitely wanted to say that the experiments have been very valuable in giving us ideas on the process of adaptation and to what extent man is able to cope with this conflicting information. The only thing that differs in the space ship from the situation in the rotating room is that, as conflicts arise in the space station, they occur much more often, and in an unpredictable fashion.

PATTERSON: You mean psychological conflicts, not physical.

LANSBERG: No, physical.

PATTERSON: Haven't you removed at least some of the otolithic stimulation in the space station?

LANSBERG: No, you can only reduce it if you want to go back from 1 G to 1/3 G. You can reduce the amount but whether that will be an advantage, I really don't know. Normal linear accelerations that occur during walking will then be superimposed upon a 1/3 static G level and these accelerations will,

if Weber-Fechner's Law is true also in this situation, have a more pronounced effect.

GRAYBIEL: It was planned that the topic of "rotating environments" would be taken up on the last day of this symposium, but the statement that the results of studies in a rotating room on earth cannot be applied to an orbiting rotating space station deserves a brief comment now as well as the thought that the two vestibular organs should be regarded as one.

Although Dr. Lansberg's main point that in the orbiting spacecraft, man, upright, is oriented at right angles to the axis of rotation and in a rotating room parallel to the axis is a good one and, indeed, is receiving careful study, it may be pointed out that subjects in the Slow Rotation Room become adapted and do not limit their head movements regardless of orientation to the axis of rotation. We believe that information already available may be applied with caution to conditions aloft and that studies with man carrying out activities at right angles to the axis of rotation will provide supplementary information.

With regard to the vestibular organs we will present some additional evidence later on which emphasizes the need not only to keep in mind the individuality of the semicircular canals and otolith organs but their collective influences as well.

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# Influence of the Otoliths on the Duration of Post-Caloric Nystagmus

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The otolith system is the oldest representative of labyrinthine function. It controls the tonus of the striated muscles for posture adjustment. Specific excitation of otolith sensorineural epithelium can be provoked by: (a) changing of the position which modifies the center of gravity; (b) linear acceleration; (c) angular acceleration around the horizontal axis; and (d) centrifugal force.

These stimuli, following the theory of Mach-Breuer (1873), exert pressure, traction or sliding of the otoliths on the macular hair cells. When the head is in the normal position, according to Magnus and De Kleyn (1920), otoliths are above the epithelium and provide pressure and minimal stimulus. When the head is upside down, otoliths are below the epithelium and provoke maximal stimulus by traction. Therefore, there is only one position for minimal reflexes. Using decerebrated cats, Magnus and De Kleyn demonstrated that postoperative rigidity is minimal when the head is in the normal position, and maximal in the upside-down position. After they performed bilateral labyrinthectomy, differences in tonus disappeared.

According to experimental studies by Versteegh (1927), Ulrich (1935), Graybiel et al. (1952), and Fernandez et al. (1960), nystagmus cannot be provoked by stimulation of the otolith system.

Maxwell (1923) was probably the first one to demonstrate that stimulation of the crista ampullaris is dependent on changes

in tension of the utricular hair cells. He sectioned the horizontal semicircular canal near its utricular end and applied different stimuli.

Marimoto (1955) and Owada et al. (1963), performing experiments on rabbits, pointed out that the otolith system has a regulatory and controlling effect upon the cupula ampullaris system. If the balance between ampulla and otolith is changed, the ampulla is spontaneously charged to elicit nystagmus. Nystagmus jerks are increased or decreased according to change in endolymphatic pressure on the otolith macula.

Bergstedt (1961) carried out caloric tests on human subjects in a centrifuge at 1.0 to 1.8 G. He was able to show that vestibular caloric tests are influenced by the strength of the gravitational field. The maximum intensity of nystagmus (eye speed in deg/sec) and duration increase at higher values of gravitational force.

The purpose of the present study was to investigate the influence of the otolith organ on post-caloric nystagmus in the cat.

## METHOD

Experiments were carried out on 22 adult cats with normal responses on caloric stimulation. Needle EEG electrodes (Type E-2B, subdermal) or chronic zygomatic electrodes were used, and fixation of the head was achieved by a bar inserted through the teeth (figs. 1 and 2). Cats were blindfolded and tested in a semidark room.



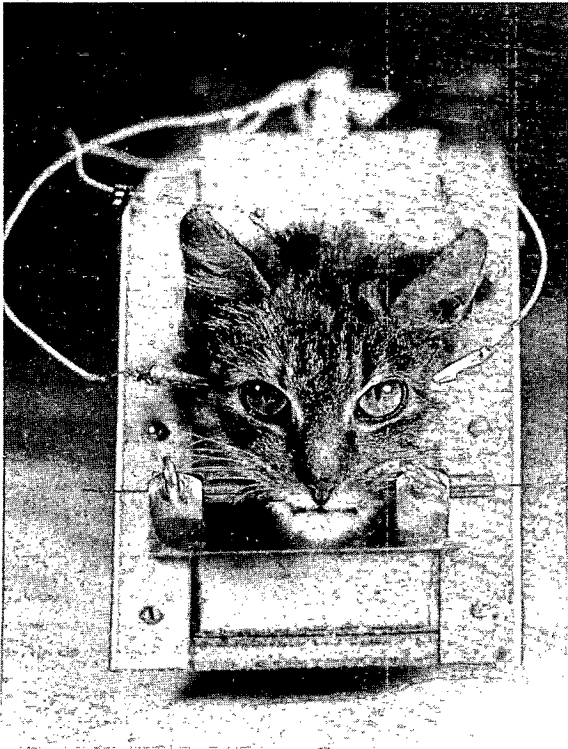


Figure 1.—Immobilization of cat's head.

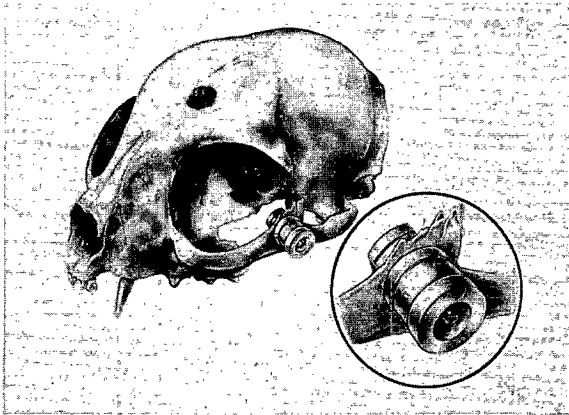


Figure 2.—Sketch showing placement of bar.

In order to place the lateral semicircular canal in a vertical position with the ampulla up for caloric testing, the head was raised from the horizontal prone position until the fronto-occipital axis reached a  $45^\circ$  angle (fig. 3). Fronto-occipital axis coincides with a position of the horizontal semicircular canal

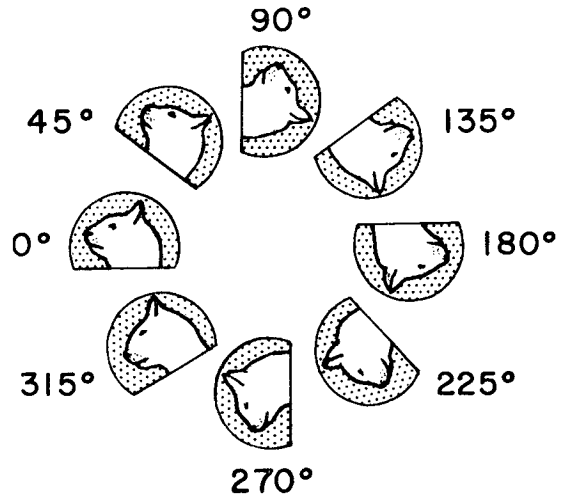


Figure 3.—Angle of fronto-occipital axis.

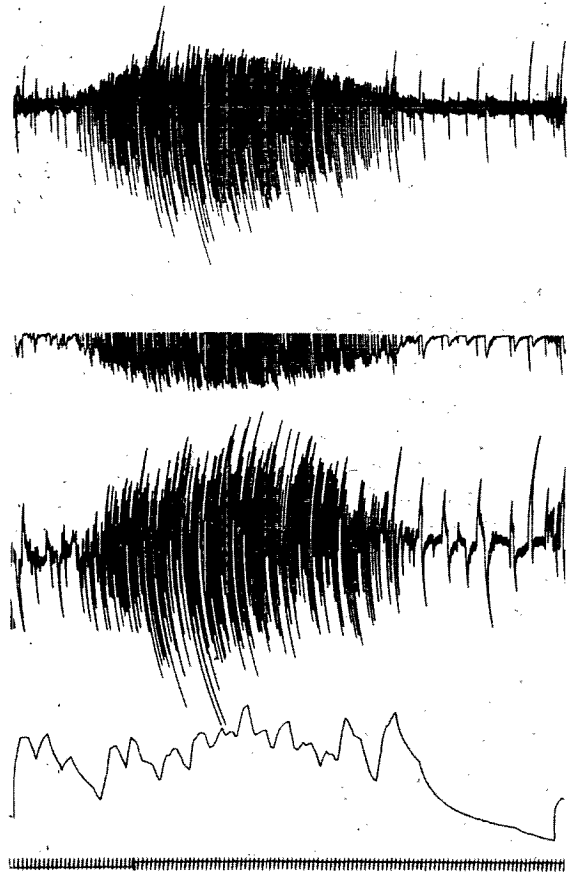


Figure 4.—Nystagmus registration.

(Rademaker, 1926). In this position caloric stimulation was performed. Water temperature ranged from 10 to 49° C, and the duration of each irrigation was 40 sec. After an interval of 10 minutes, the cat's head was raised until the fronto-occipital axis reached an angle of 135°. In this position the otoliths provoked maximal stimulation (traction) of the otolith hair cells. The caloric test was then repeated with the same water tempera-

ture and the same length of irrigation, provoking nystagmus in all 22 cats.

A four-channel recorder (Offner Type R) for nystagmus registration was used (fig. 4). Channel 1 recorded the velocity of the fast and slow eye movement, and Channel 2 registered the derivated curve of the slow phase. The actual eye movement was recorded through the third channel. Channel 4 showed the average frequency. The paper speed was 1 mm/sec. Conventional nystagmus pattern was registered by Tektronix oscilloscope and Polaroid camera (fig. 5). Duration, maximum slow phase and maximum fast phase velocity of post-caloric nystagmus were recorded and analyzed.

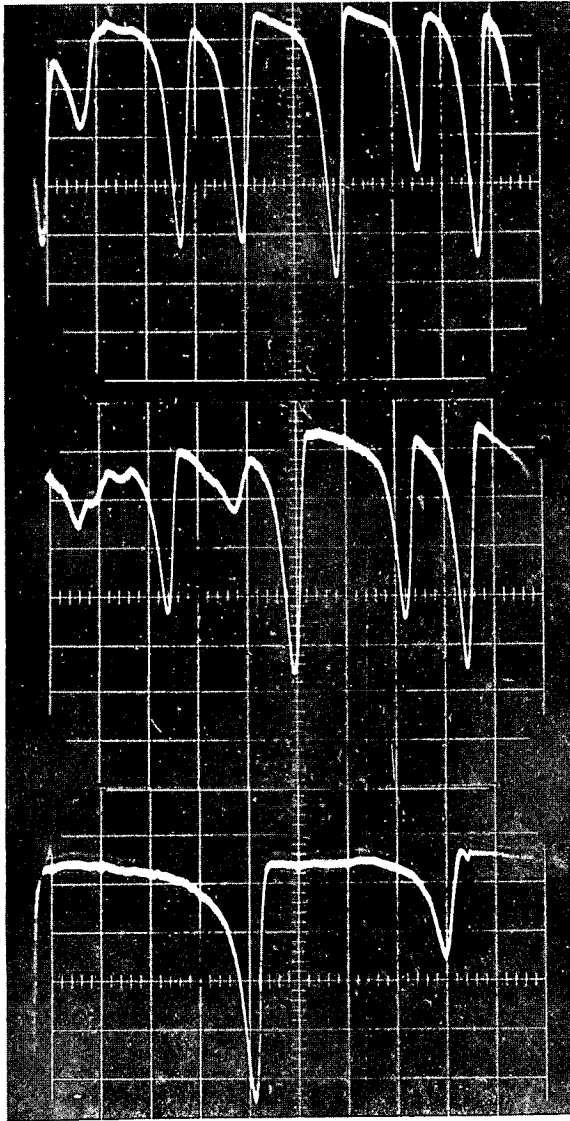


Figure 5.—Nystagmus curve as registered on oscilloscope.

RESULTS

As a preliminary to this study an experiment involving repetitive caloric testing for 18 consecutive days was carried out on 4 cats. It was found that duration of post-caloric nystagmus appeared to be a more irregular parameter than eye speed in the slow phase. The record of one cat is shown in fig. 6. Irrigation No. 6 released extremely long nystagmus duration (14 min), while eye speed showed normal values. The fast phase velocity followed the pattern of the slow phase velocity. Nystagmus duration in

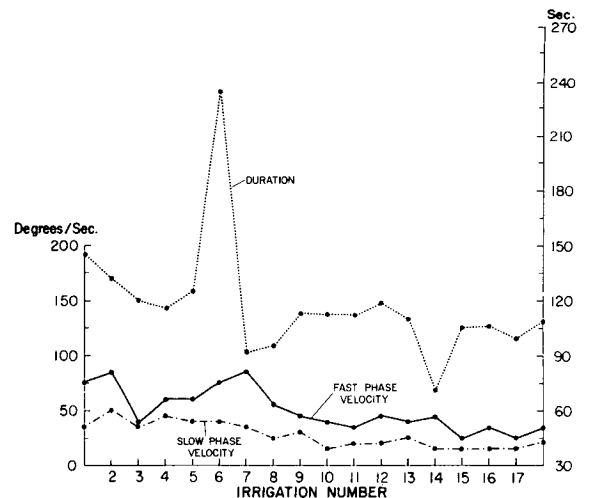


Figure 6.—Post-caloric nystagmus and eye-speed record of one cat.

caloric testing in the cat at the same physical stimulus could vary between a few seconds (fig. 7) and as long as 14 minutes (fig. 8). This indicated that duration of post-caloric nystagmus in the cat is influenced by different mechanisms than velocity of the slow and fast phase.

In the principal study, the results of the first group of 11 cats are shown in figure 9(a). Water temperature was  $16^{\circ}$  C in all caloric tests. Nystagmus duration was significantly longer in the right ear in this group when the fronto-occipital axis was placed in a  $135^{\circ}$  angle in cats 1, 2, 7, 8, and 10. Less difference was found in cats 3, 4, and 5 (20–40 sec). Two cats (9 and 11) did not indicate any difference. In the left ear, the difference in nystagmus duration was even more obvious between  $45^{\circ}$  and  $135^{\circ}$  angles (cats 5, 6, 7, 9, and 11) (fig. 9(b)).

Maximum slow phase velocity (figs. 10 (a), (b)) and maximum fast phase velocity (figs. 11 (a), (b)) failed to demonstrate any increase in the intensity of nystagmus upon caloric testing at the  $135^{\circ}$  angle.

In the second group of 11 cats (table I) the experiment was repeated with different water temperatures ( $12^{\circ}$ ,  $14^{\circ}$ ,  $19^{\circ}$ ,  $24^{\circ}$ ,  $27^{\circ}$ , and  $49^{\circ}$  C). The findings of this group were quite similar to those of the first one. Differences in duration of nystagmus for the angles of  $135^{\circ}$  and  $45^{\circ}$  were greater when a colder stimulus ( $12$  and  $14^{\circ}$  C) was applied.

Mean group values for 22 cats are pre-

sented in table II. When the fronto-occipital axis was placed at an angle of  $135^{\circ}$ , mean duration of post-caloric nystagmus was 68 sec longer than at an angle of  $45^{\circ}$ .

#### DISCUSSION

The relationship between the otolith system and nystagmus has been discussed by many authors. Borries (1920) was probably the first one to state that post-caloric nystagmus is the result of the cooling or warming of the otolith organ. De Kleyn and Lund (1924), following Witmaack's method, destroyed the otoliths by turning a centrifuge at 2,000 revolutions per minute. However, they were still able to produce caloric reaction in those animals, disproving Borries' theory.

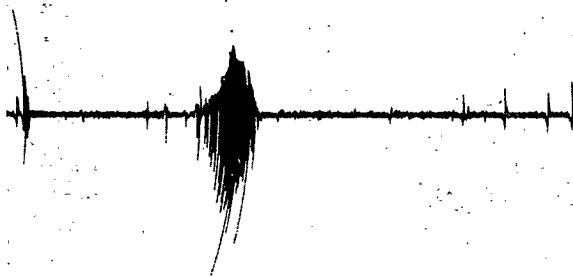


Figure 7.—Short nystagmus duration.

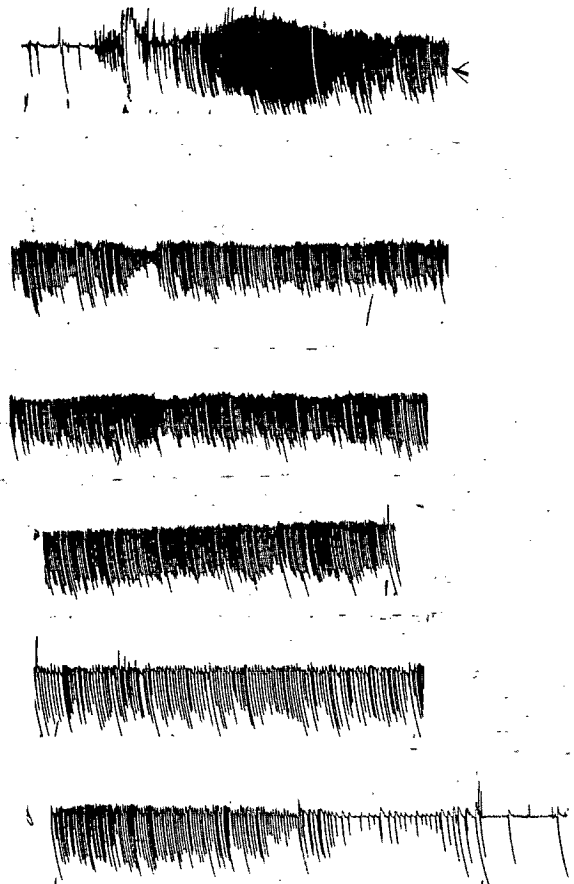


Figure 8.—Long nystagmus duration.

Table I.—*Study of Nystagmus Duration in 11 Cats*

Cat no.	Temp.	Ear	Angle	Duration, (sec)	Maximum velocity	
					Slow phase, deg/sec	Fast phase, deg/sec
12	12° C	R	45°	168	70	100
			135°	277	50	100
		L	45°	137	35	60
			135°	223	50	75
13	14° C	R	45°	92	35	45
			135°	145	20	20
		L	45°	86	20	55
			135°	112	20	30
14	19° C	R	45°	168	80	120
			135°	169	65	105
		L	45°	156	95	125
			135°	259	60	65
15	24° C	R	45°	120	80	80
			135°	228	70	70
		L	45°	117	70	75
			135°	137	75	75
16	27° C	R	45°	91	55	100
			135°	146	80	80
		L	45°	144	70	70
			135°	161	65	85
17	27° C	R	45°	126	70	115
			135°	135	70	105
		L	45°	104	75	115
			135°	120	50	70
18	27° C	R	45°			
			135°			
		L	45°	97	70	160
			135°	115	75	150
19	27° C	R	45°	120	125	150
			135°	125	100	125
		L	45°	128	70	90
			135°	186	100	155
20	27° C	R	45°	134	25	45
			135°	169	65	100
		L	45°	127	40	70
			135°	145	45	85
21	49° C	R	45°	80	35	70
			135°	100	40	70
		L	45°			
			135°	108	5	2
22	49° C	R	45°	89	20	35
			135°	159	50	70
		L	45°	83	15	35
			135°	134	35	60

Table II.—Mean Value: 22 Cats

	Angle, deg	Duration, sec
Right ear -----	45	119.15
	135	187.10
Left ear -----	45	111.52
	135	179.52

Versteegh (1927), Ulrich (1935), Jongkees (1950), Graybiel et al. (1952), Sullivan (1959), and Fernandez et al. (1960) denied any appearance of nystagmus after destruction of the otolith organ, or utricular nerve.

Maxwell (1923), Marimoto (1955), Bergstedt (1961), and Owada et al. (1963) hypothesized that the otolith system has a regulatory effect upon the cupula ampullaris system.

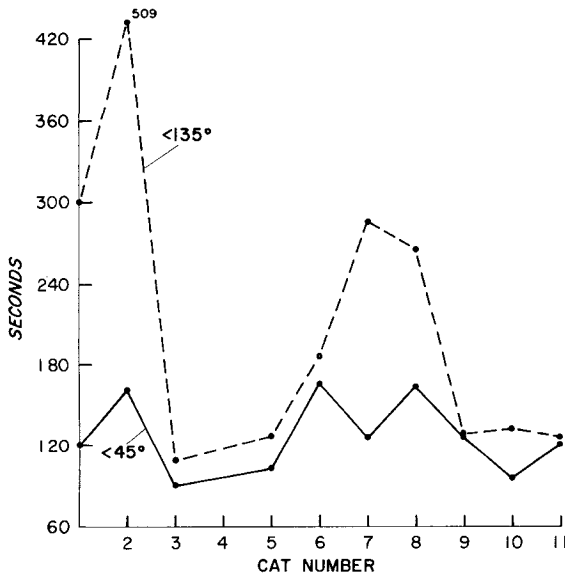


Figure 9(a).—Nystagmus duration—cat right ear.

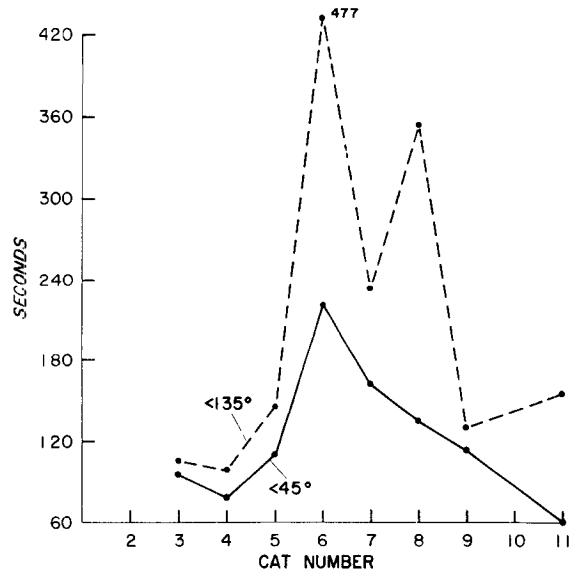


Figure 9(b).—Nystagmus duration—cat left ear.

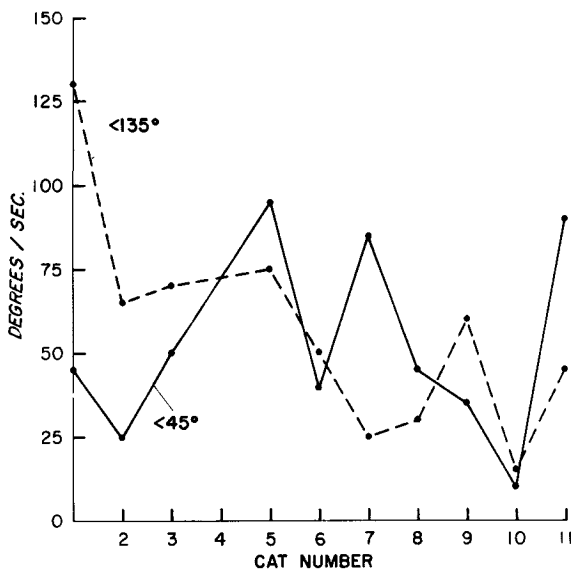


Figure 10(a).—Maximum slow phase velocity—cat right ear.

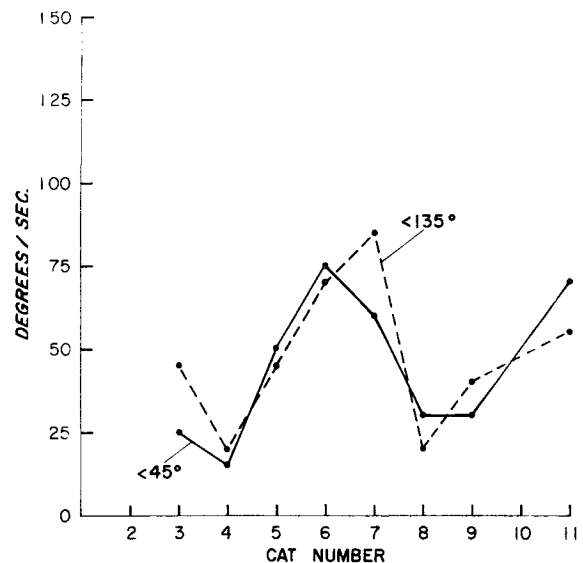


Figure 10(b).—Maximum slow phase velocity—cat left ear.

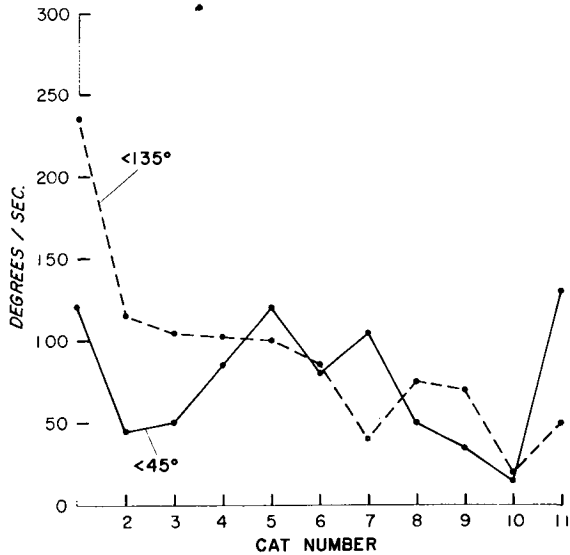


Figure 11(a).—Maximum fast phase velocity—cat right ear.

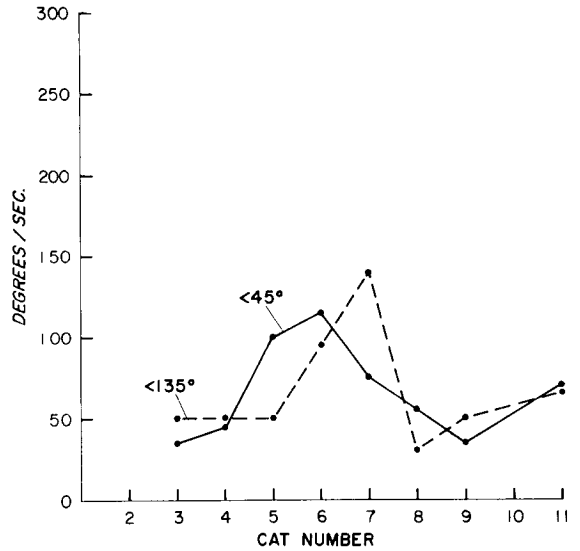


Figure 11(b).—Maximum fast phase velocity—cat left ear.

From a review of the literature it appears that the present method of stimulating the otolith system (fronto-occipital axis at  $135^\circ$  angle) with simultaneous caloric testing in the cat has never been reported before.

Enteritis, pneumonitis and otitis media are three illnesses which interfere sometimes with experimentation in the cat. Great individual differences in nystagmus response between different cats could be attributed to these illnesses, to the S-shaped form of the external meatus, to the cat's ability for closure of the ear canal by muscle contraction, and to the presence of ear wax in the meatus. The inhibition of caloric nystagmus could be explained by lack of mental activity if the cat becomes sleepy or by some other mechanism, where the otolith system apparently plays an important role. The

duration of post-caloric nystagmus is a much more irregular parameter than eye velocity. By changing the center of gravity and moving the fronto-occipital axis from  $45^\circ$  to  $135^\circ$  angle, the otoliths provoke traction and maximal physiological stimulus on the macular hair cells. If a caloric test is performed at this angle, which is not the optimal position for the lateral semicircular canal, there is a significant increase in duration of post-caloric nystagmus with no difference in maximum intensity of nystagmus. Therefore, it could be hypothesized that the otolith system has a controlling regulatory mechanism over post-caloric duration of nystagmus through central pathways. Nystagmus duration increases when the otoliths provoke stronger stimulus on the otolith membrane.

REFERENCES

MACH, E.: Physicalische Versuche über das Gleichgewicht.—Sitzungsb. akad. d. Wissensch. 68, 1873, p. 124.  
 MAGNUS; AND DE KLEYN: Experimenteller Physiologie des Vestibular-apparates. Handb. d. Neurol. d. Ohres, 1, 1920, p. 465.  
 VERSTEEGH, C.: Ergebnisse partieller Labyrinthextirpation bei Kaninchen. Acta oto-laryng., Stockholm, 11, 1927, p. 393.  
 ULRICH, H.: Die Funktion des otolithen geprüft direkte mechanische Beeinflussung des utriculustolithen am lebenden hecht. Pflüg., arch. ges. Physiol., 1935, pp. 235-565.  
 OWADA, U. et al.: The Otolithic Reaction on Nystagmus by Caloric Stimulations. Acta oto-laryng., Stockholm, suppl. 179, 1963.  
 BERGSTEDT, M.: The Effect of Gravitational Force on the Vestibular Caloric Test. Acta oto-laryng., Stockholm, 53, 1961, pp. 551-562.

## DISCUSSION

COMMENT: There are many moments that can be responsible for such changes in nystagmus response when the cat is placed in an unnatural and unpleasant position. Emotional factors could play a very important role. Measuring other parameters of induced nystagmus in sequence of time unit, as, for instance, the frequency, would give us more information.

MILOJEVIC: I agree that frequency is a very important parameter. In another study on human subjects and cats and using caloric stimulus in five ordered durations of irrigation (5, 10, 20, 40, and 60 sec), we found that duration of nystagmus could vary quite independently from frequency and velocity. Slow and fast phase velocity show a similar pattern. According to this study, we got the impression that duration is a totally different parameter and under different influences than velocity or frequency in caloric testing.

COMMENT: I was going to question if this difference in duration of caloric nystagmus in the cat by changing the fronto-occipital axis can be attributed to the otolithic action or to some other factors. By changing the position of the horizontal semicircular canal, we change the circulation of fluid in semicircular canals.

BERGSTEDT: Dr. Milojevic mentioned the study I made with caloric tests in the centrifuge. The results show a certain relation between nystagmus output and G during this test. I think this result is in line with and supporting Bárány's own concept about the caloric test, i.e., about the thermic flow in the semicircular canals. I do not, however, attribute the results to any effect from the otolith organs. I have later made further studies and these give similar results. I have syringed both ears with water of the same temperature, etc., and no unexpected results occur.

TOROK: I would like to suggest that we extend the field of observation of the provoked vestibular nystagmus beyond the duration. As it has been found and proven, a more accurate expression of vestibular sensitivity can be obtained by counting and evaluating the frequency of the nystagmus. It provides more reliable and more constant information of the state of vestibular sensitivity than nystagmus duration. Compared with the measurement of the velocity of the slow phase of the nystagmus, the frequency closely follows the former. However, it is much simpler and easier to assess the frequency characteristics by counting the number of nystagmus beats in successive 5 second time intervals. The slow component velocity measurement, on the other hand, needs elaborate electronic equipment.

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# Response of Single Cells in Cat Brain Stem to Angular Acceleration in the Horizontal Plane

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16113

## SUMMARY

Single units within the brain stem of deeply anesthetized cats were observed for long periods during which the animal was repeatedly subjected to long duration angular accelerations. The head was centered over the vertical axis of rotation and the horizontal stereotaxic plane of the head was parallel to the plane of rotation. Observations were made of adaptation, habituation, and forms of response discharge. Electrode tip locations were confirmed histologically.

1. No regular evidence for a reduction in discharge rate during the application of a long angular acceleration could be found.
2. No regular evidence for a reduction in discharge rate from acceleration to acceleration could be detected.
3. There are indications of changes in patterning of the discharge with repeated stimulation which may reflect habituation.
4. A new form of response is described which appears to act as a binary switch, in that it holds a discharge rate for long periods after a stimulation, and is turned on and off by alternating negative and positive angular accelerations.

Author

## INTRODUCTION

These observations were aimed at three of the more usual questions asked about sensory systems. What is the neural code for the various stimulus parameters? Is there adaptation during an extended application of the stimulus? And, does repeated stimulation of the sensory system produce a successive reduction in response magnitude?

These questions demand very special equipment for their investigation. The study of adaptation requires a stimulator which is capable of applying constant angular accelerations of long duration separated by long periods of constant velocity. The examination of the effects of repeated stimulation requires a stimulator capable of offering successive accelerations of reliable accuracy.

The stimulator must be so stable and free from transients and vibration that activity from a single neural unit can be recorded for periods longer than one hour. Finally, microelectrode and electronics technique is needed which is sufficient to successfully monitor single unit activity from continuously rotating preparations without intermediate adjustment.

Briefly, deeply anesthetized cats were centered over the vertical axis of rotation. Microelectrodes were advanced into the brain stem and directed by stereotaxic coordinates into the region of the vestibular nuclei. Isolated units were studied with long duration constant angular accelerations repeated several times. Strict criteria for acceptable data were established: we had to have held an



isolated unit for at least one stimulus series of eight accelerations which lasted an hour; we required full and adequate films of the response for 15 seconds prior to each acceleration to 3 minutes following the acceleration; and, we demanded a clear histological verification of each electrode tip location. Our work has only just begun and we have data from but 21 units that meet these stringent requirements. Here we offer a preliminary report on some of the salient aspects of the data, and in particular, on some response types not described heretofore.

#### METHODS

##### Stimulator

A circular turntable 1.25 meters in diameter was mounted on a vertical shaft, and driven through a friction coupling by a pneumatic wheel pressed against the rim (fig. 1). The precision-ground vertical shaft and the oil-bronze smooth radial and thrust bearings were enclosed in a column of oil. The drive system was a hydraulic pump and motor with servo amplifier control employing closed feedback loops from the slide block and a tachometer on the output. The turntable structure and the hydraulic motor were mounted within a radiofrequency shielded, lightproof, and ventilated room. The hydrau-

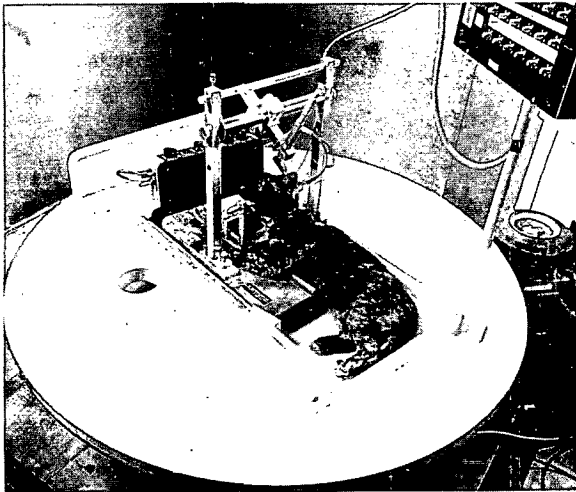


Figure 1.—Turntable structure with cat in stereotaxic apparatus and positioned with the center of head over axis of rotation.

lic pump and control console were mounted outside of the room.

##### Procedure

Cats were deeply anesthetized with Nembutal and mounted within the stereotaxic head-holder. Depending upon the procedure selected for that animal, (a) a small defect was made in the skull for the electrode entry, or (b) this small defect was made and another one placed on the contralateral side through which the eighth nerve was sectioned, or (c) the major portion of the dorsal aspect of the cerebellum was exposed, the cerebellum aspirated, and the eighth nerve sectioned on one side. The animal was then positioned on the turntable with the head centered over the axis, and the horizontal stereotaxic plane of the head parallel to the turntable surface. The electrode was tilted back  $30^\circ$  from the transverse plane in order to avoid the tentorium and then advanced in a sagittal plane.

The friction drive wheel was disengaged from the turntable rim and the table gently oscillated between successive advances of the electrode manipulator until a single cell which responded to angular acceleration was sufficiently isolated for definitive recording. The friction drive was then reengaged to the turntable rim, the shielded room closed, and the animal accelerated in total darkness by a subthreshold acceleration ( $0.22^\circ/\text{sec}^2$ ) to a clockwise (CW) velocity of either 15 rpm or 20 rpm.

The stimulus series that followed consisted of a number of constant angular accelerations, during which the turntable passed through zero velocity and terminated at a velocity, equal to but opposite in direction from that of the base velocity. For example, most units were started at 15 rpm CW and then underwent a  $4^\circ/\text{sec}^2$  negative acceleration of 45 sec duration which brought the turntable from the base velocity of 15 rpm CW, through zero velocity to 15 rpm counterclockwise (CCW). Five minutes later, a positive  $4^\circ/\text{sec}^2$  acceleration of 45 seconds duration was impressed and the turntable

passed from 15 rpm CCW through zero velocity to 15 rpm CW.<sup>1</sup> A series of trials consisted of not fewer than eight such maneuvers, four in each direction of acceleration, and each maneuver spaced at 5 minute intervals.

Units with high thresholds underwent a similar series but from 20 to 20 rpm and with accelerations of  $8^\circ/\text{sec}^2$  and 30 seconds duration. A very few low threshold units were tested at 7.5 to 7.5 rpm with accelerations of  $2^\circ/\text{sec}^2$  and 45 seconds duration. Some cells were tested at more than one acceleration level. Occasionally a cell could be held for over three hours of continuous rotation.

Note, in particular, that this form of stimulus series helps to identify those effects which may be due to linear (centripetal) acceleration or noise from the response to angular acceleration. Any effective centripetal acceleration was the same at both the beginning and the end of each angular acceleration. Noise was also related to angular velocity. Thus, responses to these extraneous stimuli could be identified because they were dependent entirely upon angular velocity and were not related to angular acceleration.

#### Recording

Electrodes were electro-polished steel insect pins according to the method of Green (1958), but insulated with successive coats of baked Formvar. Electrode tips were less than 6 microns in diameter, and tested with saline immersion under a microscope to observe if a small bubble was produced at the tip when a current was passed through the electrode. No estimate of the area of the exposed tip is available. The single-ended signal from the electrode was led through a cathode follower mounted on the stereotaxic instrument, through instrument slip rings

<sup>1</sup> A convention of analytical mechanics is observed in this terminology. The turntable, as viewed from above, undergoes a positive angular acceleration during a period of decreasing CCW velocity or increasing CW velocity. Similarly, the turntable undergoes a negative angular acceleration during a period of decreasing CW velocity or increasing CCW velocity.

and thence to amplifying and recording equipment outside of the turntable room. The signals were filtered through a frequency band between 300 and 3000 cps, and photographed from an oscilloscope at a 50 mm/sec film speed.

#### Histology

Following the recording, a small current was passed through the electrode by attaching the anode of a d.c. source to the electrode. The current in microamperes and its time of application in seconds were adjusted so that the product of the two amounted to approximately 150; more correctly, 0.00015 coulomb. The animal was then perfused with saline followed by 10% formalin to which 0.5% potassium ferrocyanide and 0.5% potassium ferricyanide had been added. The brain was removed and placed in this perfusing solution for not less than three days before subsequent processing. This procedure, adapted after the method of Green (1958) produces by virtue of the prussian blue reaction, a small blue spot at the electrode tip site which just can be detected by the unaided eye while preparing frozen sections. A series of 40-micron sections in the region of the dyed spot were then stained with chrysosleucht violet, and in some cases neutral red. Identification of electrode locations was made by reference to Brodal & Pompeiano (1957), Brodal, Pompeiano, and Walberg (1962), and Verhaart (1964).

### RESULTS AND DISCUSSION

#### Major Cell Categories

Two broad categories of types have been found. The first type is that described by Ross (1936) for the frog, Löwenstein and Sand (1936) for the dogfish, Adrian (1943), Gernandt (1949), Eckel (1954), and Duenzing and Schaefer (1958) for the cat. This cell follows the classical description for the function of the lateral canal as described by Ewald (1892) in which it was deduced that utriculopetal movement of the cupula should produce a maximum response, and that utriculofugal movement of the cupula

should produce but little response. This first category was termed Type I by Gernandt, but we prefer to term it an "Ewaldian" unit and denote that it increases its firing rate with positive acceleration and decreases its resting rate (if any) with a negative acceleration, when the electrode is on the right side and recording activity originating in the right ear. The opposite obtains for an electrode on the left side; that is, an increase in firing rate with a negative acceleration and a decrease in the resting rate (if any) with a positive acceleration.

We prefer to call cells from a second broad category "non-Ewaldian" units: units which increase their discharge rate with a negative acceleration and decrease their resting discharge (if any) with a positive acceleration when the electrode is on the right side and recording activity originating from the right ear. The opposite obtains for an electrode on the left side; that is, an increase in firing rate with a positive acceleration and a decrease in resting rate (if any) with a negative acceleration. Duensing and Schaefer (1958) found them in the vestibular nuclei and called them Type II, which confuses the issue because the new Type II does not agree with the former Type II designation of Gernandt's. These two categories fit all of the cells we have recorded.

We have not found Gernandt's Type II cell, a cell which increases its rate with acceleration of either sign. Nor, have we clearly confirmed a Type III cell; a cell that decreases its firing rate with accelerations of either sign. On one occasion we did find a cell during the preliminary examination using hand-driven stimulation which appeared to be a Type III by Gernandt's terminology; the resting discharge turned off with acceleration in either direction. A subsequent series of programmed stimulations was started but our maximum acceleration of  $8^\circ/\text{sec}^2$  was below threshold for this cell and we were unable to confirm its classification. Great care must be taken in accepting data obtained with accelerations beginning and ending at zero

velocity. An activity may be otolithic in origin and a small displacement from the center of rotation may be sufficient to alter its resting discharge in a like manner irrespective of the direction of acceleration. A symmetrical velocity program, such as used here, is clearly required to confirm Type II and Type III cells. Duensing and Schaefer (1958) found Gernandt's Types II and III and Eckel (1954) found Type II, but both investigations used velocity programs starting and ending on zero. Further, Eckel's confirmation of a Type II cell is not well supported by the reproduction of a record in which activity from not one but several cells appears (see Eckel, figs. 3(a) and 4(a), p. 495).

We are convinced that the non-Ewaldian reacting unit is reflecting activity originating in one of the vertical canals of the labyrinth on the same side as that in which the electrode is positioned. When the stereotaxic horizontal plane is parallel to the plane of rotation, it appears that all three canals have a sufficient projection on the horizontal surface to receive a significant stimulation during angular acceleration. Löwenstein and Sand (1940) have shown that a vertical canal will produce what appears to be non-Ewaldian activity with horizontal rotation of the head, and Ross (1936) observed that even a small misalignment of the vertical canal from a true orthogonal orientation with the horizontal plane led to adequate stimulation.

We have performed several supplementary experiments in which the horizontal stereotaxic plane was tipped nose-down to an angle of  $30^\circ$  with the horizontal. Whereas the population of Ewaldian and non-Ewaldian cells are about half and half with the horizontal stereotaxic plane parallel with the rotating plane, only Ewaldian units have been found with the  $30^\circ$  declination. Similarly, only once in many attempts, has a non-Ewaldian cell first found during parallel orientation continued to fire in the same manner when subsequently tipped to  $30^\circ$ . Adrian (1936) and Duensing and Schaefer

(1958) tilted the nose down from the horizontal by  $45^\circ$ , and Eckel (1954) used a  $35^\circ$  declination; yet only Duensing and Schaefer found non-Ewaldian units. In none of these three cases was the declination determined by an accurate reference to the stereotaxic horizontal, and the difference in findings is likely due to differences in tilt.

Duensing and Schaefer, employing light ether anesthesia, were inclined to believe that the non-Ewaldian units were not seen previously because of a sensitivity to narcosis and decerebration. They considered two possibilities for their origin. One was that the non-Ewaldian unit reflected Ewaldian activity from the contralateral ear conducted across the midline. A second possibility was that this reversed action was due to an inverse recoding within the vestibular nuclei themselves. Inasmuch as we employed very

deep barbiturate anesthesia, it is unlikely that the non-Ewaldian units are entirely susceptible to anesthesia and as will be seen below, our observations clearly show that these units can be found after contralateral eighth nerve section; a procedure that dispenses with the contralateral influence possibility. The possibility that there is a recoding is intriguing, but yet unproven. Parenthetically, Gernandt (1950) demonstrated several non-Ewaldian units much higher in the brain stem (inferior colliculus) and attributed them to crossed secondary fibers; the crossing at least was confirmed by caloric irrigation of the contralateral ear.

#### Response Forms

Figure 2 reproduces a recording sample from a non-Ewaldian unit that was found, in this case, not in the vestibular nuclei but

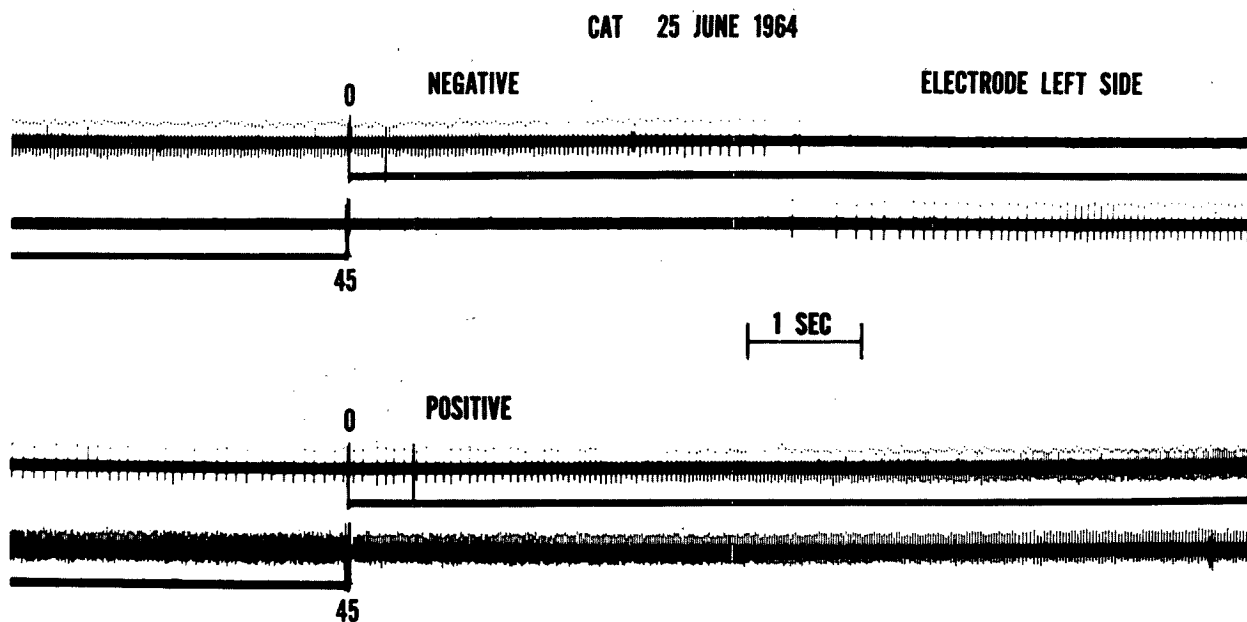


Figure 2.—Cat 25, June 1964: electrode tip in this sample recording was confirmed to be in abducens nucleus on left side. These non-Ewaldian responses are two of a series of alternating negative and positive  $4^\circ/\text{sec}^2$  accelerations of 45 sec duration. Only segments of full recordings are shown; prior resting discharge and first few seconds of acceleration, followed on second line by last few seconds of stimulus period and beginning of recovery. Relay artifacts from function generator for stimulator are prominent at both onset and end of accelerations.

in the abducens nucleus. Note that negative acceleration turned off the resting discharge, and a positive acceleration increased it.

Two new response forms are shown in figures 3 and 4. In figure 3, the non-Ewaldian cell after some latency (albeit long in this instance) rises in discharge rate and appears to be approaching an asymptote within the 45 second duration of the stimulus. Following the stimulus, the discharge declines slowly toward its beginning level. Note that in the turn-off direction, the cell shows some post-stimulus rebound. The high reliability of this cell is particularly noteworthy, but of special interest is that there is no

activity whatsoever in the four trials at 5 minutes after the negative acceleration, and there is a continuing activity in three of the four trials 5 minutes after the positive acceleration. Such a long duration effect appears to be almost "hysteresis" like and is not an altogether uncommon finding.

An even more prominent long-duration effect is seen in figure 4, in which an Ewaldian cell that takes the form of a "memory" unit in that it indicates the direction of the last acceleration for periods of at least 5 minutes. In this series, the first acceleration was of negative sign and found the cell already in its "on" state. Thereafter, however,

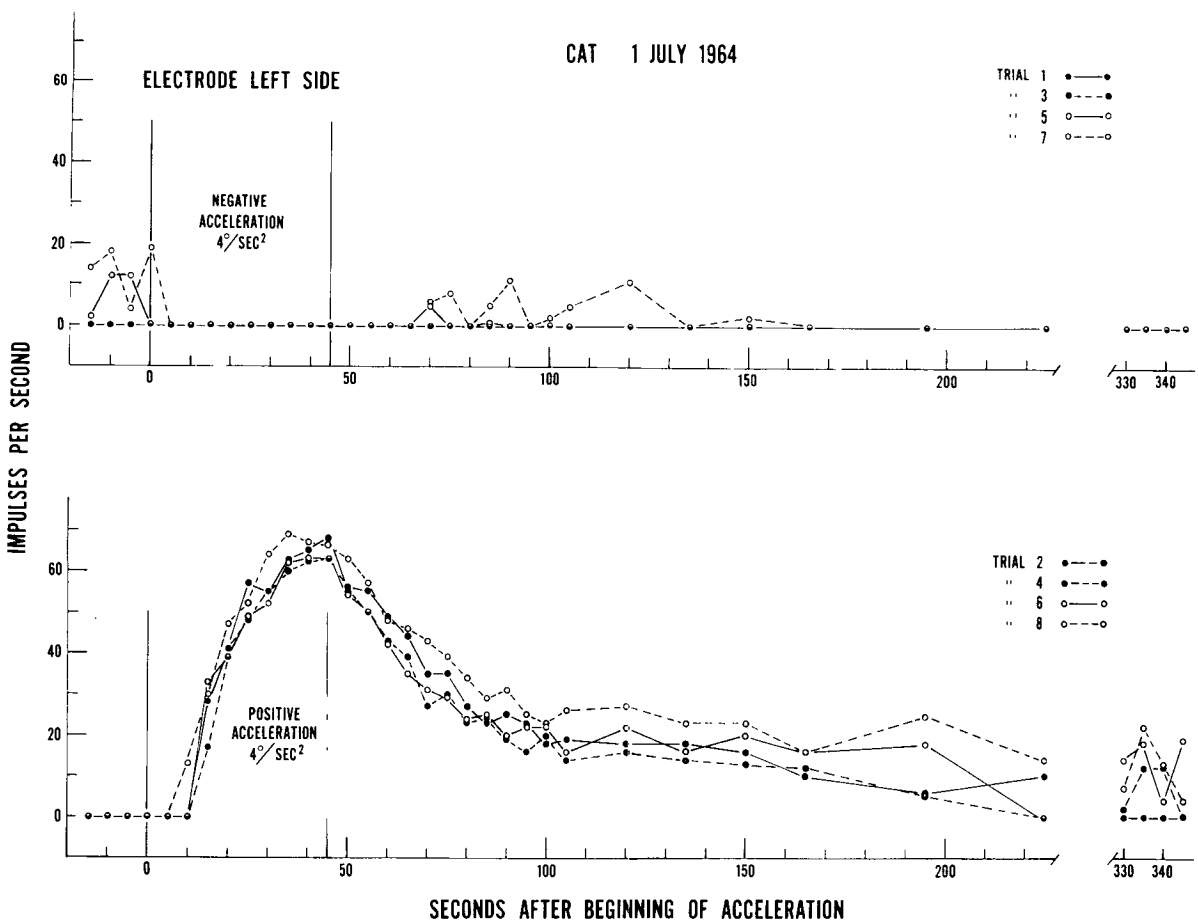


Figure 3.—Cat 1, July 1964: electrode tip was confirmed to be in medial vestibular nucleus on left side. All eight accelerations of an alternating series of  $4^\circ/\text{sec}^2$  magnitude and 45 sec duration are plotted. Five minutes separated the stimuli, of which the first 3 minutes of the intertrial period was recorded. Note the trial-to-trial reliability of this non-Ewaldian cell.

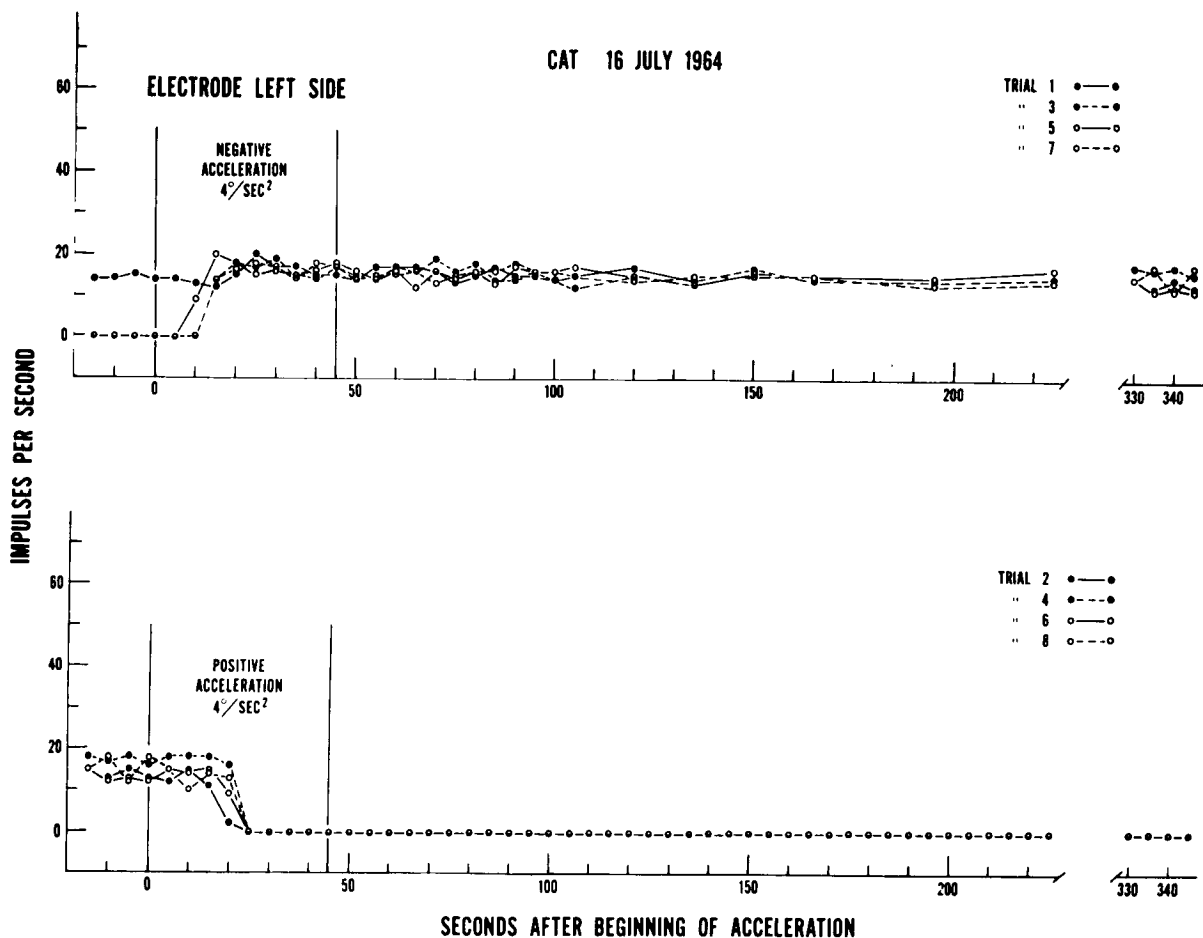


Figure 4.—Cat 16, July 1964: electrode tip was confirmed to be at ventral margin of central gray bordering direct vestibulomesencephalic fibers on left side. A stimulus series identical to that of figure 3 was followed. Note again extreme trial-to-trial reliability of response of this Ewaldian cell, and fixed manner in which its stimulus-induced state is held throughout 5-minute intertrial interval and until changed by succeeding stimulus.

the accelerations alternately switched the cell off and on. This cell is probably best called a long-term memory cell, because it is more common for these cells to “forget” in some 2 minutes after the stimulus. A good question concerns whether this is a go-no-go switched response or a graded response to a graded stimulus, acting as an integrator, and abstracting velocity information for the organism. We have seen one similar cell in which the response height reached during 45 seconds of either  $2^\circ/\text{sec}^2$  or  $4^\circ/\text{sec}^2$  acceleration was the same, indicating a simple binary switch and not a graded response.

More information is needed about a greater number of cells, but the fact that this response form exists greatly extends our basis for speculation on the data handling capabilities of the vestibular system.

In figure 5 are plotted the responses of two cells recorded after the contralateral eighth nerve was sectioned and the cerebellum removed. Both Ewaldian and non-Ewaldian units are represented in this preparation which verifies the ipsilateral origin of both activities. Of particular interest is the approximately equal response in both directions of these fairly high threshold

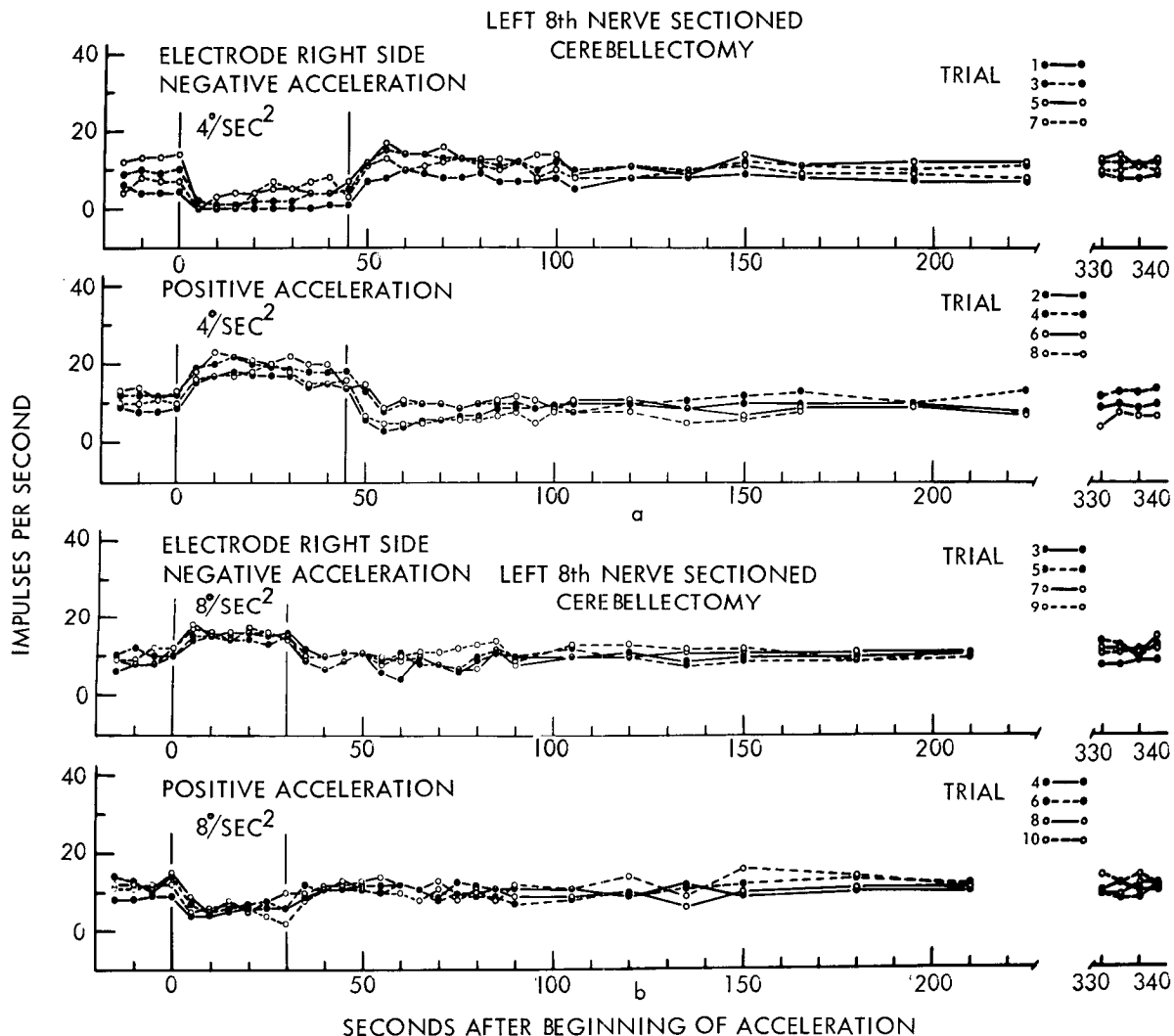


Figure 5.—Cat 1, October 1964 (a): electrode tip was confirmed to be in the interstitial nucleus of vestibular nerve on right side. The contralateral eighth nerve was sectioned and cerebellum removed. Ewaldian cell received same stimulus series as shown in figures 3 and 4. Note close stimulus-bound nature of response and trial-to-trial reliability. Cat 19, October 1964 (b): electrode tip was confirmed to be in lateral vestibular nucleus on right side. Contralateral eighth nerve was sectioned and cerebellum removed. Non-Ewaldian cell first received a negative and positive acceleration of  $4^\circ/\text{sec}^2$  intensity and 45 sec duration, which proved to be just at threshold. A stimulus series of  $8^\circ/\text{sec}^2$  intensity and 30 sec durations was then commenced and produced eight trials depicted in this graph. Here we find same stimulus-bound response and comparable reliability to that of the cell shown in upper record.

units. We have seen cell types which vary from those with no resting discharge and therefore a totally uni-directional response, to those with a resting discharge but a clear directional bias, to such as these in figure 5 with a symmetrical bidirectional discharge.

We do not yet know if the stimulus-bound

response from units in cats in which the contralateral eighth nerve was sectioned and the cerebellum removed is characteristic of that preparation, but it may very well be the case. Thus far units showing the following characteristics have had the cerebellum and contralateral eighth nerve intact: (a)

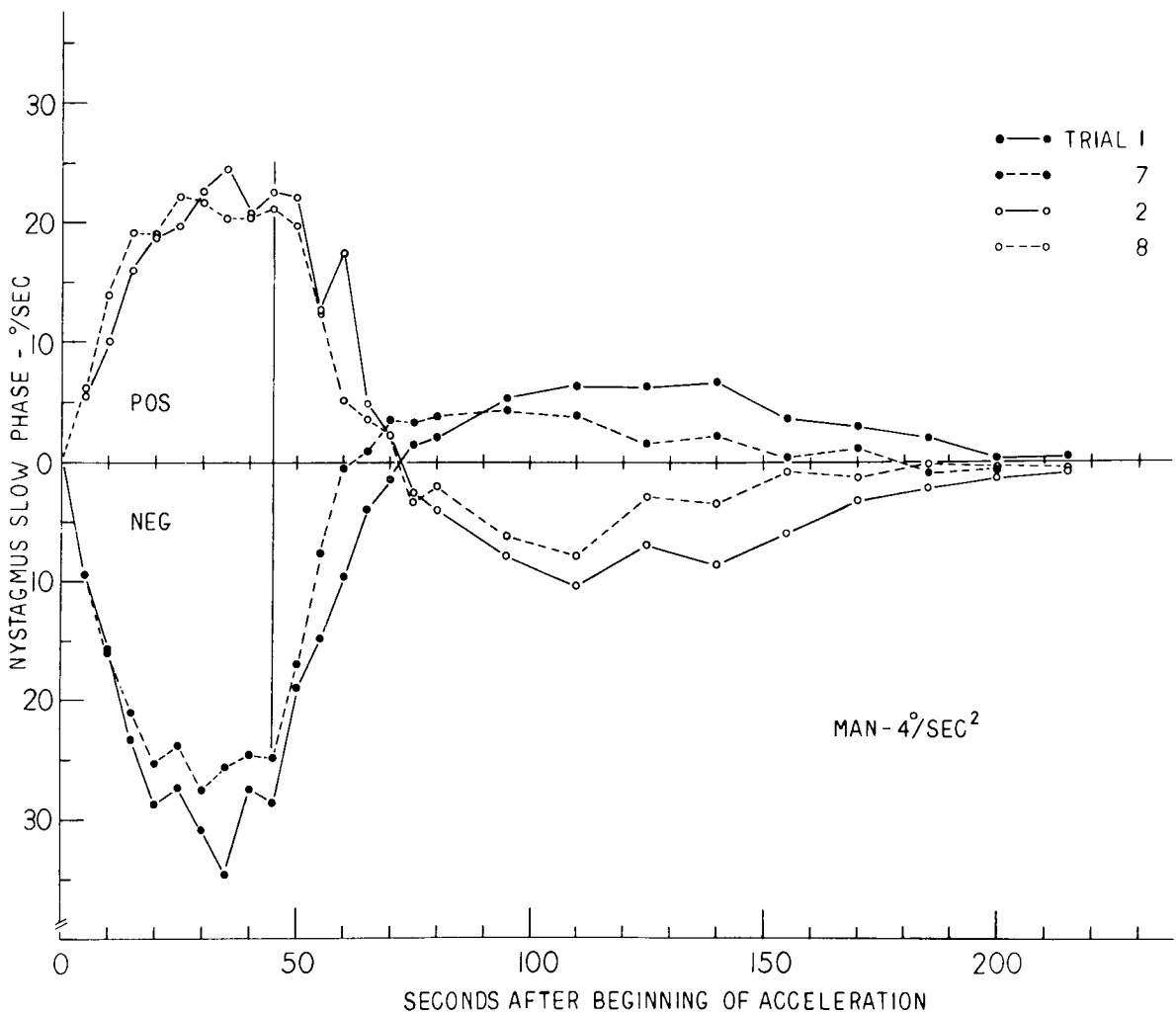


Figure 6.—Slow-phase horizontal nystagmic output of man riding in total darkness at center of rotation and performing a psychophysical task requiring judgments of angular velocity. Nystagmus was recorded electrically. Averages of responses from 10 men are plotted for the first and last two trials of an eight trial series identical to those to which cats in figures 3, 4, and 5 were exposed. Negative accelerations are odd numbered, and positive accelerations are even numbered. Full range of the response is plotted, including the reversal in sign of the slow phase after the stimulus cessation; the secondary nystagmus. This stimulator is not one used for cats, and a full description of the stimulator and the procedures employed for man may be found in Brown and Crampton, 1964.

“memory” cell types which respond with long-duration fixed states, (b) cells with widely varying periodicities of the resting frequencies, (c) units with high frequency outputs, (d) cells that show large post-stimulus rebounds or suppressions, and finally (e) units which show low trial-to-trial reliability. We do not have enough units cataloged yet to answer the above question

nor to draw conclusions about the nuclear membership of the cell types.

#### Habituation and Adaptation

Figures 6 and 7 show nystagmic outputs for man and unanesthetized cat in response to a series of  $4^\circ/\text{sec}^2$  stimuli exactly like the one employed for the neurophysiological studies. The principal features of human



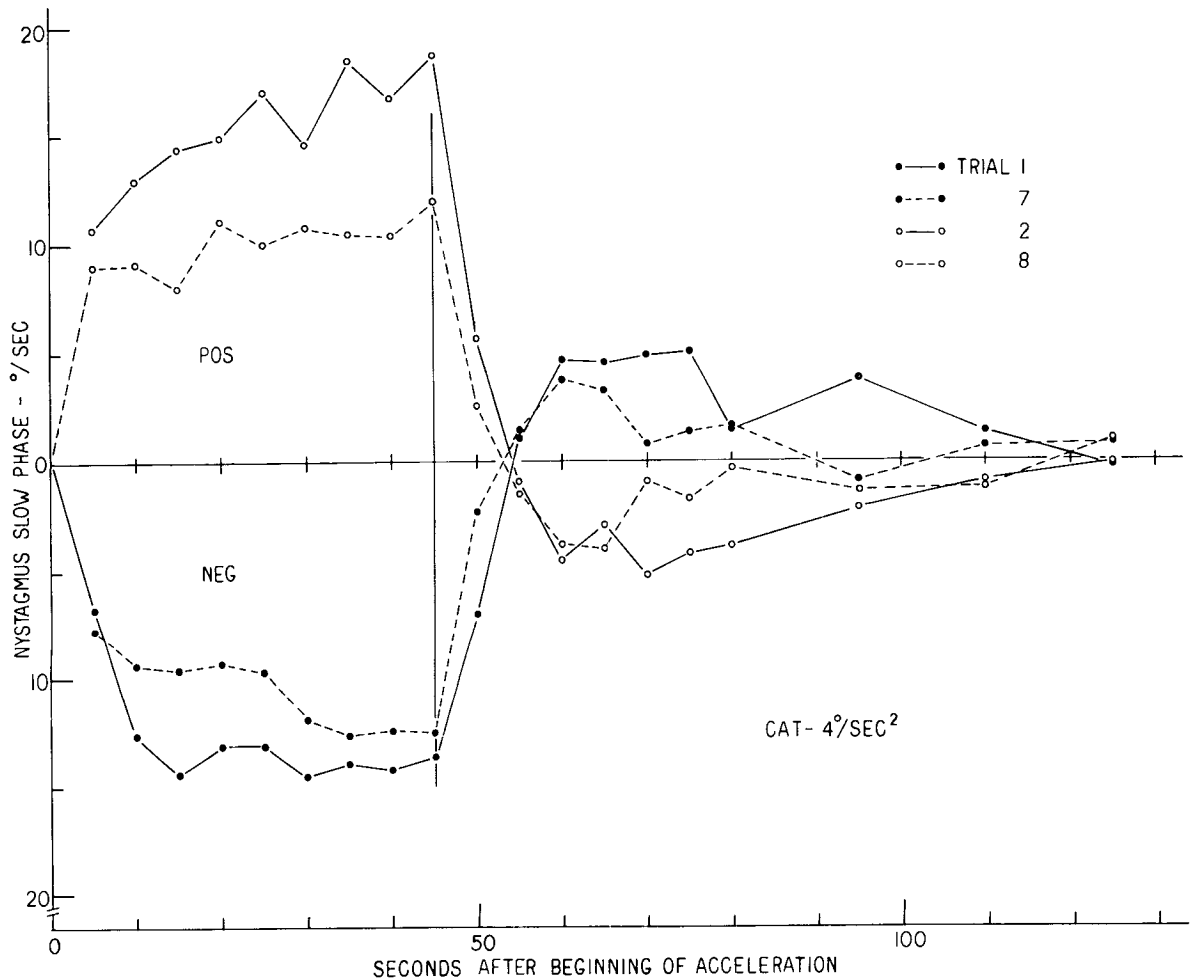


Figure 7.—Slow-phase horizontal nystagmic output for cat, riding in total darkness at center of rotation, and under medication of 2.5 mg/kg d-amphetamine to maintain arousal. Nystagmus was recorded electrically, and average responses for 10 cats are plotted as for man in figure 6. Stimulus series was identical to that used in previous figures. Stimulator is one used for electrophysiological work; a full description of the procedures employed in studies of cat nystagmic habituation may be found in Crampton and Brown, 1964.

and feline nystagmus are very much the same, except that the total response is shorter for cat, and as indicated by our calibration technique at least, the slow-phase output per second is less. Habituation is a prominent feature, more so for cat than for man. There is no adaptation-like decrement for cat, and only a little indication of it for man.

There is no good evidence for adaptation of nystagmic responses to angular acceleration when special care is taken to maintain a high state of arousal with mental tasks in man (Collins and Guedry, 1962) or with

d-amphetamine medication in cat. An examination of all of our records from the vestibular nuclei thus far shows no prominent evidence for adaptation, although some units on some occasions will show a slight decline in discharge rate near the end of a 45 second acceleration (fig. 5). The absence of clear adaptation effects in either the nystagmic response or in the single units of the vestibular nuclei places this sense organ in a unique position among sensory systems.

No regular and progressive decrease in discharge rate from trial to trial was found.

This failure to find habituation effects in single units is in accord with the absence of nystagmic habituation after accelerations during barbiturate anesthesia (Fearing and Mowrer, 1934) and is therefore no clear indication that habituation effects will not be found in units studied in unanesthetized animals. The evidence thus far indicates that habituation is a central and not a peripheral or sense organ phenomenon (Henriksson, Kohut and Fernández, 1961). Even so, habituation effects will not necessarily be found in the vestibular nuclei under any circumstances.

Special care must be taken when evaluating these few records for adaptation or habituation. We should not assume that all of the intensive information of acceleration is coded simply with a monotonic magnitude-to-frequency conversion. There are other possibilities, and adaptation and habituation effects may be elegantly disguised in superb

patterning. For example, some units will start firing with rapid pairs in which the spikes have a separation of about 10 msec. Three or four accelerations later the paired firing will have disappeared to be replaced with a more usual spaced discharge. We have seen one unit within the lateral vestibular nucleus in which the resting discharge increased by five to ten spikes per second from trial to trial and yet the absolute change in discharge rate in response to acceleration remained the same. Again, a unit from within the reticular substance initially responded to the accelerations with an increase in rate embellished with a parasitic oscillatory perturbation. The response to accelerations continued with some reduction, but the oscillations dramatically declined in amplitude with each trial. More subtle phase and amplitude changes would not be detected by our simple frequency counts, and sophisticated techniques must be applied to this important analysis problem.

#### REFERENCES

- ADRIAN, E. D.: Discharges From Vestibular Receptors in the Cat. *J. Physiol. (London)*, 101, 1943, pp. 389-407.
- BRODAL, A.; AND POMPEIANO, O.: The Vestibular Nuclei in the Cat. *J. Anat.*, 91, 1957, pp. 438-454.
- BRODAL, A.; POMPEIANO, O.; AND WALBERG, F.: The Vestibular Nuclei and Their Anatomy and Functional Correlations. Springfield: Thomas, 1962.
- BROWN, J. H.; AND CRAMPTON, G. H.: Quantification of the Human Nystagmic Response to Angular Acceleration. Prediction Formulae and Nomograph. *Acta Oto-Laryngol.*, 58, 1964, pp. 555-564.
- COLLINS, W. E.; AND GUEDRY, F. E.: Arousal Effects and Nystagmus During Prolonged Constant Angular Acceleration. *Acta Oto-Laryngol.*, 54, 1962, pp. 349-362.
- CRAMPTON, G. H.; AND BROWN, J. H.: Repeated Vertical Semicircular Canal Stimulation Does Not Habituate Horizontal Nystagmus in Cat. *Acta Oto-Laryngol.*, 58, 1964, pp. 441-448.
- DUENSING, F.; AND SCHAEFER, K. P.: Die Aktivität einzelner Neurone im Bereich der Vestibulariskerne bei Horizontalbeschleunigungen unter besonderer Berücksichtigung des vestibulären Nystagmus. *Arch. f. Psychiat. u. Z. Neurol.*, 198, 1958, pp. 225-252.
- ECKEL, W.: Elektrophysiologische und histologische Untersuchungen im Vestibulariskerngebiet bei Drehreizen. *Archiv Ohr- usw. Heilk. u. Z. Hals- usw. Heilk.*, 164, 1954, pp. 487-513.
- EWALD, J. R.: Physiologische Untersuchungen über das Endorgan des Nervus octavus. Wiesbaden: Bergmann, 1892.
- FEARING, F.; AND MOWRER, O. H.: The Effect of General Anesthesia Upon the Experimental Reduction of Vestibular Nystagmus. *J. General Psychol.*, 11, 1934, pp. 133-144.
- GERNANDT, B.: Response of Mammalian Vestibular Neurons to Horizontal Rotation and Caloric Stimulation. *J. Neurophysiol.*, 12, 1949, pp. 173-184.
- GERNANDT, B.: Midbrain Activity in Response to Vestibular Stimulation. *Acta physiol. Scand.*, 21, 1950, pp. 73-81.
- GREEN, J. D.: A Simple Microelectrode for Recording From the Central Nervous System. *Nature*, 182, 1958, p. 962.
- HENRIKSSON, N. G.; KOHUT, R.; AND FERNÁNDEZ, C.: Studies on Habituation of Vestibular Reflexes. I. Effect of Repetitive Caloric Test. *Acta Oto-Laryngol.*, 53, 1961, pp. 333-349.
- LÖWENSTEIN, O.; AND SAND, A.: The Activity of the Horizontal Semicircular Canal of the Dogfish, *Scyllium canicula*. *J. Exp. Biol.*, 13, 1963, pp. 416-428.
- LÖWENSTEIN, O.; AND SAND, A.: The Individual and Integrated Activity of the Semicircular Canals of the Elasmobranch Labyrinth. *J. Physiol. (London)*, 99, 1940, pp. 89-101.

ROSS, D. A.: Electrical Studies on the Frog's Labyrinth. *J. Physiol.* (London), 86, 1936, pp. 117-146.

VERHAART, W. J. C.: A Stereotactic Atlas of the Brain Stem of the Cat. Philadelphia: Davis, 1964.

#### DISCUSSION

M. JONES: First, I would like to endorse the fact that you can pick up almost any cell you like if you look long enough in the brain stem. Second, the so-called "vestibular nucleus" is really a rather complex structure of enormous volume having a wide variety of afferent and efferent connections: ascending, descending, cerebellar, etc. It just occurred to me that some of your "holding" units might represent descending pathways passing to the lower limb postural muscles. For a velocity modulated canal signal, when integrated, would tell the CNS that a certain dis-

placement had occurred, and this would perhaps be expected to call for a "holding" pattern of muscle activity. I wonder if you would agree with this?

CRAMPTON: Indeed. And, I wish to add that previous brain stem work in cats, so far as I know, involved removal of the cerebellum before observing single units. In our case, using a stereotaxic approach down through the cerebellum, we find a greater wealth of coding types. I think we have seen a simplified picture of the coding systems in data derived from cerebellectomized animals.

**SESSION II**

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# A New Quantitative Ataxia Test Battery

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AND

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## SUMMARY

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A new multidimensional quantitative ataxia test battery employing the "rail method" of testing was developed to assess more precisely than heretofore postural equilibrium-disequilibrium under unusual conditions and stresses such as rotating environments.

High reliability, including test-retest reliability, was demonstrated for each of two versions: a Long Version employing six rails of varying widths, and a Short Version employing two of these rails. Normative standards covering a wide age range, and age, height, and weight influences upon performance, tentative sex differences in performance, practice effects, and Test Battery relationships with several clinical-type ataxia tests were determined. Validity of the standardized test procedures in the laboratory, in the field and in clinical situations was demonstrated, present and future uses of the Test Battery in normals and auricular-involved individuals in vestibular research as well as in related research-clinical areas were outlined, and several methodological limitations were indicated.

Author

## INTRODUCTION

The disturbances of equilibrium while standing or walking are diagnostic signs with a long tradition of usefulness in clinical medicine. Many procedures have been proposed to quantify these disturbances (refs. 2-5, 8, 9, 11, 16-20, 22, 27, 31-35, 37, 39, 41, 42, 44, 45, 48-62, 65, 66) but almost as many have not stood the test of time. The procedures commonly used today, subjective estimates of disequilibrium, are valued mainly as rough screening tests to indicate lines of direction for more precise diagnostic study and, judging from the small investigative interest expressed in such tests, it must be assumed that they are adequate for all except special purposes.

Our interest in ataxia tests grew out of the fact that subjects, exposed to the unusual inertial forces in a rotating environment, initially experience ataxia then gradually

adapt and a quantitative measure of the time-course of this adaption was needed. The requirements were stringent inasmuch as it was necessary to measure small differences in postural equilibrium over the normal and abnormal range. It is the purpose of this report to describe a new ataxia test battery with numerical scores, demonstrate its reliability and validity and point out some of its uses in laboratory and clinic.

## GENERAL CONSIDERATIONS

The reader is referred elsewhere for a review of the numerous physiological mechanisms and psychological factors which govern postural equilibrium and the various pathological alterations which may affect it. Here it is important only to set forth the guidelines which were followed in devising the test battery, namely (1) selection and categorization of subjects, (2) medical evalua-

tion, (3) standardizing the test procedure, (4) objectivity in scoring, and (5) choice of "long" or "short" version of the Test Battery.

Healthy subjects (Ss) were selected to provide normative data and were categorized on the basis of age, sex, height, weight, occupation, etc. All had a recent medical examination and none complained of postural difficulties. A more comprehensive examination including tests of vestibular function was carried out in the case of groups used in validation studies. Some of the Ss were selected on the basis of labyrinthine defects (L-D), hence manifested vestibular ataxia.

All of the tests were carried out using "rails" (refs. 5, 7, 8, 10, 12, 13-15, 17-19, 21, 23, 25, 28-30, 39, 43, 46, 47, 52, 59, 64) with their advantages of flexibility in width and objectivity in scoring, i.e., the subject either remained "on" or "fell off." The subject was required to remain upright with arms folded and stand or walk heel-to-toe as the case might be. Well-fitting shoes with nonflexible soles and low heels were required. Inasmuch as the test was interesting, good motivation was the rule.

Only two measurements were made: (1) the number of seconds the subject could "stand" and (2) the number of "steps" he could take without "falling." No attempt was made to grade variations in the amount of body sway. A number of trials were given to increase reliability. The test procedures described in appendix A represent the end product of evolutionary development during which many variations of tests were tried and the various items subjected to statistical analysis. Two versions of the test evolved—a Long Version, which utilizes six rails of varying widths, and a Short Version, which utilizes only two of these rails.

The Long Version was designed to test individual performance differences over an extremely wide age range in both normal and clinical populations. Although somewhat time consuming to administer (about 45 min), the Long Version proved ideal for testing the postural equilibrium perform-

ances of the group of labyrinthine defective Ss (L-D's), who participate regularly in the vestibular research program of this laboratory, inasmuch as individual differences within this group were apparent for each of the six rails. As our samples of the higher scoring normal Ss increased, however, it became increasingly clear that a briefer version of the Test Battery was needed on the basis that about one-half of the rails afforded no performance discrimination whatsoever.

The Long Version serves the original purposes of assessing the performance capabilities of extreme age groups (children and senior citizens) and certain severe clinical cases. It offers the major advantage of establishing subtle individual differences in such samples.

The Short Version, it will be seen from our results, serves ideally the purposes of assessing individual differences in normal Ss and to fulfill the premium time-saving requirement imposed by much repeated pre-, per-, and post-testing of Ss with no vestibular dysfunctioning or with varying amounts of vestibular losses, who are exposed to unusual experimental situations.

#### TEST BATTERY (LONG VERSION)<sup>1</sup>

A total of 550 normal males, 11 labyrinthine defective males (L-D's) (college professors, graduates, or near college graduates), and 158 females widely varying in age and occupational status comprise the samples tested with the Long Version of the Test Battery. The samples include highly experienced Naval and Marine Corps test pilots and aviators, including Project Gemini applicants (one is now a Gemini astronaut), military flight surgeons, Naval and Marine Corps student aviators and "Project Astronaut Candidates" (ref. 1), military officers and enlisted personnel, firemen, college professors, college students, senior citizens, clerical and technical medical staff, physicians,

<sup>1</sup> Test Battery refers only to the three tests undertaken on the rails, viz., Walk H/T (walking heel-to-toe with eyes open), Stand E/O (standing heel-to-toe with eyes open), and Stand E/C (standing heel-to-toe with eyes closed).

medical students, nurses, scientists, and high school students.

The Test Battery, the Classical Romberg Test (ref. 50) and the Sharpened Romberg Test (SR) (ref. 4), which were undertaken by these Ss, are described fully in terms of materials, administration, and scoring procedures in appendix A.

#### Normative Data

Test Battery means and standard deviations by age classification in the samples of male and female populations are shown in table I. The ranges of scores observed in each sample and the percentile rankings are contained in table II. There are marked individual differences in performance. The two standing tests appear to be more sensitive to age increase than is the Walk H/T test. In males, standing test performances appear to decline significantly as early as age 43, and Walk H/T performance appears to decline significantly at the later age of 54. A strict assessment of sex differences was not considered practical because of great variability in the footwear of our female samples as opposed to great uniformity of footwear in our male samples. In our opinion, however, sex differences will still be

apparent when tests of this variable are carried out.

#### Reliability

Intratest correlations ( $r$ 's between best trial and second best trial) of Walk H/T ranged from 0.75 to 0.92; intratest correlations of Stand E/O and Stand E/C ranged from 0.83 to 0.96. Test-retest reliabilities ranged from moderate to high ( $r$ 's of 0.57 to 0.96) over a period of seven successive test sessions in a group of twelve normal Ss.

#### Practice Effects

Walk H/T performance plateaued at 9 percent improvement on the fourth day; Stand E/O performance plateaued at 29 percent improvement on the fourth day; Stand E/C performance plateaued at 9 percent improvement on the fifth day.

#### Interest Relationships

Correlations between Walk H/T and Stand E/O ranged from 0.37 to 0.69; correlations between Walk H/T and Stand E/C ranged from 0.13 to 0.48; correlations between Stand E/O and Stand E/C ranged from 0.41 to 0.61. It is apparent from these results that each test comprising the Test Battery

Table I.—*Test Battery (Long Version) Means and Standard Deviations by Age Classification in Samples of Male<sup>a</sup> and Female Populations*

N	Age range	Walk H/T test		Stand E/O test		Stand E/C test	
		Mean	S.D.	Mean	S.D.	Mean	S.D.
Males							
32	13-16	51.3	5.36	469.2	64.09	196.0	89.45
424	17-42	54.8	4.53	483.1	59.15	203.7	98.61
9	43-50	56.7	4.08	457.4	88.78	150.4	98.01
4	51-53	54.5	5.50	416.3	92.19	120.8	70.97
7	54-66	45.3	9.11	322.1	139.01	101.7	67.88
Females							
28	14-16	51.5	4.26	489.6	41.71	225.8	69.08
112	17-42	51.3	4.66	486.2	54.49	232.3	99.52
7	43-50	46.6	6.64	475.6	33.95	196.1	107.24
2	51-53	43.0	1.00	320.0	32.00	49.5	1.50
9	54-67	41.0	10.34	296.8	140.73	90.7	77.78

<sup>a</sup> These samples do not include 74 student military aviators and "Project Astronaut Candidates."

Table II.—*Test Battery (Long Version) Raw Scores and Their Percentile Equivalents*

Percentile	Males						Females					
	Ages 13-16 N= 32		Ages 17-42 N= 424		Ages 43-50 N= 9		Ages 14-16 N= 28		Ages 17-42 N= 112		Ages 43-50 N= 7	
	W <sup>a</sup>	S/O <sup>b</sup>	S/C <sup>c</sup>	W	S/O	S/C	W	S/O	S/C	W	S/O	S/C
99th	60	555	350	60	640	420	60	566	367	60	552	342
98th	59	550	347	59	595	402	59	559	340	59	549	340
97th	59	545	345	59	585	385	59	553	314	58	547	337
96th	58	542	342	59	575	370	58	549	287	57	545	334
95th	58	539	339	59	565	355	59	544	261	57	540	325
90th	58	525	325	59	530	320	59	539	235	57	534	321
80th	56	510	285	58	519	295	59	509	230	55	520	300
70th	54	505	244	57	510	270	59	504	192	54	510	280
60th	53	495	220	57	501	240	58	503	177	52	507	244
50th	51	485	174	55	495	200	58	500	116	51	504	233
40th	50	460	150	54	480	165	56	499	79	49	501	205
30th	48	435	140	52	460	130	55	402	77	48	478	187
25th	47	425	125	51	445	115	52	396	75	47	461	179
20th	46	410	104	50	430	90	50	361	70	47	450	166
15th	45	401	84	49	409	79	49	312	56	47	441	157
10th	43	392	80	48	402	65	48	270	41	46	420	122
9th	42	385	75	47	395	62	48	270	41	46	415	121
8th	41	370	70	47	390	58	48	270	41	45	411	121
7th	40	359	65	46	385	55	48	270	41	45	407	120
6th	39	349	55	46	380	53	48	270	41	44	405	110
5th	38	330	51	46	375	51	48	270	41	43	403	100
4th	37	315	48	45	365	49	48	270	41	43	401	90
3rd	35	305	44	44	354	47	48	270	41	43	401	90
2nd	35	305	44	43	330	45	48	270	41	43	401	90
1st	35	305	44	42	291	45	48	270	41	43	401	90

<sup>a</sup> Walk H/T (six rails).

<sup>b</sup> Stand E/O (six rails).

<sup>c</sup> Stand E/C (six rails).



relates only moderately to each other test and thereby suggests a nearly ideal distinctness desired in a battery of tests designed to measure complex performances referred to singularly as ataxia, or postural equilibrium.

#### Height and Weight Influences

All correlations with height and weight (except in highly heterogeneous samples) were very low, or zero order, and had negligible influences upon performance.

#### Validity

##### Labyrinthine Defective Group

The majority of this group scored at the 1st percentile on each of the tests. In this group virtually no improvement with extended practice was shown in their Stand E/C performances (only 3 percent), whereas Walk H/T and Stand E/O performances typically improved rather markedly—70 percent and 60 percent, respectively, over seven daily retest periods.

##### Prediction of Motion Sickness Susceptibility

In a small group of male Ss ( $N = 15$ ), who were evaluated with regard to susceptibility to motion sickness by means of a motion sickness questionnaire, a boat ride, exhaustive motion sickness-arousing air ride, and rotations on the Pensacola Slow Rotating Room and the Toronto Counter Rotating Platform (ref. 24), susceptibility to motion sickness was predicted to a moderate extent; correlations with the Test Battery ranged from 0.50 to 0.75.

#### Discriminatory Power and Limitations of the Long Version

Intercorrelations of performances by normals on each of the six rails disclosed that Rail 5 ( $\frac{3}{4}$  in. wide) showed highest communality with the remaining rails in the case of Walk H/T and Stand E/O, and Rail 2. ( $2-\frac{1}{4}$  in. wide) showed highest communality in the case of the Stand E/C test. Consequently, in the interest of economy we pilot-tested a large number of randomly selected miscellaneous normal Ss, with the modified

procedure of scoring the best three trials out of five trials and the Short Version of our Test Battery was derived utilizing these two rails.

#### TEST BATTERY (SHORT VERSION)

A total of 828 normal males, 10 of the L-D's tested previously on the Long Version, 17 male otoneurological cases, 99 normal females, and 15 female otoneurological cases comprise the samples tested. These included experienced military aviators, Naval and Marine Corps student aviators and "Project Astronaut Candidates," flight surgeons, military officers, enlisted personnel, and military and civilian scientific, clerical, and technical personnel.

The Test Battery (Short Version) and the SR test was undertaken by all Ss, and many of the Ss undertook in addition the Stand One Leg Eyes Closed test (SOLEC) (refs. 5, 8, 65), and the Walk Line Eyes Closed test (WALEC) (refs. 42, 58). These are described fully in terms of materials, administration and scoring procedures in appendix A. Score sheet is shown in table III.

#### Normative Data

Samples tested to date range 17–59 years in age. As with the Long Version, there are marked individual differences in the capabilities tapped by the Short Version, and there is considerable overlap in the performances of older and younger individuals (table IV). The ranges of scores observed and the percentile equivalents are shown in table V. In the males sampled, performance declines significantly in about the age range of 43 to 53 years. Analysis of possible sex differences in performances again was not considered practical in view of marked variability of footwear in the female samples as opposed to virtual uniformity of footwear in the male samples.

#### Intratest Reliability

Intratest correlations (best trial with second best, best trial with third best, and second best with third best) of Walk H/T scores

Table III.—*Score Sheet*

NAME (Last)					DATE	
DATE OF BIRTH (Mo.)		(Day)	(Yr.)	AGE	SEX	
HEIGHT		WEIGHT	OCCUPATION			
TRIAL	WALK	STAND OPEN	STAND CLOSED	SR	STAND ONE LEG CLOSED	
					RT.	LT.
1						
2						
3						
4						
5						
TOTALS						

POSTURAL EQUILIBRIUM TEST SERIES Navscolavmed 3930/1 (7-63)

Table IV.—*Test Battery (Short Version) Means and Standard Deviations by Age Classification in Samples of Male and Female Populations*

N	Age range	Walk H/T test		Stand E/O test		Stand E/C test	
		Mean	S.D.	Mean	S.D.	Mean	S.D.
Males							
340	17-42	12.5	2.62	37.6	31.98	103.8	58.37
471	43-50	10.4	3.10	19.1	13.47	52.0	45.90
17	51-53	9.2	3.99	13.2	5.87	24.2	14.09
Females							
41	18-29	11.5	2.66	26.7	14.05	84.6	60.92
47	30-49	9.9	2.95	18.8	10.65	49.1	43.33
11	50-59	8.8	4.00	13.5	5.90	42.6	37.61

ranged from 0.71 to 0.90, of Stand E/O scores from 0.89 to 0.96, and of Stand E/C scores from 0.82 to 0.96. Thus, performance on a given test utilizing a single rail, as opposed to utilizing six rails formerly, duplicated the high reliability established for the Long Version.

#### Test-Retest Reliability

In a group of twelve normal male Ss in the age range 18-49, who undertook ten succes-

sive daily performances (two additional daily retests were administered while Ss wore basketball shoes) on the Test Battery, test-retest reliabilities, computed by correlations of Day 1 performance with mean performances on Days 2 through 10, were 0.40, 0.86, and 0.91 for Walk H/T, Stand E/O, and Stand E/C, respectively. Other combinations of initial and early performances correlated with later, practiced performances yielded

Table V.—*Test Battery (Short Version) Raw Scores and Their Percentile Equivalents*

Percentile	Males						Females								
	Ages 17-42 N = 235		Ages 43-50 N = 360		Ages 51-53 N = 14		Ages 18-29 N = 41		Ages 30-49 N = 47		Ages 50-59 N = 11				
	W <sup>a</sup>	S/O <sup>b</sup>	S/C <sup>c</sup>	W	S/O	S/C	W	S/O	S/C	W	S/O	S/C			
99th	15	163	180	15	125	180	15	61	180	15	48	180	15	27	121
98th	15	146	179	15	66	177	15	58	175	15	44	174	15	25	118
97th	15	137	177	15	56	175	15	56	170	15	40	169	15	24	115
96th	15	128	176	15	46	172	15	54	165	15	36	163	15	23	111
95th	15	122	174	15	42	170	15	52	160	15	34	141	15	22	107
90th	15	74	173	14	30	136	14	50	153	13	32	120	14	19	103
80th	15	50	172	13	24	81	13	44	150	12	24	80	13	18	89
70th	14	39	154	12	20	54	12	32	107	11	20	60	10	17	58
60th	13	30	123	11	17	43	11	25	90	10	17	39	9	15	33
50th	13	25	92	10	15	33	10	21	64	10	16	31	8	13	30
40th	12	22	68	9	14	28	9	18	51	9	14	24	7	11	25
30th	11	19	47	8	12	23	7	16	33	8	12	20	6	9	19
25th	10	17	39	8	11	21	6	15	28	7	11	18	5	8	19
20th	10	16	35	7	10	19	4	14	26	7	10	15	4	7	18
15th	9	15	26	7	10	15	2	12	22	6	9	12	3	7	15
10th	8	13	20	6	9	14	1	12	18	6	8	11	3	7	12
9th	8	13	19	6	9	14	1	12	18	5	7	11	2	6	9
8th	8	13	18	6	8	13	1	12	18	5	7	11	2	6	6
7th	7	12	17	5	8	13	1	12	17	5	6	11	2	6	6
6th	7	11	16	5	8	12	1	12	17	5	6	10	2	6	6
5th	6	11	15	5	7	12	0	11	16	4	6	10	2	5	6
4th	5	10	15	5	7	11	0	11	15	4	6	10	1	5	5
3rd	5	9	14	5	7	10	0	11	14	4	5	9	1	5	5
2nd	4	9	13	4	6	9	0	10	13	3	5	8	1	5	5
1st	4	8	12	3	5	8	0	9	8	2	5	6	1	5	5

<sup>a</sup> Walk H/T (¾ in. wide rail).

<sup>b</sup> Stand E/O (¾ in. wide rail).

<sup>c</sup> Stand E/C (2¼ in. wide rail).

virtually identical coefficients. Substantial repeatability of standing test performances was apparent. The lower reliability of Walk H/T performance reflects the more rapid rate of learning afforded both by the locomotor aspect of this test, and, in turn, a more easily attained perfect score than is found on the standing tests. Comparisons of Days 1 and 2 (combined) with Days 9 and 10 (combined) performances revealed 26 percent improvement on Walk H/T, 77 percent on Stand E/O, and 82 percent on the Stand E/C test.

#### Practice Effects and Effects of Footwear Upon Highly Practiced Performance

These results are summarized in figure 1. In the group of 12 normal male Ss, performances improved in almost linear fashion throughout the 10-day period, although the improvements were relatively slight following the plateau points on the learning curve.

Plateaus in Walk H/T, Stand E/O, and Stand E/C performances were realized on

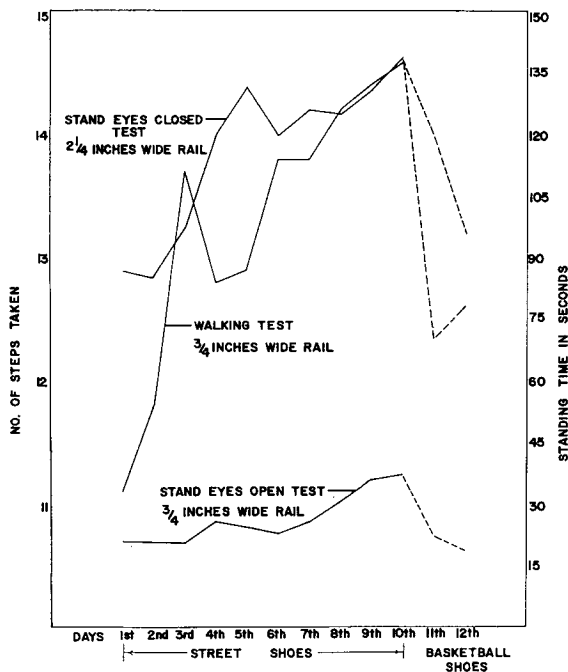


Figure 1.—Influences of practice and of type of footwear on Test Battery (Short Version) performance in a group of ( $N = 12$ ) normal male subjects.

the third day, fourth day, and fifth day, respectively. The detrimental effects of wearing basketball shoes upon highly practiced standing test performances (obtained while the Ss wore street shoes) was to the extent of a 44 percent decrease in Stand E/O performance and a 47 percent decrease in Stand E/C performance. In marked contrast, Walk H/T performance decreased a mere 6 percent, indicating that the loss of stability due to basketball shoes was almost completely compensated for on Walk H/T and very poorly compensated for on the two standing tests.

#### Interest Relationships

Correlations among the three distinct tests comprising the Short Version corresponded very nearly to those reported for the Long Version. The correlations ranged from 0.19 to 0.51.

#### Height and Weight Influences

The correlations with the Test Battery were very low, or zero order, and negligible for differentiation and prediction purposes—a finding in keeping with results on the Long Version.

#### Validity

##### The Identification of Individuals With Auricular Defects

The mean performance scores of L-D's, streptomycin-treated Ménière's cases, and clinical cases which include postural vertigo, positional nystagmus, Ménière's pseudo-Ménière's, and labyrinthitis, were compared with the mean performance scores of an equivalent number of randomly sampled, age-matched normal, symptom-free individuals. In all instances, the performances of individuals with auricular involvement were significantly poorer than the performances of the normals.

#### Relationships with Threshold Caloric Responses

The performances of 11 symptom-free male individuals with below normal threshold caloric responses ( $\cong 35.0^\circ \text{C}$ ) were compared with the performances of eleven randomly sampled normal male individuals with

normal threshold caloric responses ( $36.0^{\circ}$  to  $36.6^{\circ}$  C). Both Stand E/O and Stand E/C performances were identified with depressed semicircular canal sensitivity insofar as such sensitivity is reflected by caloric responses in the range of  $35.0^{\circ}$  C and below (0.02 level of confidence). Mean Walk H/T performances in the two groups were virtually identical.

#### Identification of Canal Sickness Susceptibility

In a sample of 20 normal male Ss the test performances of the ten most susceptible to canal sickness on the SRR were compared to the test performances of the 10 remaining, i.e., least susceptible, Ss. Susceptibility in this instance was defined as a rank ordering of the 20 Ss in terms of the number of dial sequences completed during rotation and qualitative ratings by an observer (ref. 38). Generally, on all three tests the 10 most susceptible Ss as a group attained higher performance scores than those attained by the least susceptible Ss. To this extent, the Test Battery would appear to reflect sensitivity to canal sickness, which is a finding in parallel with predictability of motion sickness from Long Version performance.

#### Effects of Prolonged Rotation in the Pensacola Slow Rotation Rooms

Prerotation Test Battery performances were compared with postrotation performances in several groups of normal Ss who were rotated at 10 rpm for 12 days in the SRR (ref. 25). Post-testing occurred immediately upon cessation of rotation, and in all instances severe declines in test performances were evidenced. Daily retesting during the post-rotation periods revealed complete recovery, within 24–72 hours, of all Test Battery performances except Stand E/C performances. The visually influenced performances on the Walk H/T and Stand E/O tests had not only recovered but improved, whereas the non-visually-influenced Stand E/C performances proved more sensitive to the influences of prolonged rotation.

#### Influences of Moderate and Severe Sea Conditions Upon Performance

In the Nova Scotia Experiment (to be published soon) twenty normal male Ss withstood a 25-hour ride on an ocean-going tug in midwinter during moderate to rough sea conditions between Nova Scotia and Newfoundland. Baseline Test Battery performances, which were obtained prior to the experiment, were compared with performances obtained within 30 minutes to 4 hours, within 16–21 hours, and at 36 hours following the sea experience. Comparisons were made by the split-half method, i.e., the ten Ss with the highest baseline performance scores on each test of the Test Battery were compared with the remaining 10 Ss having the lowest baseline performance scores. It was revealed (fig. 2) that the performances of the lowest scoring Ss were hardly affected by the sea experience; indeed, those who did not at least maintain their baseline performance levels showed significant improvements in performance. The ten initially highest scoring Ss, in marked contrast, showed significant decreases in performances. Both the Walk H/T and Stand E/O performances of these sensitive Ss recovered to baseline level within 16–21 hours, but Stand E/C performances had only partially recovered within 36 hours of the sea experience. This Stand E/C result is reminiscent of the delayed recovery of Stand E/C performances of the several groups of Ss exposed to prolonged rotation in the SRR.

Ten L-D male Ss acted as the control group in the Nova Scotia Experiment. As expected, the Test Battery performances of the L-D group were not at all affected by the sea experience. Indeed, as was the case with the low scoring (relatively insusceptible) normals, performances during the post-testing periods were either maintained or improved.

#### Relationships with Trampoline Performance

From each of several successive classes of student aviators undertaking physical training in the School of Pre-Flight, two to four men at the very top in terms of proficiency

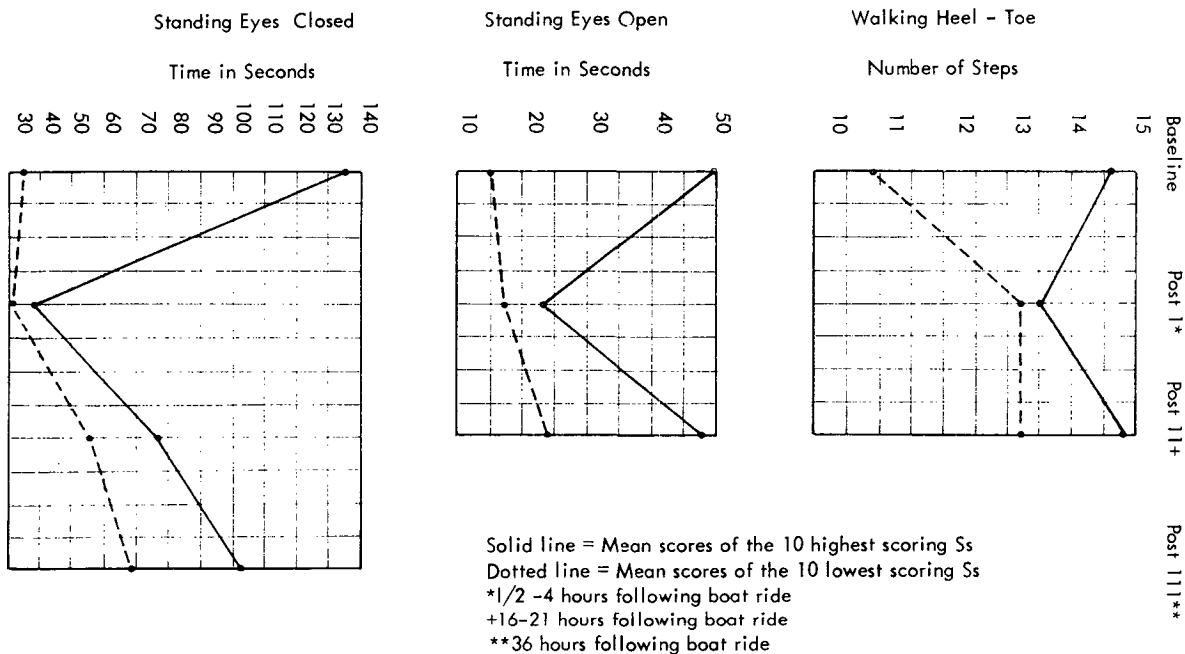


Figure 2.—Recoverability from moderate to severe sea conditions, reflected by Test Battery (Short Version) performance, as a function of baseline performance of normal males.

on the trampoline and two to four men at the very bottom in trampoline proficiency were selected for performance testing.<sup>2</sup> It was found that the top group on the trampoline scored higher on Walk H/T (0.01 confidence level) and Stand E/O (0.10 confidence level) than did the bottom group. The means of the two groups on Stand E/C were virtually identical. Inasmuch as the group mean differences observed were limited to the two visually enhanced postural equilibrium performances, it is suggested that visual-motor, primarily locomotor, factors (vs. vestibulo-motor factors) underlie the relationships found between Test Battery and trampoline performance.

Some Relationships with Several Clinical-Type Ataxia Tests

Several individuals who undertook the Test Battery undertook also the following tests described fully in appendix A: Sharp-

<sup>2</sup> Ss were very carefully selected by Joseph F. Lowder, Physical Education Instructor, Naval School of Pre-Flight and Coach, Navy "Starflights" trampoline demonstration team.

ened Romberg, Stand One Leg Eyes Closed, and Walk Line Eyes Closed. The Test Battery performances of normal male Ss who scored perfectly on these clinical-type tests were compared with the Test Battery performances of age-matched normal male subjects who had scored less than perfect on the clinical-type ataxia tests. Generally, results were in the direction of positive relationships between Test Battery scores and scores obtained on the ataxia tests. Notably, individuals with "perfect" ataxia test scores generally scored higher (better) on the Stand E/O and Stand E/C tests than did those individuals with less than perfect ataxia test scores. There were no significant differences, however, in Walk H/T performances between the two groups, and understandably, none of the correlations between Walk H/T and the ataxia tests were statistically significant.

Comparative Difficulty in Performing the Stand E/C Test and the SR Test in Relation to Age

The only procedural difference between the SR Test and the Stand E/C Test is that

the SR was performed while Ss stood on the floor, whereas the Stand E/C Test was performed while Ss stood on a 2 $\frac{1}{4}$  inches wide rail. Standing for a period of 60 seconds in position with eyes closed constituted a perfect score on each of the two tests. The two tests appeared to most subjects, deceptively, to be of equivalent difficulty. Consequently, most of our subjects expressed surprise, if not chagrin, at their considerably greater difficulty in performing on the rail than on the floor. Quantitative comparisons of the two tests in terms of difficulty in samples of male subjects ( $N=530$ ) and female subjects ( $N=211$ ) in various age ranges revealed remarkable differences between the two tests. Some 24 percent to 92 percent of the subjects scored perfectly on the first trial of the SR Test, but only 3 percent to 23 $\frac{1}{2}$  percent of the subjects scored perfectly on the first trial of the Stand E/C Test. Eighty-seven percent of the younger subjects (ages 19–26) scored perfect first trials on the SR, and only 56 percent of the older Ss (ages 43–53) did so. But greater still was the percentage differences in Stand E/C performance between the younger group and the older group—18 percent perfect first trial scores in the younger group vs. only 3 $\frac{1}{2}$  percent perfect first trial scores in the older group. These findings reflect a mean age difference between the two groups of 23 years.

#### Influence of Alcohol Upon Test Battery and Clinical Type Ataxia Test Performances

As part of a larger study (in preparation for publication in collaboration with Martin Bergstedt), which included positional alcohol nystagmus measurements and blood alcohol measurements, 13 Ss were posture-tested 30 minutes, 1 hour, 2 hours, 3 hours, 4 $\frac{1}{2}$  hours, 6 hours, and 7 hours after consuming 80-proof vodka on an empty stomach in the amount of 1 cc per lb. body wt. in the following sequence: (1) SR; (2) Walk H/T; (3) Stand E/O; (4) Stand E/C; (5–6) SOLEC L & R; (7) WALEC (each of these seven tests were administered repeatedly at the

intervals indicated). The entire experiment was duplicated 2 days later with the same subjects but with 100-proof vodka as the stimulus. The alcohol had the immediate effect of producing a marked decrement in performance by all subjects on all seven tests. Peak decrements in performance were evidenced 1 hour after alcohol intake, and performances did not recover to baseline level until 3 to 7 hours after alcohol intake. Generally, the 100-proof vodka provided greater performance decrement than did the 80-proof, and the recovery period with the 100-proof was somewhat longer than with the 80-proof. The SR Test proved least sensitive to alcohol both in terms of decrement and recovery time, whereas the Test Battery proved most sensitive in terms of recovery time although it tended to equal the SOLEC and WALEC in terms of the extent of performance decrement.

Several L-D subjects undertook, identically, this alcohol experiment with the interesting result that, unlike normal subjects, they did not suffer performance decrements on the nonvisual tests despite the adequate stimuli, suggesting that the vestibular apparatus is an essential component of ataxia due to alcohol stimulation.

#### DISCUSSION

The normal standards set forth must be regarded as tentative. The most reliable figures are those for males in the 17 to 53 age range. These values are representative of the scores obtained on subjects who not only had passed the flight medical examination on more than one occasion but also had demonstrated in the performance of their professional and recreational activities, freedom from significant disturbances of psychophysiological mechanisms governing postural equilibrium. In the great majority the functional status of the semicircular canals and otolith organs was not determined specifically and, had this been done, some would have been eliminated from the "normal" group. It is worth noting, however, that when comprehensive evaluations were car-

ried out in the case of aviators with low scores the findings usually revealed no definite abnormality. In some instances, even after practice the scores remained low suggesting either an inherited lack of skill or some cryptic disorder.

In the female populations the normative values are faulted partly on the basis of a limited medical examination and partly by the fact that in some instances the fitting of the shoes was less than ideal. Nevertheless the values are probably not far from "normal" and are included as a guide indicating that significant sex differences exist.

While the usefulness of any ataxia test is dependent on the establishment of normal values, this dependence is lessened when serial measurements are made on a single person. In the experimental situation each subject serves as his own control; in clinical evaluations, the improvements in score or lack of it in one or more test items constitutes an additional "lead."

Our experience with the Test Battery has centered mainly around its use in measuring vestibular ataxia. It was found to be a reliable indicator both of loss of function and disturbed function. With regard to the former, our findings suggest that small loss (or suppression) of semicircular canal function in the presence of normal otolith function, as revealed by the counterrolling test, is sufficient to cause slight ataxia. This is supported by the results of Igarashi, et al. (ref. 36), who produced ataxia in squirrel monkeys by the administration of streptomycin sulfate. Subsequent pathological studies revealed a significant loss of sensory epithelium of the cristae with little or no pathological changes in the maculae. The significance of these findings should be limited only to the probability that loss of semicircular canal function alone may lead to ataxia; the data are insufficient to evaluate fully the relative roles of both vestibular organs.

It was found that the ataxia in subjects with bilateral loss of labyrinthine function could be reduced with practice. This reduction was slight in the absence of visual cues,

moderate in standing with eyes open and substantial when walking with eyes open. In addition to demonstrating the role of vision and the advantage of better cues in the more dynamic test, walking compared with standing, it pointed out the desirability of training such subjects to improve their postural equilibrium. The experience with L-D subjects raises the question whether such training should be given to all persons with abnormally low performance scores. Old persons, for example, who have become "unsteady" might improve their postural equilibrium with appropriate training. Their performance level and improvement with practice could be determined easily. Such improvement might represent the loss in skill imposed by avoiding all circumstances where a test of skill is involved. That even L-D subjects can improve their Test Battery performance with practice (Stand E/C performance excepted) underscores the necessity of practice to reach performance plateaus before undertaking validation studies if maximum results are to be realized.

Further experience with the Test Battery is needed to determine its limitations and exploit its usefulness. Its adaptability to clinic or laboratory readily can be made for either general or specific purposes.

In the laboratory, it would appear to have great value in measuring adaptation in dynamic force environments, as our experiences with the Slow Rotating Rooms have shown (refs. 6, 23, 25). The time courses of adaptation and rates of recovery may be studied multidimensionally in a minimum of time with the expectation of reasonable returns for the effort. It is hoped that this, or extensions of this, approach will throw more light on the problem of understanding differential effects within the vestibular apparatus in its varied responses to differential force environments.

In the clinic, particularly otolaryngology, neurology, and geriatrics, greater attention to finer, differential details of postural equilibrium functioning may facilitate diagnostic and treatment formulations. The time



course of a disturbance or recovery of a disorder may be assessed as easily as induced disorders in the laboratory. Moreover, in the process of such continued observations additional clues to novel as well as conventional methods of rehabilitation may evolve. As reservoirs of information so obtained more or less routinely build up, unique patterns of functioning peculiar to given diagnostic categories might well become revealed and perforce aid expedition of medical situations in which ataxia is part of the problem.

Awaiting all researchers who seek a fuller understanding of postural equilibrium functioning are such problems as elaborating the practical as well as theoretical significance of appreciable differences between a given individual's visual and non-visual performance capabilities, of differentiating more carefully locomotor and other types of ataxia from vestibular ataxia, of clarifying the notion of an "otolithic ataxia" vs. a "semicircular canal ataxia," of definitively measuring differences between "dynamic" and "static" equilibrium, and of delineating apparent differences between nature- and nurture-influences upon equilibration generally.

Available vehicles for such research include the systematic study of such unusual individuals as congenitally and adventitiously blind persons with and without vestibular dysfunctioning, persons with unilateral and bilateral vestibular dysfunctioning ranging from "minimal" to "complete," persons with circumscribed neurological handicaps and disorders involving the vestibular pathways, and apparently normal individuals with extraordinarily good postural equilibrium functioning. Most desirable are longitudinal studies which systematically include vestibular evaluations as part of the complete medical evaluation (refs. 26, 40), and ideally the cross-sectional and longitudinal vestibular functional testing of large numbers of individuals at all ages whose temporal bones will, eventually, be made available for structural-functional correlational analysis. If in such studies the postural equilibrium evaluations are quantified, then data process-

ing would be facilitated considerably by modern computers.

The high reliability, in our experience, of both versions of the Test Battery, and their demonstrated validity in terms of reflecting auricular involvement and related sensory-motor functioning in widely varied situations suggests their usefulness generally as a tool for enhancing vestibular test batteries designed for more than cursory assessment of postural equilibrium functioning. In addition to high reliability and evidence of validity, each version combines uniquely the advantages of the rail method of testing, stringent body position, high ego-involving task interest and novelty, objectivity of scoring, uniformity and ease of administration (particularly the Short Version), and multi-dimensional sensitivity—locomotor vs. static, visual vs. nonvisual, sensory vs. motor, and especially visual-motor vs. vestibular-motor functioning. Moreover, there is an approaching abundance of normative data.

In the laboratory, the Test Battery (Short Version) has been found useful as a tool for the study of: (1) the influences, generally, of various types and degrees of clinically- or experimentally-induced vestibular disturbances; (2) adaptation and habituation to unusual force environments; (3) the nature of rotation and post-rotation effects (refs. 6, 23-25, 47); (4) the effectiveness of antimotion sickness drugs and other pharmacological agents (ref. 63); and (5) relationships between postural equilibrium functioning and such conventional vestibular functional tests as responses to caloric irrigation, cupulometry, counterrolling responses, etc. The Test Battery may prove useful in the study of: (1) the influences of sensory and sensory-motor alterations or stresses (isolation, deprivation, fatigue, boredom, suggestibility, etc.) (ref. 59); (2) short- and long-term neuromuscular effects (including muscle atrophy) of zero g and sub-g environments including water immersion and rotating space vehicles; (3) the influences of physiological aging and of physical fitness. In the clinic one or the other version of the Test

Battery may prove invaluable for the study of problems in otolaryngology, neurology, and geriatrics.

Methodological limitations of the Test Battery<sup>3</sup> include the following: (1) differential test sensitivity, e.g., Walking vs. Standing, or Stand Eyes Open vs. Stand Eyes Closed performances, even where subjects serve as their own controls, cannot be strictly assessed inasmuch as the rails were not equated as to

<sup>3</sup> The limitations are equally applicable to other ataxia tests including the clinical-type ataxia tests and, generally, to other types of multi-dimensional performance tests.

*APPENDIX A—POSTURAL EQUILIBRIUM TESTS AND CLINICAL-TYPE ATAXIA TESTS: APPARATUS, AND ADMINISTRATION AND SCORING PROCEDURES*

*APPARATUS<sup>4</sup>*

*Test Battery (Long Version)*

Six rails of pine wood construction, each 8 feet long and each superimposed on its 5½ inches wide plywood base, and each with width and height dimensions as follows: rail 1: 2¾ in. wide and 1 in. high (above base); rail 2: 2¼ in. wide and 1 in. high; rail 3: 1¾ in. wide and 1 in. high; rail 4: 1¼ in. wide and 1 in. high; rail 5: ¾ in. wide and 1½ in. high; rail 6: ½ in. wide and 1½ in. high. The four widest rails are attached to the top of the base, whereas the two narrowest rails are inserted within the base and held there by screws underneath to provide adequate support. Rails 5 and 6 are ½ in. higher from the base than rails 1–4 to prevent Ss from obtaining support from the base by means of overriding the feet. Also, to prevent splintering, primarily of rails 5 and 6, the top of each rail is covered by ¼ in. thick fiber glass attached by means of ordinary glue. However, any wear-resistant, nonslip surface material equivalent to fiber glass would have proved satisfactory. To prevent warping, each rail is secured to the floor with screws through the base. The rails are situated in parallel position at 22-inch intervals (fig. 3).

<sup>4</sup> Utmost safety precaution is necessary on the part of the examiner to prevent possible injury of Ss from inadvertent falling.

difficulty, and, accordingly, (2) comparative performance effects, such as exposure to prolonged rotation, between groups unmatched in performance skills, e.g., vestibular defective individuals vs. vestibular normal individuals, cannot be strictly assessed, and (3) performances on any or all of the tests comprising the Test Battery do not portend definition nor even representation necessarily of postural equilibrium (or ataxia) however it may be defined, subjectively or objectively, by any other test or battery of tests.

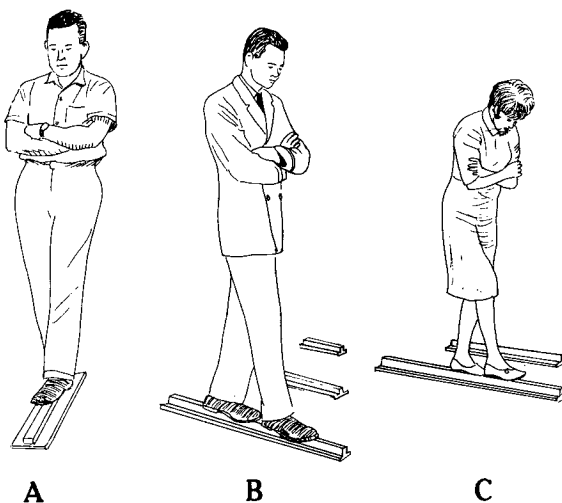


Figure 3.—*Test Battery (Long Version): A, Walk H/T Test (walking with eyes open); B, Stand E/O Test (standing with eyes open); and C, Stand E/C Test (standing with eyes closed).*

A more durable version of the apparatus consists of a singular metal base (with the same dimensions as each wooden base) with in each of six metal rails (with sand-blasted top surfaces and with dimensions identical with the wooden version) may be inserted readily by simple turning of two rigid-securing hand screws. Four pairs of set screws within the base permit leveling on uneven floors.

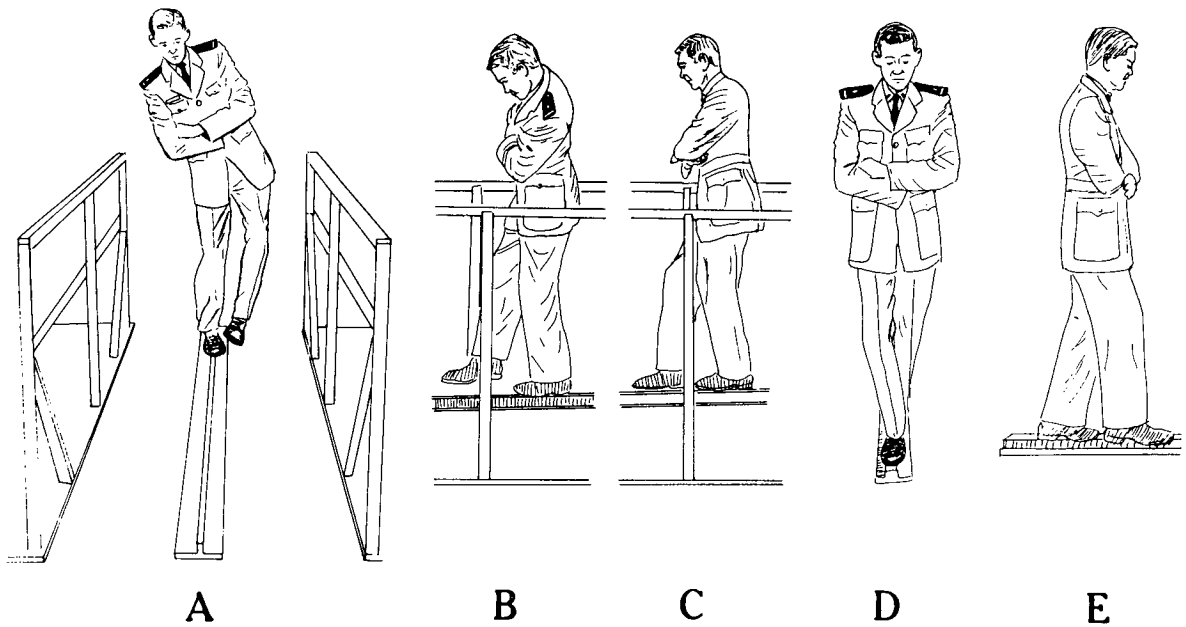


Figure 4.—*Test Battery (Short Version)*: A–B, *Walk H/T Test* (on  $\frac{3}{4}$ -inch-wide rail); C, *Stand E/O Test* (on  $\frac{3}{4}$ -inch-wide rail); and D–E, *Stand E/C Test* (on  $2\frac{1}{4}$ -inch-wide rail).

#### Test Battery (Short Version)

Rail 2 ( $2\frac{1}{4}$  in. wide, 30 in. long) and rail 5 ( $\frac{3}{4}$  in. wide, 8 ft. long) of the Long Version (wood version or metal version) (fig. 4), or a portable, foldable metal unit specific to the Short Version.

#### METHOD

##### Test Battery (Long Version)

The tests were performed with *shoes on*. Most male Ss wore a military or military-type shoe with relatively thick soles, whereas most female Ss wore relatively thin-soled flats. Prior to testing, all Ss read the following instructions:

##### Test Battery (Long Version)

###### Instructions

###### Test Sequence:

- (a) Walking with eyes open on each of six rails of varying width
- (b) Standing with eyes open on each of the six rails<sup>5</sup>

<sup>5</sup> Normal Ss began on rail 3, and if a perfect score was attained S was credited with perfect performances on rails 1 and 2. If score on rail 3 was less than perfect, testing was undertaken on rail 2 (and rail 1 if necessary) and upon completion S proceeded to rail 4.

- (c) Standing with eyes closed on each of the six rails

###### Body Position for All Tests:

- (a) Body erect or nearly erect
- (b) Arms folded against chest
- (c) Feet in heel-to-toe position
- (d) Feet tandemly aligned

Scoring: The *best two out of three* trials constitutes the scoring of each test.

- (a) *Walk H/T Test*—The first two steps, which are necessary for positioning on the rail, are not scored. A trial begins when the third step is taken.
- (b) *Stand E/O Test*—Timing begins as soon as correct position on the rail is assumed.
- (c) *Stand E/C Test*—You may take unlimited time for positioning yourself on the rail first with your eyes open. Timing will begin as soon as you close your eyes. Examiner will observe your eyes carefully, so that signaling the examiner is unnecessary.

###### General:

As there does not appear to be any single "best method," you must develop (rap-

idly) your own techniques. You may position your head up or down and/or forward or backward; you may lean forward or backward slightly if you do not prefer a perfectly erect position; *between trials*, alternation of the feet is permissible; you may place more weight on your front foot than on your rear foot or vice versa, or you may distribute your weight equally. However, a stooping position should be avoided.

After S read the instructions the examiner demonstrated all procedures and attempted to answer all questions raised about the manner of performing. The brief demonstration included illustrations of correct vs. incorrect body and foot positions, two or three demonstrations of walking one or more rails with emphasis that speed of walking should be considered secondary to negotiating the rails, and one or more demonstrations of appropriate positions for standing with eyes open and closed. The importance of maintaining the tandem, heel-to-toe position was re-emphasized as often as necessary. Ss were requested to avoid signaling the examiner upon closing the eyes to minimize losses of assumed position(s) on the rail(s).

Not included on the score sheet were the "false starts," defined as inadequate initial positioning on the rail leading to immediate loss of equilibrium on any trial, or a low time score (usually 2 or 3 seconds) not in keeping with a given S's generally higher level of performance. (Faulty techniques may be distinguished from inability even by inexperienced examiners.) The scoring procedures were as follows:

#### Scoring Procedures

##### Walk H/T Test

- (a) Each correct step is scored as one (step)
- (b) Maximum trial score equals five (steps)
- (c) Maximum rail score equals ten (steps), or total of the two best trials
- (d) Total score equals 60 (steps), the sum of all six rail scores.

##### Stand E/O Test

- (a) Timing, to the nearest second, begins when S assumes correct and balanced position on the rail, and timing ends at 60 seconds, or when S violates his position or falls off the rail
- (b) Maximum trial score equals 60 (seconds)
- (c) Maximum rail score equals 120 (seconds), the sum of the two best trials
- (d) Total score equals 720 (seconds), the sum of all six rail scores.

##### Stand E/C Test

- (a) Timing begins as soon as positioned S closes his eyes, and timing ends at 60 seconds or when S violates his position, or opens his eyes, or falls off of the rail
- (b) Maximum trial score equals 60 (seconds)
- (c) Maximum rail score equals 120 (seconds), the sum of the two best trials
- (d) Total score equals 720 (seconds), the sum of all six rail scores.

*Classical and Sharpened Romberg Procedures.*—Prior to undertaking the Test Battery, Ss were administered one trial of the Classical Romberg Test with eyes closed. Ss who failed to stand the required 60 seconds were then administered one trial of the Classical Romberg Test with eyes open. These Ss then were administered one trial in the Sharpened Romberg position with eyes closed. (The Sharpened Romberg position refers to the following: S attempts standing on floor for 60 seconds in arms-folded-against-chest, feet tandemly aligned and heel-to-toe.) Ss who failed to stand the required 60 seconds then attempted to stand in the Sharpened Romberg position for a period of 60 seconds with eyes open.

#### Test Battery (Short Version)

As with the Long Version, the tests were performed with *shoes on*. Again, most of the males wore a military or military-type shoe, whereas most female Ss wore relatively thin-soled flats. Prior to testing, all Ss read the following instruction sheet:

## Test Battery (Short Version)

*Instructions*

## Test Sequence:

- (a) Walking with eyes open on a  $\frac{3}{4}$  in. wide rail
- (b) Standing with eyes open on a  $\frac{3}{4}$  in. wide rail
- (c) Standing with eyes closed on a  $2\frac{1}{4}$  in. wide rail

## Body Position for All Tests:

- (a) Body erect or nearly erect
- (b) Arms folded against chest
- (c) Feet in heel-to-toe position
- (d) Feet tandemly aligned

Scoring: The *best three out of five* trials constitutes the scoring procedure.

- (a) Walk H/T Test—The first two steps, which are necessary for positioning on the rail, are not scored. A trial begins when the third step is taken.
- (b) Stand E/O Test—Timing begins as soon as correct position on the rail is assumed.
- (c) Stand E/C Test—You may take unlimited time for positioning yourself on the rail first with your eyes open. Timing will begin as soon as you close your eyes. Examiner will observe your eyes carefully, so that signaling the examiner is unnecessary.

## General:

As there does not appear to be any single "best method," you must develop (rapidly) your own technique. You may position your head up or down and/or forward or backward; you may lean forward or backward slightly if you do not prefer a perfectly erect position; *between trials*, alternation of the feet is permissible; you may place more weight on your front foot than on your rear foot or vice versa, or you may distribute your weight equally. However, a stooping position should be avoided.

As with the Long Version, after S read instructions the examiner demonstrated all procedures and answered all questions raised about the performance procedures. Examiner gave two or three demonstrations of

walking the  $\frac{3}{4}$  in. wide rail and one or two demonstrations of standing on each of the two rails. The scoring procedures were as follows:

*Scoring Procedures*

## Walk H/T Test

- (a) Each correct step is scored as one (step)
- (b) Maximum trial score equals five (steps)
- (c) Maximum test score equals fifteen (steps), the sum of the three best trials.

## Stand E/O Test

- (a) Timing, to the nearest second, begins when S assumes correct and balanced position on the rail, and timing ends at 60 seconds, or when S violates his position or falls off the rail
- (b) Maximum trial score equals 60 (seconds)
- (c) Maximum test score equals 180 (seconds), the sum of the three best trials.

## Stand E/C Test

- (a) Timing begins as soon as positioned S closes his eyes, and timing ends at 60 seconds or when S violates his position, or opens his eyes, or falls off the rail
- (b) Maximum trial score equals 60 (seconds)
- (c) Maximum test score equals 180 (seconds), the sum of the three best trials.

Sharpened Romberg Test (SR)<sup>6</sup>

All Ss prior to undertaking the Test Battery undertook the SR test. Ss who failed to stand for the required period of 60 seconds on the first trial were administered a second trial. Ss who failed to stand 60 seconds on the second trial were administered a third trial before proceeding with the Walk H/T test. Ss who failed to stand 60 seconds on the third trial were administered an additional (fourth) trial upon their completion of their Stand E/C test.

<sup>6</sup> Indicates standing in the Sharpened Romberg position with eyes closed for a maximum of four trials.

SR performance was scored as follows: a perfect score of 60 seconds on the first trial was weighted 4, and a score of 240 ( $60 \times 4$ ) was assigned; a perfect score on the second trial was weighted 3, and 180 ( $60 \times 3$ ) plus the number of seconds stood on the first trial became the assigned test score; a perfect score on the third trial was weighted 2, and 120 ( $60 \times 2$ ) plus the number of seconds stood on the first two trials became the assigned test score; with Ss requiring a fourth trial, the total number of seconds stood on the four trials became the assigned test score.

In addition to undertaking the SR test, a clinical-type ataxia test, on the occasion of undertaking the Test Battery, our most recently tested Ss undertook two additional clinical-type tests; (1) Stand One Leg Eyes Closed Test (SOLEC), and (2) Walk Line Eyes Closed Test (WALEC).

#### SOLEC

Ss undertook this test upon completion of the Test Battery. The task as a "static" test consists of standing on each leg (SOLEC-R and SOLEC-L) with arms folded against chest and with eyes closed for a period of 30 seconds. Ss were not permitted to make this a dynamic test by virtue of moving the standing foot in any way. Rather, it was required that the standing foot remain stationary. However, any amount of movement of the opposite leg or of the body was permitted so long as the body was maintained in an erect or near erect position. Ss were permitted to close their eyes at any time after assuming a correct standing position. Ss who violated the static foot requirement were stopped immediately, and the number of seconds stood prior to violation constituted the trial score. Ss began the test on the leg of their choice. Ss who required more than

one trial on each leg (for the perfect score criterion of 30 seconds) were requested to alternate legs on additional trials in the interest of reducing fatigue.

SOLEC performance was scored as follows: a perfect score on the first trial was weighted 5, and a score of 150 ( $30 \times 5$ ) was assigned; a perfect score on the second trial was weighted 4, and a score of 120 ( $30 \times 4$ ) plus the number of seconds stood on the first trial was assigned; a perfect score on the third trial was weighted 3, and a score of 90 ( $30 \times 3$ ) plus the number of seconds stood on the two previous trials was assigned; a perfect score on the fourth trial was weighted 2, and a score of 60 ( $30 \times 2$ ) plus the number of seconds stood on the three previous trials was assigned; with Ss requiring a fifth trial, the total number of seconds stood on the five trials became the assigned test score.

#### WALEC

Ss undertook this test upon completion of the SOLEC. The test consists of walking as straight as possible a 12-foot-long line on the floor at a typical (to the S) speed with eyes closed, arms folded against chest, and feet heel-to-toe. Ss alternated their starting positions from trial to trial. Each scorable trial required that S walk the entire length of the line. The number of inches of deviation from the line at the end of its 12-foot length constituted a trial score, and the total of the *two best trials out of three* (best equaled least deviant from the line) constituted the test score. A major limitation of the WALEC procedure is that in notably ataxic individuals the qualitative performance is often more deviant than the individual's score would indicate. Accordingly, the WALEC would appear to be more of a test of spatial orientation than of ataxia or of postural equilibrium.

#### REFERENCES

1. AMBLER, R. K.; AND BERKSHIRE, J. R.: A Forward View of Astronaut Personnel Procurement. Paper Presented at 70th Annual Convention of American Psychological Association (St. Louis), Aug. 30-Sept. 5, 1962.
2. ALDEN, F. D.; HORTON, M.; AND CALDWELL, G. M.: A Motor Ability Test for University Women for the Classification of Entering Students into Homogeneous Groups. Res. Quart. Amer. Ass. Health Phys. Ed., 3, 1932, pp. 85-131.
3. BABSKII, E. B.; GURFINKEL, V. S.; AND ROMEL', E. L.: Nova a metodika i nekotorye resul'taty issledovaniia vestibulomotornogo refleksa che-

- loveska prineadekuatom razdrazenii vestibularndgo apparata. Vestnik Oto-Rino-Laringologii (Moskva), 14, 1952, pp. 19-22.
4. BARBEY, E.: À propos du signe de Romberg et de sea variantes comme tests de l'équilibration statique. *Confin. Neurol.*, 6, 1944, pp. 162-168.
  5. BECK, K.: Untersuchungen über den statischen apparat von gesunden und taubstummen. *Z. Psychol. U. Physiol. D. Sinnesorg.*, 46, 1911-1912, pp. 362-378.
  6. BERGSTEDT, M.: Step Wise Adaptation to a Velocity of 10 RPM in the Pensacola Slow Rotation Room. Paper Presented at NASA Sponsored Symposium: "The Role of the Vestibular Organs in the Exploration of Space" (Pensacola, Fla.), Jan. 20-22, 1965.
  7. BIRREN, J. E.: Static Equilibrium and Vestibular Function. *J. Exp. Psychol.*, 35, 1945, pp. 127-133.
  8. BRUCK, A.: Ueber die beziehungen der taubstummheit zuzogenannten statischen sinn. *Pflügers Arch. f.d. ges. Physiol.*, 59, 1895, pp. 16-42.
  9. BULLARD, W. N.; AND BRACKETT, E. G.: Observations on the Steadiness of the Hand on Static Equilibrium. *Boston Med. & Surg. J.*, 119, 1888, pp. 595-603.
  10. CALLAHAN, F. E.: A Study of the Effect of the Loss of Visual Cues and of Flat-footedness on Locomotor Ability in Relation to Hearing Loss. Master's Thesis, Univ. of Kansas (Lawrence), 1958.
  11. COLLINS, V. D.; AND HOWE, E. C.: The Measurement of Organic and Neuromuscular Fitness. *Amer. Phys. Ed. Rev.*, 29, 1924, pp. 64-70.
  12. COURTNEY, D. M.; AND JOHNSON, B.: Skill in Progressive Movements of Children. *Child Develop.*, 1930, pp. 35-347.
  13. CURETON, T. K.: Physical Fitness of Champion Athletes. Univ. of Ill. Press, 1951.
  14. DAIN, C. A.: The Measurement of Dynamic Balance in Boys Between Ages 13 to 18 With a Beam-walking Test. Master's Thesis, Springfield College Library, Mass., 1938.
  15. DAVEY, P. R.: Clinical Note: Observation on Equilibrium in Deaf Children. *J. Laryng. & Otol.*, 68, 1954, pp. 329-331.
  16. FEARING, F. S.: The Experimental Study of the Romberg Sign. *The J. Nerv. Ment. Dis.*, 61, 1925, pp. 449-465.
  17. FLEISHMAN, E. A.: The Dimensions of Physical Fitness—The Nationwide Normative and Developmental Study of Basic Tests. Tech. Report 4, Yale Univ., 1962.
  18. FLEISHMAN, E. A.; THOMAS, P.; AND MUNROE, P.: The Dimensions of Physical Fitness—A Factor Analysis of Speed, Flexibility, Balance, and Coordination Tests. Tech. Rpt. 3, Yale Univ., 1961.
  19. FRISINA, R.: Motor Ability. *The Hlth. Phys. Ed. & Recreat. Rev.*, 1, 1957, pp. 3-6.
  20. FUKUDA, T.: The Stepping Test—Two Phases of the Labyrinthine Reflex. *Acta Otolaryng.*, 50, 1959, pp. 95-108.
  21. GOETZINGER, C. P.: A Re-evaluation of the Heath Railwalking Test. *J. Educ. Research*, 54, 1961, pp. 187-191.
  22. GRANT, W. T.: Romberg Test; Graphic Method of Recording Disturbances of Equilibrium. *Bull. Los Angeles Neurol. Soc.*, 8, 1943, pp. 25-30.
  23. GUEDRY, F. E.; KENNEDY, R. S.; HARRIS, C. S.; AND GRAYBIEL, A.: Human Performance During Two Weeks in a Room Rotating at Three RPM. *Aerospace Med.*, 35, 1964, pp. 1071-1082.
  24. GRAYBIEL, A.; AND JOHNSON, W. H.: A Comparison of the Symptomatology Experienced by Healthy Persons and Subjects With Loss of Labyrinthine Function When Exposed to Unusual Patterns of Centripetal Force in a Counter-Rotating Room. *Ann. Otol. Rhin. Laryng.*, 72, June 1963, p. 357.
  25. GRAYBIEL, A.; ET AL.: The Effects of Exposure to a Rotating Environment (10 RPM) on Four Aviators for a Period of Twelve Days. *Aerospace Med.*, 36, 1965, pp. 733-754.
  26. HARLAN, W. R., JR.; GRAYBIEL, A.; AND OSBORNE, R. K.: Longitudinal Study of Healthy Young Men Followed Over an Eighteen-Year Period. NSAM-829, 1962.
  27. HEAD, H.: The Sense of Stability and Balance in the Air. *The Medical Problems of Flying. Med. Research Council, Spec. Rpt. Series*, No. 53, Chap. 11, 1920, p. 217.
  28. HEATH, S. R.: Clinical Significance of Motor Defect, With Military Implications. *Amer. J. Psychol.*, 57, 1944, pp. 482-499.
  29. HEATH, S. R.: Rail-Walking Performance as Related to Mental Age and Etiological Type Among the Mentally Retarded. *Amer. J. Psychol.*, 55, 1942, pp. 240-247.
  30. HEATH, S. R.: The Military Use of the Rail-Walking Test as an Index of Locomotor Coordination. *Psychol. Bull.*, 40, 1943, pp. 282-284.
  31. HELLEBRANDT, F. A.; AND BRAUN, G. I.: Influence of Sex and Age on the Postural Sway of Man. *Amer. J. Phys. Anthropol.*, 24, 1939, pp. 347-360.
  32. HELLEBRANDT, F. A.; AND FRANSEEN, E. B.: Physiological Study of the Vertical Stance of Man. *Physiol. Rev.*, 23, 1943, pp. 220-255.
  33. HENMON, V. A. C.: Air Service Tests of Aptitude for Flying. *J. App. Psychol.*, 3, 1919, pp. 103-109.
  34. HERTZBERG, C. E.: The Relationship of Motor Ability to the Intelligence of Kindergarten

- Children. *J. Educ. Psychol.*, 20, 1929, pp. 507-519.
35. HINSDALE, G.: The Station of Man, Considered Physiologically and Clinically. *Amer. J. Med. Sci. N.S.*, 93, 1887, pp. 478-485.
  36. IGARASHI, M.; MCLEOD, M. E.; AND GRAYBIEL, A.: Clinical Pathological Correlation in Suppression of Semicircular Canal Function by Streptomycin Sulfate. *Acta Otolaryngol.* In press.
  37. JAMES, W.: The Sense of Dizziness in Deaf Mutes. *Amer. J. Otol.*, 4, 1882, pp. 239-254.
  38. KENNEDY, R. S.; AND GRAYBIEL, A.: Validity of Tests of Canal Sickness in Predicting Susceptibility to Air Sickness and Sea Sickness. *Aerospace Med.*, 33, 1962, pp. 935-938.
  39. KREIDL, A.: Beiträge zur physiologie des ohr-labyrinths auf grund von versuchen an taubstummen. *Pflügers arch. f.d. ges. Physiol.*, 51, 1892, pp. 119-150.
  40. MCFARLAND, R. A.; AND FRANZEN, R.: The Pensacola Study of Naval Aviators—Final Summary Report. CAA Div. Rsch. Rpt. No. 38, 1944.
  41. MILES, W. R.: Static Equilibrium as a Useful Test of Motor Control. *J. Indust. Hygiene*, 3(111), 1922, pp. 316-331.
  42. MONRAD-KROHN, G. H.: The Clinical Examination of the Central Nervous System. Harper and Bros., (7th ed.), 1938.
  43. MORSH, P. E.: A Comparative Study of Deaf and Hearing Students. *Amer. Ann. Deaf*, 82, 1937, pp. 223-233.
  44. MORTON, J. P.: Equilibrium Tests. *Canad. Pract. & Rev.*, 36, 1911, pp. 602-607.
  45. MOSS, F. S.: New Riding-Comfort Research Instruments and Wobble Meter Applications. *S. A. E. Jour.*, 30, 1932, pp. 182-184.
  46. MYKLEBUST, S. R.: Significance of Etiology in Motor Performance of Deaf Children With Special Reference to Meningitis. *Amer. J. Psychol.*, 59, 1946, pp. 249-258.
  47. NEWSOM, B. D.; BRADY, J. F.; AND GOBLE, G. J.: Equilibrium and Walking Changes Observed at 5½, 7½, 10 and 12 RPM in the Revolving Space Station Simulator (General Dynamics Corp., San Diego, Calif.), Presented at Thirty-fifth Annual Meeting, Aerospace Medical Association Meeting (Miami Beach), May 11-14, 1964.
  48. PIERON, H.: Interpretation of Labyrinthine Troubles Manifested in Tonic Reflectivity in Standing and Walking. *C. R. Soc. Biol.*, 81, 1918, pp. 663-675.
  49. RICALDONI, M. A.: Plate-Forme Mobile pour l'observation et l'enregistrement graphique du phenomene du Romberg et d'autres troubles de la statique. *Soc. Med.d. Hsp. de Paris*, 52, 1928, pp. 1138-1145.
  50. ROMBERG, M. H.: Manual of the Nervous Diseases of Man. London: Sydenham Trans., 2, 1853, p. 396.
  51. ROSENFELD, M.: Zur methodik der untersuchung auf gleichgewichtsstörungen. *Arch Psychiat. Nerven-Krankh.*, 59, 1918, pp. 287-300.
  52. SEASHORE, H. G.: The Development of a Beam-Walking Test and its Use in Measuring Development of Balance in Children. *Res. Quart.*, 18, 1947, pp. 246-259.
  53. SKOGLAND, J. E.: A Quantitative Study of Normal and Pathological Station in Human Subjects. *Medical Record*, 155, 1942, pp. 15-22.
  54. SMITH, K. U.: Sensory Feedback Analysis of Man-Like Machine Systems: A New Behavioral Theory for Human Engineering. Special Report to the Nat. Sci. Foundation (Project 3785) and to the NIH (Project M-4469). Presented before a Symposium on Motor Skill and Vehicle Design of the Southern Psychol. Assoc., Apr. 13, 1963.
  55. TRAVIS, R. C.: A New Stabilometer for Measuring Dynamic Equilibrium in the Standing Position. *J. Exp. Psychol.*, 34, 1944, pp. 418-424.
  56. TRAVIS, R. C.: An Experimental Analysis of Dynamic and Static Equilibrium. *J. Exp. Psychol.*, 35, 1945, pp. 216-234.
  57. VAGLINI, F.: La prova del cammino a stella in soggetti normali. *Boll. mal. Orecch.*, 79, 1961, pp. 360-366.
  58. VAZUKA, F. A.: Essentials of the Neurological Examination. Smith, Kline and French Laboratories, Philadelphia, Pa., 1962.
  59. VERNON, J. A., ET AL.: Effect of Sensory Deprivation on Some Perceptual and Motor Skills. *Percept. Mot. Skills*, 9, 1959, pp. 91-97.
  60. VIERODT: Physiologie Des Menschen. Tubingen, 1862.
  61. WAPNER, S.; AND WITKIN, H. A.: The Role of Visual Factors in the Maintenance of Body-Balance. *Amer. J. Psychol.*, 63, 1950, pp. 385-408.
  62. WITKIN, H. A.; AND WAPNER, S.: Visual Factors in the Maintenance of Upright Posture. *Amer. J. Psychol.*, 63, 1950, pp. 31-50.
  63. WOOD, C. D.: Review of Antimotion Sickness Drugs From 1954-1964. Special Report 64-2, Naval School of Aviation Medicine, 1964.
  64. WHITNEY, E.: A Beam-Walking Test of Dynamic Balance. Master's Thesis, Springfield College Library, Mass., 1937.
  65. WORCHEL, P.; AND DALLENBACH, K. M.: The Vestibular Sensitivity of Deaf-Blind Subjects. *Amer. J. Psychol.*, 61, 1948, pp. 94-99.
  66. WORCHEL, P.; AND DALLENBACH, K. M.: Vestibular Sensitivity in the Deaf. *Amer. J. Psychol.*, 63, 1950, pp. 161-175.



*DISCUSSION*

CHAIRMAN: I'm interested in how soon the police, for example, might use your battery instead of alcohol determination as criteria for "drunkenness."

FREGLY: It would be quite interesting to see if one could do that.

MANN: Have you made any comparison between this test and the Dallenbach test, a very simple one of standing with your eyes closed first on your right foot, then on your left foot, then on your right foot?

FREGLY: We did indeed look very seriously at Worchel's and Dallenbach's studies with the deaf and blind, and felt that our method was the preferred method.

WHITE: We have incorporated this new equilibrium test in a series of studies that we're doing now on

sleep and centrifugation. We have twelve subjects in this program. They did their baseline walks the other night, and certainly this matter of footwear is a point of concern. Have you given any thought to the possibility of a clog shoe or something of that sort that might be standardized with respect to this?

FREGLY: Yes, we did. We have several kinds on the drawing board and this remains as a perplexing problem for scientific research. We had in mind here particularly a clear device to offer maximum cutaneous cues in the learning process in the case of blind individuals. But we had looked at this variable of footwear very carefully, and with everything considered the present scheme presents the least variable factor.

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# Otolith Function as Measured by Ocular Counterrolling

EARL F. MILLER II

AND

ASHTON GRAYBIEL

*U. S. Naval School of Aviation Medicine*

## SUMMARY

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This report summarizes the results of several studies in which the effect upon otolith activity of change in magnitude or direction of the gravito-inertial force was determined in man by measuring the particular vestibulo-ocular reflex of counterrolling. In these studies hypergravic forces were generated by means of centrifugation while periods of partial or complete weightlessness were produced by ballistic flights in specially equipped aircraft. Since the available periods of reduced gravity were relatively short, the rapid adjustment in counterrolling position in response to the acting gravitational stimulus made this index of otolith activity particularly well-suited for study. A photographic method developed for quantitatively studying counterrolling movement in normal and labyrinthine-defective subjects provided the accuracy required for measuring the extremely small response changes found under reduced G loading. The average data of several normal subjects indicated that the relationship between otolith activity and gravitational force expressed in  $\log_{10}$  units was linear, i.e. obeyed Fechner's law, from approximately 0.6 G up to at least 1.0 G and probably beyond. Below 0.6 G the otolithic response began to deviate from linearity and asymptotically approached its zero level at some infinitely small G value. The possible application of these findings to manned space flights was outlined.

Author

## INTRODUCTION

This report will summarize some investigations carried out at the U.S. Naval School of Aviation Medicine which have considered the action of the otolith organs under the influence of unusual force environments with particular emphasis upon weightlessness. The initial stimulus for these researches stemmed from the need to evaluate the role of gravito-inertial force (GIF) in causing disorientation in pilots (ref. 1) for it was learned early that exposure to unusual force environments greatly affected the pilot of conventional aircraft through the sensory receptors in his vestibular organs. Now space flight has provided an added impetus to such studies by introducing the bizarre condition of weightlessness and the possibility that

astronauts will be exposed to a rotating environment. Without the adequate stimulus of gravitational force, the otolith apparatus might be expected to convey unusual neural messages to the central nervous system. For this reason it was predicted by various writers (refs. 2 to 5) that the integration of the resultant changes in gravireceptor input might affect man's behavior. Concern was expressed that gross difficulties would be encountered, but these, largely based upon assumptions and hypotheses, would appear exaggerated from the reports of the participants in the Mercury (ref. 6), Vostok and Voskhod flights (ref. 7). Carefully selected individuals have functioned relatively well under agravic conditions with only slight disturbances which can be specifi-

cally related to an influence of weightlessness upon the vestibular system. The problem must still be considered unsettled, however, until more prolonged flights can be accomplished and experimental studies of vestibular function are made throughout this weightless exposure.

For the investigation of changes in basic otolithic response to decreased G loading it has not been necessary to await availability of time and space in an orbiting capsule or laboratory. Through the cooperation of the U.S. Air Force, experiments were carried out within special aircraft capable of flying precise Keplerian trajectories. Although relatively short, the time of these transient periods of reduced gravity was found to be entirely adequate for studying otolithic function as determined by measurements of a particular vestibulo-ocular reflex known as counterrolling.

#### COUNTERROLLING

Counterrolling may be defined as the involuntary conjugate rolling movement of the eyes around their lines of sight in the direction opposite to the lateral inclination of the head with respect to gravity. "Inclination of the otolith organs" could be substituted for that of the "head" since it is implied in the definition. The mechanics of stimulation is revealed by the microscopic anatomy and the topography of these organs.

#### Otolith Organs

Within two sac-like structures in the inner ear are found tiny calcareous crystals (otoliths) which are supported by mucous or gelatinous material. The otoliths being higher in specific gravity are relatively displaced in the surrounding matrix by changes in direction and magnitude in gravity or inertial forces relative to the macular plates. Without entering into the controversial theories of the exact mode of stimulation, it is sufficient to say that the hair-like projections of sensory cells embedded within the gelatinous layer are bent by slight relative displacement of the otoliths resulting in a transducer effect converting mechanical into

neural energy. The frequency pattern of nerve impulses traveling over the eighth nerve to the brain probably depends upon the anatomical spatial arrangement of the two pairs of otolith organs (utricle and saccule) and their orientation with respect to the acting GIF. Among the nerve pathways traveled by these impulses are those to the extraocular muscles and as such these otolith organs represent a source of tonic innervation to these muscles. When the GIF is altered in the subject's lateral plane, a change in tonicity normally results which is reflected in a torsional shift of both eyeballs around their lines of sight as the axis. Measurement of the amount of counterrolling associated with a change in lateral direction or magnitude of GIF provides the best known objective means of determining correlative changes in otolithic activity in man.

#### Measurement of Counterrolling

The distinct advantage of having an external indicator of otolith function in the human in the form of the counterrolling reflex has been outweighed in the past by the great difficulty in obtaining precise measurements of this response (ref. 8). Throughout the long colorful history of counterrolling studies, several methods of measurement have been used. All have as a common basis the selection of anatomical landmarks on the eye to establish a reference plane containing the line of sight for specifying rolling of the eye. Most of these methods were found to be insufficiently sensitive or difficult to employ in certain experimental situations.

A method involving photography of natural landmarks on the iris was devised to meet the requirement of greater precision in measurement. A solution to the problem of measuring very small amounts of movement of these landmarks was found in simple magnification. In this procedure a 35 mm film image of the entire eye is enlarged over 300 times the actual size by projection onto a distant screen. Measurement of angular torsional movement around the center of the pupil is then accomplished by superimposing

upon each test image in succession a second projected image of the subject's eye serving as a standard of comparison. More complete details of this measuring technique have been published (ref. 8). It is sufficient for this discussion to point out that a high degree of accuracy and reliability in measurement ( $\pm 5$  minutes of arc) is possible with this procedure.

#### Normal Subjects

Counterrolling measurements using the photographic technique have been made on many normal individuals tilted up to  $\pm 75^\circ$  from the gravitational vertical (fig. 1). Normal subjects typically reveal qualitatively similar counterrolling response to lateral head inclination but quantitatively there are certain interindividual differences (ref. 9). There are also significant right-left differences in some individuals but not in others. A more extensive study (ref. 8) in which measurements were made at every 15 degrees of body tilt within the subject's frontal, sagittal, and two intermediate planes revealed that maximal compensatory torsional eye movement occurred in the frontal plane, was somewhat less in the intermediate planes, and was not present in the sagittal plane. This study revealed that counterrolling increased fairly rapidly up to maximum at a head inclination between 60 and 70 degrees. From this point on counterrolling decreased, but at a lesser rate than it increased, reaching about zero when the head was positioned vertically downward. A considerable amount of variability among individual measurements far greater than the measuring error has been found in almost every subject tested, indicating that a certain amount of physiological unrest exists with respect to the anteroposterior axis of the eye. This variability has been observed by several authors (refs. 10 to 12) using various measuring techniques and requires that several measurements be made for each GIF condition investigated. This requirement was considerably more difficult to meet in the weightless state since only a limited number of measurements could be made for each parabolic flight.

#### Labyrinthine-Defective Subjects

The photographic technique of measurement was found to be particularly useful in accurately measuring smaller than normal amounts of counterrolling that are manifested by individuals with disease or otherwise damaged vestibular organs (ref. 9). Successful use of this particular technique in these studies paved the way for accurately determining the decreased level of activity of normal otolith organs under reduced GIF, as will be described. Investigators with less sensitive measuring devices had the difficult task of differentiating between a relatively large measuring error and a possible small residuum of otolith function in their labyrinthine-defective subjects. For the same reason studies of human otolith function in subgravic states would have been difficult if not impossible with most of these former methods.

Information gained from precise measurements of otolith organ activity is needed to evaluate completely inner ear function. There is evidence as described below (ref. 9) that even complete loss of hearing and semicircular canal function does not necessarily indicate that otolithic function is similarly affected. Also it has been found (unpublished data from "Effects of Partial Suppression of Vestibular Function Ten Years After Treatment of Ménière's Disease With Streptomycin Sulfate" by A. Graybiel, et al.) that in subjects with Ménière's Disease treated with streptomycin sulfate, suppression of semicircular canal function can occur with little loss of otolith function. These results indicate that all subjects used in studies involving stimulation of the vestibular apparatus should be given specific quantitative tests of these organs. A standardized counterrolling test is now used routinely at the U.S. Naval School of Aviation Medicine to complement audiometric, threshold caloric irrigation (ref. 13) and other tests of the other two auricular organs.

The use of counterrolling as a specific indicator of otolith activity is based upon the assumption that the level of function is re-

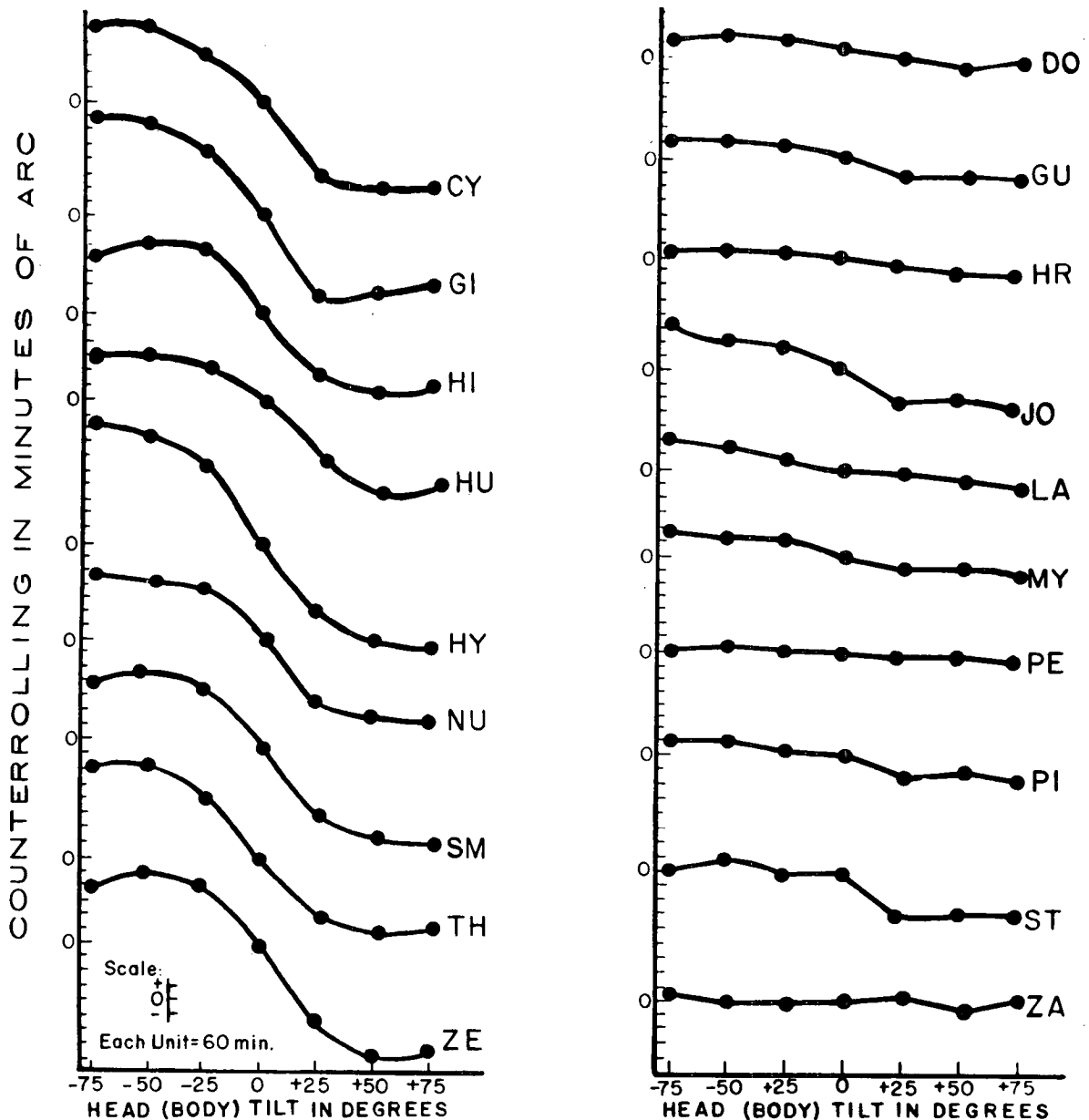


Figure 1.—Mean counterrolling values plotted as a function of leftward and rightward tilt. Left: Normals; right: L-D subjects.

vealed by the character of this response. We examined this theory in an experiment involving 10 deaf subjects with severe and complete bilateral loss of the semicircular canals (ref. 9). As can be seen in figure 1 these labyrinthine-defective (L-D) subjects did not disclose the characteristic pattern found in normal subjects in most instances. The magnitude of the response was in all cases less than in a comparable normal

group. In some instances, there was no definite evidence of counterrolling, in others it was limited to one direction, and in still others there was a small but regular dependence of counterroll with the successive increase in bodily tilt. The highly significant differences between the normal and L-D groups must have been due to loss of function of the sensory organs of the inner ear. More specifically, since there is no evidence

that the counterrolling reflex is released by the organ of Corti, and insufficient evidence that it originates in the semicircular canals, but good evidence that it is released by the otoliths, it was concluded that the reduction in counterrolling in these cases was the result of injury to the otoliths. Interindividual differences in the L-D group were therefore best explained by the presence of some residual otolith function.

#### HYPERGRAVITATIONAL FORCE

Within the Earth's gravitational environment counterrolling is useful in determining the functional loss of the otolithic gravireceptor organs or their nerve pathways. Once it has been established that an individual has normal otolithic function, his counterrolling response can be used to indicate changes in otolithic response to variations in magnitude of GIF.

In hypergravic states counterrolling was found (ref. 14) to increase directly with the magnitude of force and its limit was not reached even with a lateral force of 2.25 G. Data of this and other studies (ref. 15 and unpublished data, E. F. Miller II and A. Graybiel) demonstrate the important fact that counterrolling is normally stimulus bound. Counterrolling can therefore be used as an indicator of otolith response to hypergravic stimulation up to limits as yet undetermined. In the Woellner and Graybiel study the effect of changing the magnitude of the lateral G force was determined by tilting the subject with respect to gravity. The magnitude effect in the range from zero to 1 G was thus estimated under terrestrial conditions by varying the orientation of the otolith organs with respect to the ever present force of gravity. In order to study the effect of a reduction in magnitude of G force, *per se*, however, it is necessary to keep the direction of force constant. This requirement for subgravity states can only be accomplished aloft in parabolic or orbital flight.

#### HYPOGRAVITATIONAL FORCE

In several experiments involving special aircraft (C131B) equipped to fly Keplerian

trajectories the normal gravitational pull of the Earth was counteracted partially or completely in specific amounts. In the initial study (ref. 16) using a tilt chair device and photographic equipment to record eye position, the counterrolling response of six normal and six L-D subjects was measured at five different tilt positions under zero G, one-half G, and standard G conditions. Although the subgravity periods obtained in this experiment were necessarily relatively short (5-6 seconds) the shifts in torsional eye position in response to changes in gravitational force occurred much more rapidly. The average results of the normal group of subjects are portrayed in the figure 2. It can be seen that otolith activity as indicated by counterrolling decreased in a regular fashion as the force was reduced. In the weightless condition tilting the normal individual would appear to have little, if any, effect upon the activity of the otolith organs under the conditions of the test. These data, as far as we know, provide the first quantitative evidence that the otolith organs in man are physiologically deafferentated when he is exposed to hypogravitational environments. The L-D subjects manifested a greatly reduced pat-

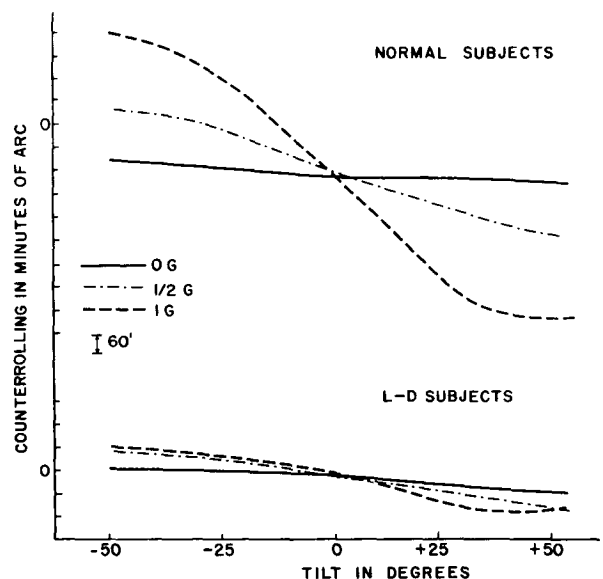


Figure 2.—Mean counterrolling response to tilt of normal and L-D subjects under 0, 0.5, and 1.0 G conditions.

tern of response which resembled that of the normals. This could be interpreted either as actual change in the already greatly reduced otolith function of the L-D subjects or as an effect of stimulation of extra-labyrinthine source(s) of tonic innervation to the extra-ocular muscles. The former explanation seems more reasonable based upon the care exercised to eliminate cervical, fixational, or binocular sources of cyclorotational eye movement. Even if such factors were involved, their importance is not great as evidenced by the small changes in counterrolling measured in the L-D group; it would appear therefore that when certain variables are adequately controlled, otolith organ activity as a function of subgravity forces is revealed by ocular counterrolling.

It was interesting to note that under one-half G conditions the normal subjects manifested the characteristic counterrolling response, but quantitatively this reflex movement on the average was markedly reduced and substantially less than the level midway between zero and 1 G. These data suggested that the otolith activity is not linearly related to G force at least throughout the complete range between zero and the Earth's standard level. A followup study was made to determine more exactly the nature of this function by exploring several subgravity levels.

In carrying out this second experiment (unpublished paper entitled "Otolith Activity as a Function of Gravitational Acceleration from Zero to One G" by E. F. Miller II, R. S. Kellogg, and A. Graybiel) the same equipment and aircraft type (C131B) were used. The parabolic maneuvers were designed to yield subgravity periods which were approximately twice as long as those of the first study. Three young healthy pilots served as subjects. Each was placed, in random order by means of the tilt-chair device, at visual upright or tilted from this position counterclockwise 25 or 50 degrees. Two usually successive parabolas were flown for each tilt position and for each subgravity level which was induced in the following

order: 0.75, 0.50, 0.33, 0.20, 0.17, 0.10, 0.05, 0.00, and 1.00 G. Four recordings of eye position were made during the latter portion of the subgravity phase of each parabola flown. This experimental design would have yielded for each individual up to eight eye recordings at each of his three tilt positions under nine gravitational conditions in the original test session and the same number in the re-test session on a separate day. As it happened, the numerous difficulties in flying "perfect" trajectories made it impossible to achieve always the exact and constant level of subgravity force desired. This result was unimportant to the experiment since the on-board accelerometer recordings were used to define G force to within  $\pm 0.001$  G. The continuous write-out of the linear accelerometer system was marked each time the camera fired in order to correlate the manifested counterrolling precisely with the gravitational force existing at that moment. The small variations in the planned flight maneuver had the effect of increasing the number of G levels investigated and reducing considerably in some cases the number of records at each level. For this reason the counterrolling measurements were grouped according to their correlative G value in steps of 0.05 G as indicated in figure 3 which summarizes the results; the test-retest data of each subject were quite similar, and therefore only the average data of the two test sessions are presented.

Since averaging counterrolling measurements for data reduction purposes has the effect of masking the variability inherent in this response, it should be borne in mind that the empirical points in figure 3 represent the mean positions of the eye about which small apparently continuous rolling adjustments occur. Although not specifically investigated in this study, variability in eye position including occasional spontaneous rolling "jerk" movements of considerable amount ( $1^\circ$  or more) were not any more apparent at the lower than at the normal end of the G range tested.

Qualitatively, it can be seen that the sub-

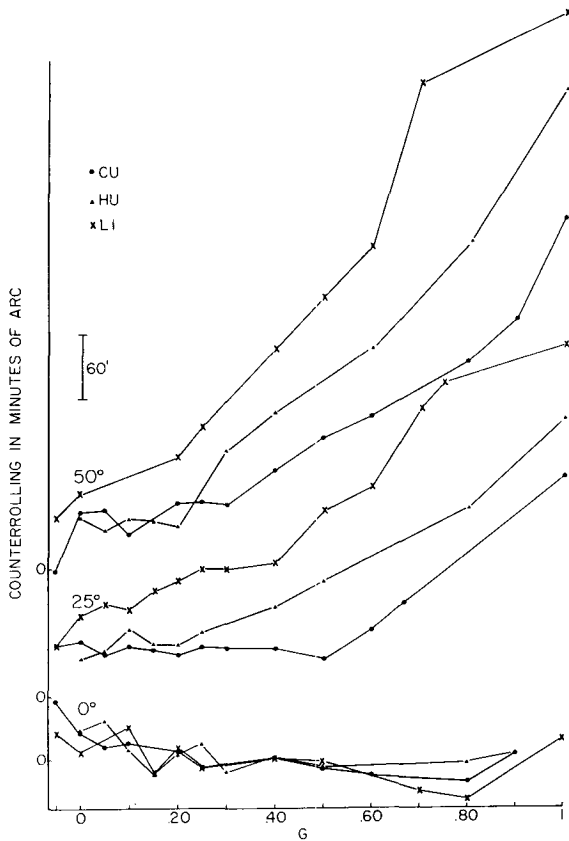


Figure 3.—Ocular counterrolling data of three subjects positioned 0°, 25°, and 50° from visual upright as a function of G force.

jects revealed essentially the same results. The substantial interindividual quantitative differences in counterrolling as shown in the figure 3 are well within normal range. At upright, the usual position for determining the basic (zero) mean eye position, the data revealed a possible slight increase in counterrolling (clockwise movement) of the eye as the G loading was reduced. Under weightlessness and slight negative G force conditions counterrolling in the 25 and 50 degree positions, although approximately at the same level, was slightly higher than in the upright position. The reasons for this are not known but may reflect a residual hyper-tonicity of the extraocular muscles resulting from the heightened eye rolling response to the hypergravic force (2½ G) that preceded the weightless period. Artifact or not, the

effect is quite small and unimportant in considering relative changes. As the gravitational force increased above zero there was initially for the two constantly held positions of tilt only rather weak counterrolling response which tended to increase at an ever increasing rate to reach a maximum at 1 G.

An alternative method of plotting the data was used to explore linearity and therefore possible compliance with Fechner's law as expressed by the formula:

$$\psi = \log_{10} G + b$$

where  $\psi$  represents counterrolling (otolith activity), G, gravitational force, and b, a constant. The counterrolling data were averaged for all three subjects in each of the two tilt positions and transformed into an arbitrary scale system in which the unit equals the average maximum counterrolling exhibited in this study (50° tilt, 1.0 G). The counterrolling data in relative units were plotted as points (open circles, 25°; closed circles, 50°) in relation to the logarithm of the acting G force as shown in figure 4. The respective data points for both the 25° and 50° tilt positions could be readily fitted with two straight lines between 1.0 and 0.06 G as shown in figure 4, but below this gravitational level the lines of apparent fit had to be continuously bent to reach finally the basic, resting level of otolith activity in weightlessness. The asymptotic character of the resultant curves indicates without precise definition that the absolute threshold of the otolith system is quite small. This would be in general agreement with the estimates of the high sensitivity reported for the otolith receptors in man (ref. 17), 0.000344 G, cats (ref. 18, after data of Adrian) 0.017 G, and indicated for the lateral line organ of fish by Granit's (ref. 19) observation that the gentlest tapping of the table or even someone walking in his laboratory stimulated his preparation. Although apparently stimulated by only a very small fraction of the normal gravitational pull, the curves of figure 4 reveal that, as the G load is increased above threshold, otolith activity is initially raised only slightly; it should be noted that



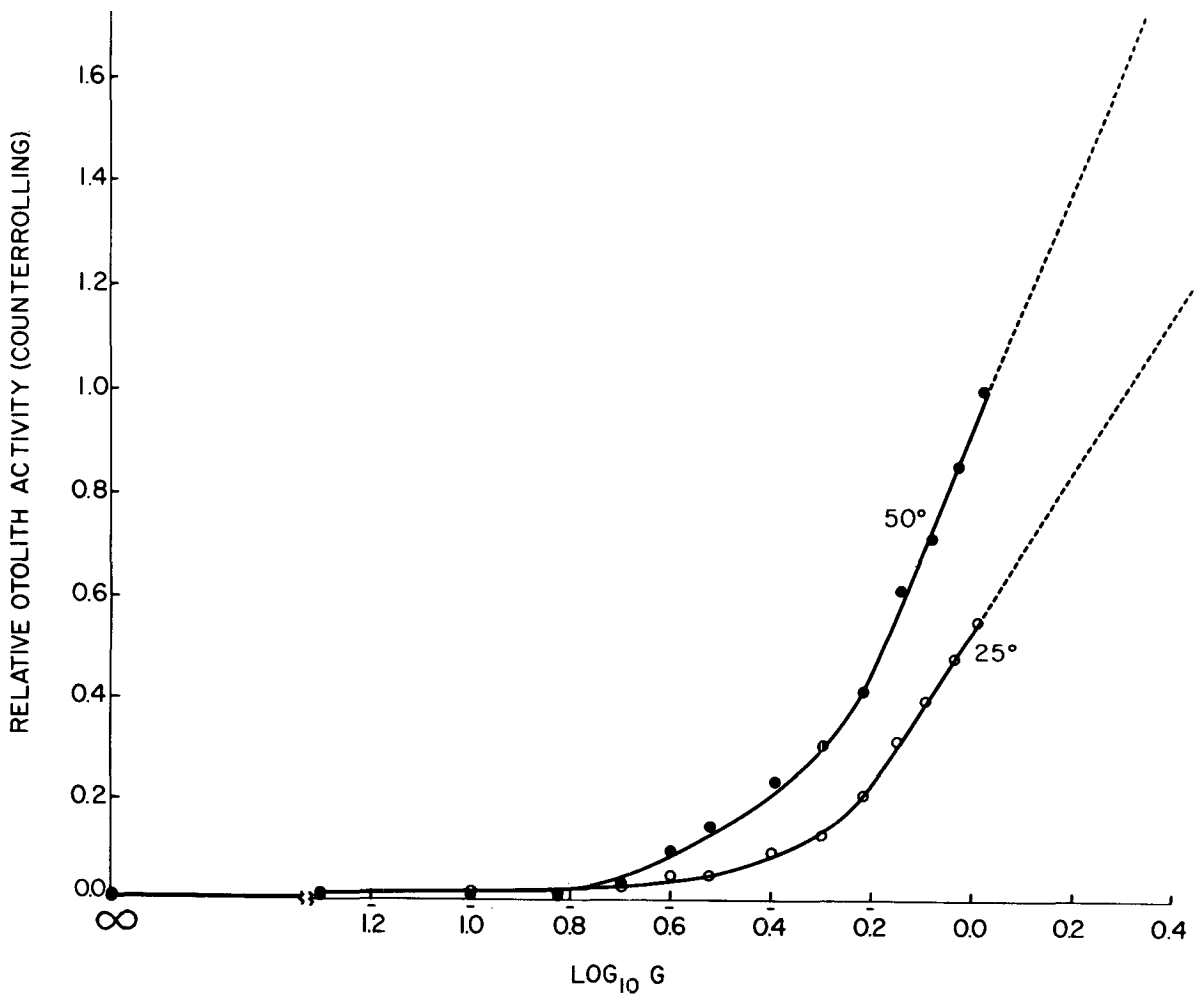


Figure 4.—Relative otolith activity (mean counterolling response of three subjects, fig. 1) as a function of the logarithm of the gravitational stimulus.

the range of greatly reduced activity includes that for lunar gravity. The change in otolith activity increases steadily and in ever increasing amounts as a direct function of this load. Above approximately 0.6 G otolith activity becomes proportional to the logarithm of G force at least up to 1.0 G. Beyond this value a continuation of this linear function is suggested by certain other counterolling data which were collected under hypergravic conditions. Colenbrander (ref. 15), for example, using the subjective location of the blind spot to specify torsional eye position, found that the counterolling response was linear between 1.0 and 2.0 G;

specifically, he based his conclusion on the fact that the difference in his counterolling measurements taken at 1.0 and 2.0 G was twice that difference found between 1.0 and 1.5 G. Certain unpublished data by Miller and Graybiel involving counterolling measurements for 50° tilt at 1.556 resultant G force revealed that the average increase in counterolling as measured on seven normal subjects seated upright on a constantly revolving centrifuge was greater than 1.3 times the comparable 1.0 G value. As shown in figure 4 a straight line (dotted) extension of the 50° curve intercepts a relative value at 1.556 G which is only slightly higher

(1.45) than this ratio. Further extension up to 2.0 G yields a ratio of about 1.7 which is similar to that indicated in the data of Woellner and Graybiel (ref. 14). It would appear, therefore, that Fechner's law is a valid description of otolithic response to gravitational stimulation from about 0.6 G up to some hypergravic level. It is interesting to note that the lateral line organ of fish obeys this law: Neural activity (spike frequency) was found to be proportional to the logarithm of the mechanical stimulus (rate of flow). Additional data are required to determine at what level, possibly above 2.0 G, logarithmicity ends. If, as expected, a gradual deviation from the Fechnerian function occurs in the hypergravic range as it did below 0.6 G, an S-shape curve would be generated which would be similar to that found to describe the full range of other biological transducers.

Haber and Gerathewohl (ref. 3) originally suggested that the relation between sensation and gravitational stimulation might be described by the Weber-Fechner law. An important consequence of their hypothesis, assuming strict adherence to this law, would be that the gravity sense is particularly sensitive in the proximity of G zero and strong sensations would be caused by minute changes in acceleration at this G level. These authors pointed out, however, that such an analysis is based upon assumptions and conclusions by analogy, and only experiment will decide. Gerathewohl (ref. 20), in a later study, suggested that this psychophysical relationship be represented by a "modified" Weber-Fechner function which yields an S-shape curve. If this be the case they argued that only slight changes in sensation would result from small inertial forces under gravity-free conditions. This appears likely on the basis of the evidence acquired in the counterrolling studies already described. Thus, small linear accelerations which may be produced during an orbital or

space flight probably would not be significant as stimuli to the otolith organs. This response characteristic of the otolith organs may be of an advantage in space flight. On the other hand, if some damping effect of inertial force acting on the otolith apparatus is found to be required to defend against adverse effects of prolonged weightlessness, it may be a disadvantage.

#### *APPLICATION OF COUNTERROLLING DATA TO MANNED SPACE FLIGHTS*

From the studies presented summarily in this report there are certain results concerning otolith activity that may be directly applicable to future manned space flights: (1) the otolith organs are physiologically deafferentated; i.e., otolith activity is reduced to its resting level by weightlessness; (2) the threshold of these organs is a very small fraction of normal gravity; (3) relatively little change in otolithic response to slight increases in inertial stimulation would result in a weightless environment; (4) little benefit could be expected from otolith activity as stimulated by lunar gravity alone; (5) the frequently proposed levels (0.2-0.3 G) of artificial gravity for manned space vehicles would provide very little additional otolithic input. Counterrolling data such as presented might be useful in estimating the approximate effect upon the otolithic gravireceptors of any inertial force that may be generated in a spacecraft.

It is essential to keep in mind that the effect of prolonged weightlessness upon the otolith organs is still unknown, and that the above statements are based upon experimental data obtained during transient exposures to zero G. Whether these factors hold true when the time factor is extended can only be answered through further experiments designed to measure changes in otolithic response of the astronaut throughout his prolonged flight.

## REFERENCES

1. GRAYBIEL, A.: Spatial Disorientation in Flight. *Milit. Surg.*, 108, 1951, pp. 287-294.
2. ARMSTRONG, H. G.; HABER, H.; AND STRUGHOLD, H.: The Aero Medical Problems of Space Travel. *J. Av. Med.*, 20, 1949, pp. 383-402.
3. HABER, H.; AND GERATHEWOHL, S. J.: On the Physics and Psychophysics of Weightlessness. *J. Av. Med.*, 22, 1951, pp. 180-189.
4. LEVINE, R. B.: Zero Gravity Simulation. In: *Weightlessness-Physical Phenomena and Biological Effects* (E. T. Benedikt, ed.), Plenum Press (New York), 1960, pp. 135-153.
5. SLATER, A. E.: Sensory Perceptions of the Weightless Condition. In: *Realities of Space Travel* (L. J. Carter, ed.), Selected Papers of the British Interplanetary Society, London, 1957, pp. 266-274.
6. HENRY, J. P., ET AL.: Effects of Weightlessness in Ballistic and Orbital Flights. *Aerospace Med.*, 33, 1962, pp. 1055-1068.
7. GAZENKO, O.: Medical Tests on Cosmic Ships Vostok and Voskhod. Presented at the Third International Symposium on Bioastronautics and Exploration of Space, San Antonio, Texas, Nov. 16, 1964.
8. MILLER, E. F., II: Counterrolling of the Human Eyes Produced by Head Tilt with Respect to Gravity. *Acta Otolaryng. Stockh.*, 54, 1962, pp. 479-501.
9. MILLER, E. F., II; AND GRAYBIEL, A.: A Comparison of Ocular Counterrolling Movements Between Normal Persons and Deaf Subjects with Bilateral Labyrinthine Defects. *Ann. Otol.*, 72, 1963, pp. 885-893.
10. FISCHER, M. H.: Messende Untersuchungen über die Gegenrollung der Augen und die Lokalisation der scheinbaren Vertikalen bei seitlicher Neigung (des Kopfes, des Stammes und des Gesamtkörpers). Neigungen bis zu 40°. v. Graefes *Arch. Ophth.*, 118, 1927, pp. 633-680.
11. GRAHE, K.: Otolithenprüfung beim Menschen. *Z. Hals-Nas. -u. Ohrenheilk.*, 18, 1927-1928, pp. 411-422.
12. GRAYBIEL, A.; AND WOELLNER, R. C.: New and Objective Method for Measuring Ocular Torsion. *Amer. J. Ophthal.*, 47, 1959, pp. 349-352.
13. MCLEOD, M. E.; AND MEEK, J. C.: A Threshold Caloric Test for the Horizontal Semicircular Canal. NSAM-834, NASA Order No. R-47. Naval School of Aviation Medicine, 1962.
14. WOELLNER, R. C.; AND GRAYBIEL, A.: Counterrolling of the Eyes and Its Dependence on the Magnitude of Gravitational or Inertial Force Acting Laterally on the Body. *J. Appl. Physiol.*, 14, 1959, pp. 632-634.
15. COLENBRANDER, A.: Eye and Otoliths. *Aeromed. Acta*, 9, 1963-1964, pp. 45-91.
16. MILLER, E. F., II; KELLOGG, R. S.; AND GRAYBIEL, A.: Otolith Organ Activity within Earth Standard, One-Half Standard, and Zero Gravity Environments. NSAM-943, NASA Order No. R-93. Naval School of Aviation Medicine, 1965.
17. GRAYBIEL, A.; AND PATTERSON, J. L., JR.: Thresholds of Stimulation of the Otolith Organs as Indicated by the Oculogravic Illusion. *J. appl. Physiol.*, 7, 1955, pp. 666-670.
18. MARGARIA, R.; AND GUALTIEROTTI, T.: Body Susceptibility to High Accelerations and to Zero Gravity Condition. *Advances in Aeronautical Sciences*, 3-4, 1961, pp. 1081-1102.
19. GRANIT, R.: *Receptors and Sensory Perception*. Yale University Press, (New Haven), 1955.
20. GERATHEWOHL, S. J.: Comparative Studies on Animals and Human Subjects in the Gravity Free State. *J. Av. Med.*, 25, 1954, pp. 412-419.

## DISCUSSION

STEELE: You might say you were comparing the information reported by the otolith organ with the force vector, which has direction and length, and checking the Weber-Fechner relationship by varying the length of the vector. However, any major change in the angle reported by it need not be expected to follow logarithmically with the length of the vector.

MILLER: In these studies, we have attempted only to keep the direction of the force vector constant, which varied its magnitude, and this did yield, at least throughout that part of the G range tested, a Fechnerian function.

MAYNE: Dr. Miller, it seems to me I remember from your paper that the counterrolling of the eyes was proportional to the component of gravity perpendicular to the longitudinal axis of the body. Your

present record indicates a logarithmic relation. Did I misread your earlier paper?

MILLER: In my paper on counterrolling, the subject was tilted in the lateral plane. As typically found for normal subjects, his eyes counterrolled up to a maximum with about 60 or 70 degrees bodily tilt; with tilting beyond this amount counterrolling gradually decreased, and finally the eye returned to its original position in its socket. Now by rather arbitrarily choosing, as we did, a certain angle of tilt with respect to the resultant force vector, and keeping it constant, the effect upon otolith activity of magnitude of the force, per se, can be studied. For at least two angles of tilt, counterrolling was found to be a logarithmic function of magnitude of force between about 0.6 and 1.0 G and probably

beyond. If several angles were investigated, the entire counterrolling response curve could probably be seen to rise and fall with respective increase or decrease in the magnitude of the acting force.

MAYNE: If I recall properly the experimental results given in one of your papers, the relation between the component of gravity normal to the longitudinal axis of the body is linearly related to counterrolling in that counterrolling was a sine function of the angle of rotation of the body.

MILLER: This wasn't our approach in this investigation or in my original paper. I believe Woellner and Graybiel in one of their counterrolling studies considered the counterrolling response as a function of the lateral component of force which varied as the sine of the angle of tilt. Thus, in the presentation of their data they indicated in effect the relationship between counterrolling and magnitude of force from zero to 2.2 G. However, these results obtained by changing simultaneously the magnitude and direction of force were quite different from those of the present study in which magnitude was varied independently of direction and therefore orientation of the otolith receptors. The typical sinusoidal type curve that you refer to is revealed when counterrolling is related to the angle of tilt with respect to gravitational vertical.

QUALTIEROTTI: I have results that can compare very directly with what Dr. Miller says; namely, I have been recording the activity of a single otolith unit during the same kind of flight. I would like to discuss what actually happens to the single otolith during this plane flight. A record was made of the discharge of the single otolith unit on the tape record, and accelerations were recorded. Before the period of zero G and during this period the excitation of this particular otolith unit is achieved by increasing the positive acceleration in the A-X axis only. We had a pilot who could do that and very well. We can directly compare the effect of the responsive single otolith unit at roughly 1 G in level flight with the one in zero G by accelerating by the same amount in the two conditions, that is, immediately after going into the parabolic flight and in level flight. The response is nearly normal; namely, the frequency of discharge increases according to the increase of the acceleration in the A-X direction only. There is no effect on this unit during changes of the vertical acceleration. When we are in the zero G state, at least three important phenomena take place: First, the spontaneous rate of firing increases before any acceleration is applied; second, there is an initial response which is much larger than the one that should be expected at the 1 G gravitational level;

then a third unexpected phenomenon takes place, a suppressory effect, during which there is no more response even if you increase the stimulus remarkably. This suppressory phenomenon blocks the firing rate at a constant low value, independently from excitation. It disappears when the 1-G condition is restored. On profiles in which the longitudinal acceleration and the response of the unit in rate of firing are plotted, there is a close following up of the rate of firing according to the change in A-X acceleration. At the beginning of the zero G condition, there is an increase of the rate of spontaneous firing and a much higher response for the same amount of linear acceleration along the A-X axis. After this the suppressory phenomenon is observed and this disappears when zero G condition is discontinued. In a direct comparison between the response of the otolith unit before the suppressory effect takes place in the 1-G condition and in the zero G condition, the response of the unit under normal conditions is a logarithmic curve. During zero G the initial response is much higher for the same amount of acceleration. So, we have to take into account the fact that we have a number of very complex phenomena when we go from 1 G or passing through 2.5 G to zero G. Now, how this series of phenomena are directly concerned with zero G and which are concerned with the transit from high acceleration to zero acceleration, we cannot tell. But probably the fact that the spontaneous firing increases, not immediately during the transient or immediately after, but some seconds, 1 or 2 seconds, after the zero-G state is achieved, seems to indicate that the increase of spontaneous firing is a phenomenon directly connected with weightlessness.

VON GIERKE: Has there been any attempt made to simulate on the centrifuge, under hypergravic conditions the same G-time transition pattern which you had in the hypogravic experiments of the zero G flights? For example, did you have subjects at 3 G's for the same time period you were at 2 G during the zero G flight and then bring them down to 1 G for a short period equivalent to the zero G period to measure counterrolling and to see if there were any consistent influences resulting from the transition period of the G history?

MILLER: We have not done this, but we have made some follow-up studies using the C-135 aircraft with less G force preceding the subgravity exposure and with much longer periods than in the studies reported. These data have not been analyzed so I cannot comment about them. However, further investigation of the effect upon counterrolling of pre-exposure to higher G should be done, and your suggestion of using a centrifuge seems excellent.

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# The Nature of Adaptation to Oscillatory Rotation

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## INTRODUCTION

More than 30 years ago I proposed an explanation for habituation to rotation that was part of a larger behavior theory (refs. 1 and 2). The proposal was that nystagmus was lost when it was replaced by a competing eye movement system, a wandering type of movement. This is often nonconjugate, similar to that found in dreamless sleep. Its appearance is facilitated by relaxation and reverie, and prevented by alertness and arousal.

The present paper is a progress report on a further experimental examination of this theory begun last year. I had not been completely happy with the very general terms in which I couched this application of a theory of competition between behavior systems to the specific problem of loss of vestibular nystagmus during habituation. In 1931, 1950, 1951, and 1964 I suggested, briefly and, rather enigmatically, that the competing system of wandering eye movements made use of the neural mechanisms which control the fast phase of nystagmus (refs. 1, 4-6). I did this hoping that others would pick up the suggestion, but no one has. It would be an interesting lead to follow. The neural mechanisms of the fast phase are to a strong degree under cerebral cortical control, and modifiable by learning. I see the attractive possibility of a theory which might encompass both habituation to rotation and such phenomena as adaptation to the rotating room. It may be that both make use of the neural mechanisms of the fast

phase in order to control the slow phase of nystagmus and the ocular fixation mechanism. So, for this Symposium, I decided to lay aside my inhibitions and speak out with some speculations.

## METHODS

I need to say a few brief words about methods and the relative advantages and disadvantages of those we are using. We use either electrical or photographic recording of eye movements. For photography we use the Dodge mirror recorder method. A small wooden cube, pressed lightly against the closed lid, is swiveled so as to remain tangential to the cornea as the eye moves from side to side. A mirror on the cube focuses a line of light on a photokymograph. The method yields accurate, highly magnified, independent records of the movements of each eye, largely free of artifacts. Its greatest advantage over the electrical method is its simultaneous and independent recording from each of the two eyes. This seems essential to show some of the features of the habituated response I will show you. The defect of the method is that it is beyond the mechanical aptitude of many.

The electrical record, which makes use of the electrical polarity of the eye, has the advantage of ease of application and economy of time. Its disadvantages are that separate and simultaneous records of each eye are obtainable only from some subjects and various extraneous potentials limit the identification of rapidly alternating eye movements.

Further, to the extent that the eyes move in opposite directions, the movements cancel one another on the record.

Both methods are applicable to monkeys or humans; the photographic method is not applicable to cats.

Our stimulation device is a horizontally rotating platform with the subject seated at the center of rotation. Horizontal oscillation of the platform is the stimulus which is the subject of the title of this paper. Recently we have been using sinusoidal oscillation through  $20^\circ$  of arc at a rate of 18 cycles/min. Such a stimulus has two advantages. The first is that it keeps the labyrinths under continuous stimulation for any desired period of time. Thereby, one can get a continuous record of adaptation. The second is that the alternation of direction of rotation makes it possible to see whether the habituation process results in a compensatory opposite reaction. As you will see later, it does.

#### RESULTS

I said in introduction that this is a progress report because we are continuing to gather data. I should also like to say that my detailed interpretations of what is happening during habituation are tentative. These interpretations are derived from qualitative aspects of the eye movement records. My confidence in my interpretations will presumably rise or fall as I see more and more records which do or do not fit what I now suspect to be the case. If the records continue to confirm my suspicions, final proof would need to wait on neurophysiological approaches to the centers involved.

Let us first look at some normal, unhabituated records of ocular response to  $20^\circ$  of

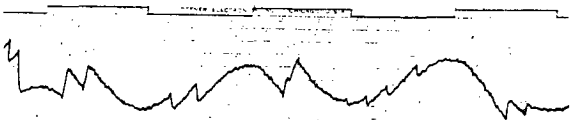


Figure 1.—Human nystagmus during  $20^\circ$  horizontal oscillation at 18 cycles/min. Ocular potential recording; both eyes in circuit.

horizontal oscillation at a rate of 18 cycles/min.

Figure 1 is an electrical record where both eyes are in the circuit. The eyes are open; the subject is in total darkness. The turning points of the oscillation are indicated by the changes in position of the event marker. Note that the slow phase of the ocular response mirrors the sinusoidal nature of the oscillation. Note also that there are relatively few fast phases, about two per second.

Figure 2 is an electrical record taken separately for each eye. Again, eyes open; total darkness. This subject's fast phases are more frequent. Note also that at the right side of figure 2 the compensatory eye movement is lost. This is possibly the brief entrance of a period of the habituation response.

Figure 3 shows old photographic records, made many years ago but never published, which shows both unhabituated and habituated segments of records. All are made with closed eyes. The top example is from a habituated record. At no point in this record is there any reflex compensatory eye movement. Instead, the eyes move mostly toward the rotation. Note that the movements may be different for the two eyes. It seems that the competing process is over-compensating so that the eyes mostly move ahead of the rotation. Note also that for the first four turns the eyes turn roughly synchronously with the rotation. Thereafter, in turns 5 to 9, they are more or less out of phase with the oscillation of the platform.

The third record in figure 3 is on a slower time scale but the oscillation is nevertheless

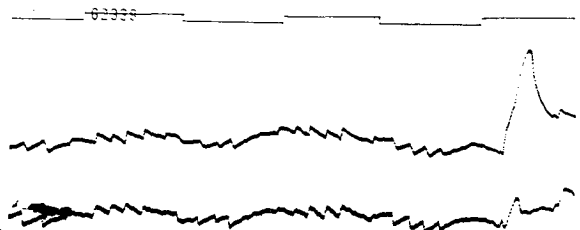


Figure 2.—Electrical record of human response to oscillation. Right and left eye recorded separately.

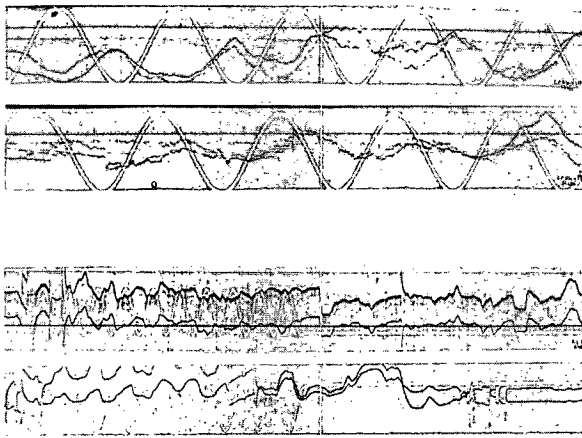


Figure 3.—Human and monkey nystagmus during  $15^\circ$  horizontal oscillation at 20 cycles/min. Photographic records made through closed lids; each eye recorded. Movements up on record are leftward movements of eyes, rightward movements of the platform; down on record is converse. Top record: Human; habituated; second record: Human; nystagmus for first half, then habituated; third record: Monkey; nystagmus for two cycles, then habituated; bottom record: Monkey; nystagmus for one cycle, then habituated.

at the same rate, three seconds per oscillation. This differs in that the subject was a monkey. If you look closely you will see that from the onset of oscillation until after the fourth turn he had normal reflex compensatory eye movements. Thereafter, the competing tendency oscillated the eyes in movements which mostly went *toward* the rotation. The last record is also from a monkey; it is an habituated record. It again shows the eyes moving in the same direction as the platform. This figure additionally illustrates something fairly commonly encountered; the eyes continue a horizontal oscillation after the oscillation of the platform stops.

At a later point I will call attention to the possible theoretical significance of the features shown in these records.

Most of the figures still to be shown were selected to repetitiously demonstrate the qualitative features which led me to the suspicion that it is the fast phase mechanism which is being subverted to the production of a competing behavior system, and thus to the production of habituation.

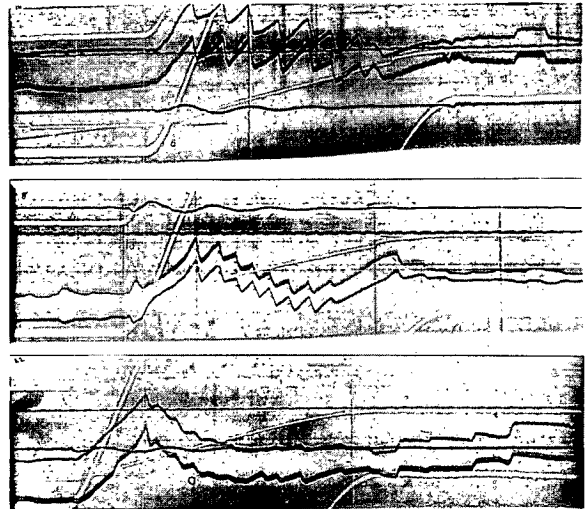


Figure 4.—Human nystagmus during  $65^\circ$  turn in two seconds. Photokymographic recording through closed lids. Records show three early stages in habituation process.

Figure 4 shows three stages of habituation to a single turn of  $65^\circ$  (this record was made in 1931). The  $65^\circ$  turn took two seconds. Human subject; eyes closed. Note, in the top record, made at an intermediate stage of habituation, that while nystagmus is still intact, the fast phases are frequent, about 7 per second, and regular in spacing, instead of the unhabituated rate of 2 to 4 per second, irregularly spaced. In the middle record, at a more advanced stage, the fast phases have increased to perhaps 10 per second, with nystagmus still intact. In the bottom record, nystagmus is no longer intact, but a degenerate fast phase is easily visible in the "habituated" portion of the record. Thus, the records show increasingly more numerous fast phases until the original form of nystagmus is lost.

Figure 5 is a highly magnified portion of a further stage of the habituation process, also during a  $65^\circ$  turn. This portion of the record covers a period of one second. Note the small tremulous movements. The amplitude of these eye movements goes down to perhaps 10 minutes of arc. They clearly look like a very rapid alternation of slow phase (in the direction required by the



Figure 5.—*Transitional stage of habituation. Records of each eye through closed lids during 65° turn. Time marker shows 1/10 second. Vertical lines are separated by 1/120 second.*

stimulation) and fast phases, with the maximum alternation rate being about 60 per second. These movements are exactly synchronous in the two eyes, but their amplitude may differ. The overall result is a somewhat irregular wandering movement of the eyes.

For some subjects the final stage of habituation may be a nontremulous wandering improvement such as that seen in figure 3 which showed habituated response to oscillation in human and monkey subjects. There remained no trace of an alternation of slow and fast phases. Figure 6 shows a very slightly tremulous record and a nontremulous record for the 65° arc of rotation.

One aspect of figure 6 should be remarked

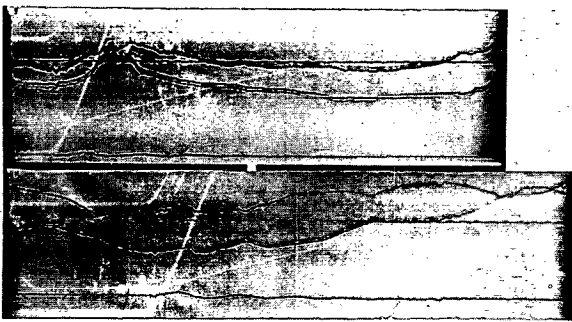


Figure 6.—*Human eye movement records during 65° turn. Top record is transitional stage of habituation. Bottom record is late stage of habituation. Apparent convergence and divergence of eyes is cardiac pulse artifact.*

on, since it is pertinent to theorizing. This is that the wandering character of the eye movement is present both before the onset of oscillation or rotation and after its cessation, that is, it is present in the resting, non-stimulated state. In other words, the "alert" state of the eyes is replaced by wandering at times other than rotation. Whatever the competing process is, it also competes with the alert state fixation mechanism, not alone with vestibular nystagmus.

The next three figures of response to oscillation, 7, 8, and 9, all show the same phenomenon: transition from nystagmus, through increasingly frequent fast phases, to a relatively smooth drift of the record in the "wrong" direction, that is, toward the direction of rotation. They are recent records from human subjects. The eyes are closed; recording is photographic.

Figure 7 shows, at the left, nystagmus with increasingly greater rates of fast phase. At the certain points in the tracing it is clear that a rapid alternation of fast and slow phase, with the fast phases bigger than the slow phases, causes the eye to move toward the rotation. In a less magnified record this might look like a smooth drift of the eyes.

Figure 8 begins with a rapid rate of fast

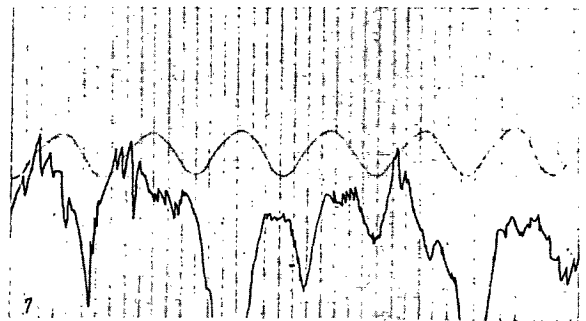


Figure 7.—*Tracing of photographic record of human nystagmus through closed lids during 20° horizontal oscillation at rate 18 cycles/min. Records made in 1964. The tracings (which are never exactly correct because of hand tremor and other distortions) were necessary because of unevenness of exposures. This is a transition stage between very rapid nystagmus and a habituated (non-nystagmoid) section. Because of lack of temporal resolution record cannot show the detail of figures 4, 5, and 6.*



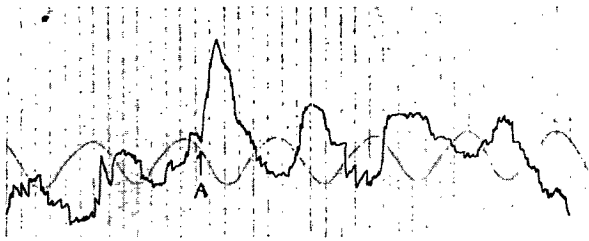


Figure 8.—Tracing of photographic record of human nystagmus through closed lids during 20° horizontal oscillation at rate of 18 cycles/min. Shifts between nystagmus and tremulous drifting movements.

phases and, at "A" and thereafter, shows either an apparently smooth drift toward the rotation or a very rapid alternation of slow and fast phases. Figure 9 shows more of the same, with some back and forth shifts between nystagmus and habituated eye movement tracings.

The final record, in figure 10, is included to show how large the "wrong way" response can be. This is considerably larger in the wrong direction than would be a reflex nystagmus in the right direction. By exercise of only a little imagination, one can convince oneself that here, too, fast and slow phases alternate. The frequent darkening and broadening of the line of the record may well be due to a momentary intrusion of a bit of slow phase. A very fast record with a high degree of photographic resolution would be required to decide this. We plan to take a few such records.

As a final comment about the records shown to accompany this paper, I would like to remark that these were not carefully

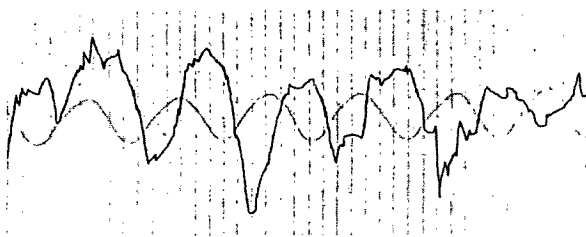


Figure 9.—Tracing of photographic record of human nystagmus through closed lids during 20° horizontal oscillation at rate of 18 cycles/min. Some nystagmus for short periods in cycles 1, 4, and 5, otherwise tremulous drifting movements.

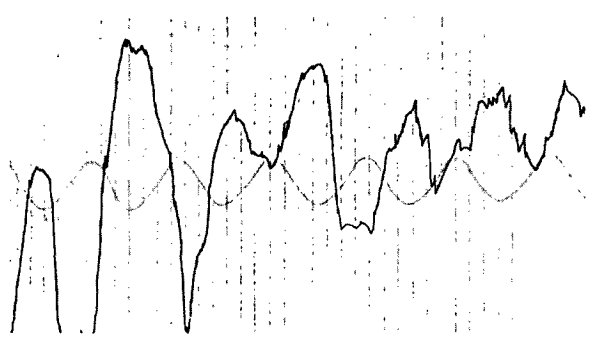


Figure 10.—Tracing of photographic record of human nystagmus through closed lids during 20° horizontal oscillation at rate of 18 cycles/min. Extraordinarily large drifts of eyes toward rotation.

selected records or hard to find. Innumerable instances could easily have been used.

#### DISCUSSION

I now wish to move on to a consideration of what these data appear to mean and of what needs to be done to further clarify interpretations. I am aware that my scientific colleagues do not look with great favor on making inferences about neurology from behavior data (although the converse, inferences about behavior from neurological data, seems quite respectable). Nevertheless, I am going to do so, because it has worked out fairly well for me in the past. I am deliberately refraining from use of any neurological literature because it seems to me that such use should be the next step in the thinking process. This is a case where too little sure knowledge of neurology can be worse than doing without any knowledge.

Initially, I will make some neurological assumptions. First, is that the control centers for coordinated fixation of the two eyes and for coordinated slow phase movements of the two eyes are the same centers. Next, I will assume that the center producing the fast phase controls the output of the slow phase centers and is connected with them by reciprocal inhibitory feedback circuits controlling outputs of each. Third, I will assume that both the fixation—slow phase centers and the fast phase centers are under the control of other outside centers. For

example, the fixation—slow phase centers are under the control both of centers receiving vestibular input (for the slow phase of vestibular nystagmus) and of centers receiving visual input (for the slow phase of optokinetic nystagmus). The fast phase centers are obviously under control of other centers, probably both cortical and non-cortical, receiving visual and auditory input, in addition to numerous other inputs.

After these assumptions, let us go back to my data. I will number the facts to be explained and suggest a speculative neural mechanism.

*Fact 1.*—During habituation the fast phases increase in frequency, reaching rates of alternation with the slow phase as high as 60 per second. The onset and cessation of each fast phase is exactly synchronous in the two eyes, even though amplitude may differ somewhat. Eventually this becomes a smooth wandering movement.

From these facts I speculate that some center (or centers) outside the fast phase centers is triggering the onset of the fast phases. The rate of the triggering impulses increases until at some point they become essentially asynchronous so that fast phases are no longer detectable, only an apparently smooth wandering movement.

*Fact 2.*—As a subject is repeatedly exposed to the same pattern of oscillation or turning, and as the reflex nystagmoid response is lost, a frequent response is an ocular oscillation or deviation which is opposite to the reflex, that is, toward the direction of rotation.

From these facts of general synchrony of response and of over-compensation, I speculate that the process is one of learning. The learning may be either or both cortical and noncortical. (Another reason to speculate that it is a learned process will be mentioned in a moment.)

*Fact 3.*—The wandering eye movement, while normally in phase with the oscillation, may at other times be out of phase.

My crystal ball becomes slightly clouded for seeing the implications of this fact. I suggest, however, that an asynchronous in-

put to the fast phase centers, from the centers originating the compensating or competing system, is not strongly stimulus-bound and varies in amount.

*Fact 4.*—At the cessation of oscillation of the platform, oscillation of the eyes may continue for a few seconds.

This again causes me to speculate that a learned process, cortical or noncortical or both, is responsible for the competing process, and further, that it is not firmly stimulus-bound. It does not seem possible that the process is controlled strongly by the vestibular input.

*Fact 5.*—During periods when no vestibular or visual stimuli are present, in the non-alert, nonaroused, relaxed subject, the eyes no longer show a fixed position or conjugate fast phases, but wander irregularly.

From this fact I speculate that of, on the one hand, the reciprocal centers controlling vigilance and alertness, and, on the other hand, those controlling revery, relaxation and sleep, the latter become dominant. I also speculate that they act on the fast phase centers and that these in turn act on the fixation—slow phase centers.

This is the end of my speculations. Now I should like to list some things I would like to do (or see someone else do) to clarify the picture.

(a) Most of my experience (but not all) has been with rotation of subjects whose eyes were closed. Closed eyes and sleep are in association as a behavior system. Habituation with closed eyes may be an early transition phase into sleep. Habituation with open eyes is slower, and may be different. This could limit the generality of application of my speculation.

To investigate this I intend to take photographic records of one closed eye while the other is open. While I have some such, I want to see more to assure myself that the phenomena are the same as with closed eyes.

(b) Our photographic records do not have sufficient resolution. This can be obtained from a very sharp focus of the recording line,

a large magnification, and a very fast record. I hope that we can achieve these.

(c) I want to investigate the kind of compensation for rotation that takes place in an active person, such as the subjects in the Pensacola rotating room. I will use subjects already thoroughly accustomed to an active compensatory process. My approach will be to take records from highly experienced ballet dancers and figure skaters. Fortunately we have a good supply of each in Rochester.

(d) A thorough search of the neuroanatomical and neurophysiological literature for possible locales for my speculative "centers" must be made. This can guide experiments

with electrophysiological records from chronically implanted electrodes (using monkeys), and with stimulation after lesions. These must be correlated with simultaneous eye movement records.

(e) Finally, we intend, with human subjects, to make quantitative measures of the amount of habituation or, conversely, the ability to maintain alertness or vigilance during rotation, and search for personal attributes which correlate with susceptibility to habituation. We will have approximately 75 twenty-one year old college students on whom very extensive personal data will be available.

#### REFERENCES

- WENDT, G. R.: Negative Adaptation as an Active, Positive Antagonism. *Psychol. Bull.*, 28, 1931, pp. 681-682.
- WENDT, G. R.: An Interpretation of Inhibition of Conditioned Reflexes as Competition Between Reaction Systems. *Psychol. Rev.*, 43, 1936, pp. 258-281.
- WENDT, G. R.: The Form of the Vestibular Eye-Movement Response in Man. *Psychol. Monog.*, no. 212, 1936, pp. 311-328.
- WENDT, G. R.: Some Characteristics of Vestibular Eye Movements. In *ONR Symposium: Psychological Factors in Spatial Orientation* (Pensacola, Fla.), 1950, pp. 36-41.
- WENDT, G. R.: Vestibular Functions. In *Handbook of Experimental Psychology*. (S. S. Stevens, ed.), 1951, pp. 1191-1223.
- WENDT, G. R.: Man and Motion. In *Handbook of Physiology*. Sect. 4. *Adaptation to the Environment*. (D. B. Dill, ed.), 1964, pp. 999-1005.

#### DISCUSSION

CRAMPTON: Your records were taken during continuous oscillation, and I am interested if some kind of anticipatory if not voluntary activity by the subject is involved. What would happen if he were in darkness, unaware of the direction and time of onset of the displacement, and he receives only one-half of a cycle?

WENDT: These early records were made in the days when I was interested in what the nature of the conditioned reflex was. There are no conditioned slow phase movements, but there are conditioned fast phases. The only anticipatory responses we got were fast phases, never a slow phase. I investigated this with a large number of different situations.

MAYNE: Dr. Wendt, it seems to me that there are two modes of response in the eye motion you recorded. The first seems to correspond to fixation. In this mode your record indicates that the displacement of the eyes is exactly 180° out of phase with head motion corresponding to a compensatory motion; this would allow a person to fixate a particular object while the head is moving. In the other mode the phase shifts to about 90°, so that the eye motion is roughly in phase with velocity and corresponds to modes such as recorded by Dr. Niven. At the same

time saccadic nystagmus appears. There seemed to be also an indecision as to which mode the organism must choose to operate so that the response may be a mix of the two modes. Does that correspond to your thought?

WENDT: No, that isn't the way the records actually look. While they are usually roughly in phase with the oscillation, when they get out of phase, the out of phaseness wanders from cycle to cycle, going ahead and dropping behind. I think the out of phaseness has no significance except to demonstrate that the compensatory response is not strictly stimulus bound, but affected by some other factors from the central nervous system.

The chief weakness in my speculations is that my experience has been mostly with closed eyes. When the eyes are closed, you have a response which is a part of the behavior system of sleep and we may here be dealing with a situation in which alertness is competing with an early stage of sleep. Obviously, in order to prove this, I'll need to see more records with the eyes open. It can be done only with a photographic method; the electrical method isn't going to reveal these features. The electrical method would obscure the tiny alternations of the eye movements since muscle potentials and other potentials would be confused with the tiny fast phases.

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# Repeated Caloric Stimulation of the Human Labyrinth and the Question of Vestibular Habituation

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## SUMMARY

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Forty unilateral caloric irrigations were administered in a habituation series to each of two groups of subjects. One group was tested in total darkness. The second group was stimulated in illumination and actively attempted to control and suppress their eye movements by means of visual fixation. Pre- and post-tests were administered (always in total darkness) in which both directions of response were elicited. In all cases, tasks were assigned to subjects to maintain alertness. The nystagmic reaction was altered as a result of the habituation series, but the change was different for the two groups. After one month of rest, there was no apparent recovery of the response toward the pretest level for either group. Subjective reactions declined in intensity for the two groups, but showed recovery after a 1-month rest period.

Author

Recent studies have been concerned with the effects of repeated unilateral caloric irrigations on human nystagmus and vertigo. Lidvall (1961a,b; 1962) recorded nystagmus from behind closed eyes and reported habituation (a response decline) with as few as four repeated stimulations. Number of nystagmic beats and duration of the response both declined, while latency of response and degree of dysrhythmia increased. In addition, subjective responses (vertigo) declined markedly. Fluor and Mendel (1962a,b) used the duration of nystagmus recorded with the subjects' eyes closed as their primary measure and obtained a shortening of nystagmus time after 8 to 12 unilateral irrigations. The latter authors also examined before-and-after responses to stimulation of each ear with warm and with cool water and reported that subjects fell into two groups: (1) those who exhibited a directional preponderance in the unhabituated direction, and (2) those who showed reduced responses in both directions (1962a). A later study indicated that the directional

preponderance following a habituation series could be reversed by then habituating the opposite direction (1962b).

Forssman, Henriksson, and Dolowitz (1963) in darkness and Forssman (1964) in light investigated the effects of repeated unilateral calorizations on nystagmus, laterotorsion, and vertigo. Results for the two studies were similar. Twelve stimulations produced declines in vertigo in excess of 80%, laterotorsion declined about 40%, and the maximum eye-velocity and the duration of nystagmus were reduced by 55% and 25%, respectively.

The importance of alertness or arousal on the process of habituation was noted in several of the above studies (e.g., Lidval, 1962; Forssman, 1964) and has received extensive attention elsewhere (e.g., Collins, 1963; 1964). It has been demonstrated that nystagmic output (Collins, 1963) and evidence of dysrhythmia in records of nystagmus can be readily manipulated by instructions given to the subjects. Subjective states of arousal result in brisk nystagmic responses. Although

acknowledgment is made of the effect of mental states on production of nystagmus, few researchers seem to control for this factor.

The present study was designed to examine the effects on nystagmus and subjective experience of repeated unilateral caloric irrigations administered under conditions of total darkness and of visual fixation. Tasks were assigned to subjects in an attempt to control subjective states. Pre- and post-tests comprised stimuli which elicited unilateral and bilateral nystagmic responses in both directions.

#### METHOD

##### Subjects

Subjects were 20 males between the ages of 21-29. All were experimentally naive and were free of any history of ear difficulties, dizziness, loss of consciousness, or unusual reactions to linear and angular accelerations. On each day, prior to testing, subjects' ears were examined and cleaned.

##### Apparatus

Two water baths were equipped with Bronwill constant temperature circulators. Tubing from each bath terminated in its own plastic nozzle which was to be inserted in the external auditory meatus. Water temperature was maintained such that it was 30° C from one bath, and 44° C from the other, upon introduction to the ear.

Horizontal components of eye-movements were obtained by means of an Offner Type TC recorder. A 3-second RC time constant was employed in amplification. Signals were picked up from electrodes taped near the outer canthi. An indifferent electrode was located on the forehead. All nystagmus recordings were obtained in total darkness with the subject's eye open. Subjective reactions were signalled by means of a buzzer manually controlled by the subject.

A special device (Caloric Irrigation Receptacle) was constructed to collect, in a convenient fashion, return flow of water from the ear and to provide, simultaneously, proper head elevation. The device, set on the

examining table, was a rectangular basin into which the subject's head was placed and anteverted approximately 30° by means of a head rest. A drain was located at the back of the basin. A tube extended from the drain into a large water receptacle placed underneath the examining table. The return flow of water thus was collected in the basin and was led to the receptacle beneath the examining table by means of the tubing. A large number of irrigations could be performed without disturbing the subjects and without concern for collecting the water.

##### Procedure

Subjects were divided into two groups of 10 each. Both groups were tested on 7 occasions. Six of these occasions were on consecutive days; the seventh was one month later. The first, sixth, and seventh days were termed pre-test, post-1, and post-2 tests respectively, and each comprised 6 trials, the order of which was varied as indicated in table I. Two of the 6 trials were bilateral irrigations (warm and cool water were delivered simultaneously to the two ears) testing both directions of response. The remaining 4 trials consisted of unilateral irrigations (a warm and a cool stimulus applied to each ear separately). Days 2 through 4 consisted of 10 daily habituation trials. In all 40 of the latter, a unilateral stimulus of 30° C was applied to the right ear. Twenty to thirty minutes of rest occurred between successive stimulations.

The two groups of subjects were differentiated on the basis of the habituation trials. The Dark Group received all trials in total darkness. The Visual Fixation Group was given all pre- and post-tests in total darkness, but in the habituation series only trials 1 and 10 on each day were in darkness. The remaining daily trials for the Visual Fixation Group were in illumination with the subjects actively fixating on the center marker of the calibration device on the ceiling and consciously trying thereby to reduce and prevent the nystagmic movement. Thus, the Dark Group received a total of 40 habituation trials in total darkness while the Visual

Table I.—*The Order of Stimulus Presentation*

[R and L refer to the right and the left ear; c and w indicate cool (30° C) and warm (44° C) stimuli. Bilateral stimuli were administered for 15 sec, unilateral stimuli for 30 sec. All pre- and post-tests were conducted in total darkness.]

Groups	Subjects									
Dark	BY	PH	RA	BE	GR	JB	HL	JL	BM	JG
Fixation	TV	FN	GP	FS	CW	DM	FG	FK	RV	VV
Pre-tests	R <sub>c</sub> L <sub>w</sub> R <sub>w</sub> L <sub>c</sub> L <sub>c</sub> R <sub>w</sub> L <sub>w</sub> R <sub>c</sub>	R <sub>w</sub> L <sub>c</sub> R <sub>c</sub> L <sub>w</sub> L <sub>c</sub> R <sub>c</sub> L <sub>w</sub> R <sub>w</sub>	R <sub>c</sub> L <sub>w</sub> R <sub>w</sub> L <sub>c</sub> R <sub>c</sub> L <sub>c</sub> R <sub>w</sub> L <sub>w</sub>	R <sub>w</sub> L <sub>c</sub> R <sub>c</sub> L <sub>w</sub> L <sub>c</sub> L <sub>w</sub> R <sub>w</sub> R <sub>w</sub>	R <sub>c</sub> L <sub>w</sub> R <sub>w</sub> L <sub>c</sub> R <sub>c</sub> R <sub>w</sub> L <sub>c</sub> L <sub>w</sub>	R <sub>w</sub> L <sub>c</sub> R <sub>c</sub> L <sub>w</sub> L <sub>w</sub> R <sub>w</sub> L <sub>c</sub> R <sub>c</sub>	R <sub>c</sub> L <sub>w</sub> R <sub>w</sub> L <sub>c</sub> L <sub>w</sub> R <sub>w</sub> L <sub>c</sub> L <sub>c</sub>	R <sub>w</sub> L <sub>c</sub> R <sub>c</sub> L <sub>w</sub> L <sub>w</sub> R <sub>c</sub> L <sub>c</sub> R <sub>w</sub>	R <sub>c</sub> L <sub>w</sub> R <sub>w</sub> L <sub>c</sub> R <sub>w</sub> R <sub>c</sub> L <sub>c</sub> L <sub>w</sub>	R <sub>w</sub> L <sub>c</sub> R <sub>c</sub> L <sub>w</sub> L <sub>w</sub> L <sub>c</sub> R <sub>w</sub> R <sub>c</sub>
Habituation tests	Dark group: 10 unilateral trials (all R <sub>c</sub> ) per day for 4 days. All trials in total darkness. Fixation groups: 10 unilateral trials (all R <sub>c</sub> ) per day for 4 days. Trials 1 and 10 on each day in total darkness. All other trials in illumination with visual fixation.									
Post-1 and post-2 tests	L <sub>c</sub> R <sub>w</sub> L <sub>w</sub> R <sub>c</sub> R <sub>c</sub> L <sub>w</sub> R <sub>w</sub> L <sub>c</sub>	L <sub>c</sub> R <sub>c</sub> L <sub>w</sub> R <sub>w</sub> R <sub>w</sub> L <sub>c</sub> R <sub>c</sub> L <sub>w</sub>	R <sub>c</sub> L <sub>c</sub> R <sub>w</sub> L <sub>w</sub> R <sub>c</sub> L <sub>w</sub> R <sub>w</sub> L <sub>c</sub>	L <sub>c</sub> L <sub>w</sub> R <sub>c</sub> R <sub>w</sub> R <sub>w</sub> L <sub>c</sub> R <sub>c</sub> L <sub>w</sub>	R <sub>c</sub> R <sub>w</sub> L <sub>c</sub> L <sub>w</sub> R <sub>c</sub> L <sub>w</sub> R <sub>w</sub> L <sub>c</sub>	L <sub>w</sub> R <sub>w</sub> L <sub>c</sub> R <sub>c</sub> R <sub>c</sub> L <sub>w</sub> R <sub>w</sub> L <sub>c</sub>	R <sub>w</sub> L <sub>w</sub> R <sub>c</sub> L <sub>c</sub> R <sub>c</sub> L <sub>w</sub> R <sub>w</sub> L <sub>c</sub>	L <sub>w</sub> R <sub>c</sub> L <sub>c</sub> R <sub>w</sub> R <sub>w</sub> L <sub>c</sub> R <sub>c</sub> L <sub>w</sub>	R <sub>w</sub> R <sub>c</sub> L <sub>c</sub> L <sub>w</sub> R <sub>c</sub> L <sub>w</sub> R <sub>w</sub> L <sub>c</sub>	L <sub>w</sub> L <sub>c</sub> R <sub>w</sub> R <sub>c</sub> L <sub>w</sub> R <sub>w</sub> L <sub>c</sub> R <sub>c</sub> L <sub>w</sub>

Fixation Group received 8 habituation trials in total darkness and 32 under conditions of visual fixation.

During all "total darkness" trials, the irrigation period was also in the dark. The rate of flow of the water was relatively high, approximately 15cc/sec, to permit ready monitoring of the irrigation by touch alone. Irrigation time was 15 sec for bilateral and 30 sec for unilateral stimuli.

Tests were conducted in the following manner: Subjects assumed a supine position on the examining table with their heads elevated and anteverted approximately 30°. Prior to each trial, eye-movement calibrations were obtained by means of a ceiling panel with a center and two lateral markers. For all pre- and post-tests, subjects signalled the onset, point of maximum intensity, and

cessation of their subjective vestibular experiences by means of a manually operated microswitch. Several seconds after their "cessation of sensation" signal, subjects were given mental arithmetic (MA) problems (continuous division) to perform throughout the remainder of the trial. The MA task was used purely as an alerting technique (Collins, 1963; 1964). At the conclusion of each pre- and post-trial, subjects described their sensations and rated them.

Subjective ratings were made in the following fashion. During the pretest, the first two trials for all subjects were bilateral irrigations. Each subject was asked which of the two bilateral stimulations produced the most intense subjective experience. His selection was then given a rating of 100 and all other subjective ratings were made with

the "100 intensity" experience as a reference point.

During the habituation trials in total darkness, several tasks were presented to the subjects in accordance with the schedule in table II. All used the microswitch as a signaling device. The tasks included:

- (1) Reaction Time (RT)—Subjects responded to a series of 500 cps tones of  $\frac{1}{10}$  sec duration by depressing the microswitch as quickly as possible at the onset of each tone.
- (2) Temporal Estimation of Sound-Filled Intervals (FI)—The 500 cps tone was sounded for periods ranging between 0.5 and 6.5 sec. At the conclusion of a tone, subjects attempted to reproduce its duration by depressing the microswitch for the same length of time as that occupied by the tone.
- (3) Temporal Estimation of Sound-Bounded Intervals (BI)—The 500 cps tone was sounded for  $\frac{1}{10}$  sec to indicate the start and end of an intervening period of silence. Silent periods

between the two brief tones ranged from 0.5–6.5 sec. After each pair of tones, subjects depressed the microswitch for a length of time which they considered to be equal to that of the period of silence between tones.

- (4) Temporal Comparison of Sound-Filled Intervals (FIC)—Pairs of 500 cps tones were successively presented for comparison. A given tone would be of a duration ranging from 0.5–6.5 sec. Its companion tone would differ by no more than 2 sec. Subjects indicated which tone of the pair was longer in duration by signaling with either one or two depressions of the microswitch.
- (5) Temporal Comparison of Sound-Bounded Intervals (BIC)—A period of silence was delimited by two  $\frac{1}{10}$  sec, 500 cps tones and was followed by another pair of tones with an intervening period of silence. Silent periods ranged from 0.5–6.5 sec, but in each pair of silent periods the maximum temporal difference did not ex-

Table II.—*The Order of Task Presentation for the Dark Group and for the Visual Fixation Group*

[RT = reaction time; FI and BI = temporal estimation of sound-filled and of sound-bounded intervals, respectively; FIC and BIC = temporal comparison of sound-filled intervals and of sound-bounded intervals, respectively.]

Trials	Habituation sessions			
	I	II	III	IV
	Dark group			
1	FI	FIC	BI	BIC
2	FI	FIC	BI	BIC
3	FI	FIC	BIC	BI
4	RT	RT	BIC	BI
5	BI	BIC	RT	RT
6	BI	BIC	FIC	FI
7	BI	BIC	FIC	FI
8	RT	RT	RT	RT
9	FI	FIC	FI	FIC
10	BI	BIC	FI	FIC
	Visual fixation group			
1	FI	FIC	BI	BIC
2–9		Illumination with visual fixation		
10	BI	BIC	FI	FIC

ceed 2 sec. Subjects depressed the microswitch once or twice to indicate whether the first or second silent period was longer in duration.

For each of the above tasks, a whole series of stimuli was presented throughout a trial. The tasks were used purely to maintain a state of alertness in the subjects. However, subjects were encouraged to perform as accurately and as attentively as possible and were impressed with the fact that their performance was of significance. Records of their signals were obtained.

RESULTS AND DISCUSSION

Nystagmus

Eye-movement recordings (see figs. 1 and 2) were scored and analyzed for duration of nystagmus, slow-phase displacement, and number of nystagmic beats as indicated else-

where (Collins, 1964). Mean data for both the Dark Group and the Visual Fixation Group appear in table III. Percentage data relating the post-test scores to those of the pretest appear in table IV. In both tables, scores have been rounded off to the nearest whole number.

The data were treated statistically by analysis of variance and yielded the following results. For the Dark Group, the habituation series produced no significant change in duration or slow-phase activity. The number of beats of nystagmus, however, rose significantly from pre- to post-1 and showed no evidence of normalizing after one month with no stimulation (post-2).

The Visual Fixation Group was affected differently. Whereas there was no change in the number of beats of nystagmus from the

Table III.—Mean Duration, Number of Nystagmic Eye Movements, and Slow-Phase Displacement of the Eyes to Preliminary, Post-1, and Post-2 Tests for the Two Groups

[Each value is a mean for 10 subjects.]

Stimuli	Dark Group			Fixation Group		
	Pre	Post-1	Post-2	Pre	Post-1	Post-2
Duration (sec)						
R <sub>c</sub> L <sub>w</sub>	172	182	173	191	191	172
R <sub>w</sub> L <sub>c</sub>	168	153	163	193	186	170
R <sub>c</sub>	154	152	158	174	169	155
L <sub>w</sub>	136	156	147	174	180	163
R <sub>w</sub>	123	130	124	172	156	155
L <sub>c</sub>	135	139	136	180	179	154
Number of beats						
R <sub>c</sub> L <sub>w</sub>	250	326	285	287	316	291
R <sub>w</sub> L <sub>c</sub>	215	248	255	272	318	294
R <sub>c</sub>	193	231	242	233	242	215
L <sub>w</sub>	174	242	223	227	245	218
R <sub>w</sub>	136	200	192	223	234	220
L <sub>c</sub>	158	177	176	246	249	228
Slow-phase displacement (deg.)						
R <sub>c</sub> L <sub>w</sub>	1991	2124	1926	2997	2403	2557
R <sub>w</sub> L <sub>c</sub>	2016	1644	1847	3000	2629	2374
R <sub>c</sub>	1389	1271	1433	2166	1537	1430
L <sub>w</sub>	1331	1403	1236	1907	1512	1488
R <sub>w</sub>	961	1203	1128	1976	1793	1521
L <sub>c</sub>	1196	1130	1071	2174	1920	1525



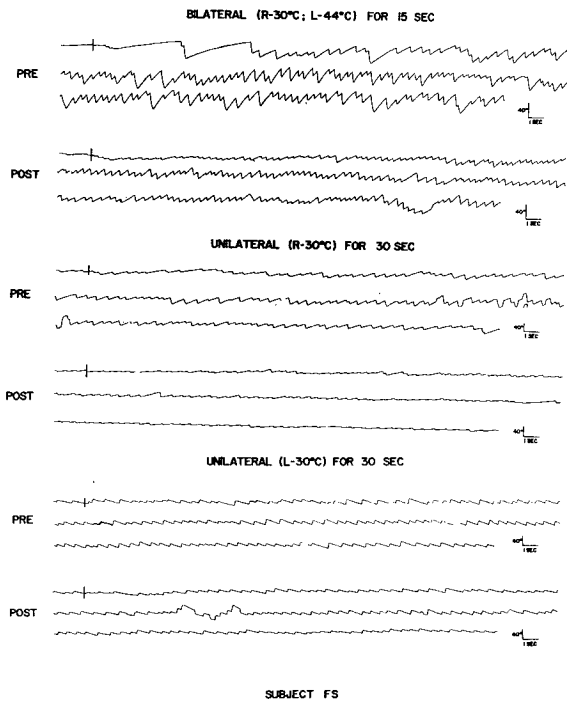


Figure 1.—Examples of nystagmus recorded from a subject in the Visual Fixation Group. Vertical bars through records indicate point at which irrigation was terminated. The post-test presented is post-1.

pre- to the post-tests, slow-phase displacement declined significantly from pre- to post-1 and, after one month of rest, the response showed some further decline. The duration of nystagmus for the fixation group showed no pre- to post-1 change, but a statistically significant decline occurred in post-2.

Dysrhythmia occurred on several occasions during the habituation trials. However, any alerting stimulus would restore a brisk nystagmus. Moreover, based upon results obtained prior to the experiment, it appeared that some of this dysrhythmia may have been related to the gaze-level of the subjects' eyes. That is, with repeated testing in the dark, there is a tendency for some subjects to lower their gaze (and probably half-close their eyelids). At least for some subjects, this appears to produce dysrhythmia. With eyes closed it is possible that subjects roll up their eyes and produce a like effect.

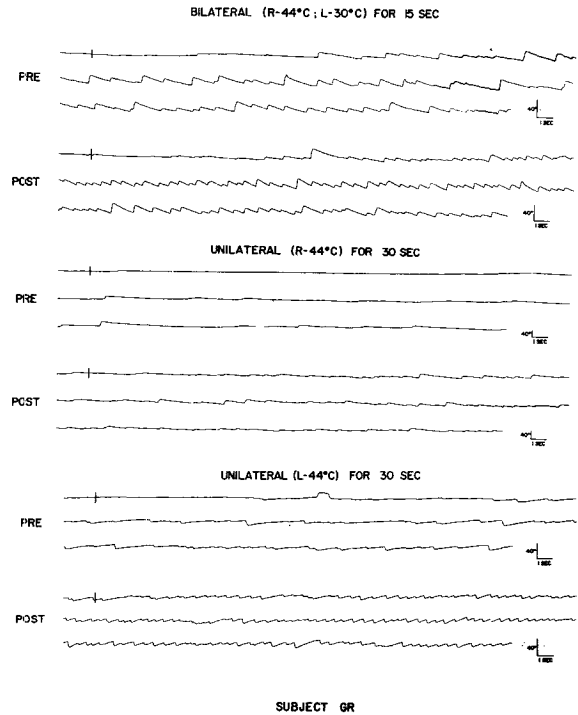


Figure 2.—Examples of nystagmus recorded from a subject in Total Darkness Group. Markings are same as in figure 1.

#### Subjective Responses

The mean intensity ratings reported by the subjects appear in table V. In addition, for both groups, the percentage the post-test ratings were of the pretest is included. For the Dark Group, a decline in the subjective vestibular experiences due to the irrigations was most marked for the  $R_c$  and  $R_cL_w$  stimuli. However, the  $R_w$  and  $L_c$  stimuli, eliciting unilateral responses in the direction opposite that of the habituating stimuli showed increases. The Visual Fixation Group reported subjective declines for all post-1 stimuli. Further, a directional consistency was evident. The greatest drop occurred to the habituation stimulus ( $R_c$ ), next to the  $R_cL_w$ , and next to the  $L_w$  stimuli. The forementioned all produced nystagmus with fast-phase to the left. The remaining three stimuli, all of which elicit a nystagmus to the right, declined much less. After one month with no intervening tests, recovery was evident for

Table IV.—*Relation of Data From Post-1 and Post-2 Tests to Preliminary Test Scores*  
 [All values are percentages calculated from data in Table III.]

Stimuli	Dark Group		Fixation Group	
	Post-1/Pre	Post-2/Pre	Post-1/Pre	Post-2/Pre
	Duration			
R <sub>c</sub> L <sub>w</sub>	106	101	100	90
R <sub>w</sub> L <sub>c</sub>	91	97	96	88
R <sub>c</sub>	99	103	97	89
L <sub>w</sub>	115	108	103	94
R <sub>w</sub>	106	101	91	90
L <sub>c</sub>	103	101	99	86
	Number of Beats			
R <sub>c</sub> L <sub>w</sub>	130	114	110	101
R <sub>w</sub> L <sub>c</sub>	115	119	117	108
R <sub>c</sub>	120	125	104	93
L <sub>w</sub>	139	128	108	96
R <sub>w</sub>	147	141	105	99
L <sub>c</sub>	112	111	101	93
	Slow-Phase Displacement			
R <sub>c</sub> L <sub>w</sub>	107	97	80	85
R <sub>w</sub> L <sub>c</sub>	82	92	88	79
R <sub>c</sub>	92	103	71	66
L <sub>w</sub>	105	93	79	78
R <sub>w</sub>	125	117	91	77
L <sub>c</sub>	96	90	88	70

both groups with some directional effects still in evidence however.

General Discussion

These findings indicate that, when mental states are controlled, remarkable declines in nystagmic output and duration do not necessarily occur. In partial agreement with a previous study using angular acceleration as the habituating stimulus (Collins, 1964), an increase in number of nystagmic beats may occur in total darkness. It is, then, not always appropriate to consider habituation (a response decline) as the only effect of repeated stimulation. The form of the response may change.

The studies cited earlier which used a relatively small number of irrigating stimuli and yet found high rates of nystagmus loss in total darkness (whereas here there were no losses and, in the case of number of beats,

a significant response increase) differed in two major respects from the present work: (1) state of arousal was not controlled; (2) data were obtained in darkness with the subject's eyes closed. These two conditions probably interact to lower nystagmic output.

Contrary to data reported by Forssman and his associates (1963; 1964), the effects of darkness and light on the nystagmic response appear different. Neither duration nor frequency showed any pre- to post-1 decline for the Visual Fixation Group, whereas the slow-phase output dropped significantly with fairly clear directional differences evident. The importance of the light condition is almost certainly not the mere presence of visual objects. It seems most likely that what is required is an active attempt on the part of the subject to control and suppress his eye movements.

In agreement with the previously cited

Table V.—*Mean Scores for the Intensity of the Subjective Experience Resulting From Caloric Irrigation*

[Whichever of the two bilateral irrigations in the preliminary test produced the strongest sensation was given a rating of 100. All other ratings were made by comparing a new sensation with the standard of 100.]

Stimuli	Dark Group			Fixation Group		
	Mean Subjective Rating					
	Pre	Post-1	Post-2	Pre	Post-1	Post-2
R <sub>c</sub> L <sub>w</sub>	87	41	50	95	47	54
R <sub>w</sub> L <sub>c</sub>	84	59	113	73	66	75
R <sub>c</sub>	36	19	27	31	8	19
L <sub>w</sub>	56	39	47	45	29	49
R <sub>w</sub>	39	66	53	44	37	46
L <sub>c</sub>	30	35	29	48	41	46
% Post-1 and Post-2 Are of Pre Rating						
	Post-1		Post-2		Post-2	
R <sub>c</sub> L <sub>w</sub>	47		57		57	
R <sub>w</sub> L <sub>c</sub>	70		135		103	
R <sub>c</sub>	53		75		61	
L <sub>w</sub>	70		84		109	
R <sub>w</sub>	169		136		105	
L <sub>c</sub>	117		97		96	

study of the effects on nystagmus of repeated angular accelerations (Collins, 1964), a one-month rest period did not restore the alterations in the response produced by the repeated stimulation. Thus, for the Dark Group, frequency remained at a higher level than in the preliminary tests and, for the Visual Fixation Group, a further decline in slow-phase output and in nystagmus duration was evident rather than a return toward the preliminary response level.

Although some directionally specific effects are suggested by the nystagmus data, they are neither consistent nor striking. This too agrees with the rotation findings noted above.

Subjective responses seem also to be differently affected by the total darkness and visual fixation conditions. The differences, however, are primarily directional and earlier studies examined only responses to the same stimulus as that employed in the habituation trials. Unlike the nystagmus response, recovery is clearly evident following the one-month rest period. These results also agree with our previous rotatory work.

Thus our data indicate that repeated, simple vestibular stimulation results in a modified nystagmic response (which may reflect increased activity rather than a reduction) and that the modification is not capricious; the response shows little or no recovery toward its original form after a month of rest. The changes are bidirectional (although the repeated stimulation was unidirectional) and the nature of the changes depends upon whether the response was repetitively elicited in darkness or with fixation. The subjective response declines for the practiced direction; subjective reactions to the unpracticed direction may decline or increase in intensity depending upon whether the total darkness or the visual fixation condition was present during stimulus repetition.

It is sometimes implied that the arousal factor represents a kind of artifact in habituation. Actually, the view propounded by Wendt (1951) some years ago seems most appropriate; namely, that there can be habituation to the test situation which produces a response decrement when alertness

is not controlled. But, by simple instructions designed to relax a subject and reduce his mental activity, the same response decrement can be produced on the very first trial. Hence, the decline is not necessarily related to repeated vestibular stimulation. The changes that do occur as a result of repeated stimulation must be evaluated in terms of arousal level.

A final point is worthy of note. Researchers are frequently interested in the question of whether changes in vestibular responses occasioned by repeated stimulation of one type (e.g., rotation) will carry over or transfer to another type of stimulation (e.g., caloric). Fluor and Mendel (1963) reported alterations (declines) in caloric nystagmus after a number of unidirectional rotation trials. McCabe (1960) noted no caloric responses (by direct observation) from professional figure skaters who, as part of their occupational routine, are daily exposed to a large number of high velocity angular accelerations. In figure 3 are presented records of caloric nystagmus recorded

from two professional figure skaters tested in total darkness with eyes open. Clear, brisk responses are evident. We are currently completing a study of the vestibular reactions of a number of figure skaters and plan to present a complete report in the near future.

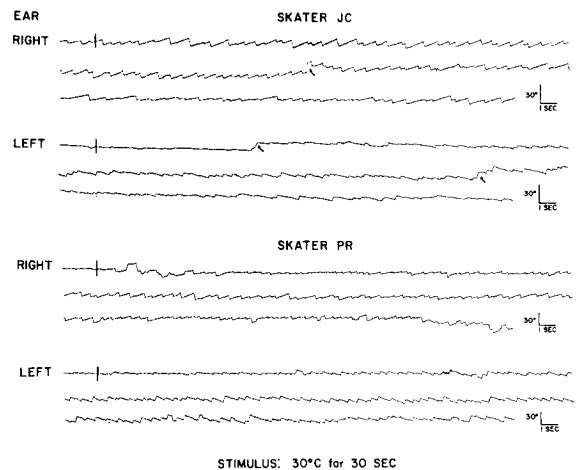


Figure 3.—Nystagmograms obtained from a female (JC) and a male (PR) figure skater. Recordings are DC. Arrows indicate points at which recording pen was manually repositioned.

#### REFERENCES

- COLLINS, W. E.: Manipulation of Arousal and its Effects Upon Human Vestibular Nystagmus Induced by Caloric Irrigation and Angular Accelerations. *Aerospace Med.*, 34, 1963, pp. 124-129.
- COLLINS, W. E.: Task-Control of Arousal and the Effects of Repeated Unidirectional Angular Acceleration on Human Vestibular Responses. *Acta Otolaryngol. Suppl.* 190, 1964.
- COLLINS, W. E.; CRAMPTON, G. H.; AND POSNER, J. B.: Effects of Mental Activity on Vestibular Nystagmus and the Electroencephalogram. *Nature*, 190, 1961, pp. 194-195.
- COLLINS, W. E.; AND POSNER, J. B.: Electroencephalogram Alpha Activity During Mild Vestibular Stimulation, *Nature*, 199, 1963, pp. 933-934.
- FLUOR, E.; AND MENDEL, L.: Habituation, Efference and Vestibular Interplay. I. Monaural Caloric Habituation. *Acta Otolaryngol.*, 55, 1962a, pp. 65-80.
- FLUOR, E.; AND MENDEL, L.: Habituation, Efference and Vestibular Interplay. II. Combined Caloric Habituation. *Acta Otolaryngol.*, 55, 1962b, pp. 136-144.
- FLUOR, E.; AND MENDEL, L.: Habituation Efference and Vestibular Interplay. III. Unidirectional Rotation Habituation. *Acta Otolaryngol.*, 57, 1964, pp. 81-88.
- FORSSMAN, B.: Studies on Habituation of Vestibular Reflexes. VII. Habituation in Light of Calorically Induced Nystagmus, Laterotorsion and Vertigo in Man. *Acta Otolaryngol.*, 57, 1964, pp. 163-176.
- FORSSMAN, B.; HENRIKSSON, N. G.; AND DOLOWITZ, D. A.: Studies on Habituation of Vestibular Reflexes. VI. Habituation in Darkness of Calorically Induced Nystagmus, Laterotorsion, and Vertigo in Man. *Acta Otolaryngol.*, 56, 1963, pp. 663-674.
- LIDVALL, H. F.: Vertigo and Nystagmus Responses to Caloric Stimuli Repeated at Short Intervals. *Acta Otolaryngol.*, 53, 1961a, pp. 33-44.
- LIDVALL, H.: Vertigo and Nystagmus Responses to Caloric Stimuli Repeated at Short and Long Intervals. *Acta Otolaryngol.*, 53, 1961b, pp. 507-518.
- LIDVALL, H.: Specific and Non-Specific Traits of Habituation in Nystagmus Responses to Caloric Stimuli. *Acta Otolaryngol.*, 55, 1962, pp. 315-325.
- MCCABE, B. F.: Vestibular Suppression in Figure Skaters. *Trans. Amer. Acad. Ophthalm. Otolaryngol.*, 64, 1960, pp. 264-268.
- WENDT, G. R.: Vestibular Functions. In *Handbook of Experimental Psychology* (S. S. Stevens, ed.), John Wiley & Sons, 1951, pp. 1191-1223.

## DISCUSSION

BECK: This is very interesting to us. We've been trying to make some cardiovascular measurements during stimulation of the labyrinth both on the Pensacola SRR and by caloric stimulation; this is being done on primates. We have been consistently thwarted by this habituation, blocking, or inhibition, whichever you like to call it, and to demonstrate this, we obtained some EEG's and nystagmograms on several monkeys. As the nystagmus disappears, slower waves appear on the EEG and vice versa. The animals seem to come in and out of this inhibiting state quite voluntarily.

C. CLARK: How did you record your EEG's?

BECK: From surface electrodes.

COLLINS: We have sought to relate the differences in nystagmic output occasioned by our instructions regarding mental activity to changes in EEG patterns (Collins, Crampton, and Posner, 1961; Collins and Posner, 1963). We have not been successful, however. It appears as though the eye-movement response is more sensitive to our instructions than brain wave activity. However, our EEG recordings were taken with only a single pair of electrodes and our analysis consisted only of a "percent time alpha" score.

TOROK: It is surprising that an increase of nystagmus frequency was found after repeated stimulation. It is difficult to evaluate nystagmus intensity when frequency is counted through the entire time of response. If, however, the peak of the frequency appears in a plotted form, more characteristic data of nystagmus intensity will be available and I would not expect an increase in this respect. To score nystagmus frequency in consecutive 5-sec time units is a simple procedure and obtainable from the conventional nystagmogram. In our experience, nystagmus frequency maximum, termed also as the "culmination phenomenon," is not only the most characteristic expression of intensity

of sensitivity, but the most stable feature of vestibular nystagmus. Because of this, an increase is unlikely.

COLLINS: Well, you noticed in some of Dr. Wendt's records a clear increase in frequency of nystagmus with repeated testing. In addition, we have obtained increases in number of nystagmic beats after 200 unidirectional accelerations (Collins, 1964). Moreover, those increases were limited to the stimulus period and 5-7 sec immediately thereafter. Although I have presented no curves here, the increased frequency in the caloric response appears to occur throughout the time-course of the reaction.

M. JONES: Dr. Collins, do you think that the changes associated with the increasing arousal could be reciprocally related to the changes associated with decrease of arousal. We recently undertook a study of spontaneous changes in the oculomotor response to oscillatory stimulation of the semicircular canals in high decerebrate cats. The oscillatory stimulus was established by means of a servo-controlled turntable which could be maintained in continuous oscillation for indefinitely long periods. Respiration was kept constant by overventilation and, as far as one knows, everything else in the cat's external environment remained constant also. Yet there were enormous variations in the oculomotor response; everything from complete ocular compensation to zero response. The variations took no particular pattern, but were usually slow, taking hours rather than minutes. Do you think that these very gross changes in the decerebrate cat could be in any way related to the changes you have described?

COLLINS: Well, I can't provide a direct answer. Changes in oculomotor patterns do occur over time in relation to activation—during sleep for example. I have no experience with decerebrate cats, but it seems possible that the changes you described may reflect some alteration in states of arousal.

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# Some Factors Contributing to the Delay in the Perception of the Oculogravic Illusion

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## SUMMARY

The purpose of the five experiments reported here was to study some variables which influence the lag in the perception of the oculogravic illusion following a change in the direction and magnitude of resultant force acting on the subject. All the observations were made in The Pensacola Slow Rotation Room with the subject facing the direction of rotation while he observed a collimated, luminous line. Five normal and nine labyrinthine defective men acted as subjects. The subjects' task was to set the luminous line to the apparent horizontal following different conditions of preexposure to a visual framework, of different exposure to the luminous line, and for different periods of constant rotation of the room. The inertial-lag effect was found to occur in both normals and labyrinthine defective subjects, but the oculogravic illusion was significantly greater in the normal subjects for short periods of observation. But the change in the perception of the horizontal continued systematically for the labyrinthine defective subjects for about 1 hour at the end of which time the magnitude of the oculogravic illusion was not significantly different for the two groups. Preexposure to a visual field was found to be of little importance in producing the lag effect, but setting the line to horizontal during rotation does reduce the lag effect. The results are discussed in terms of Adaptation Level Theory.

## INTRODUCTION

It is a truism of everyday experience that highly complex objects such as living things, scientific publications, musical compositions, television programs, fine wines, and all manner of complex stimulus situations may be judged as if they were simple unitary wholes. Individuals with specialized knowledge or experience can make reasonably dependable judgments of such complex objects because of the ability of the judge to pool or fuse these many attributes into a unitary impression. Indeed, this principle is so important in psychology that Helson (1964) in his recent book, "Adaptation Level Theory,"

has listed pooling and interaction effects as one of his "seven basic characteristics of behavior." Furthermore, Helson has incorporated into his theoretical framework, a second closely related basic characteristic of behavior, the weighting of stimuli in perception.

These basic notions regarding perception emphasize two facts which have long been axiomatic in psychology. The first of these is that the spatial attributes of objects depend not only on the focal stimulus but also upon other stimuli in the perceptual field, e.g., brightness and color contrast effects, geometrical illusions where nonveridical perceptions

result from interactions of spatial attributes of figures, the Bezold-Brücke effect where brightness influences hue, and the influence of intensity on pitch discrimination. The second fact is that the spatial attributes of objects in the perceptual field are affected by stimuli from all other sense modalities to a greater or lesser degree. In the practical experimental situation both of these effects are so ubiquitous that it is practically impossible to study a single sensory dimension in complete isolation.

The type of pooling of concern in this paper is cross-modality interaction, e.g., the interactions of taste and smell, sight and sound, and vision and kinesthesia. Detailed reviews of such studies have been reported by Ryan (1940), Gilbert (1941), and Dember (1960). In addition, a review of the Soviet literature in this field by London (1954) ten years ago cited 506 Russian references. The results of these studies make it obvious that cross-modality pooling is commonplace in perception.

Cross-modality interactions of particular interest to this symposium are those involving the interaction of vestibular processes with other sensory mechanisms. These are of special importance in flight in aircraft and spacecraft because of the unique force environments which may occur under these conditions involving changes in both angular and linear accelerations. Auditory localization has been found to be influenced by a change in resultant force acting on a subject (Graybiel and Niven, 1951) and by semicircular canal stimulation (Clark and Graybiel, 1949a). Many studies have shown that visual space perception is influenced by stimulation of the semicircular canals, e.g., in producing the oculogyral illusion (Graybiel & Hupp, 1946) and in producing changes in the localization of the median plane (Morant, 1959). Furthermore, it has been known for 100 years that body position influences visual space perception as illustrated by the A- and E-phenomena. It has also been known since the time of Mach that centripetal force influences visual space perception producing

what has been termed more recently the oculogravic illusion (Graybiel, 1952; Witkin, 1950). Special cases of this have been called the merry-go-round illusion, the oculogravic illusion (Gerathwohl and Stallings, 1958), and the elevator illusion (Niven, Whiteside, and Graybiel, 1963).

The influence of a change in direction ( $\phi$ ) and magnitude of resultant force on visual perception (i.e., the oculogravic illusion) has been studied extensively at Pensacola. It has been shown that the thresholds for the effects are very low both on the centrifuge (Graybiel and Patterson, 1954) and in flight (Clark and Graybiel, 1949b). The effect is minimal with a full visual framework and maximal in darkness (Graybiel, 1952). The otolith organs appear to play a dominant role in producing the effect in normal subjects, but labyrinthine defective (L-D) subjects exhibit a minimal amount of interaction effects (Graybiel and Clark, 1962). Adaptation effects are lacking at least up to 4 hours (Clark and Graybiel, 1962), and the full effects of change in direction and magnitude of resultant force do not take place immediately, but there is a clearly defined lag effect. A recent investigation (Clark and Graybiel, 1962) has shown that, following a rapid change in direction and magnitude of resultant force acting on the subject, the perceived visual horizontal in darkness changed from the static line-of-subjective-horizontal rather rapidly for 60 sec and then more slowly for an additional 60-sec period. In this process, the subject *slowly* changed his orientation from a visual frame of reference to the direction of the resultant force acting on him. This phenomenon has been termed the inertial-lag effect. It was the purpose of these studies to consider some factors which may contribute to this inertial-lag phenomenon.

#### METHOD

##### Apparatus

All the observations were made in the Pensacola Slow Rotation Room which is, in simple terms, a carousel which rotates counterclockwise (fig. 1). The subject (*S*)

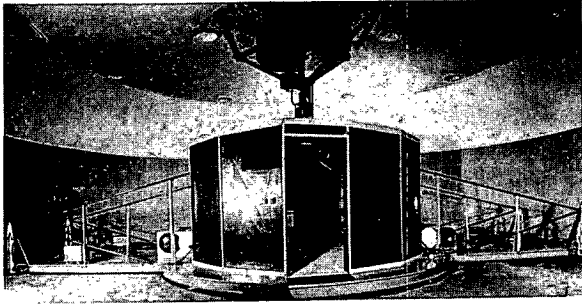


Figure 1.—Pensacola Slow Rotation Room.

was seated 5.5' from the center of rotation and for each trial was accelerated quickly to a velocity to produce a change in the direction of resultant force of either  $20^\circ$  or  $30^\circ$  within 3–6 sec. The *S* was seated in a lightproof cubicle within the room facing the direction of rotation. He was strapped into position in a rigid chair and a Fiberglas head holder was used to maintain the head firmly in position. A collimating device mounted directly in front of him contained a luminous line which could be rotated about its center by either *S* or the experimenter. The experimenter was seated in the room directly behind *S* where he could read the position of the luminous line to the nearest  $\frac{1}{2}^\circ$ .

#### Subjects

Five, experienced, normal *Ss* including one of the authors were studied. All of them had normal nystagmus to caloric stimulation, normal responses to rotation, and experienced the oculogyral illusion. A group of labyrinthine defective (L-D) men also served as *Ss* in certain experiments. All were deaf, had abnormal labyrinthine responses, and had acquired their deafness in childhood as a sequela of meningitis.

#### General Procedure

The same general procedure was used throughout the experiments to be reported with appropriate modifications to investigate the various variables under study. Each series of observations began with the room stationary. The line was then offset for a series of trials, and *S* set it to horizontal in

darkness. The mean of these settings gave a static point-of-subjective-horizontal (PSH) from which deviations during rotation could be measured. The *S*'s task in each experiment was either to maintain the line at his PSH or to reset it to his PSH after it had been offset. Two minute rest periods were given between trials. The extent of the oculogyric illusion was determined for each trial by computing the deviation of *S*'s setting from the mean of his static settings. Using this procedure, *S* could indicate his perception of the visual horizontal as a function of time following a rapid change in the magnitude and direction of resultant force and subsequent constant force environment.

#### Experiments 1 and 2

The purpose of these experiments was to determine whether the nature of the pre-exposure field would influence the inertial-lag effect. It was predicted that pre-exposure to the lighted room would produce a greater inertial-lag effect than pre-exposure to darkness.

*Procedure.*—In Experiment 1 the normal and L-D *Ss* made continuous settings of the luminous line to their PSH while the room rotated at a constant velocity to produce a change in resultant force of  $20^\circ$  or  $30^\circ$ . The settings were made under two conditions: following a 2-minute preexposure to the lighted cubicle or after a 2 minute pre-exposure to darkness. Experiment 2 was identical with 1 except that only the five normal *Ss* were used and that instead of permitting *S* to set the line freely throughout the trial, the line was offset clockwise every 15 sec, and *S*'s task was to reset it.

*Results of Experiments 1 and 2.*—Since the results of the first two experiments are similar, they will be considered together. The data show the inertial-lag effect clearly both in normal and L-D *Ss*. In figure 2, which presents the data for normals with continuous settings, there was a tendency for the oculogyric illusion to be greater following pre-exposure to darkness, but the curves are similar and overlap at both velocities.



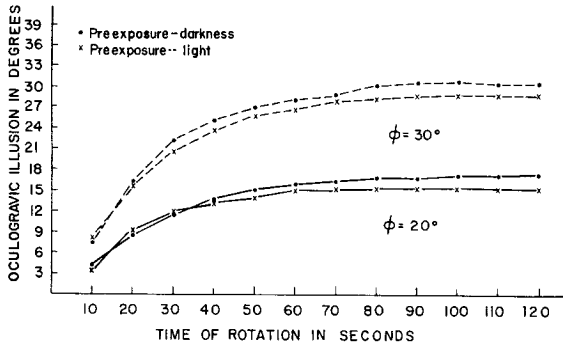


Figure 2.—Mean values for oculogravic illusion in five normal subjects: continuous settings.

Similar lag effects are shown for the L-D Ss (fig. 3), but the preexposure to the lighted cubicle shows a greater illusory effect at the slower velocity, making the results ambiguous. When the offset method was used for normal Ss (fig. 4), the curves overlapped at both velocities. To determine the significance of the differences between the light and dark conditions, the mean performance for each S for the final 30 sec of the trials was determined. It was found that only one of the six comparisons was significant at the 0.01 level while for all the others

the *p* values were greater than 0.10. Thus, the only significant difference was in the predicted direction, but all of the differences were quite small. For example, in the case of the lower velocity for Experiment 1 for the normal Ss, it can be said at the 0.05 level of confidence that the difference is between 0.5° and 3.2°.

Experiment 3

The purpose of this experiment was to study the effects of delay in presenting the luminous line following acceleration to a constant velocity. Some incidental observations had suggested that the inertial-lag effect would be longer if *S* was exposed to darkness without viewing the line than if the line was visible continuously throughout the trial.

*Procedure.*—The procedure was similar to that followed in the previous experiments. Immediately after the static observations were completed, the *S* was exposed to two minutes of darkness, and then the room was quickly accelerated to produce a change in the direction of resultant force ( $\phi$ ) of 20° or 30°. In these trials, however, seven conditions of delay in exposure of the luminous

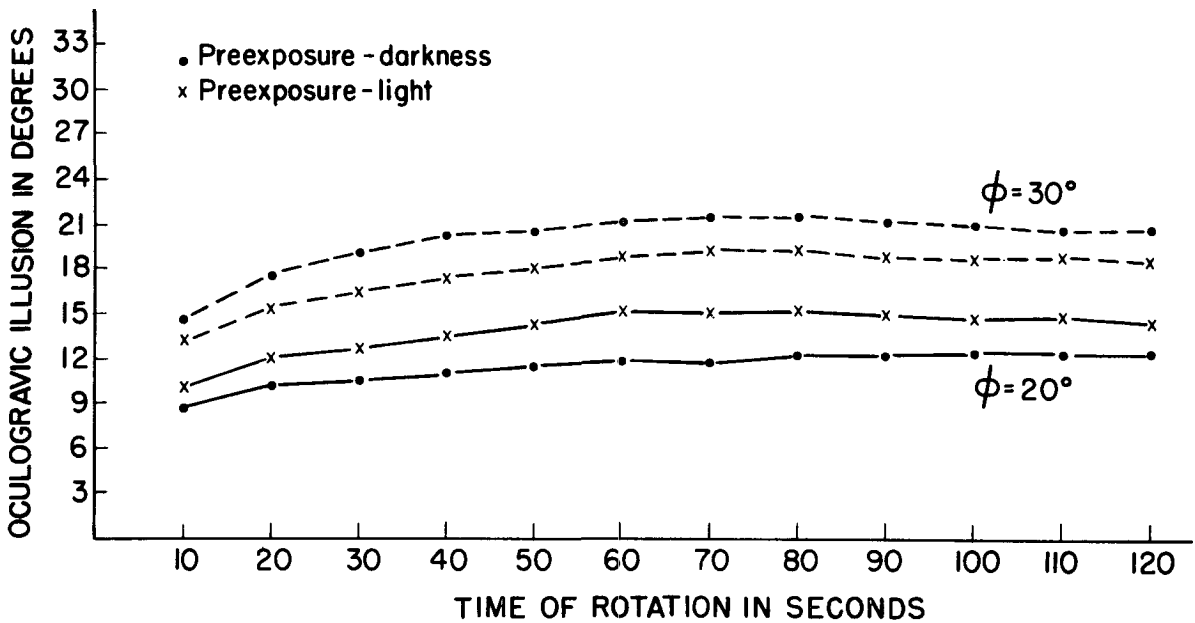


Figure 3.—Mean values for oculogravic illusion in nine L-D subjects: continuous settings.

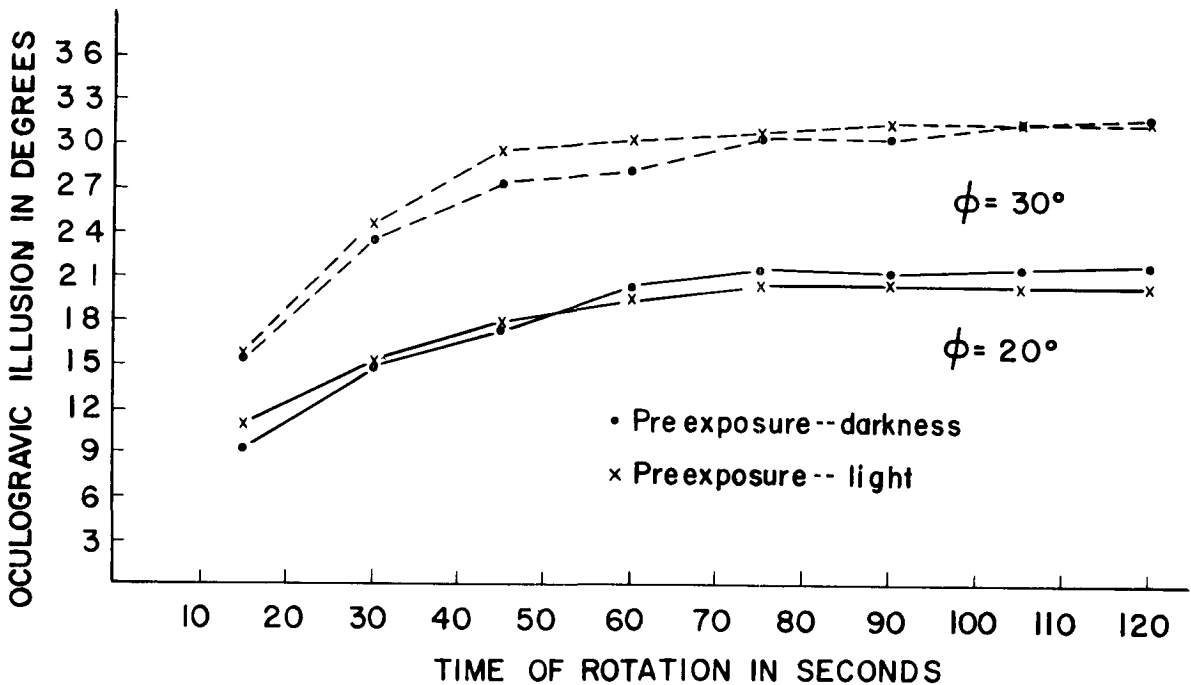


Figure 4.—Mean values for oculogravic illusion in five normal subjects: offset method.

line were used. In Condition 1, the luminous line was turned on as soon as the room began to rotate, as in the previous experiments. For Condition 2, the room rotated with the subject in darkness for ten seconds, and then the luminous line was turned on and readings began ten seconds later at 20 seconds. The seven delay conditions were: 0, 10, 20, 30, 50, 70, and 120 seconds. The room rotated for 3 minutes during each trial. The order of presentation of the conditions was randomized, and five trials were taken for each delay period at each of the two velocities of rotation.

*Results of Experiment 3.*—As in the previous experiments, the inertial-lag effect was clearly evident (fig. 5). The results also show that observation and setting of the luminous line has a clear effect on *S*'s perception of the horizontal under these conditions. As the period of delay in presenting the line increased, three things happened: First, the value of the initial setting increased in accordance with the change in the direction of resultant force acting on the subject. Second, the first setting of the line,

when it was illuminated, fell below the corresponding setting for the zero delay condition by an increasing amount, being about  $1^\circ$  at the 10-second delay and about  $4.5^\circ$  at the 120-sec delay. Third, the final reading at the end of the 3 minutes tended to be less than that when *S* observed the luminous line throughout the trial. For a change in direction of resultant force of  $20^\circ$ , all of the means of the seven delay conditions were significantly less than those for the zero delay condition ( $p \leq 0.05$ ). The differences at the greater velocity were less convincing, but the differences at 50 and 120 sec were significant ( $p \leq 0.05$ ). Three of the remaining four were in the predicted direction, but none of the differences was significant. These data make it clear that the inertial-lag effect is a function of the interaction of labyrinthine cues and visual cues associated with setting the line to horizontal.

#### Experiment 4

The purpose of this experiment was to study the effects of two types of fixation during the period of delay before *S* began to

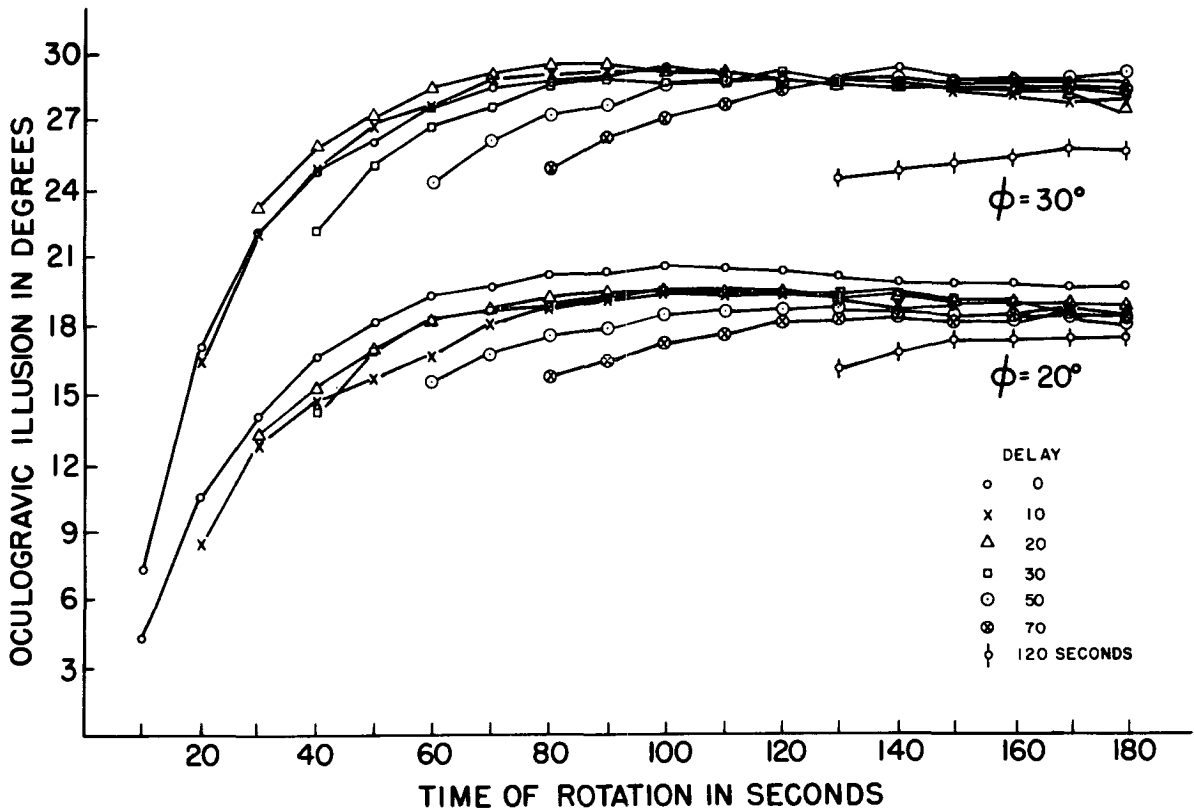


Figure 5.—Mean values for oculogravic illusion in normal subjects with progressively longer delay time in presenting target.

make settings of the luminous line to horizontal.

*Procedure.*—The procedure was similar to Experiment 3 except that only the zero and 2-minute delays were compared, and two conditions of visual fixation during the delay were used. During the first condition, the Ss fixated a luminous spot during the 2-minute delay period before the settings began, and during the second delay condition, they viewed the luminous line set to the mean of the preliminary, static settings; i.e., they viewed the line which appeared to be rotated clockwise from the PSH without setting it. Again, comparisons were made between the zero delay setting at 130 sec and the first setting following the period of observation of the spot or line.

*Results of Experiment 4.*—In each case, the mean of the settings following the 2-minute delay period was less than the corre-

sponding setting at 130 sec for the zero delay condition. These differences were, however, not significant following the fixation of the spot ( $p > 0.10$ ). Following fixation of the line at  $20^\circ$  change in direction of resultant force, the difference was also not significant ( $p > 0.05$ ), but at the higher velocity it was barely significant ( $p < 0.05$ ).

#### Experiment 5

In the previous experiments for normal Ss when an abrupt acceleration to a constant velocity occurred, S's visual orientation changed slowly, and the oculogravic illusion reached a maximum value in about 2 minutes. Following this inertial-lag effect there is little or no systematic change in the perceived visual horizontal at least up to 4 hours (Clark and Graybiel, 1962). The lag effect also occurs in L-D Ss, but it is less clearly defined, and no data are available for pro-

longed periods of rotation. It was the purpose of this experiment to make frequent, periodic observations of the perceived visual horizontal in both normal and L-D Ss during one hour of constant rotation to identify any systematic changes in the visual horizontal.

*Procedure.*—Following five static settings of the line to horizontal in darkness, the room was accelerated quickly to produce a change in direction of resultant force of  $20^\circ$ . The line was immediately offset, and *S* set it to horizontal and maintained it in that position. At 15 sec following the beginning of rotation and every 15 sec up to 2 min, a reading was made and immediately thereafter the line was offset as before. The line was turned off after the 2 min reading and remained off for 45 sec. During this period it was offset to a prearranged position, and when the luminous line was turned on, *S* set it to horizontal. *E* made a reading at 3 min and turned the light off again for 45 sec. Readings were then made every minute for 60 min. The offsets were prearranged to make *S*'s task fairly difficult. The reference point was always his last setting. For the 2 min setting

it was offset from the previous setting clockwise (+)  $10^\circ$ ; at 3 min it was offset counterclockwise (—)  $10^\circ$ . Succeeding offsets were  $+20^\circ$ ,  $-20^\circ$ ,  $+30^\circ$ , and  $-30^\circ$ . This sequence was then repeated until 58 additional settings were made. The oculogravic illusion was determined by computing the deviation of *S*'s observations from the mean of his static settings. This procedure obtained a record of *S*'s perception of the horizontal as a function of time following a change in the magnitude and direction of resultant force acting on him.

*Results.*—The results showing the change in settings to the horizontal for one hour for the five normal Ss and the nine L-D Ss are presented in figure 6. There is a rapid increase in the oculogravic illusion for about 60 sec and then a slight reduction with a leveling off thereafter for both groups. It is worth noting that the 2 min setting for both groups is greater than the 3 min setting. This is to be expected since the former setting followed 2 min of continuous viewing of the line whereas the 3 min setting followed 45 sec of darkness (cf., Ex-

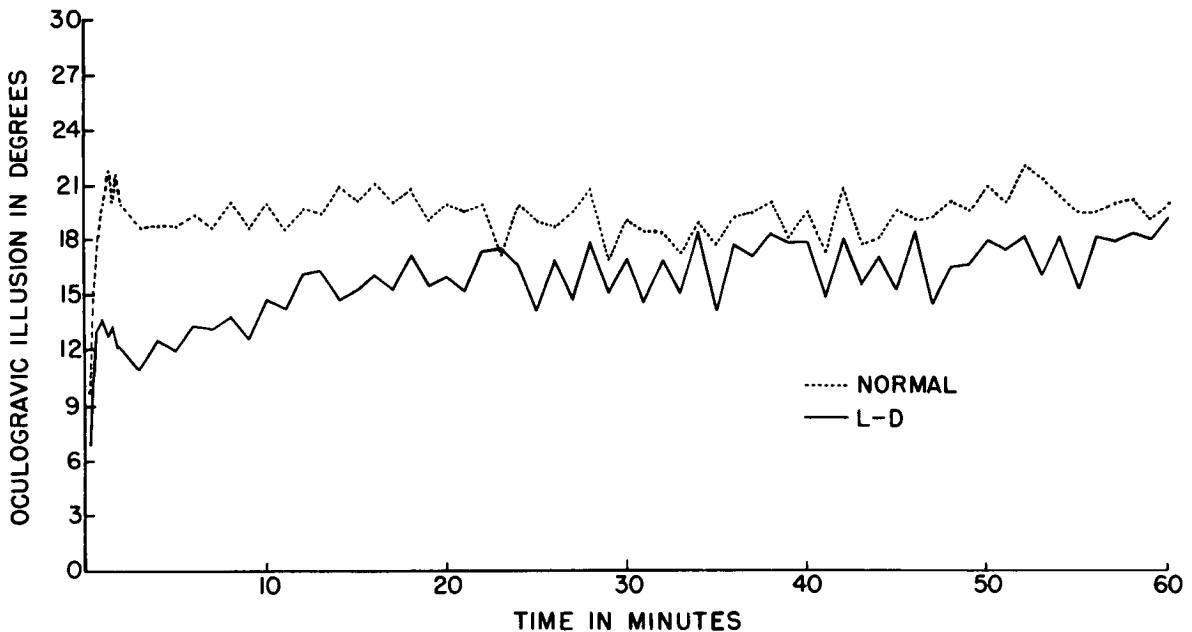


Figure 6.—Mean values for oculogravic illusion (angle  $\phi$   $20^\circ$ ) for five normal subjects and nine L-D subjects rotated continuously for 1 hour.

periment 3). The normal and L-D Ss differ from each other in two significant ways: First, at the termination of the rapid phase of the increase in the illusion, the normal group made settings to the horizontal which were closely in accord with the direction of the change in the direction of force; i.e., the oculogravic illusion was approximately  $20^\circ$  while the illusion was only about  $13^\circ$  for the L-D group (fig. 6); second, the settings of the normal Ss did not show a systematic change following the inertial-lag effect whereas the L-D Ss showed a continuous increase in the illusion throughout the 1-hour period of the observations. A trend analysis (Kurnow, Glasser, and Ottman, 1959) was performed for the two groups independently, combining the data in blocks of five 1-min trials. The test for trend for the normal Ss was *not* significant ( $p > 0.10$ ), while the L-D Ss exhibited a significant trend in the direction of an increase in the oculogravic illusion throughout the hour of testing ( $p < 0.01$ ). Over the first block of five 1-min settings, a median test showed that the normal group had a significantly greater illusion ( $p = 0.03$ ), but the means of the settings from 56–60 min were not significantly different ( $p = 0.37$ ). Thus, although the normals showed a consistently larger mean oculogravic illusion throughout the hour of constant rotation, during the final 5 min, the difference was only  $1.4^\circ$ . It should be noted that nonparametric analyses have been used here because of the gross difference in variance between the two groups.

#### DISCUSSION

The results of the five experiments reported give additional evidence of the presence of the oculogravic illusion and at the same time shed some light on the factors involved in the inertial-lag effect. In searching for factors contributing to the prolonged latency or lag between the change in the force environment acting upon *S* and the end point of the change in his orientation to the visual vertical, an obvious possibility in the normal Ss would be to consider the transducer mechanism of the otolith organs.

Loewenstein and Roberts (1950) have shown, however, that the time necessary for individual utricular fibers to assume a new discharge rate following a change in position is only a few sec. Consequently, it becomes necessary to look elsewhere for factors contributing to the inertial-lag effect.

The data (Experiments 1 and 5) showing a greater oculogravic illusion for normal Ss than for L-D Ss for short periods of observation are well known (Graybiel and Clark, 1962). But the finding that after an exposure of 60 min to a constant change in the direction and magnitude of resultant force, the L-D Ss do not differ significantly from normals has not been reported elsewhere in the literature to our knowledge and warrants special consideration.

It has been widely assumed that the otolith organs play a crucial role in the perception of the *postural* vertical as well as the *visual* vertical in normal Ss. At the same time the evidence shows that although L-D Ss make greater errors in setting themselves to the *postural* vertical than normals for initial settings, the difference between the average settings for the two groups is not significant after practice (Clark and Graybiel, 1963a). This makes it clear that other gravitational receptors than the otoliths are involved in this task. The fact that the oculogravic illusion increases throughout the one hour of constant rotation for the L-D Ss also suggests that information from other sensory mechanisms than the otoliths becomes important in their perception of the horizontal over time. The gravitational receptors of the tactile and proprioceptive sensory modalities are highly complex (Rose and Mountcastle, 1959). It has been shown that for both of these sensory mechanisms, there are individual fibers which adapt slowly and others which adapt rapidly. For example, Loewenstein (1956) has found that fibers from certain stretch mechanoreceptors in the skin of the frog "... show no signs of adapting to zero over the period of 40 min of observation," whereas Gray and Matthews (1951) report that fibers from the Pacinian cor-

puscles in the cat's toe adapt in about 2 min. Thus, it would appear that for the L-D Ss, there would be variable synergistic information from several tactile receptors and proprioceptors over a short period of time, but that information from several other tactile and proprioceptive receptors would be available for a prolonged period of time as in the case of the otoliths for normals. This continuous information from these gravitational receptors may become more heavily weighted over time, and cause a change in the perception of the visual horizontal in darkness.

Another line of evidence which bears on this problem of the delay in the effects of a change in direction of resultant force acting on *S* is to be found in changes in the perception of the *visual* horizontal attendant upon changes in the visual framework in passing from a condition of a lighted room to a darkened room *during constant rotation*. It has been reported by Clark and Graybiel (1963b) that an effect similar to the inertial-lag effect also occurs when *S* makes settings to the horizontal with the room illuminated and then shifts to settings in total darkness. For normal Ss the oculogravic illusion increases rapidly for about 60 sec and more slowly for an additional 60 sec as it does immediately following a change in the direction and magnitude of the resultant force acting on *S*. These data support the results of Experiment 3 in indicating an interaction of visual and gravitational factors.

In general terms these experiments present evidence of the direct influence of otolith, tactile, and proprioceptive stimuli on the visual perception of the horizontal. The data suggest the presence of central pooling across these sense modalities with a differential weighting of the stimuli under different stimulus conditions and between normal and L-D subjects. The data tend to be in

accord with Helson's (1964) Adaptation-Level Theory of perception which conceives of perception as being the pooled effect of focal, contextual, and residual sources of stimulation. Under this concept visual and the various gravitational cues would be considered to combine to produce the resultant perception. For normal and L-D Ss the visual perception of the horizontal with full visual framework would be heavily weighted toward visual cues. In the absence of a full visual framework the normal *S* places heavier weight on the gravitational, probably primarily on the otolith, cues and in the experiments reported here, after a substantial latency *S* perceives the horizontal in terms of heavy weighting on otolith stimulation. The data from Experiment 5 suggest that a similar differential weighting also occurs with the L-D Ss with the important difference that the latency in weighting the nonlabyrinthine cues is about six times as great. This difference may be found in part in differential adaptation rates in tactile receptors and proprioceptors as indicated above, but it is suggested that the primary variable is in central nervous system factors in which there is a differential weighting process for the limited sensory cues available to the L-D subjects. The possibility of neural interaction between central sensory areas has, of course, been suggested for a variety of perceptual and learning phenomena. Nevertheless, the specific neurological basis for these effects is unknown. Particularly difficult of explanation by simple neurological interaction or pooling is the prolonged duration of the inertial-lag effect for the L-D Ss. However, in terms of the Adaptation-Level Theory the inertial-lag effect may be described in terms of a differential rate of change in weighting of otolith as compared to other gravitational receptors.

#### REFERENCES

- CLARK, B.; AND GRAYBIEL, A.: The Effect of Angular Acceleration on Sound Localization: the Audiogravic Illusion. *J. Psychol.* 28, 1949a, pp. 235-244.
- CLARK, B.; AND GRAYBIEL, A.: Apparent Rotation of a Fixed Target Associated With Linear Acceleration in Flight. *Amer. J. Ophthal.*, 32, 1949b, pp. 549-557.
- CLARK, B.; AND GRAYBIEL, A.: Visual Perception of the Horizontal During Prolonged Exposure to Radial Acceleration on a Centrifuge. *J. Exp. Psychol.*, 63, 1962, pp. 294-301.

- CLARK, B.; AND GRAYBIEL, A.: Perception of the Postural Vertical in Normals and Subjects With Labyrinthine Defects. *J. Exp. Psychol.*, 65, 1963a, pp. 490-494.
- CLARK, B.; AND GRAYBIEL, A.: Contributing Factors in the Perception of the Oculogravic Illusion. *Amer. J. Psychol.*, 76, 1963b, pp. 18-27.
- CLARK, B.; AND GRAYBIEL, A.: Perception of the Postural Vertical Following Prolonged Bodily Tilt in Normals and Subjects with Labyrinthine Defects. *Acta Otolaryng.*, 58, 1964, pp. 143-148.
- DEMBER, W. N.: *Psychology of Perception*. New York: Henry Holt, 1960.
- GERATHEWOHL, S. J.; AND STALLINGS, H. D.: Experiments During Weightlessness: A Study of the Oculo-Agravic Illusion. *J. Aviat. Med.*, 29, 1958, pp. 504-516.
- GILBERT, G. M.: Inter-Sensory Facilitation and Inhibition. *J. Gen. Psychol.*, 24, 1941, pp. 381-407.
- GRAY, J. A. B.; AND MATTHEWS, P. B. C.: Responses of Pacinian Corpuscles in the Cat's Toe. *J. Physiol.*, 113, 1951, pp. 475-482.
- GRAYBIEL, A.: The Oculogravic Illusion. *Arch. Ophthalm.*, 48, 1952, pp. 605-615.
- GRAYBIEL, A.; AND BROWN, R. H.: The Delay in Visual Reorientation Following Exposure to a Change in Direction of Resultant Force on a Centrifuge. *J. Gen. Psychol.*, 45, 1951, pp. 143-150.
- GRAYBIEL, A.; AND CLARK, B.: The Validity of the Oculogravic Illusion as a Specific Indicator of Otolith Function. NSAM-849. NASA Order No. R-37, Naval School of Aviation Medicine, Feb. 17, 1962.
- GRAYBIEL, A.; CLARK, B.; AND ZARRIELLO, J. J.: Observations on Human Subjects Living in a 'Slow Rotation Room' for Periods of Two Days. *Arch. Neurol.*, 3, 1960, pp. 55-73.
- GRAYBIEL, A.; AND HUPP, D. I.: The Oculogyral Illusion. *J. Aviat. Med.*, 17, 1946, pp. 3-27.
- GRAYBIEL, A.; AND NIVEN, J. I.: The Effect of a Change in Direction of Resultant Force on Sound Localization: The Audiogravic Illusion. *J. Exp. Psychol.*, 42, 1951, pp. 227-230.
- GRAYBIEL, A.; AND PATTERSON, J. L., JR.: Thresholds of Stimulation of the Otolith Organs as Indicated by the Oculogravic Illusion. *J. Appl. Physiol.*, 7, 1955, pp. 666-670.
- HELSON, H.: *Adaptation-Level Theory*. Harper & Row, 1964.
- KURNOW, E.; GLASSER, G. J.; AND OTTMAN, F. R.: *Statistics for Business Decisions*. R. D. Irwin, Inc. (Homewood, Ill.), 1959.
- LOEWENSTEIN, O.; AND ROBERTS, T. D. M.: The Equilibrium Function. *J. Physiol.*, 110, 1950, pp. 392-415.
- LOEWENSTEIN, W. R.: Excitation and Change in Adaptation by Stretch Mechanoreceptors. *J. Physiol.*, 133, 1956, pp. 588-602.
- LONDON, I. D.: Research on Sensory Interaction in the Soviet Union. *Psychol. Bull.*, 51, 1954, pp. 531-568.
- MORANT, R. B.: The Visual Perception of the Median Plane as Influenced by Labyrinthian Stimulation. *J. Psychol.*, 47, 1959, pp. 25-35.
- NIVEN, J. I.; WHITESIDE, T. C. D.; AND GRAYBIEL, A.: The Elevator Illusion: Apparent Motion of a Visual Target During Vertical Acceleration. Joint Report. FPRC 1213, NSAM-876. NASA Order No. R-93. Flying Personnel Research Committee, Air Ministry, and Naval School of Aviation Medicine, October 18, 1963.
- ROSE, J. E.; AND MOUNTCASTLE, V. B.: Touch and Kinesthesia. In: *Handbook of Physiology* (Field, J., ed.), Section 1: Neurophysiology. Volume 1. Amer. Physiol. Soc., 1959.
- RYAN, T. A.: Interrelations of the Sensory Systems in Perception. *Psychol. Bull.*, 37, 1940, pp. 659-698.
- WITKIN, H. A.: Perception of the Upright When the Direction of the Force Acting on the Body is Changed. *J. Exp. Psychol.*, 40, 1950, pp. 93-106.

#### DISCUSSION

BERGSTEDT: I have two short comments. One, I made a study on positional nystagmus in a human centrifuge and found just the same kind of lag, which I think is a thing that is characteristic for that receptor system for gravity. The second is that I have seen a paper by Captain Wing of the Air Force, who has picked up signals from Scarpa's ganglion with a lag of 40 to 60 seconds. If this is verified, there is electrophysiological evidence of this lag effect. He published his paper about two years ago in *Acta Otolaryngol.* I have been in contact with him but he doesn't seem to have continued his interesting study. So there do seem to be certain cells with such long lag effect from the receptor itself, which is tremendously interesting.

CLARK: Your comments, of course, refer to normal subjects with normal labyrinths. That still leaves us with the L-D subjects for explanation for the very long, apparently about an hour, lag. I would be interested in your comment on this.

BERGSTEDT: I agree with you that the otolith organs mean most for this phenomenon but there are other mechanisms too. The human body doesn't only rely on one mechanism; other mechanisms can substitute or be trained up.

MAYNE: Dr. Clark, it seems to me there is another mechanism that may be considered in the difference between your L-D subjects and the normal subject; namely, the difference in resolution of the stimulus by somatic and vestibular organ, using the word

resolution in the engineering sense and nearly synonymous to just noticeable difference. Both organs are sensitive to the same stimulus but provide a different rate of information. With a lower information rate the signal-to-noise ratio is reduced, and a greater smoothing time may be taken by the organism to arrive at a proper solution in the way of a subjective perception of horizontality.

CLARK: It would seem to me that in setting a line, the subject is given a full opportunity to set it to what he considers his point of subjective horizontal, and I am not quite clear how the differences, the different levels would affect this ability.

MAYNE: I could think of a process where the detection of differences in the direction of the forces on the body would be just slightly above the threshold, and with all the noise that may be present, the evidence is lacking to dispose of the feeling that the subject is sitting up straight, since he has experienced no sensation from the semicircular canals that he has been rotated. Under these conditions the weaker evidence of a change of orientation which has occurred takes a longer time to affect other evidence that the body has not been rotated.

GUALTIEROTTI: I would like to make some comments about the delay of the receptors' response. Recording directly from the otolith units, you find different kinds of effects. First of all, you get an immediate response. Then you have some accommodation effect, by which a steady state in the activity of the receptors is achieved only after 3 to 10 seconds. Then you get a further, later effect which is a suppressory effect. This may be delayed by up to 30 seconds or more. I would emphasize that the delayed effect might result from events happening directly at otolith level. However, there should be an immediate response in a correlated function if this is directly and continuously related with the otolith activity because you have an immediate change as soon as you start tilting your head.

WENZEL: I have a slide of an experiment that shows how many of the other sensory organs may come into play. The subject is on a centrifuge and is walking a curved wall while lying in a sling used to counter Earth's gravity. We did not perceive an oculogravic illusion at all. Now, if the subject lay on a cot, then he got a 45° oculogravic illusion. He felt the sliding effect of friction on the cot and he felt like he was lying on the side of a slope of 45°. But in the sling, the centrifugal force is taken up by the feet and there was no oculogravic illusion at all.

GRAYBIEL: How did you measure the oculogravic illusion?

CLARK: Thank you. I was going to ask the same question.

WENZEL: The illusion was not measured by any instrumentation. I was the subject in this case and I experienced two very distinct, different sensations. One, I felt exactly as though I was in a space station.

There was no tilt whatsoever, and in the other case, on the cot, I definitely felt that the room was tilted and disorientated with the centrifuge.

GRAYBIEL: This was not a visual perception, I take it, hence, not the oculogravic illusion. Is that right?

WENZEL: That's right. My total perception was that of the room being in the normal orientation with the centrifuge when I was in the sling, but when I was on the cot, I felt the room was tilted 45°.

M. JONES: I would like to suggest that the time course of the gradual approach to steady-state might reflect the time course of a short-term memory loss of semicircular canal information. If one considers the equivalent experience to which the mechanisms of attitude perception would be exposed in natural life, we have a real angular tilt as on a tilt-table. In this case both the gravity and the rotation sensing (canals) devices will tell the central nervous system there has been such and such a change in angle of tilt, say 20°. In the centrifuge experiments only the gravity sensing elements sense the change in direction of resultant vector. Hence the CNS presumably receives two opposing signals; one from gravity sensation which says "20° tilt left" and the other from the canals which says (equally positively) "no tilt has occurred."

One might imagine that the "no tilt" canal signal, being derived from an organ which only responds to a dynamic rather than a static stimulus, would initially subtract from the gravity signal, leading to an underestimate of the angle of tilt. But with time the steady d.c. gravity signal would remain in evidence whilst the memory of the transient "no tilt" canal signal would be expected to fade along a particular time course. In this case the perceived angular tilt could be imagined as progressively moving towards the information from gravity sensation, as the opposing canal information is progressively "forgotten" by the CNS computer.

Perhaps the "forgetting" time course corresponds to your observed time course towards a steady state of perceived angle of tilt. If this were indeed the case, the method might be made to yield interesting quantitative data on short-term retention of stored information, or in effect on short-term memory processes.

CLARK: I would merely like to comment that, on this one point, there are some very real differences between just tilting the body in a tilt chair and the situation on a centrifuge. One of them obviously is the G level. We always have not only a change in the direction of force, we have a change in the magnitude of the force. It seems to me that it is clear that the oculogravic illusion either on elevation or rotation of the target, depending on the bodily orientation, increases pretty much as the velocity increases or the tilt increases whereas if you tilt the body, for example, one gets first the E-phenomenon in one direction and then the A-phenomenon in the



other direction. We have discussed this some and at least I am not clear on the point. Maybe Dr. Graybiel would like to discuss it.

CRAMPTON: With a very fast onset of angular acceleration designed to quickly produce a  $20^\circ$  tilt when riding at a radius, I have experienced in total darkness a transient-like sensation of immediately tipping further than  $20^\circ$  and then more slowly returning to  $20^\circ$ . It has occurred to me, of course, that this is due to over-shooting of the otoliths and I want to ask if you ever see this overshooting in your L-D subjects. I appreciate that oftentimes it is difficult to communicate with them.

GRAYBIEL: I would make two comments. First, a normal person who is experiencing this change in direction of the force vector doesn't experience the full amount of the tilt immediately. There is a lag, a so-called postural lag effect. We have tried to determine if this was experienced by our L-D subjects. We have not established that they experience such a lag, although failure might have been due to communication difficulties. They do experience a postural illusion of tilt which differs from that indicated by normal subjects mainly in being less precise. With eyes closed and using a "swivel-rod" to indicate the direction of the gravito-inertial upright they make grosser corrections than do normal subjects during the change in the force vector and, in the steady state, are more inclined to under or overcorrect.

One other point that I think might clarify the role

of the peripheral end organs in the perception of the oculogravic illusion is to point out that, if during perception of the illusion you turn the light on in the cubicle, you nearly abolish it; that is, you will reset the luminous line nearly to the gravitational vertical. Then if you turn the light off again, the luminous line slowly appears to rotate to accord once more with the force upright. Inasmuch as the force environment has never changed, this "second" lag effect must be due to central mechanisms.

CLARK: I would like to emphasize the variance in performance of the L-D subjects. There were very marked individual differences among these people.

MONEY: I would like to mention, unless we forget, that there are also some extremely rapid labyrinth dependent responses to linear acceleration and gravity. For example, if a blindfolded animal is accelerated downward, the toes spread and the limbs extend, certainly in less than half a second, and probably much faster than that.

STEELE: I want to put in a word for perception being a central phenomenon not directly residing in the sense organs. It is a synthesis of available sensory data weighted by some estimate of relative reliability if various inputs are in conflict. From personal observations, on a merry-go-round with full visual observations of the world, I have found that acceptance of the stationary geographic or the rotating tilted horizon is almost a matter of choice. I simply switch back and forth as convenient.

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# The Vestibular Contribution to Stabilization of the Retinal Image

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## SUMMARY

The vestibulo-ocular reflex system is examined in the context of an essential backup to the visual fixation reflex. The vestibular stimulus (in this case, mainly the semicircular canal) is seen as the output from an angular velocity transducing hydrodynamic system, feeding the central nervous system with angular velocity modulated information, provided the mechanical stimulus is confined within a specified frequency range. This general interpretation appears to apply over a wide range of different animal species. The central nervous angular velocity signal is envisaged as driving an angular velocity control system through the physiology of the oculo-motor system. It appears that the dynamic response of the vestibulo-ocular system improves as that of the visual tracking systems fails, to the extent that the former operates with a gain of 1 at frequencies associated with failure of the latter. The overall system is apparently superimposed by a pattern of response which insures that compensating eye muscles are stretched on an average by an amount directly related to the compensatory eye angular velocity required for image stabilization at every instant. In addition, the system response is substantially dependent upon the axis of rotation referred to the head, in the sense that the rate of intrusion of errors attributable to cupular restoration is greater in the vertical than in the horizontal planes. Moreover, in the roll plane there appears to be relatively poor visual tracking, and this permits the virtually unopposed operation of the vestibulo-ocular reflex when the latter is at variance with the real event.

These mechanisms, nicely adapted to man's natural environment, are not so nicely adapted to the environments of flight and space. In three-dimensional space the natural constraints to rotational head movement may be removed and rotational movements may be faster, have longer transients, and extend over a wider frequency range than normal. In consequence, errors due to cupular restoration, predominance of an anti-compensatory response, and dissociation between the movement of the visual image and the vestibulo-ocular drive in this plane can arise.

Author

## INTRODUCTION

In order to "see" a visual scene clearly, the corresponding image on the retina of the eye must be stabilized, at least intermittently, even during short sharp head movements. With the head stationary this can usually be adequately achieved through the oculo-motor response to visual information; that is, by means of visual fixation achieved through the visual tracking system. But with the introduction of head movement, the visual tracking system alone may become quite

inadequate. The maximum relative angular velocity for good visual tracking is around 30°/sec (ref. 1) while the maximum frequency of good visual following is around 1 cps (refs. 2 and 3). Yet when running, for example, a fundamental frequency of about 4 cps is commonly generated. Moreover, assuming a 5° amplitude of resulting head movement, a maximum head angular velocity around 125°/sec would then be encountered. Thus both the frequency cut-off and the maximum tracking angular velocity

attainable from vision alone may frequently be far exceeded. Evidently, since clear vision is possible while running, a powerful "back-up" system is brought into play, namely the vestibulo-ocular reflex mechanism.

With the eyeball constrained to move only in the rotational degrees of freedom relative to the skull, it seems rational to assume that the rotation sensing vestibular components, namely the semicircular canals, provide the main drive to the vestibulo-ocular reflex. Consequently, this account will be largely confined to the part played by these organs. It must be noted in passing, however, that it is becoming progressively more evident that the oculomotor response to canal stimulation can in some circumstances be substantially modified by otolithic stimulation (ref. 4).

#### THE VESTIBULAR STIMULUS

##### Mechanical Considerations

Mechanically each semicircular canal essentially comprises a thin tube, containing fluid having density and viscosity approximately the same as water, with continuity of fluid flow round a circuit conducted through the utricle, except insofar as there is a watertight transducer, the cupula, inserted at one point in the circuit. The cupula acts as a watertight, spring loaded, swing door, hinged at its base and generating a neural signal, which is some function of the angular deflection of the cupula. Thus, for small angles of deflection the cupular angle directly reflects the volume flow of fluid round the closed circuit. The relationship between cupular deflection and angular movement of the head is therefore determined by hydrodynamic factors (moment of inertia and viscosity of the fluid) and the elastic characteristics of the cupula itself.

In this connection it is important to appreciate the significance of the very small size of the hydrodynamic system. At the dimensions of the human semicircular canal (internal diameter of the tube approximately  $\frac{1}{3}$  mm) the viscosity of the fluid plays a dominant role, which fact essentially deter-

mines the mode in which the semicircular canal functions as a transducer.

As the size of a hydrodynamic system decreases so does the Reynold's number, which amounts to the fact that the ratio of inertial to viscous forces decreases, or conversely the ratio of viscous to inertial forces increases. Thus, in the very small human canal, the mode of relative fluid flow is substantially governed by the fluid viscosity. Three important points emerge from this. First, the actual amount of relative fluid flow is exceedingly small; sufficiently small that is for the cupular system to follow all normal flows without leakage and hence to measure faithfully the relative fluid displacement incurred. Second, the relative flow being very small, it is permissible to assume, to a close approximation, that the inertial force tending to cause fluid flow around the circuit during angular acceleration of the canal is the same as it would be if there was no fluid slip. Third, such flow as does occur must be laminar, with the consequences that the velocity of flow will be proportional to the inertial force driving it.

From these considerations it can be seen that the inertial driving force is proportional both to the angular acceleration of the canal in its own plane (Newton's laws of motion) and the velocity (very small) of relative fluid flow. Thus the velocity of flow is seen to be proportional to the angular acceleration of the system, with the consequence that the fluid displacement, and hence also the angle of cupular deflection, are proportional to the angular velocity of the system.

Looked at in this way it seems obvious that the canal hydrodynamics perform one integration on the angular acceleration to which it is exposed, and by so doing turn the mechanical elements at least, into an angular velocity transducer. In this connection it is significant that at the very small Reynold's numbers involved the hydrodynamic integration would be expected to be very precise.

However there are other points to be considered; in particular, the elastic restoring

force of the cupula and the rate of achieving steady flow conditions in response to a given angular acceleration. Perhaps the simplest and most meaningful approach to an understanding of the normal physiological part played by these factors is that of a frequency response analysis, first performed as such on the canal by Mayne (ref. 5), so far as the author is aware. Other authors have since reappraised the matter, for example, Niven & Hixson (ref. 6) and M. Jones and Milsum (in "Spatial and Dynamic Aspects of Visual Fixation," in press). Figure 1 gives the frequency response data obtained by the latter authors from a combination of physical analyses based on canal dimensions given by Jones and Spells (ref. 7), and the experimental data of Van Egmond, Groen and Jongkees (ref. 8). The curves in this figure show the relations between the response, chosen as cupular deflection and the stimulus, chosen as *angular velocity* of the

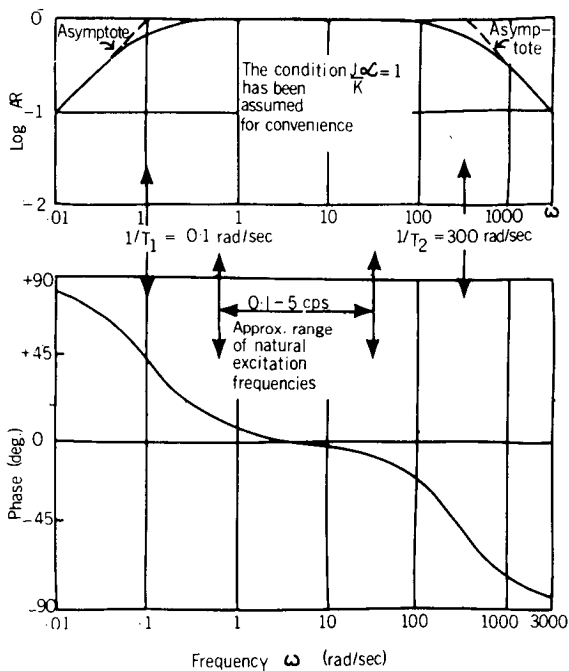


Figure 1.—Theoretical frequency response of mechanical components of semicircular canal, based partly upon numerical data in references 7 and 8. This frequency response diagram relates to transfer function (angle of cupular deflection/angular velocity of canal in its own plane). AR = amplitude ratio.

canal. It appears from the figure that the amplitude ratio (AR) chosen in this way (cupular angle/angular velocity of the canal) is constant and the phase shift negligibly small, over a range of frequencies extending roughly from 0.1 to 5 cycles per sec. If this conclusion is correct, then we may say that the mechanical components of the canal do indeed act as an angular velocity transducer so long as angular movements contain only frequencies within this range; which is tantamount to saying that this is the case for virtually all naturally occurring patterns of head movement. At slower frequencies, including of course the case of steady angular velocity, time permits the intrusion of elastic restoration of the cupula, and then the well known errors in canal response ensue, generating both illusions of motion, and inappropriate postural and oculomotor responses.

It might have been a matter of chance that the size of the human canals should be such as to lead to these conclusions. But the study of Jones and Spells (ref. 7) referred to above strongly suggests that this is not the case. Over a wide range of species there is little change of canal dimensions with body weight, approximately 7 log cycles of weight change being required to bring about 1 log cycle of the relevant linear dimensions (fig. 2). Indeed, these authors have shown that such very small dimensional changes as do occur are of an appropriate magnitude

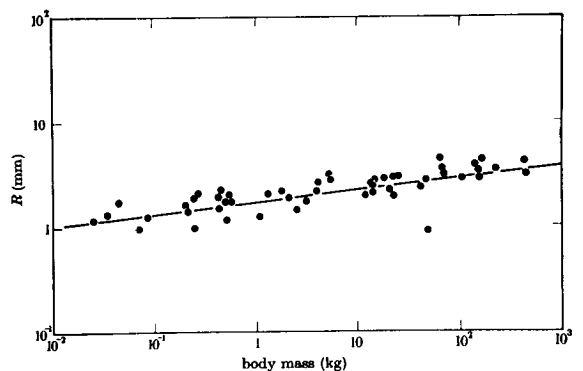


Figure 2.—Relationship between radius of canal (R) and body mass obtained from 46 different mammalian species. (From ref. 17.)

for bringing about a change in velocity gain which would be nicely matched to the likely changes in head angular velocity to be expected from change in animal weight. It seems that evolution has brought about the development of a velocity transducing hydrodynamic sensing element, despite the fact that to do so has called for marked divergence from what Julian Huxley has termed "natural growth trends" in evolution. Evidently the velocity transducing characteristic has proved highly influential in natural selection.

#### Neural Considerations

Of course, a knowledge of mechanical behavior of the canal does not necessarily tell us the characteristics of the neural signal received by the brainstem. However, Groen, Löwenstein, and Vendrik (ref. 9) have shown in the Ray fish that at least some of the primary afferent neurons from the canals contain frequency modulated signals which bear a virtually direct relation to head angular velocity during sinusoidal head movement, within the frequency range of velocity transduction in the mechanical components. It is important to note that these authors emphasized the fact that in this investigation they knowingly discarded afferent neural units not showing this pattern of linearity. In one figure, reproduced here as figure 3, they show the response of one nonlinear unit (curve I) together with both the response of an approximately linear unit (curve II) and the angular velocity of the canal (curve III). They suggest that the nonlinear pattern of response might be attributable to neural adaptation, a point which will be referred to again below. But whatever the cause, it is interesting to note that if the main frequency contents of this nonlinear curve are considered in a "push-pull" configuration, the response has very greatly improved linearity. Thus it seems that, considered in this way, the main neural input to the central nervous system probably does essentially represent the relevant component of head angular velocity, despite apparent non-linearity in some individual neural elements.

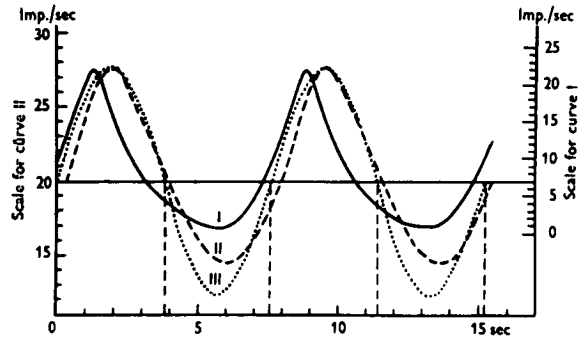


Figure 3.—Nerve impulse frequency derived from two primary afferent neural units recorded against time during sinusoidal oscillation of the canal. I. Distorted curve. II. Relatively undistorted response. III. Sincwave representing angular velocity of the canal. (From ref. 9.)

### THE OCULOMOTOR RESPONSE

#### Velocity Control

Such a velocity modulated input to the CNS is altogether appropriate for generating the patterns of oculomotor response required for stabilization of the image of the outside world on the retina of the eye. For accurate image fixation the angular velocity of the eye must, at every instant, be equal but opposite to the angular velocity of the head, both in magnitude and direction. Thus the angular velocity signal presented to the CNS by the semicircular canal can be envisaged as driving a velocity servo-system, the output of which is effected through the oculomotor nerves and the extrinsic eye muscles which they innervate. That this is effectively the case is supported by the fact that when a semicircular canal signal is generated by the method of post-rotational stimulation, the angular velocity of slow phase compensatory ocular nystagmus decays along an exponential time course corresponding broadly to that to be expected from the exponential pattern of elastic restoration of the cupula. It would be surprising indeed to find an alternative main cause for this pattern of decline in ocular angular velocity, when in these special circumstances the head is in practice stationary and the signal source has been generated by making use of the elastic restoration itself.

In this connection it is noteworthy that if the eye angular velocity is plotted against time elapsed after the stopping stimulus on a log-linear plot as in figure 4, then backwards extrapolation of the straight line relationship obtained usually intersects the ordinate at an angular velocity well below the original angular velocity of stimulus. From this it might be inferred that in normal circumstances the velocity gain due to canal stimulation is substantially less than 1. Yet one can easily demonstrate that, at least when the frequency of stimulus is greater than about 1 cps, this is not the case. The reader has only to oscillate his head through small angles about a vertical axis at 2 to 3 cps with eyes open to observe that the image seen is quite clear; that is, it has been stabilized on the retina. That this is a valid argument can be simply verified using the "radial line disc" described by M. Jones and Drazin (ref. 2) when indication of significant image slip over the retina does not arise until a

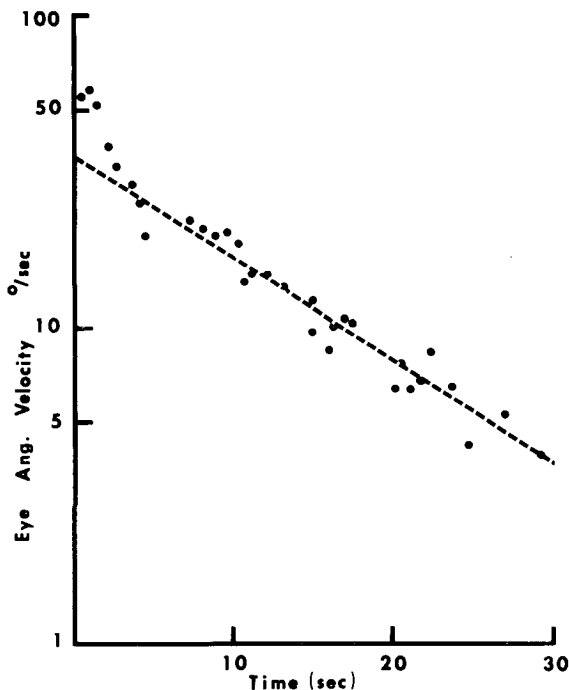


Figure 4.—Log-linear plot of compensatory eye velocity against time elapsed after suddenly imposing a post-rotational stimulus of 60°/sec. Note that initial points lie well above regression line.

frequency of 4–5 cps is achieved. Evidently, since these frequencies are well above the cutoff value of 1 cps for visual tracking, the vestibulo-ocular reflex must be virtually operating on its own at a gain of 1.

A clue to this apparent discrepancy is perhaps to be found in the upward trend of *initial* points in post-rotational log-linear plots of compensatory eye angular velocity such as figure 4. In this figure the initial eye angular velocities lie substantially above (note the ordinate is a logarithmic scale) the regression line drawn through the main body of points, and this is not infrequently the case when predominance of the anticompany response (ref. 10) (referred to below) does not mask the early compensatory one. If, as suggested by the nonlinear curve of neural response in figure 3, neural adaptation occurs in some units, then an early decline in eye angular velocity after post rotational stimulation of the canal might also be attributed to such adaptation.

In which case an overall picture emerges whereby at frequencies sufficiently low to be within the scope of the visual tracking system, the vestibular contribution to visual fixation is manifest as a velocity servo acting with a gain less than 1. But when the frequency of head movement rises above the visual tracking cutoff value, the additional effect of bringing adaptive units into play boosts the gain until eventually it reaches unity with considerable precision. If this interpretation is correct, then it seems that in reality the gain, and in effect with it the frequency responses in the vestibulo-ocular velocity servo-system, are dependent on the type of movement incurred. The vestibulo-ocular reflex system is thus seen to act in the sense that as the vestibular contribution to fixation of the retinal image becomes progressively more necessary, so the dynamic response of the system becomes progressively more effective.

#### Optimization of Control

Recent experiments have indicated that there are other effects contributing to optimization of the available nerve-muscle dy-

namics. When the head is oscillated in a rotational sense about a vertical axis, DC recording of the eye movement relative to the skull reveals a slow waveform of eye movement superimposed on the customary nystagmoid pattern of compensation (from "Evidence for a Noncompensatory Vestibulo-Ocular Reflex by G. Melvill Jones and S. Mishkin, in press). The waveform is evident both with eyes open and eyes closed, and when averaged appears to adopt a rather stereotyped pattern summarized in figure 5. This figure gives the averaged eye position relative to the skull (intermittent line) plotted simultaneously with skull position and shows that the former is  $90^\circ$  phase advanced with respect to the latter. Note that, for reasons which need not be here considered, the polarity of the two records has been reversed in this figure. When the results from a number of different subjects exposed to a range of different frequencies and amplitudes of head movement were normalized, it transpired that the overall averaged eye position relative to the skull executed a sine-wave of motion which could be very closely superimposed on the sine wave defining the angular velocity of the head.

One might imagine that this relationship would easily be subject to voluntary overlay. But surprisingly, it is remarkably difficult to

force oneself to look in the lagging quadrant during oscillatory head movement when this approaches a frequency of 1 cps. It seems that the eye is forced to perform its nystagmoid pattern of compensation in a direction of gaze which leads that of the head. Two interpretations are given by these authors to the functional role of this phenomenon. First, it is obviously relevant to cause the eyes to lead the head when the head is quickly turned towards an intended target, thereby advancing the moment at which the intended target is seen. Indeed, when the head is twisted rapidly to one side the eye is violently thrown out in the same direction by a large saccadic movement (ref. 10).

But a second, perhaps more fundamental interpretation, is given, namely, that the  $90^\circ$  phase advancement ensures that the compensatory eye muscles will be placed in a degree of stretch which is directly related to the velocity at which they will have to contract for the next image stabilization movement. One can imagine that the dynamic response of the overall velocity control system would be automatically optimized by this procedure to meet the requirements of the movement. More recent experiments (G. Melvill Jones and J. Rubin: "Dependence of Compensatory Vestibulo-Ocular Response Upon the Position of the Eye Relative to the

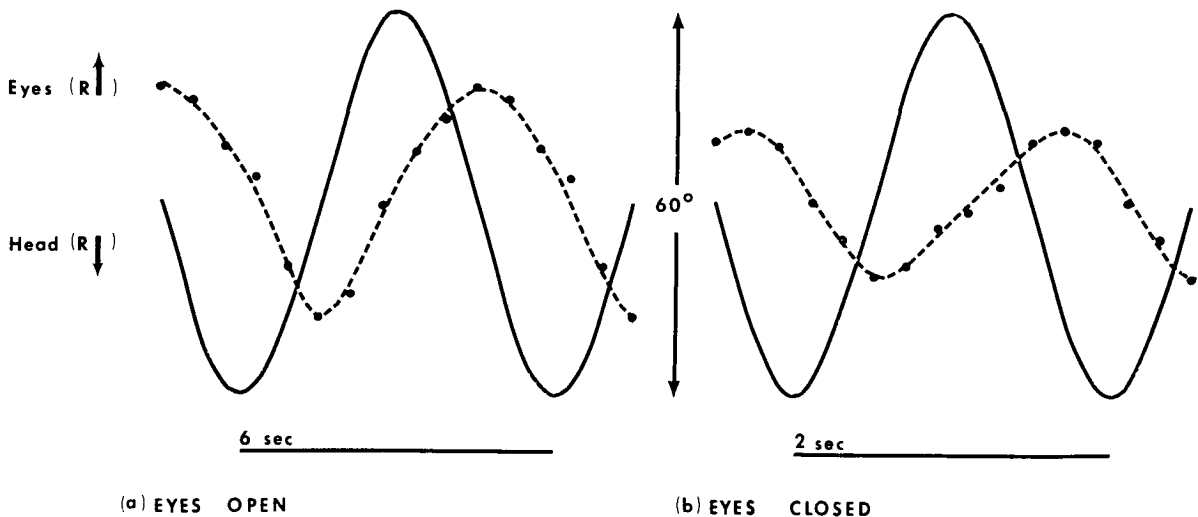


Figure 5.—Averaged curve (from 10 cycles) of eye displacement relative to the skull (intermittent line) plotted simultaneously with skull displacement.

Skull," in preparation) show that the velocity gain is indeed dependent upon the position of the eye in the skull.

The fact that the superimposed slow waveform of eye position is in phase with the angular velocity of the head suggests that a velocity modulated source of information may be driving it. From earlier considerations the semicircular canal evidently provides just such a source of information, and it is tempting to assume that the semicircular canal signal is responsible for the observed effect. Additional support is given to this interpretation by the fact already referred to that, when the head is suddenly turned, the *initial* response of the oculomotor system is to throw the eyes out in the leading direction, often before significant compensatory movement has occurred. In this case, with the head fixed to the body, it is difficult to postulate any other source of information than the vestibular organs. As an outcome of these and other observations, Melvill Jones and Mishkin suggest that the vestibular signal, in addition to generating the compensatory angular velocity of eye movement, also generates an output mediated through the saccadic (anticompensatory) mode of eye movement, which on the average takes the eye muscles to their most appropriate lengths for execution of the ensuing controlled compensatory angular velocity.

As a result of more recent experiments (G. Melvill Jones and N. Sugie: "Dependence of Compensatory Vestibulo-Ocular Response Upon the Preceding Saccadic Flick During Nystagmus in the Cat," in preparation), additional significance is attached to this conclusion, since it now appears that each saccadic movement itself contributes an important component to the ensuing slow phase compensatory sweep. In which case it appears that the vestibular signal exercises its stabilizing influence through operation on both the slow and the quick phase components of nystagmus.

#### Dependence of Vestibulo-Ocular Control Upon the Axis of Turn

The optimizing processes discussed above apply generally to the vestibulo-oculomotor

system. But for two main reasons it now appears that there is substantial dependence of response dynamics upon the axis of turn, referred to the skull. First, the results of Melvill Jones, Barry, and Kowalsky (ref. 11) have shown considerably greater rates of what they termed "effective cupular restoration" during rotation in the vertical planes (time constant approx. 5 sec) than in the horizontal plane (t.c. approx. 15 sec). Functionally this implies that the system cannot tolerate such low frequencies of head movement in these planes as in the horizontal plane. This state of affairs is nicely matched to our everyday life, in which much lower frequencies of head movement are normally experienced in the horizontal than the vertical planes since the whole body can turn about the vertical axis. But for reasons discussed later it is not so nicely matched to the special circumstances of flight.

Second, in the context of visual image stabilization, rotation about the roll, or visual, axis is unique, on account of the relatively poor visual tracking capability associated with rotation about this axis (ref. 12). In normal life this deficiency presumably does not signify, owing to the short sharp character of usual head movements in the roll plane leading to the vestibulo-ocular reflex acting in its high gain mode of operation. Moreover, the rate of circumferential image slip over the foveal retina is much lower for a given angular velocity in this plane, than in those of yaw and pitch. However, as will emerge below, the phenomenon has important implications in the flight environment.

#### APPLIED CONSIDERATIONS

All the effects described above, and presumably many more not yet known, appear to be aimed at matching the physiology of the vestibulo-ocular responses to the requirements of natural life. But, not unnaturally, when man chooses suddenly to expose himself to new environmental conditions of motion, it turns out that the physiological mechanisms concerned are not so nicely matched to the new patterns of movement.



In three-dimensional space the natural constraints to rotational movement are removed, and the velocities incurred, the duration of the transients, and the range of frequency of movement may all extend well beyond the natural limits. From a knowledge of the basic physiological processes concerned it is often possible to infer the causal factors in some of the consequences which arise.

First, as is well known, the effect of cupular restoration quickly becomes significant when turning movements are prolonged beyond about 3 seconds, as is frequently the case when turning in a conventional aircraft. In this connection it is important to bear in mind that the rate of intrusion of such error is greater for the vertical planes than the horizontal. The significance of this in conventional aviation lies in the fact that nearly all turning movements are constrained by the aerodynamics of the aeroplane to take place in the vertical planes of pitch and roll. Thus the shorter time constant of "effective cupula return" attributed to the vertical planes holds the pilot of an aircraft at a disadvantage which is not evident in natural life. Numerical examples of the penalty occurred are given in reference (11).

Again, what has been described as the optimizing process whereby the eye is, on the average, moved out in a leading direction relative to the head movement, tends to bring about a highly undesirable effect when transients are large. The leading, or anti-compensatory drive apparently predominates when the initial stimulus is large, to the extent that it may even abolish, temporarily, the useful compensatory response (ref. 10).

Again, the virtual absence of visual tracking in the roll plane can lead to an oculomotor response in this plane which follows the erroneous vestibular drive associated with prolonged rotation, unopposed by an effective visual fixation reflex. For example, during an aerodynamic spin, the pilot may fail completely to achieve image stabilization on the retina, with the imposition of difficulties both in establishing adequate visual acuity and in interpretation of the direction of such relative movement as is observed.

These latter observations may be conveniently summarized by reference to records from these experiments (Melvill Jones: "Human Factors in Spinning Aircraft," in preparation). Figure 6 represents the collected data from one pilot while he was performing an eight-turn spin in a Vampire T-11 jet trainer aircraft. The continuous line gives the angular velocity of eye movement required for image stabilization in each plane, the intermittent line giving the actual angular velocities of compensatory eye movement achieved, measured parallel to these planes. Even in the horizontal plane, image slip occurs after the vestibular signal has virtually decayed to zero (16 sec). In the roll plane the decay in compensatory eye angular velocity is much more rapid, presumably being attributable to both the increased rate of "effective cupular restoration" and to the virtual absence of visual tracking in the roll plane. At the point of recovery, the reversed vestibular drive is particularly effective in generating a reversed eye movement in the roll plane, since the oculomotor response goes largely unopposed by an effective visual fixation reflex. Lastly, when the stimulus is large, that is, on approach to the angular velocity peaks of stimulus (continuous line), predominance of the anti-compensatory response occurs, leading to temporary but often substantial, interference with the generation of the useful compensatory mode of eye movement.

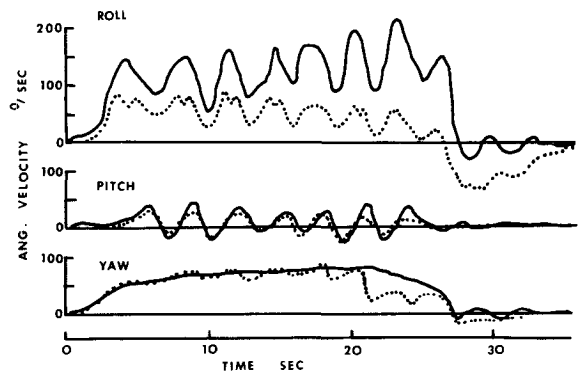


Figure 6.—Comparison of required angular velocities of eye movement for retinal image stabilization (continuous lines) with those actually achieved during an 8-turn aerodynamic spin.

## REFERENCES

1. MILLER, J. W.: Study of Visual Acuity During the Ocular Pursuit of Moving Test Objects. *J. Opt. Soc. Amer.*, 48, 1958, p. 803.
2. JONES, G. MELVILL; AND DRAZIN, D.: Oscillatory Motion in Flight. Proc. of the 5th European Congress of Aviation Medicine, London, August (1960). Also in "Human Problems of Supersonic and Hypersonic Flight" (A. B. Barbour and H. E. Whittingham, ed.). Pergamon Press (London), 1962, pp. 134-151.
3. YOUNG, L.: A Sampled Data Model for Tracking Eye Movements. Sc.D. Thesis, Instrumentation, M.I.T., June 1962.
4. CORREIA, M. J.; AND GUEDRY, F. E.: Influence of Labyrinth Orientation Relative to Gravity on Responses Elicited by Stimulation of the Horizontal Semicircular Canals. NSAM-905. NASA Order No. R-93. Naval School of Aviation Medicine, 1964.
5. MAYNE, R.: The Dynamic Characteristics of the Semicircular Canals. *J. Comp. & Physiol. Psychol.*, 43, 1950, p. 309.
6. NIVEN, J. I.; AND HIXSON, W. C.: Frequency Response of the Human Semicircular Canals: I. Steady-State Ocular Nystagmus Response to High-level Sinusoidal Angular Rotations. NSAM-459. NASA Order No. R-1. Naval School of Aviation Medicine, 1961.
7. JONES, G. MELVILL; AND SPELLS, K. E.: A Theoretical and Comparative Study of the Functional Dependence of the Semicircular Canal upon its Physical Dimensions. *Proc. Roy. Soc. (B)*, 157, 1963, p. 403.
8. VAN EGMOND, A. A. J.; GROEN, J. J.; AND JONGKEES, L. B. W.: The Mechanics of the Semicircular Canal. *J. Physiol.*, 110, 1949, p. 1.
9. GROEN, J. J.; LOEWENSTEIN, O.; AND VENDRIK, A. J. H.: The Mechanical Analysis of the Responses From the End-Organs of the Horizontal Semicircular Canal in the Isolated Elasmobranch Labyrinth. *J. Physiol.*, 117, 1952, p. 329.
10. JONES, G. MELVILL: Predominance of Anti-Compensatory Oculomotor Response During Rapid Head Rotation. *Aerospace Medicine*, 35, 1964, p. 995.
11. JONES, G. MELVILL; BARRY, W.; AND KOWALSKY, N.: Dynamics of the Semicircular Canals Compared in Yaw, Pitch and Roll. *Aerospace Medicine*, 35, 1964, p. 984.
12. JONES, G. MELVILL: Oculomotor Responses to Simultaneous Rotational Stimuli About Two Orthogonal Axes. Proceedings of the XXII International Congress of Physiological Sciences, Leiden, 1962, p. 1017.

## DISCUSSION

**FIELDS:** I would like to just make a plea for one other thing, Dr. Jones, and that is a consideration of certain other mechanisms in this reflex arc, mainly cerebellar. I think, certainly, when one talks of the vestibular ocular reflex, one must also assume that there is a vestibulo-cerebellar-ocular reflex which brings into play proprioceptive mechanisms in the muscles of the neck, for instance. I came across, fortuitously, a phenomenon in a patient, a boy 7 years old, that led me to suspect that we had some clue as to where this mechanism is through the cerebellar circuit. This boy had a very peculiar posture, walked in a sort of anthropoid position without the usual associated movements of his arms. He bent forward, head forward, and, when seated, if you would get him to turn his head to follow, he would have something I have only seen with this lateral bending, namely the rolling movement. But on moving his head in the horizontal plane to the right, he had an extreme clockwise movement and to the left, a counter-clockwise movement. I have an excellent color film strip of this, and the thing that really clinched it was an air study we did which showed a deformity that the radiologist interpreted as being due to a cyst in the posterior fossa, and exploration showed us the congenital defect. There was a complete absence of the vermis of the cerebellum and no roof nuclei. So, I just wonder whether it is not a circuit through the ancient part of the

cerebellum and the roof nuclei that may play a role in this as well.

**JONES:** A very good point.

**FIELDS:** I would be glad to send you this film if you would like.

**JONES:** Thank you very much. I'd like to see it. I too am aware of the importance of the cerebellum in this context. In our experiments on cats we have taken the precaution of interfering as little as possible with the functional integrity of the cerebellum. Any microelectrode work undertaken by us at present leaves the cerebellum intact, our approach to the brain stem being through a small hole in the skull employing stereotaxic penetration with a long, rigid microelectrode.

**WENDT:** I would like to make two comments. One is that, according to my records and those of other investigators, in ocular compensation during short arcs of rotation, such as is characteristic in normal life, there is only about two-thirds the displacement of that of the body. If you add a visual factor to it, the compensation rises to about 0.8 to 0.9 but is still not perfect. So it would appear that the visual system can tolerate a certain amount of slippage on the retina without losing clarity. As a matter of fact, you can't imagine how optokinetic nystagmus could continue unless this were a fact. In optokinetic nystagmus there is a certain amount of after discharge. This accounts for the baseline, but you have to have

continued input. And the only way you can get that continued input is by allowing slippage on the retina. In other words, by allowing movement of the retinal images across the retina.

The other point that you made was that, when you shake your head from side to side, you get perfect fixation. This may well be, but there is another factor which comes in here which is very important. That is, that the same impulses that operate the neck muscles also start the eye muscles. They start them without latency. This is not a vestibulo-ocular reflex. This is a compensatory movement which is controlled by the same neural controls that are at that moment controlling your neck muscles. So that particular illustration is not relevant to the argument.

JONES: No doubt proprioceptive information from the neck contributes when present; but the phenomenon referred to occurs when the body is turned with the head. With regard to your second point. The rather short time suggested for the vestibulo-ocular response was obtained by measuring eye movement following a tap with a light hammer to a lightweight metallic bar held in a dental bite.

MAYO: Dr. Jones, have you applied this very beautiful quantitative approach to resolving emotional versus objective character of inside versus outside controversy with two inadequate instruments; for example, gyro horizon versus low wing aircraft, both of which give insufficient information but have produced violent counter information?

JONES: Yes. If you stick a little radial lined disc onto the cockpit turning of an airplane, you have a lot of information from a very simple measuring device. We set up experiments where the pilot oscillated the aircraft in a roll at different frequencies, and he had an instrument to tell him when he was right and when he was wrong, and when it was a properly controlled experiment. We had him do a number of tests like calculations on a logarithmic navigational computer where you have nonlinear scales and where they couldn't interpolate or extrapolate. We also asked him to perform various visual acuity tests while at the same time giving the roughest estimate and how much slip there was compared to what was happening to the airplane.

CRAWFORD: On this subject of retinal slips. We did some work a few years ago in which we wondered just how much slip could occur. So we deliberately introduced into our system position errors and looked at visual acuity; then we introduced velocity errors and looked at visual acuity. We found that the visual acuity would go down when you were 1° off. It would also go down if you were 1° per second off. If you compounded both of these and had a velocity error and a position error simultaneously, you went blank if you did not have good acuity. I wonder, as I always do when I hear Melvill Jones, that he tends

to treat the whole body as a thing. If you are doing a visual acuity study of some extent and someone stands on your toe, you don't see very well. But if you look at the whole body response, and looking at eye movement and simultaneous head movement in pursuit of a moving target, we find electromyographic activity building up in neck muscles some 50 to 75 milliseconds before the eyes start moving.

JONES: I am glad you mentioned the significance of neck muscle actuation. I feel sure these muscles play an important role in stabilization of the retinal image, both in response to optokinetic and vestibular stimuli.

GUEDRY: You have just answered part of what I was going to ask, but I wondered if, in passive movements at the high frequencies, the one-to-one following occurs immediately or if there isn't some delay before the one-to-one following comes in.

JONES: I don't know. We did not examine the transients. But I think they are very important and may give a clue as to the way in which visual tracking capability improves with increasing predictability of target movement.

WENZEL: I would like to tell you about another experiment we did. I am an engineer and I would like to get your comments on this and perhaps get some explanation on just what happened here. With a subject seated on a Bárány chair, we put a cardboard box over him so that he had the same sensation that he had in the centrifuge, and he had the same experiences. He received the Coriolis stimulation and got sick after three or four head motions and began to get pallid. We found that, without this cover on him, he did not experience any stimulation or get sick. Therefore, we thought we might simulate the uncovered condition by putting a light down through a hole right on the axis of the rotation and putting a paint can over the light bulb with three holes drilled in the paint can 120° apart. The subject would follow these spots of light as the chair rotated around this paint can. We found that he could nod and tilt his head and not get sick at all; this was at 12 rpm. The amazing thing here is that, if we should pull out the plug and turn the lights off, the sickness response did not return; he stayed in this so-called neutralized state.

JONES: Can you tell me again what the spots were doing? Were the spots stationary relative to space?

WENZEL: In inertia they were stationary.

JONES: I think here that we have something akin to the case of the helicopter simulator in which the pilot remains still but the visual scene moves. Unaccustomed subjects are relatively unaffected, but helicopter pilots who have become accustomed to certain relations between vestibular and visual cues while flying a real helicopter tend to become nauseated, sometimes to the point of vomiting very quickly in the simulator.

***SESSION III***

***Chairman: JOHN BILLINGHAM***  
***Manned Spacecraft Center, NASA***

***Cochairman: RANDALL CHAMBERS***  
***U.S. Naval Air Development Center***

Perhaps before this session begins it may be of interest to describe briefly the work that we are trying to do at NASA's Manned Spacecraft Center in connection with vestibular physiology.

Most of the jobs we have at the Center are applied. They consist of laying down the physiological requirements for space vehicle design and for mission planning (its goals being crew safety and mission success) with respect to the stresses on the body; and of course one of these stresses is that imposed on the vestibular system. We have another job, which is to assist in the design of equipment which will tend to protect the man against stresses which become unduly severe. Another job we have is to mount, on Gemini and Apollo missions, medical and physiological in-flight experiments.

First, I would like to agree with the statement that Dr. Lansberg made that it is impossible to extrapolate directly from any vehicle you can build on Earth (any rotating type of vehicle) to a rotating space vehicle. The forces involved are always different; I think the approach should be to learn the fundamental details of the mechanisms from all the experimental work that is going on, and then put these back together and predict what will happen in the space environment. That's the situation at this stage. Later on, of course, there may be opportunities for actually carrying out rotation in the real situation.

Mr. Johnston has referred to some of the problems and programs in the Crew Systems Division of the Manned Spacecraft Center, and, in passing, to medical experiments. I would like now just to summarize the experiments we have planned for the Gemini and Apollo missions in the vestibular area. I am not going into any detail because, of course, I can't give any results at this stage.

The experimental programs, in the Gemini and Apollo missions, are run by the NASA Headquarters, Office of Manned Space Flight, and we at the Manned Spacecraft Center carry them out in conjunction with the principal scientific investigator. In the case of the vestibular experiments for the fifth Gemini flight, which is a 7-day one, and for the seventh Gemini flight, which is probably a 14-day one, we plan to do an experiment to measure the effects of weightlessness on the otolith organ. In the Apollo missions we plan to develop a much more sophisticated experimental approach which would include, once again, a reexamination of the effects of weightlessness on the otolith, but now include also some experiments concerned with interaction between otolith and semicircular canals, and experiments on semicircular canal function that we feel will be most relevant to any possible future design criteria for rotating space stations. These experiments would involve rotating the Apollo vehicle in Earth orbit and then performing the appropriate maneuvers.

We recognize that the space environment is an important laboratory in which to do space vestibular research. It is an awfully expensive one, and this is something which is not often apparent to people who are not actually tied into the operational aspects of space missions. It requires an enormous organization, and the logistic problems are almost overwhelming. The further we get down the road and have the space and experience to run experiments, the easier these problems will become, but at the moment the Gemini and Apollo missions are primarily exploratory. Scientific investigations are part of them, but not as large a part as they will be in the later manned orbiting research laboratories. The final point is that a considerable benefit comes, I feel, in advances in understanding of basic physiologic mechanisms as a result of some of our practical problems and those of our associates in the vestibular physiology area, and it might perhaps be appropriate to describe some of these results as a scientific "spin-off."

JOHN BILLINGHAM

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# Radiosensitivity of the Vestibular Apparatus of the Rabbit

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AND

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## SUMMARY

16/20

- (1) A method of radiating the inner ear of the rabbit with an alpha particle beam without significant exposure of the brain has been devised.
- (2) Long lasting changes in semicircular canal function of the rabbit have been demonstrated with doses of 500 rads.
- (3) The approaches to determine the threshold of radiosensitivity of the semicircular canal function and the radiosensitivity of the macula utriculi are described.
- (4) Structural changes are discussed which may account for the functional alterations observed. Work being undertaken to study the structural changes is described.

Author

## INTRODUCTION

Experiments showing the labyrinth to be sensitive to the effects of ionizing radiation date back at least to 1905 when Ewald (ref. 1) showed that glass beads containing 3 mg of radium bromide, when placed in the vicinity of the labyrinth of the pigeon for a few hours, caused effects of removal of the labyrinth. Thielemann (ref. 2) made observations of the inner ear after X-ray exposure of mice. As confirmed by other later experiments perilymphatic hemorrhage was seen with higher doses, especially in the cochlea and about the ampullae. There have been studies of hearing in patients receiving Cobalt<sup>60</sup> gamma irradiation for tumors of the head and neck (ref. 3). These showed some decrease in the hearing threshold and some perceptive hearing loss with temporary recruitment attributed to transient vasculitis. With doses above 500 r, Kelemen (ref. 4) regularly found perilymphatic edema about the vestibular part of the inner ear of the rat. The cupula was resistant until doses of 1000 to 2000 r were reached, when disinte-

gration of the cupulae and cristae was seen. It was not possible from his studies to determine a precise threshold above which changes always occurred. Generally the cochlea showed more evidence of change than the cupula and crista. No recent references to studies of vestibular function following radiation in man have been found, except for those of Moskovskaya (ref. 5), who reported observations that he interpreted as indicating increased excitability of the vestibular apparatus. These observations consisted of shakiness in gait, dizziness, nausea on turning the head, pallor, tendency toward perspiration, reduction of blood pressure, and changes in post-rotational nystagmus. These symptoms were reduced in 2-3 weeks but persisted and were still present after 5 years. The increased excitability of the vestibular apparatus was thought to be due to weakening of the inhibitory action of the cerebral cortex. The total tumor radiation dose given was 5000 to 12000 r.

Sveshnikov (ref. 6) made a study of the effects of proton beams and Co<sup>60</sup> gamma rays

on the function of the labyrinth of dogs. Six dogs were irradiated with the proton beam (apparently whole body) and 10 with a single dose or fractionated doses of  $\text{Co}^{60}$   $\gamma$ -irradiation. The testing for vestibular function was done by rotational methods with recording of vestibulo-somatic and autonomic reflexes. In addition caloric tests were used. More details about these experiments would be of interest. Sveshnikov interpreted the findings as showing a diminution of the threshold and reactivity of the vestibular apparatus. With the proton beam (510 MeV) the changes were pronounced with doses of 350 to 500 rads only at the climax of radiation sickness. Somewhat similar effects were observed with  $\gamma$ -radiation in doses of 200 r, 350 r, and 500 r, more marked effects being seen with single exposures.

The preceding literature review is not intended to be complete but is given to show the previous approaches to the study of the sensitivity of the vestibular apparatus to ionizing radiation. The review of papers in the past 4 years is complete with regard to the vestibular apparatus as distinct from the cochlea. In all of the previous studies the radiation dose received by the vestibular apparatus has been poorly defined as in the radium studies where no attempt was made to determine a dose effect relationship, or the radiation was given as whole body or whole head irradiation. In the latter case it was never certain whether the effects were due to direct effects on the vestibular apparatus or due to abscopal effects and radiation sickness.

Knowledge of the radiosensitivity of the vestibular apparatus is needed so that appropriate precautions may be taken in the therapeutic irradiation of the head and neck and so that the risk of damage to the vestibular apparatus of man in space travel may be estimated. In addition there is a need for knowledge of the effect of other stressful conditions, such as high temperatures, high and low G forces and general status of nutrition and hydration, on the radiosensitivity of the vestibular apparatus.

Our ultimate interest is in the effects of radiation on the vestibular apparatus of man. It would be less hazardous to project results of animal experiments to man if the study is limited to only the vestibular apparatus. Since the morphology of the vestibular apparatus in man is nearly identical to that of mammalian species in general, the effects in the experimental animal should qualitatively be like those in man. Previous studies have failed to limit the radiation to the inner ear and have also failed to explore the radiosensitivity of the vestibular apparatus down to the threshold levels both for functional and ultrastructural damage.

The present study was undertaken to determine the sensitivity of the rabbit semi-circular canal system to high energy alpha particle beam irradiation limited to the inner ear. These initial studies are restricted to testing the effect of such radiation on post-rotational nystagmus. Morphological studies of the crista ampularis are to be made now that methods of irradiation and functional testing have been established. Later studies are planned to study the radiosensitivity of the otolith organ of the utricle.

#### MATERIALS AND METHODS

Male New Zealand white rabbits weighing between 1 and 2 kg (7-10 weeks old) were used throughout this study. In order to localize the labyrinth in radiographs, a 1.5 kg animal was heparinized, anesthetized with pentobarbitol, and the head and upper extremities of the animal were perfused for about 30 minutes with neutral buffered formalin. The head was then removed and frozen in liquid nitrogen. The head in the frozen state was sawed into serial frontal sections through the vicinity of the inner ear. Each section was 0.5 to 0.7 cm thick. The cochleae and semicircular canals were located in these sections and fragments of lead were placed in both cochleae. The semicircular canals were located 2 mm behind and 1 mm above the location of the lead fragments. The sectioned head was allowed to thaw and the sections were all taped together and radiographs taken of the head

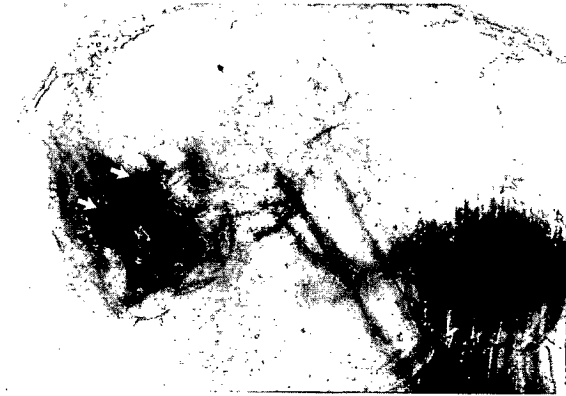


Figure 1.—Radiograph of reconstructed rabbit head showing fragments of lead in cochlea bilaterally. This is a slightly oblique lateral view. Tips of the arrows are in semicircular canals.  $\times 4$ .

as shown in figure 1. The beam of the cyclotron was directed through the points indicated by the arrows.

Radiations were done with a 910 MeV alpha particle beam produced in the 184-inch cyclotron at Berkeley. A  $\frac{3}{8}$  inch beam-defining aperture was used. The beam was passed through the labyrinth at an angle of  $40^\circ$  anterior and  $20^\circ$  superior to the transverse line joining the two labyrinths. At this angle the cerebellum and medulla are not included in the radiation field. No rotational techniques were used in the irradiation, and irradiation was done only along the plateau portion of the Bragg curve. The dosimetry methods were similar to those described by Birge, Anger, and Tobias (ref. 7). Unilateral right sided doses of 475, 998, and 1512 rads were given to different animals. Bilateral doses of 500 rads were given to two animals.

Post rotational nystagmus was tested using the apparatus shown in figure 2. Except for experimental animals number 41 and 42, all rotations of the experimental group were counter-clockwise. In earlier groups of animals, rotation rates were 40, 20 and 15 seconds for 10 revolutions. In later rotations which include the last two graphed rotations of animals number 3, 4, 5, 41, 42, 118, and 120, somewhat slower rotation rates were included. All rotation rates are given

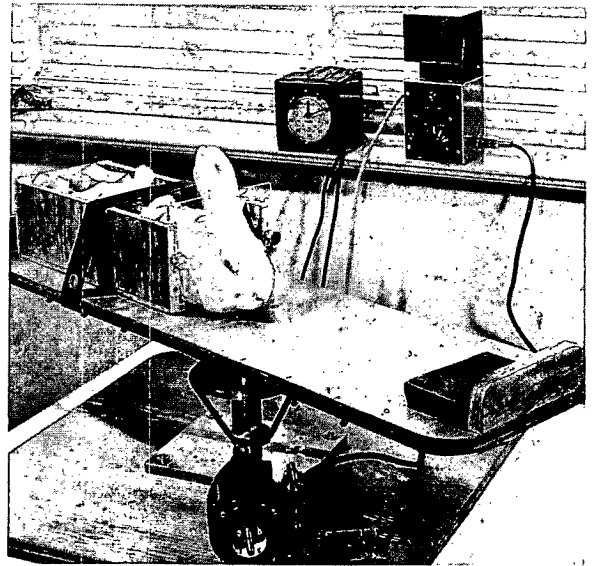


Figure 2.—Animal rotating device. Reversing switch and variable speed rheostat are located to right of timer.

as reciprocals in seconds per 10 revolutions. The animals were rotated until all nystagmus stopped and then rotation was stopped by turning off the electric power and catching the rotor of the device with one hand. The time from stopping of the rotation to the last sweep of the slow component of the nystagmus was taken as the duration of the nystagmus. Seven control animals were tested every one or two weeks for periods ranging from 3 to 5 weeks (see figs. 3 and 4). Irradiated animals were tested at 1 or 2 days to 1 week following irradiation and then generally at weekly intervals (fig. 5).

### RESULTS

The results of rotation of control animals are shown graphically in figures 3 and 4. Rotations in figure 4 were done in both directions and only maximum durations of nystagmus are shown regardless of the direction of rotation. In figure 5 are plotted the results of the experimental animals. Here only the last rotation of animals numbered 3, 4, and 5 was in both directions and again only the maximum duration of nystagmus is recorded. For animals number 41 and 42 only the pre-irradiation testing was in one



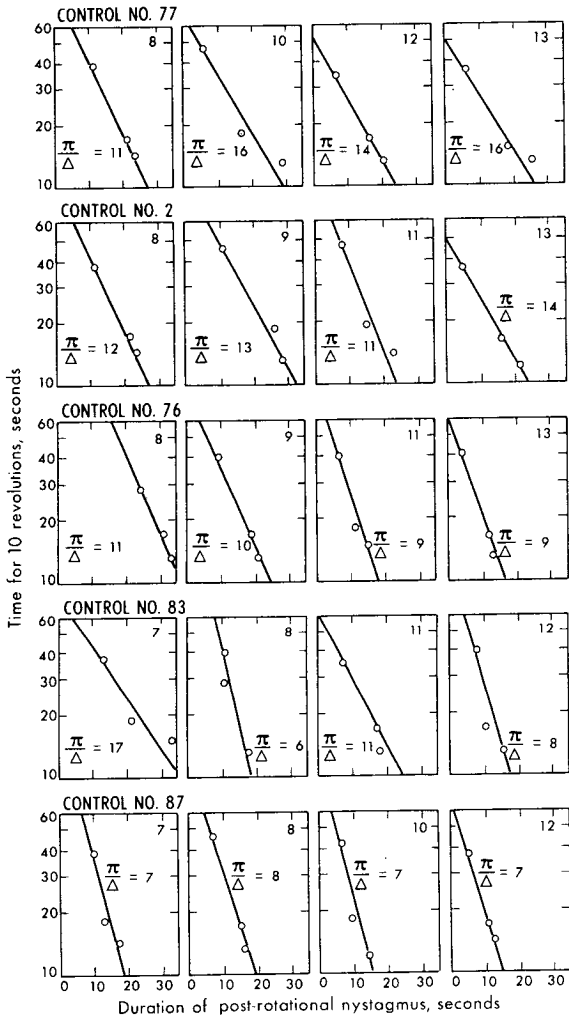


Figure 3.—All rotations were counterclockwise in this group of control animals.  $\pi/\Delta$  is a factor of reciprocal of slope of each curve. Number in upper right corner of each graph is the age of the animal in weeks.

direction only (counter-clockwise), and all other rotations were in both directions, again only the maximum duration of nystagmus being shown in the graph.

DISCUSSION

In these studies, we have found the highly damped torsion pendulum model of function of the semicircular canal to be applicable. In this model as proposed by Van Egmond et al. (ref. 8) the crista and cupula act as a tight swinging door (fig. 6) which prevents any actual flow of the endolymph, at least

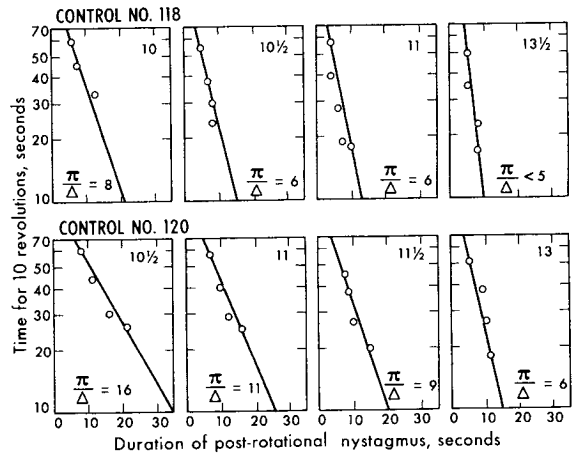


Figure 4.—All rotations were done in both directions in this group of control animals. Maximum duration of nystagmus is that plotted in each instance, regardless of direction of rotation. Number in upper right corner of each graph is age of animal in weeks.

if the system is functioning at its most efficient levels. Angular acceleration produces a deflection of the crista and cupula by the rotational shift of the mass of the fluid of the endolymph in the semicircular canal. The restoring force is in the elasticity (and possibly smooth muscle tonus) of the crista and cupula.

According to Van Egmond (ref. 8) the differential equation of a torsion pendulum as applied for a model of the function of the semicircular canal may be given as follows:

$$\theta \ddot{\xi} + \pi \dot{\xi} + \Delta \xi = 0$$

where the terms are defined as:

- $\theta$  moment of inertia of endolymph
- $\pi$  moment of friction at unit angular velocity
- $\Delta$  directional momentum at unit angle caused by cupula
- $\xi$  angular deviation of endolymph in relation to the skull
- $\dot{\xi}$  1st derivative of  $\xi$  with respect to time, i.e., angular velocity of the endolymph
- $\ddot{\xi}$  2nd derivative of  $\xi$ , i.e., angular acceleration of endolymph

All angles are taken at the center of the semicircular canal. The approximate solu-

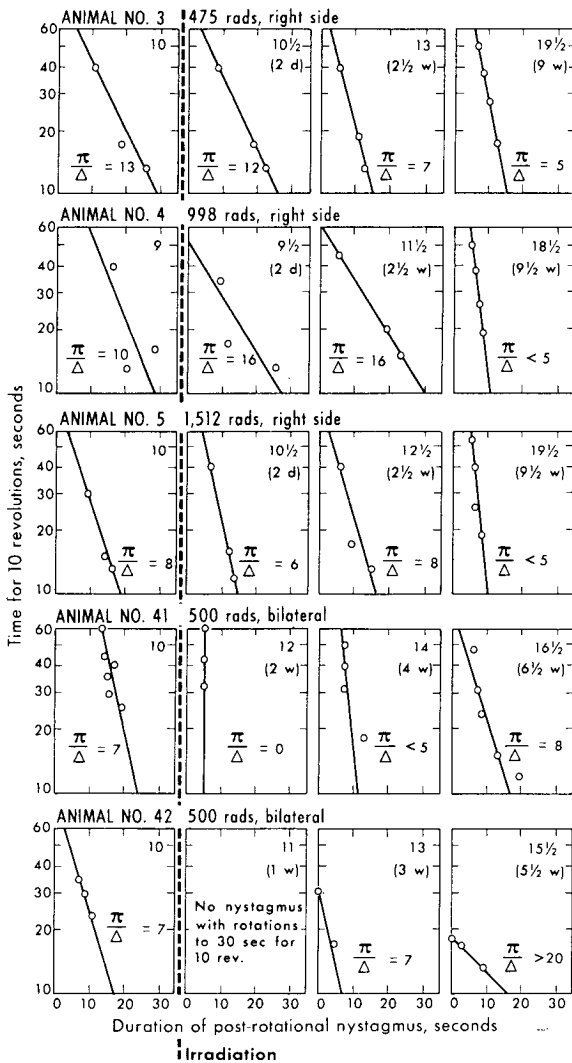


Figure 5.—Irradiated animals with doses as indicated. Rotations were counterclockwise for animals number 3, 4, and 5, except for last rotation where rotations were in both directions. For animals number 41 and 42, only pre-irradiation testing was in one direction. The other testing was with rotations in both directions. Only maximum duration of nystagmus is recorded, regardless of rotation direction. Number in upper right corner of each graph is age of animal in weeks. Number in parentheses is the time post-irradiation in days (d) or weeks (w).

tion of this second order linear differential equation as given by Van Egmond is:

$$\xi = \gamma \frac{\theta}{\pi} (e^{-\Delta t/\pi} - e^{-\pi t/\theta})$$

where  $\gamma = \xi$ , the angular velocity of the en-

dolymph, and with the limiting conditions of  $\xi = 0$  when  $t = 0$ .

The impulse given to the endolymph when rotation is stopped is equivalent to some factor of the rotation rate. At some minimum deviation of the endolymph ( $\xi_{min}$ ) nystagmus will stop.

Since the moment of friction for the endolymph  $\pi$  is fairly large, the moment of inertia  $\theta$  is small and  $t$  is near a maximum, the term  $e^{-\pi t/\theta}$  will be very small at  $\xi_{min}$ , and may be neglected. By taking logarithms, rearranging the terms of the equation and lumping all constant terms into a single constant  $K$ , the equation becomes:

$$\log \gamma = -K + \frac{\Delta}{\pi} t$$

$$\log \frac{1}{\gamma} = K - \frac{\Delta}{\pi} t$$

$$\log R = -K - \frac{\Delta}{\pi} t$$

where  $R =$  seconds per 10 revolutions.

The constant  $\pi/\Delta$  for man is, from Van Egmond's work (ref. 8), about 8 seconds. This was determined in man using the subjective sensation of rotation as the end point for  $\xi_{min}$ . The average for the testing of the seven control rabbits shown is 11 seconds. This is a little higher than the value for man (different end point used), but much lower than the value found by Groen et al. for the elasmobranch fish (*Raja clavata*) (ref. 9). As can be seen from figure 5, both animals receiving bilateral radiation of 500 rads to the labyrinth have a marked reduction in  $\pi/\Delta$  (i.e., the slope is markedly increased as the data are plotted). In addition there is a shift of the curves to the left following irradiation (i.e., the duration of nystagmus has decreased for all rates of rotation). However, in the case of animal number 42, the last testing 5½ weeks following irradiation shows  $\pi/\Delta > 20$ . The large value here is due to testing error when the duration of nystagmus is less than 5 seconds and we think that actually  $\pi/\Delta$  decreased. The decrease in  $\pi/\Delta$  can also be seen in the animal (number 5) receiving 1512 rads to the right labyrinth only.



Figure 6.—Section of normal vestibule showing ampulla (a) with crista (c), utricle (u), perilymphatic space (p), nerve to ampulla (n), vestibular ganglion (g) in internal acoustic meatus and petrous portion of temporal bone (b). Cupula of crista has been lost in this preparation. Stubs of cilia appear as irregular serrations on convex surface of crista. Semicircular canal joins ampulla at left margin of photomicrograph. Neutral buffered formalin perfusion fixation, decalcified with EDTA and stained with H and E.  $\times 110$ .

We recognize that if  $\pi$ , the moment of friction, is greatly reduced as a result of irradiation, then the approximation used above for evaluating the constants is not valid, since  $e^{-\pi/10}$  might become comparable to  $e^{-2/\pi}$ . The accuracy of observations however does not allow us at present to distinguish between various other possible models.

The zero intercept varies greatly among both experimental and control animals. Most values range between 30 and 51 deg/second (70 to 120 seconds per 10 revolutions). This is over 10 times greater than the value of 2.5 deg/second found for man by Van Eg-

mond. This difference probably reflects a much reduced sensitivity of the testing methods used in these rabbits. No effect of radiation on this intercept can be made out from the data. From the two bilaterally irradiated animals it appears that 500 rads are well above the threshold for an effect of particle radiation upon the function of the semicircular canal. More animals irradiated at lower doses will be needed to determine a precise threshold.

In further studies it is planned to determine the threshold of the radiosensitivity of the semicircular canal function and in addition to determine at what time follow-

ing irradiation the functional changes appear. During these further studies electro-nystagmography will be used, and it is planned to determine if ocular counter-rolling can be used satisfactorily as a test of macula utriculi function in the rabbit. If so, the radiosensitivity of this vestibular function will also be determined.

To make a comparison of particle and electromagnetic ionizing radiation, we also plan to irradiate the whole head of some animals with  $\text{Co}^{60}$   $\gamma$ -radiation at doses comparable to those of the particle irradiation. The animals will be tested for vestibular function to compare the biological effectiveness of the two types of ionizing radiation. In addition, the Bragg peak will be placed in the inner ear to compare the effect of very high LET radiation.

Exploration of the exact nature of the change in the labyrinth which produces the functional effect also remains to be done. Since the constant  $\pi/\Delta$  appears to be reduced with irradiation, it must be that either  $\pi$  (moment of friction) decreases or  $\Delta$  (directional momentum caused by cupula) increases or both. It is known that the protein content of the endolymph is higher than that of spinal fluid (ref. 10). Any dilution of the endolymph would lead to a decrease in viscosity and a decrease in  $\pi$  (moment of friction). Since radiation is known to increase the loss of fluid from the capillary bed, this might be a mechanism of the action, but this fluid escape is accompanied by large amounts of protein which would produce the reverse effect.

The perilymphatic edema with a dose of 500 r observed by Kelemen (ref. 4) would cause a decrease in the cross-sectional area of the semicircular canal with an increase in the surface relative to the cross-sectional area. This relative increase in surface would increase  $\pi$  (moment of friction) which is a reverse effect of what has been observed in the present experiments.

A change in the elasticity of the crista and the cupula with an increase in  $\Delta$  (directional momentum caused by the cupula)

seems to be a more likely cause of the functional change. Small changes in the degree of polymerization of ground substance mucopolysaccharides, smooth muscle fibrils and basement membrane proteins could have a profound effect on the elasticity of the crista and cupula.

On the other hand, it is possible that there is a decrease in the radial length of the cupula-crista structure with a loss of efficiency due to endolymph leaking over the cupula, effectively decreasing the moment of friction, but also decreasing the directional momentum ( $\Delta$ ) caused by the cupula, so that a change in  $\pi/\Delta$  can not be predicted.

A final possibility is that the effect might be directly on neural tissue such as the ciliated sensory cells, the nerve fibers or on Scarpa's ganglion which is also in the radiation field. Such a direct effect would not be in keeping, however, with the known effect of radiation on the function of other nerve tissue, although electroencephalographic changes have been observed with doses well below 500 rads.

A number of experimental studies could be undertaken to determine what the important structural alterations might be. At the present time only one such study is being planned and this is to examine the crista ampullaris and cupula with both the light and electron microscope. The problems involved in such studies are difficult because the delicate soft tissues of the vestibule are completely embedded in the temporal bone. Glutaraldehyde perfusion techniques which preserve the fine structure well have been worked out here, but suitable methods for embedding in plastic and then localizing the crista for ultrathin sectioning remain to be devised. Several technical approaches appear feasible.

From the present studies it appears that the vertigo as distinct from nausea of acute radiation sickness may be due to a direct effect on the functional state of the vestibular apparatus rather than effects on the central nervous system, gastrointestinal tract, or indirect effects due to changes in the state

of hydration, tissue necrosis, etc. The experimental work of Graybiel (ref. 11) and his associates indicates that the nausea and vertigo of motion sickness in man are entirely vestibular in origin and he prefers the term

vestibular sickness. Thus it seems likely that all vertigo may have its origin in the vestibular apparatus. Radiation sickness has of course other manifestations which we know can be produced without head irradiation.

#### REFERENCES

1. EWALD, C. A.: Zentr. f. Physiol., 19, 1905, p. 298.
2. THIELEMANN, M.: Fortschr. Gebiete Roentgenstrahlen, 37, 1928, p. 563.
3. BORSANYI, S.; BLANCHARD, C. L.; THORNE, B.: Ann. Otol., Rhinol. Laryngol., 70, 1961, p. 255.
4. KELEMEN, G.: Acta Otolaryngologica Suppl. 184, 1963.
5. MOSKOVSKAYA, N. V.: Vestn. Otorinolaring., 21, 1959, p. 59.
6. SVESHNIKOV, A. A.: Med. Radiol., 8, 1963, p. 48, JPRS Translation TT: 63-41278.
7. BIRGE, A. C.; ANGER, H. O.; AND TOBIAS, C. A.: In Radiation Dosimetry (G. J. Hine and G. L. Brownell, eds.), Academic Press, 1958.
8. VAN EGMOND, A. A. J.; GROEN, J. J.; AND JONGKEES, L. B. W.: J. Physiol. 110, 1949, p. 1.
9. GROEN, J. J.; LOEWENSTEIN, O.; AND VENDRICK, A. J. H.: J. Physiol. 117, 1952, p. 329.
10. CITROEN, L.; EXLEY, D.; AND HALLPIKE, C. S.: Brit. Med. Bull. 12, 1956, p. 101.
11. GRAYBIEL, A.: In Neurological Aspects of Auditory and Vestibular Disorders. (W. S. Fields and B. B. Alford, eds.), Chas. C Thomas Co. (Springfield, Ill.), 1964, p. 248.

#### DISCUSSION

KELLOGG: I presume you did histological studies afterwards. Is that correct?

MCDONALD: We did some gross tissue examination to reassure ourselves that the beam location was correct, with very high doses of 20 000 rads. Histological studies remain to be done. This is one of the major things we want to do.

KELLOGG: What I was wondering was if you saw any more effect in one area than in another?

MCDONALD: No, this hasn't been done. This remains to be done. Some of this has been done for X-ray by Kelemen (Acta Otolaryngol. Suppl. 184, 1963) for whole head radiation, but the animals were sick from the whole body radiation at the same time.

BERGSTEDI: For 3 years I have followed a group of patients who have received heavy radiation for tumors in the epipharynx nearby the labyrinth. As Dr. McDonald said, they have a decrease in function in bone conduction hearing and vestibular function compared with before the X-ray treatment, but it doesn't seem to bother them so much. It seems that, to a decreased function on both sides, there is no pronounced preponderance and there are no especially strong vestibular disturbances. About the radiation sickness, this seems to be a complicated question concerning the etiology and I don't think it has so much to do with the labyrinth. To sum up, I am astonished how little they are affected from heavy radiation.

MCDONALD: I think that this is a good point. Many of these patients are treated by fractionated doses for a period of weeks. I think this may have some importance, a gradual delivery of dosage, allowing for adjustment to changes in vestibular function without clinical vertigo developing.

J. MILLER: I have heard a number of comments which seem to suggest that motion sickness can be caused only by motion. We ran some experiments here seven or eight years ago involving a helicopter simulator in which we were asked to try to ascertain why 65% of the instructors were getting sick and showing all the subjective symptoms, as well as objective symptoms, of motion sickness when indeed there was no motion. It was a fixed platform. Among other things we came to the conclusion that the symptoms were due to a combination of a poor perceptual display in terms of distortion and a conflict of cues. While on this simulator all perceptual cues indicated you were moving and yet your vestibular mechanism indicated you were not. I would like to hear some discussion as to whether there is any cerebral ischemia involved in such things or whether anyone has indeed looked into the problem of motion sickness with no motion.

BILLINGHAM: From the point of view of the Manned Spacecraft Center we are watching Dr. McDonald's results with the utmost interest because of the vital importance of combined stresses. We are looking specifically at the type of radiation which will be met in a solar flare, and there exists as well the potential problem of some abnormal vestibular stimulation in a weightless condition. The two effects could combine to produce nausea and, if there is a direct effect of the radiation on the vestibular mechanism itself, that may increase or decrease the effect of the vestibular contribution to the nausea, depending on the particular circumstances of the combination of the two stresses.

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# Preliminary Studies of Vestibular Damage in Guinea Pigs Following High Acceleration

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## SUMMARY

Guinea pigs were exposed to high impact deceleration on a sled and short-duration acceleration on a centrifuge. Behavioral examination of swimming ability and the righting reflex revealed evidence of vestibular damage following exposure to peak acceleration in the range of 200 to 400 g for periods of 14-20 sec. Histological examination of the temporal bones demonstrated extensive structural damage for the same animals which exhibited behavioral deficiency.

No evidence of behavioral damage has been observed following exposure to impact deceleration. Also, histological evidence of damage is considerably less following impact deceleration than short-duration centrifugation.

## INTRODUCTION

At the present time we cannot specify the levels and durations of acceleration which will produce permanent damage to the vestibular apparatus. The possibility of exposure of human beings to unusual acceleration environments such as long-duration buffeting in a high performance aircraft or sharp onset high acceleration in escape from a space vehicle indicates the necessity for obtaining information regarding the response of the vestibular apparatus to intense stimulation.

This report is concerned with our preliminary investigations into the effects of high linear acceleration, primarily on the otolithic system. Guinea pigs have been subjected to impact deceleration on a sled and

short-duration acceleration on a centrifuge. Deceleration profiles with peaks as great as 314 g and durations up to 10 msec have been obtained with the sled. Centrifugation has produced acceleration profiles with peaks between 100 and 400 g and durations from 10 to 21 sec. Assessment of varying degrees of vestibular end-organ damage following exposure to these intense stimuli has been accomplished with histological and behavioral techniques.

The major purpose of these preliminary investigations was the development of procedures for assessment of vestibular damage in animals. In order to increase the chances of producing damage, intense acceleration exposures were employed. A discussion of the various manifestations of vestibular

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damage which we have observed will comprise the bulk of this report.

Currently, investigation of the effects of exposure to a wide variety of acceleration profiles is being pursued. Also, more sensitive techniques for the measurement of vestibular damage are in the process of development. A complete picture of the response of the vestibular apparatus to overstimulation will be possible only after an extensive series of studies.

#### METHOD

##### Impact Deceleration

*Subjects.*—Six guinea pigs were exposed to impact decelerations in the following positions: 2 forward facing, 2 backward facing, and 2 supine-transverse. Figure 1 illustrates the subject's orientation with respect to the decelerative force.

*Apparatus.*—The apparatus employed to obtain the impact decelerations was modified from that described by Lombard, Close, Thiede, and Larmie (ref. 1). This apparatus consists of a container for holding the animal, a sled assembly with means for mounting the container, a track upon which the sled runs, a bungee cord to accelerate the sled, a backstop to halt the sled, and instrumentation to detect the acceleration profile. All of the animals were impacted at the Northrop Space Laboratories, Hawthorne, Calif., where the apparatus was designed and constructed.

*Procedure.*—For each run, the subject was placed in a molded container which provided dorsal support. Nylon webbing held the animal firmly in place, and a conical nylon nose piece restrained the head. The animals, which were restrained in this fashion, were subjected to impact decelerations measured on the sled ranging from 240 to 314 g peak with an average peak value of 259 g and durations of about 10 msec. Differences between the deceleration profiles were a function of uncontrolled apparatus variables. A typical deceleration profile is illustrated in figure 2.

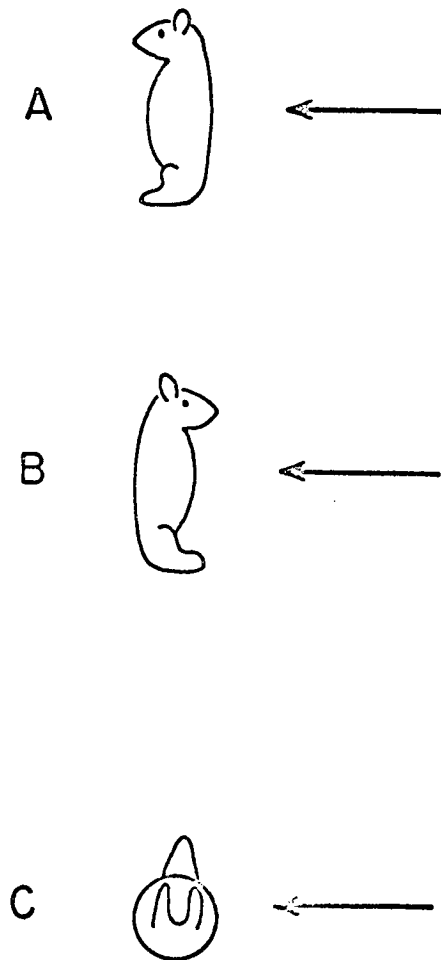


Figure 1.—Orientation of subject with respect to forces generated by sled and centrifuge. Direction of decelerative or centripetal force relative to subject is indicated by the arrows. A: backward facing ( $+G_x$ ); B: forward facing ( $-G_x$ ); C: supine transverse ( $\pm G_y$ ).

Following exposure the animals were allowed to recover for a period of 24 hours. The animals were then transported to Washington University, St. Louis, Mo., where histological processing was performed. No attempt was made to obtain behavioral data from these animals. At the time of these first exposures the investigators did not realize the ability of the guinea pig to survive intense acceleration and deceleration of this magnitude.

Celloidin sections of the temporal bones

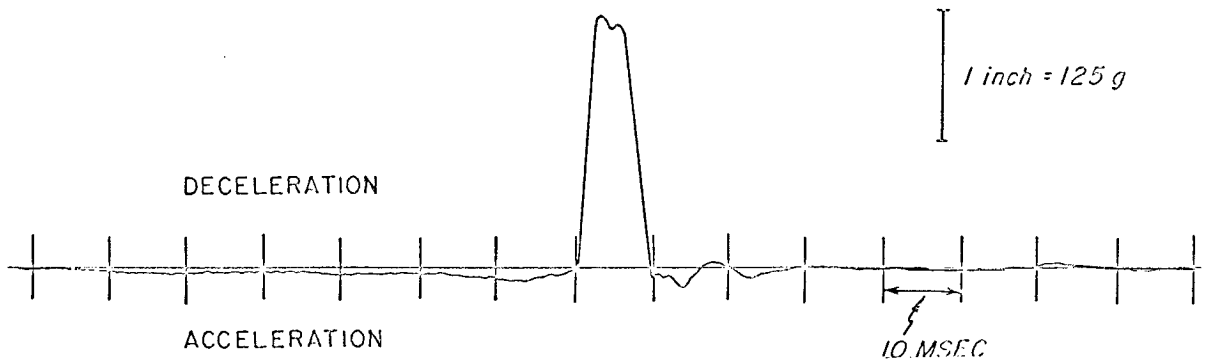


Figure 2.—Profile of impact deceleration for sled bearing guinea pig. Profile peak is 240 g and duration is approximately 10 msec.

were prepared following the techniques described by Covell and Eldredge (ref. 2). Serial sections of 15 micra in thickness were made and each fifth section was stained with hematoxylin and eosin and mounted in balsam.

**Short-Duration Centrifugation**

*Subjects.*—Six guinea pigs were exposed to a variety of acceleration profiles with a

unique centrifuge (ref. 3). All animals were run in the backward facing position.

*Apparatus.*—The apparatus employed to accelerate the subjects was the Space Flight Acceleration Profile Simulator (SFAPS), located at the Space Defense Corporation, Birmingham, Michigan. A preliminary account of SFAPS characteristics is contained in an article by Pine and Barr (ref. 3).

Essentially the SFAPS is a centrifuge. The SFAPS consists of a 92 cm primary arm, ro-

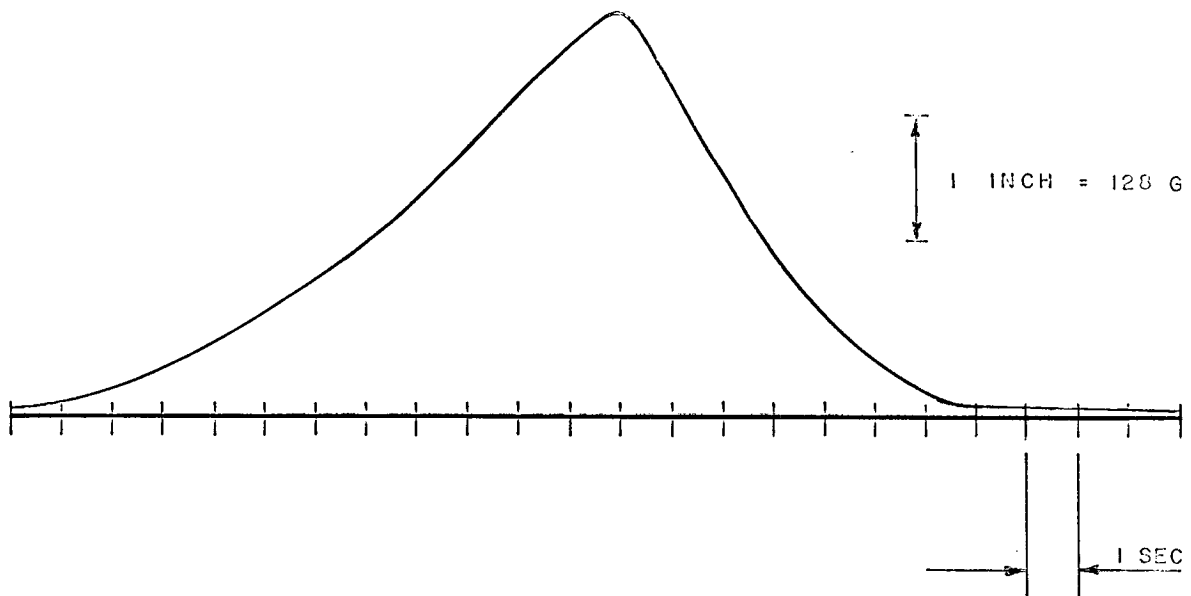


Figure 3.—Acceleration profile for centrifuge (SFAPS) bearing guinea pig. The profile has a linear acceleration peak of 410 g, a rise time of 11.5 sec, and a decay time of 8.3 sec. Rotatory acceleration component associated with this run was about 7 g.



tational center, mass balance, payload capsule, motive power source, support structure encasing the machine, and instrumentation to detect the acceleration profiles. Special design features allow onset and decay rates up to 40 g/sec. An example of the acceleration profiles obtainable with this device is presented in figure 3.

*Procedure.*—Prior to acceleration the experimental animals were subjected to a variety of behavioral tests in order to obtain an estimate of vestibular end-organ sensitivity. Our experience indicates that the most useful of the behavioral tests which we have employed are righting reflex and swimming ability. The results of pre-exposure examination with these tests are presented in table I.

The phrase “righting reflex” refers to the complex series of movements whereby an animal which is dropped in an inverted position achieves a four-point landing. Prior to exposure, guinea pigs are able to right themselves consistently (5 for 5) when dropped from heights of 10 to 25 cm. In order to determine swimming ability, the guinea pigs were placed in a 45 by 100 cm tank which was filled with water to a depth of 25 cm. Normal guinea pigs are excellent swimmers and move in straight lines, unless presented with a barrier.

For each run the subject was placed in a coffin-like box which provided dorsal support. The subject’s nose was taped to a molded head piece which assured proper

Table I.—*Behavioral Indicators of Vestibular Damage*

Behavioral indicator	Subject/peak acceleration					
	PC11-1	PC12-2	PC13-3	PC14-4	PC15-5	PC16-6
	95g	105g	195g*	200g	397g	410g
Pre-exposure: Righting	5/5 20-25cm	5/5 10-15cm	5/5 20-25cm	5/5 20-25cm	5/5 10-15cm	5/5 20-25cm
Swimming	Straight	Straight	Straight	Straight	Straight	Straight
Immediately post-exposure: Nystagmus	No	No	3 min	3 min	2.5 min	None
Position reflex	No	No	Yes	Yes	Yes	Yes
Shaking	No	No	Yes	Yes	Yes	Yes
Rolling	No	No	Yes	No	Yes	Yes
24 hours post-exposure: Righting	5/5 20-25cm	5/5 15-20cm	0/5 40cm	5/5 20-25cm	0/5 40cm	0/5 40cm
7 days post-exposure: Righting	5/5 20-25cm	5/5 15-20cm	2/10 40cm	5/5 15-20cm	0/5 40cm	0/5 40cm
Swimming	Straight	Straight	Circles, lists	Straight	Tight circles	Tight circles, lists

\* Sacrificed on 6th day.

alinement of the temporal bones during acceleration.

The 6 animals were exposed to peak accelerations of approximately 100, 200, and 400 g. Two animals were exposed to each acceleration level. The acceleration profiles, which were nearly triangular in shape, covered temporal durations of about 10 sec for the 100 g peak, 14 sec for the 200 g peak, and 20 sec for the 400 g peak.

Immediately after each exposure the subject was examined to determine general orientation and nystagmus. All but one of the animals were subjected to further behavioral testing at periods of 1 and 7 days following exposure in order to help determine the presence and extent of vestibular damage. One of the 200 g animals developed respiratory difficulty and was tested and sacrificed on the sixth day. On the seventh day the remaining animals were transported to St. Louis where the temporal bones were prepared for histological examination as previously described.

## RESULTS

### Behavioral Observations

*Impact deceleration.*—As previously noted, no behavioral studies were performed on the group of animals which was exposed to high impact deceleration. However, subsequent study of the righting reflex and swimming ability of a group of ten animals which had been exposed to similar impact decelerations resulted in little evidence of behavioral deficiency.

*Short-duration centrifugation.*—The results of behavioral examination following short-duration centrifugation are summarized in table I. Immediately following exposure the animals which were in the 200 and 400 g groups exhibited a variety of behaviors indicative of disorientation. Three of the four animals in the 200 and 400 g groups manifested nystagmic eye movements for 2.5 to 3.0 min following centrifugation. Interestingly, the plane of the nystagmus did not remain constant, but shifted from horizontal to vertical in a seemingly random

fashion. All four of the 200 and 400 g animals exhibited a shaking or tremor which diminished in amplitude and frequency over a period of 5 to 10 min following exposure. This movement resembles that seen in shaker mice. Also, three of the four 200 and 400 g animals rolled or fell over the front shoulder when they attempted to stand. The animals rolled as readily to the left as to the right. This rolling seems to be related to a position reflex which is depicted in figure 4. In the case illustrated, the animal's head is flexed and rotated to the right. The hind leg on the contralateral side is extended. After rolling had ceased, the animals would maintain this position for periods up to 2 or 3 min.

Vestibular sensitivity was determined 24 hours after exposure with the righting reflex. Three of the four 200 and 400 g animals were unable to perform the righting reflex in five trials from a height of 40 cm.

The final behavioral testing was performed seven days after exposure and included tests for righting reflex and swimming. Three animals were unable to perform the righting reflex consistently although one of the 200 g animals gave evidence of recovery by righting two times in 10 trials. Also, the animals' swimming abilities were assessed. The three animals which were unable to perform the righting reflex also manifested difficulties in swimming. Contrary to normal animals which generally swim in straight lines, the

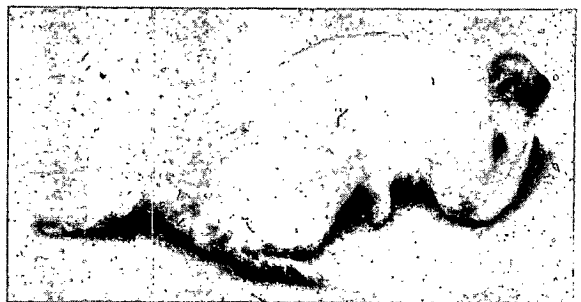


Figure 4.—*Position reflex following centrifugation. Position reflex is typically assumed following exposure to accelerations of 200 to 400 g. Animal's head is flexed and rotated right. Hind leg contralateral to side of nose displacement is extended.*

three animals which apparently sustained vestibular damage swam in circles and, in two cases, listed rather severely.

#### Histological Observations

The stained and mounted serial sections of each temporal bone were examined under the microscope at different magnifications for any changes in the vestibular end organs and labyrinth. These were compared with the series of slides for control animals, and any alterations that appeared not to be artifacts due to processing and sectioning the bones were given a rating of one to four plus depending upon the extent of injury. At the same time any changes that occurred in the cochlea were also noted.

Criteria for judging injuries are pre-

sented in table II. The most marked or severe damage is rated as four plus and gradations as one, two, and three plus. This does not imply that the entire crista or macula is damaged to the same extent. Usually it is one end and/or margins of a macula of a utricle that show the changes, and the remainder may be within normal limits. The same applies to the cristae. Changes that can be attributed to the exposure are usually to be found at the base and lateral margins of a canal crista and the apical areas may appear uninjured.

The ratings for Scarpa's ganglion are not included in table II because the problem is somewhat different. A few days following exposure some loss of Nissl's substance is

Table II.—Criteria for Rating Histological Changes

Rating	Cristae	Macula	Collapse and/or rupture of membranes	Edema of connective tissue	Cellular debris in endolymphatic channels
One plus (+)---	Barely discernible changes in supporting and sensory cells, reversible (?)	Barely discernible changes in supporting and sensory cells, reversible (?)	-----	Slight, if any	Small amount, if any
Two plus (++)-	No apparent loss of cells, but definite evidence of injury. Some evidence of loss of alinement in supporting and sensory cells	No apparent loss but definite evidence of injury. Otoconia may show some slight loss or rearrangement. Also alinement of cells disrupted for certain areas	A partial collapse, usually	Slight to moderate	Slight to moderate
Three plus (+++)-----	Loss of some cells and evidence of areas of disorderly arrangement, swelling of apical ends of cells	Loss of some cells and evidence of disorderly arrangement, loss of otoconia in margins, etc.	A marked collapse, usually	Moderate	Moderate
Four plus (++++)-	Severe injury and loss, many globules between crista and cupula. Malalinement	Severe injury and loss, globules between macula and otoconia; marked loss of otoconia	Complete collapse of most membranes	Marked	Marked



Figure 5.—Macula utriculi of left labyrinth of a guinea pig with some of epithelium of superior canal crista also shown. Otoconia are missing over a small area of lateral edge (arrow) of macula. There are a few changes in sensory cells throughout. These changes were given a rating of 2 plus. 50× Harris hematoxylin and eosin.

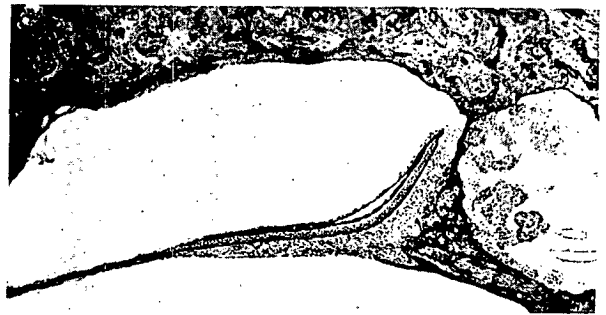


Figure 7.—Macula utriculi of right ear of a guinea pig showing collapse and partial adherence of utricular membrane. Otoliths over posterior one-half and extreme anterior end appear to be in contact with sensory epithelium, for which there is somewhat disorderly arrangement throughout. 53× Harris hematoxylin and eosin.

apparent and an eccentric position of the nucleus is discernible. After about 2 weeks the degenerating cell shows loss of nucleus and neuronophagia. The rating of one to four plus refers to relative number of cells showing degenerative changes. Four plus would infer that all cells in the ganglion are in various stages of degeneration. Nerve fiber loss is not included in the present study since special staining methods have to be used for this study. Examples of the various types of damage encountered are presented in figures 5-10.

*Impact deceleration.*—In table III are shown the ratings of histological changes in the vestibular labyrinth for the guinea pigs



Figure 6.—Macula sacculi of left ear of a guinea pig with some loss of otoconia at lower margin. Some of these can be seen in one end of saccule. There are a few changes in sensory and supporting epithelium. These changes were given a rating of 2 plus. 50× Mallory's Triple C.T. Stain.

which were exposed to impact deceleration.

The vestibule, except for the appearance of a protein coagulum in some instances, was without any significant changes.

Scarpa's ganglion cells revealed changes ranging from one to three plus. Eccentric nuclei and loss of some Nissl's substance were in evidence as early as 3 days following exposure.

The utricular membrane was rarely collapsed but the macula of the utricle showed changes with some loss and/or displacement of otoconia in only a few specimens. The degree of cell changes ranged from one to four plus. The macula of the saccule not only showed a greater loss of otoconia but

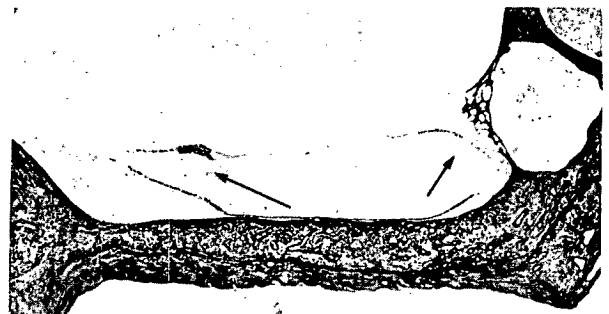


Figure 8.—Macula (shorter arrow) of a utricle showing many degenerative epithelial changes and complete loss of otoconia. A group of these (longer arrow) can be seen in left hand corner. 30× Harris hematoxylin and eosin.

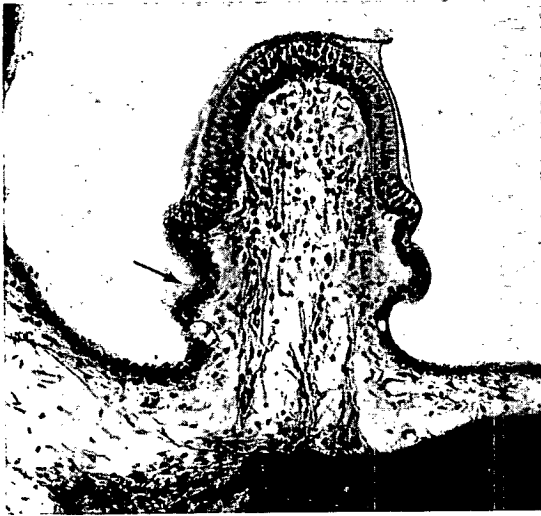


Figure 9.—Posterior canal crista of a guinea pig showing a few altered sensory cells, a very much shrunken cupula (artifact?) and some changes in transitional epithelium of one side (arrow) as compared to the opposite side. This rating was judged as 2 plus. 130× Harris hematoxylin and eosin.

in many instances more changes in sensory and supporting cells than the macula of the utricle.

The crista of the lateral canal revealed supporting and sensory cells and other changes ranging from negative for one specimen to three plus for several others. The findings for the cristae of the superior canals were about the same but with a tendency to reveal less injury than for the cristae of the lateral canals. The posterior canal crista in some instances revealed more injuries than did either the superior or lateral canal crista.

*Short-duration centrifugation.*—Table IV presents the findings for histological changes in the ears of the six guinea pigs which were exposed to short-duration centrifugation. Minor changes were usually in evidence for exposures of peak accelerations in the vicinity of 100 g. In one animal (PC11) the macula of each saccule was without otoliths while the maculae of the utricles showed some loss of otoliths and only a few changes in the sensory epithelium. When the exposure



Figure 10.—Lateral canal crista of a guinea pig showing partial collapse of ampullary membrane, and presence of some cellular debris. Arrows indicate areas of sensory cell changes. 53× Harris hematoxylin and eosin.

was increased to a peak g of 200 or thereabouts, hemorrhage into the cochlea and perilymphatic spaces occurred in one ear for each of two different animals. Loss of otoliths was usually only partial. The utricular macula in PC13R showed considerable degeneration and total loss of otoliths while the saccular macula was relatively in good condition. This is contrary to most findings. With an increase of the peak g exposure to approximately 400, macular injuries became marked, extensive hemorrhage was present in the cochlea and all perilymph-containing areas, and degenerative changes were found in the sensory and supporting cells of the organ of Corti.

#### EVALUATION OF DAMAGE ASSESSMENT PROCEDURES

*Relationships between measures.*—Examination of tables I and IV indicates a rather good correlation between the behavioral indicators of swimming ability and righting reflex and the histological rating of damage to the maculae. Of particular interest are the results for subjects PC13-3 and PC14-4,

Table III.—*Ratings of Histologic Changes in Guinea Pigs Exposed to High Impact Deceleration*

[Position: F, forward; B, backward; S, supine. Coagulum: Co. Debris: D. Otoconia loss: OL. Membranes: A, adherent; B, ballooned; C, collapsed; R, ruptured; W, wavy. Hemorrhage: H. Infection: O.M., otitis media; S.L., serous labyrinthitis. (+) Mild to extreme changes are rated on the basis of one to four plus. (—) No changes.]

Animal no.	PC1 Rt.	PC1 Lt.	PC2 Rt.	PC2 Lt.	PC3 Rt.	PC3 Lt.	PC6 Rt.	PC6 Lt.	PC7 Rt.	PC7 Lt.	PC8 Rt.	PC8 Lt.
Position -----	F	F	F	F	B	B	B	B	S	S	S	S
Peak g -----	214	214	236	236	252	252	251	251	240	240	262	262
Duration (msec) -----	4.0	4.0	4.1	4.1	4.0	4.0	3.5	3.5	4.0	4.0	3.5	3.5
Weight (gm) -----	327	327	305	305	312	312	325	325	325	325	330	330
Post-exposure (days) -----	24	24	3	3	17	17	17	17	10	10	3	3
Vestibule -----	—	—	Co.	—	—	Co.	Co.	Co.	Co.	—	Co.	Co.
Scarpa's ganglion -----	+	+++	++	+++	+++	++	+	++	+++	+++	+++	+++
Utricle and macula -----	+	++	++	++	+D	++	+	++	+++C	+++	++	+
Sacculae and macula -----	+	+++	+	+++	+++C, OL	++OL	++C, OL	+++C, OL	+++C, OL	+++C, OL	+++	+
<i>Cristae</i>												
Lateral canal -----	—	+++	++	++	+++	+++	++	++	++C	+++	+	+
Superior canal -----	—	++	+	+	++	++	++	++	++C	++	+	+
Posterior canal -----	++	+++	++	+	+++	+++D	++	++	++C	++	+++	++
<i>Cochlea</i>												
Scala tympani -----	H.	—	H.	H.	—	—	Co.	Co.	Co.	—	H	H
Scala vestibuli -----	H.	—	H.	H.	Co.	Co.	Co.	Co.	Co.	—	H	H
Organ of Corti -----	—	—	—	—	—	—	+	+	—	+	—	—
Reissner's memb. -----	B.	C.	—	—	—	—	B	B	BR	R	—	—
Infection -----	O.M., S.L.	—	—	—	—	—	O.M., S.L.	O.M., S.L.	O.M., S.L.	—	—	—

both of whom were exposed to about 200 g peak acceleration. Animal PC14-4 exhibited no evidence of damage with the righting reflex and swimming ability, and its total rating for histological changes in the maculae was four plus; on the other hand, PC13-3 demonstrated clear behavioral signs of vestibular damage, and for the temporal bones of this animal the total rating of damage to the maculae was 12 plus. The animals which were exposed to 400 g peak accelerations exhibited histological and behavioral signs of severe vestibular damage.

*Limitations of measures.*—The behavioral techniques employed in this investigation are considered to provide only a gross estimate of vestibular damage. Judgment of varying degrees of swimming ability is difficult. Also, it is difficult to obtain a satisfactory number of trials with the righting reflex because a fatigue effect seems to take place in the damaged animals following repeated falls on the neck or back.

Histological rating of injury to the vestibular labyrinth is traditionally subject to artifact. Perhaps this is due to the inability

of the sensory epithelium of the cristae and maculae to fix as satisfactorily as the organ of Corti by the standard methods of intravascular perfusion. Also, the processing of the temporal bone for sectioning removes the mineral salts in the otoconia so that only the "ghosts" of their outlines remain and stain blue with Harris hematoxylin. These are subject to artifacts; even in control specimens they may reveal differences in distribution, staining, and so forth. Judgment of extent to which otoconia may be lost in some of the sectioned material of the present series is not always easy. However, when there was a complete loss for a particular area or the entire macula, the judgment was readily determined. The otoconia are embedded in a gelatinous substance to which the hairs of the sensory cells penetrate. Fixation leaves a clear space between the apical ends of the cells and the otoconia containing substance. This is probably an artifact. In some exposed animals this space was greatly exaggerated in width and the hairs of the sensory cells appeared to be free. In other specimens the otoconia seemed to lie on the

Table IV.—Ratings of Histologic Changes in Guinea Pigs Exposed to Short-Duration Centrifugation

[For abbreviations, see table III]

Animal no.	PC11R-1	PC11L-1	PC12R-2	PC12L-2	PC13R-3	PC13L-3	PC14R-4	PC14L-4	PC15R-5	PC15L-5	PC16R-6	PC16L-6
Exposure { Rise Dwell Fall	5.3 <0.1 3.8		5.5 <0.1 4.3		7.6 <0.1 5.8		7.6 <0.1 5.9		11.1 <0.1 8.5		11.2 <0.1 8.3	
Peak g	105		95		195		200		395		410	
Post-exposure (days)	13		12		10		13		13		13	
Body weight (gm)	315		315		295		265		295		295	
Vestibule					Co.		Co.		Co.		Co.	
Scarpa's ganglion	++	++	+	+	++	++	++	++	+++	+++	+++	+++
Utricule and macula	+Some O.L.	++Some O.L.C.	+C.	+C.	+++O.L.	+++Partial O.L.	+++Some O.L.	+++Some O.L.	+++O.L.	+++O.L. C.D.	+++O.L.	+++O.L.
Sacculle and macula	+++O.L.	+++P.C.- O.L.	+C.	+C.	++	+++O.L.	+++Some O.L.	+++Some O.L.	+++O.L.	+++O.L. C.D.	+++O.L.	+++O.L.
<i>Cristae</i>												
Lateral canal crista	++	++	+	++	+	+	++	++	++	++	+++	+++
Superior canal crista	+	++	+	+	+	+	++	++	++	++	++	++
Posterior canal crista	+	++	+	++	++	++	+++	++	++	+++	+++	+++
<i>Cochlea</i>												
Scala tympani												
Scala vestibuli							H. (apex)	ppt.	H.	H.	H.	H.
Organ of Corti							H. (apex)	ppt.	H.	H.	H.	H.
Reissner's membrane												
Spinal ganglion												
Middle ear												
				New bone			A.O.M., some S.L.			Edema mucosa	Some O.M., some H.	Some O.M., some H.
Other findings						Some H. into perilymph spaces	Some H. into peri- lymph spaces		Hemor- rhage into perilymph spaces	Hemorrhage into perilymph spaces	Hemor- rhage into all perilymph spaces. Fibrosis in some perilymph area of canal. Degeneration of stria vascularis	Hemor- rhage into all perilymph spaces. Fibrosis in some perilymph area of canal. Degeneration of stria vascularis

apical ends of the hair cells with no, or very little, intervening substance. Each of the findings could be interpreted as a result of the exposure, but further study is needed before their significance can be appraised.

The cupula of a crista undergoes considerable distortion and shrinkage with fixation. It is impossible to judge any changes in posi-

tion or damage that might be present as a result of the trauma. In this way it resembles the tectorial membrane to which it is similar histochemically. The only information that at present would be of interest is the total loss of a cupula from its crista. This has not been encountered in the present series.

#### REFERENCES

1. LOMBARD, C. F.; CLOSE, P.; THIEDE, F. C.; AND LARMIE, F.: Impact Tolerance Related to Orientation and Containment. *Aerospace Med.*, 35, 1964, pp. 1-6.
2. COVELL, W. P.; AND ELDRIDGE, D. H.: Injury to Animal Ears by Intense Sound. AF Technical Report No. 6561, WADC, Wright-Patterson Air Force Base, 1951.
3. PINE, B. W.; AND BARR, N. L.: Some Responses of the Squirrel Monkey to High G Brief Duration Acceleration Profiles. *Aerospace Med.*, 34, 1963, pp. 752-757.

#### DISCUSSION

POLLACK: I'd like to make a plea for preexposure studies on the subjects who are to ride in the centrifuges in the future so that we have a baseline of data and, at best, we can interpret what happens subsequently. People may have opinions, but I think we should have some facts to back up opinions or else to lay the ghost low once and for all. When general publications such as *Popular Science* can have a front page article, "What Happened to John Glenn—Did He Get Dizzy Before He Fell or Otherwise?", this is merely a manifestation of the thinking of a great many people. No one can answer these questions positively in spite of opinions which people may hold. I think it is incumbent upon us, in the future, to have baseline data on vestibular functions of all individuals before we subject them to the dynamic stress of linear impact or centrifuge.

ENGSTRÖM: In these cases where you have problems with the time factor in the histological study of the maculae when looking for the crystals, after exposure to G-forces, wouldn't it be very easy just to open the labyrinth as we regularly do in animals and observe the position of the crystals in each animal? In this way you can, within about 45 seconds, state if the crystals are in normal position or not.

PARKER: This has been suggested and I think we will try this procedure.

WHITE: Dr. Pollack, we have made about 2000 centrifuge runs in the past year. What would be the recommendation as to a minimum battery of labyrinthine tests to follow up on your suggestion? We do some, of course, but I would like to improve the test battery.

POLLACK: Dr. Graybiel and I have a project going on at the moment where we are recalling a large group of men who have been centrifuged in the past over a number of years and they are now being studied, going through the whole gamut of tests. I think

that, after the first series of tests has been performed, we will be able to pick more definitive ones that would set up a program for more general use.

FIELDS: Dr. Pollack, are you implying that events are taking place during the astronaut training program which might have some tendency to increase the individual's susceptibility because of some influence on or damage to the vestibular mechanism? I thought at least what I had to say in respect to the one specific incident concerning Colonel Glenn was quite clear.

POLLACK: What I am saying is that the possibility of the training programs leading to destructive effects, I think, is a real one. This has been indicated by the Johnsville reports of 1953, 1955, 1956. One sees periodic reference to petechial hemorrhages in the vestibular apparatus. One sees periodic references to the residual symptomatology for 6 to 8 weeks after the runs are over, so that there is some indication in the background that a certain group of people particularly are more sensitive to the centrifuge trauma than others. While one can't generalize, it would appear from the Johnsville reports that some of the people who have been on the centrifuge have had a residual symptomatology for a variable period of time after the runs.

BERGSTEDT: One explanation for Titov's experience could be that he had some slight deviation from the normal within his equilibrium system. A careful "vestibular profile" of people involved in space travel should be of interest. I agree with Dr. Pollack that this holds also for test subjects in vestibular and especially centrifuge experiments.

WHITE: As a centrifuge rider for about 15 years, I always get concerned when people start worrying about vestibular damage. I am very worried about this from another point of view also. As a person who hires subjects and tests them on a centrifuge



it is incumbent upon me to insure their health and welfare as best I can. I use as a guide the study of Meehan, Galambos, and Hiatt, published by NAS-NRC. This is a survey of the very broad aspects of monitoring, testing, and CNS involvement as a result of repeated centrifugation. These authors report no destructive effects of human exposure to acceleration. I am curious and worried by your general remarks.

**SPOENDLIN:** Three years ago we planned to expose some squirrel monkeys to high G's. Of 12 animals, we exposed eight to 12 G and four to 6 G from 1 to 10 minutes. The animals with 6 G tolerated 10 minutes, whereas the animals exposed to 12 G died usually after 3 minutes of exposure, and survived only after 1 minute exposure. Some of them showed ataxia for a certain time after the exposure. Histological and electronmicroscopic evaluation of the maculae did not show any changes whatsoever.

**PARKER:** We've had a brief discussion here as to whether the men who have ridden the centrifuge for many years are showing vestibular damage. Also, it has been pointed out that we're getting into new types of environments: vibration, low frequency

noise, etc., which may be damaging to the vestibular apparatus. It seems that we're going at this problem backwards. It would be advantageous to have data from animals to determine the limits of vestibular damage at a broad spectrum of levels and to various types of acceleration environments.

**VON GIERKE:** Several years ago there were some studies done at Fort Knox in which monkeys were exposed to long duration vibrations. Dr. M. Lawrence did the histology on these monkeys which were exposed to levels of 2 to 10 G's of sinusoidal vibrations for many hours; I think it was 8 or 10 hours. As far as I recall the histology was somewhat similar to the figures we saw on the guinea pigs in Dr. Parker's study in which the animals were on the centrifuge for a few seconds. The vibration study gives us an indication that exposure time or repetition of exposure is probably a very important factor. A 5 G vibration exposure over a very long time period might do something to the vestibular system whereas we know that 5 G's for a short time period does nothing.

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# Dynamic Counterrolling of the Eye in Normal Subjects and in Persons with Bilateral Labyrinthine Defects

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## INTRODUCTION

Studies in dynamic counterrolling have been in progress at Wright-Patterson Air Force Base for the past year. For purposes of this discussion, dynamic counterrolling is defined as rotation of the eyes about the X-axis while the entire body is being rotated about its X-axis at the level of the canals. Measurements have been made on both normal and bilaterally labyrinthine defective (L-D) subjects and at rotation rates from zero (static) to 15 rpm in both clockwise and counterclockwise directions. Although complete data on normal subjects at some rotation rates and directions are still lacking, the trend of response is very clear.

## EQUIPMENT

The experimental equipment consists of a modified F-104 ejection seat bolted to a 2.5 ton truck rear axle and differential assembly (fig. 1). The seat is rotated by a 1/2 h.p. electric motor, coupled directly to a variable speed (0 to 90 rpm output) transmission. The transmission output is coupled to the truck differential by two pulleys and a V-belt.

Two synchronized Flight Research 35mm motion picture cameras are mounted on the device. Camera 1 is mounted on a platform suspended in front of the subject's head with the lens aligned directly with the subject's eye. The camera remains fixed in this position and rotates with the subject through 360°. The camera and lighting system are

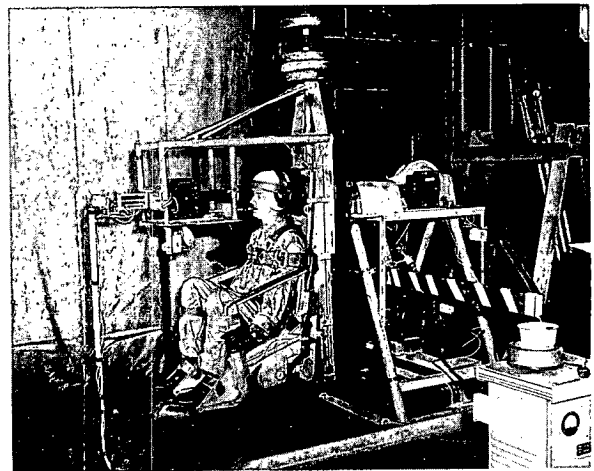


Figure 1.—Experimental equipment.

powered through 18 slip rings mounted on a horizontal shaft in front of the camera platform. Camera 2 is mounted behind the seat on the motor frame and is focused on the periphery of a disk, marked off in degrees, which rotates with the chair. The cameras have a capacity of 100 feet of film (1600 frames) and an operating speed of 10 frames per second. The shutter openings are synchronized to within 2° ( $\pm .75$  m/sec). Neon indexing lights are located in both cameras to insure correct alignment of the film in data reduction. Apochromatic Kinoptik f/2 100mm focal length lenses are used on both cameras. The eye camera has an object distance of 7 1/2 inches and an image distance of 8 1/2 inches. The depth of field for an image

object ratio of 1:1 at the  $f$  stop used,  $f/8$ , was  $\pm 0.016$  inch. The total depth of field is 0.032 inch, assuming a circle of confusion of 0.001 inch. This limited the amount of forward-backward movement of the head to  $\pm \frac{1}{64}$  inch.

The seat is equipped with reinforced lap belt, shoulder harness, chest straps, and Velcro straps for head, arms, and legs. A bite bar is positioned in the subject's mouth and is held there securely by a dental impression.

#### EXPERIMENTAL PROCEDURE

The first step in preparing a subject for the run is the application of one drop of 1% Pilocarpine on the right cornea. This procedure was found necessary to stabilize the iris since, even in the presence of high and constant illumination levels, there were wide fluctuations in pupil size.

After the subject is strapped securely in the chair and the dental bite positioned correctly, the static counterrolling measurements are made. Beginning in the upright position, with the room darkened, 10 frames of film are taken. The subject is then rotated  $20^\circ$  and locked in position and 10 more frames are taken. The rotation is continued at  $20^\circ$  intervals until shots have been taken throughout  $360^\circ$ .

The dynamic measurements are then begun. The subject is accelerated slowly in a clockwise direction up to a velocity of 5 rpm and after stabilizing for 3 revolutions, films of 2 complete revolutions are made. The subject is then accelerated up to 10 and then 15 rpm and the same procedure followed. The chair is then stopped and the same procedure is repeated in the counterclockwise direction.

#### DATA REDUCTION

The data reduction device used in this study is based on the technique used by Earl Miller, Navy SAM. The technique involves projecting two superimposed eye images on a screen. The images must be the same size, and must be aligned horizontally, vertically, and rotationally. A specially designed slide

projector was built so that a reference slide could be held fixed with respect to a rotating reference. A vernier scale allows adjustment of the reference slide to an accuracy of 1 minute of arc. The apparatus is shown in figure 2. Each frame of the run is labeled and selected frames are measured for degree of counterroll.

#### FINDINGS

The findings of this study are illustrated in figures 3 through 9.

1. *Static Values.*—Figure 3 shows the mean static counterrolling curves for 6 normal subjects. Even with such a small number of subjects, the curve is very well stabilized and at most points is symmetrical to the third decimal place. Static curves for L-D subjects show virtually zero counterrolling.

2. *Dynamic Counterroll for L-D Subject.*—Figure 4 shows the results from an L-D subject rotated in the clockwise direction at 10 rpm. The greatest excursion from the zero point is  $-1.8^\circ$ , which is the same range that was obtained on this subject under static

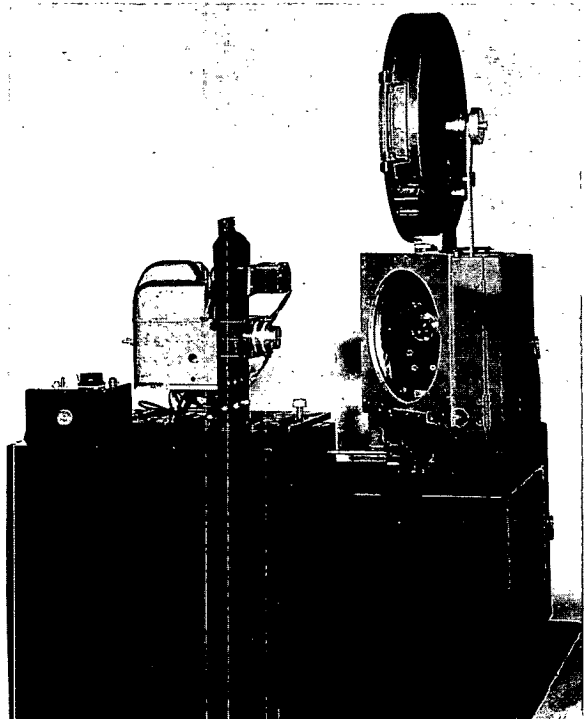


Figure 2.—Data reduction device.

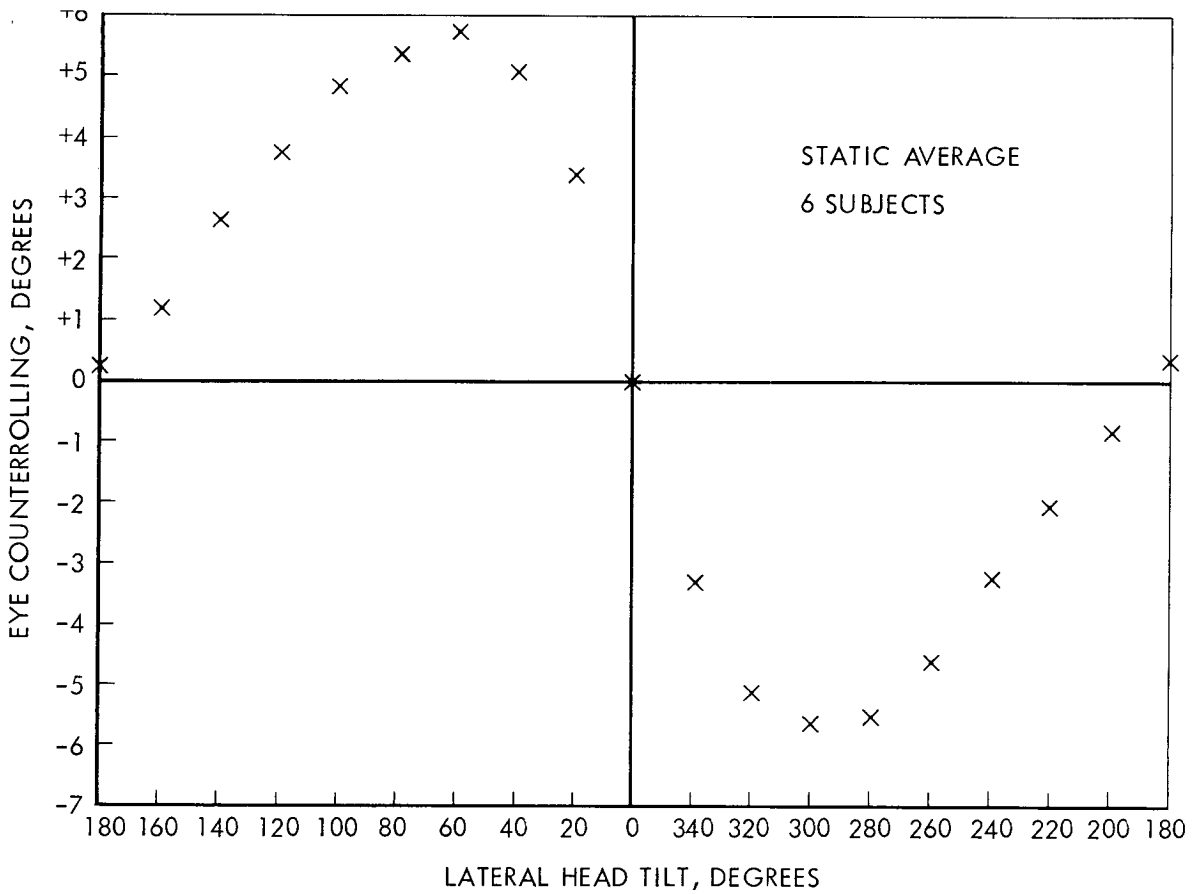


Figure 3.—Mean static counterrolling curves for six normal subjects.

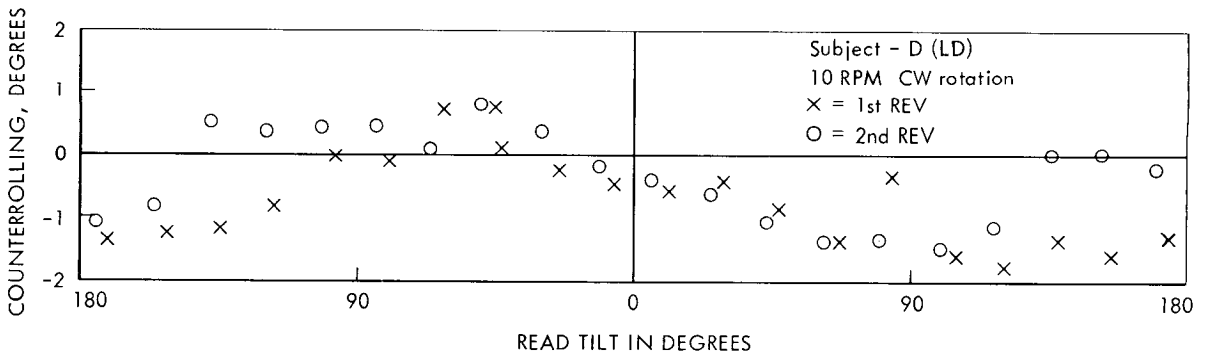


Figure 4.—Dynamic counterroll in L-D subject rotated clockwise at 10 rpm.

conditions. Figure 5 shows the same subject under prestatic conditions; that is, the subject was kept seated in the upright position and a strip of film of 50 frames was run off. These 50 frames were compared with a reference upright slide and the deviations in

minutes of arc are shown. Note that there is as much as 45 minutes of arc counterroll, without change of head position.

3. *Dynamic Counterrolling for Normal Subjects.*—Figures 6 and 7 show dynamic counterrolling curves for individual, normal

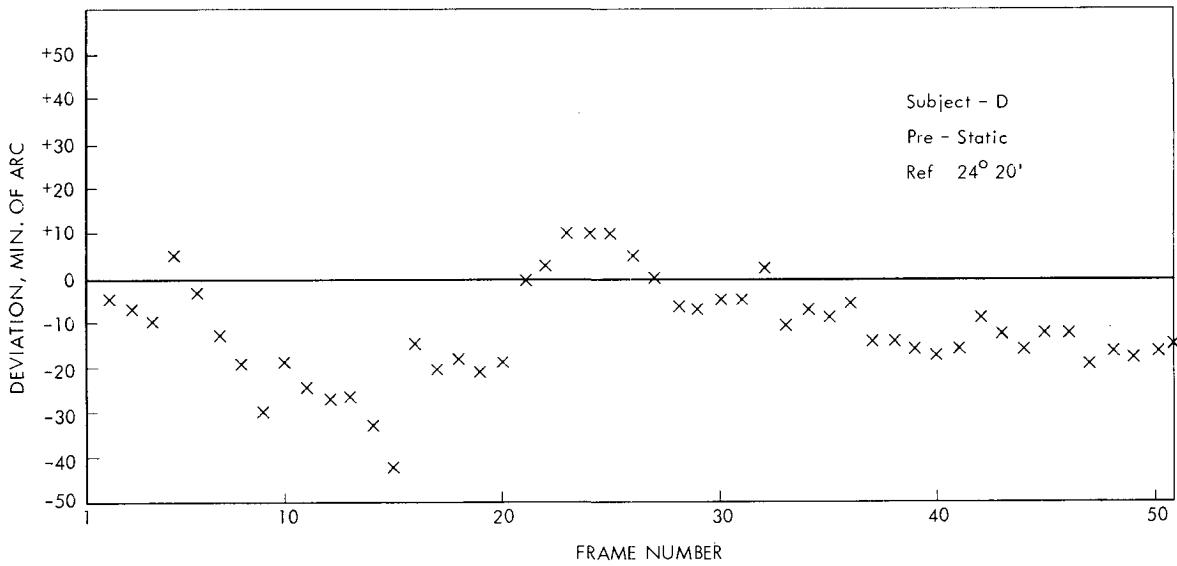


Figure 5.—*Dynamic counterroll in L-D subject under prestatic conditions.*

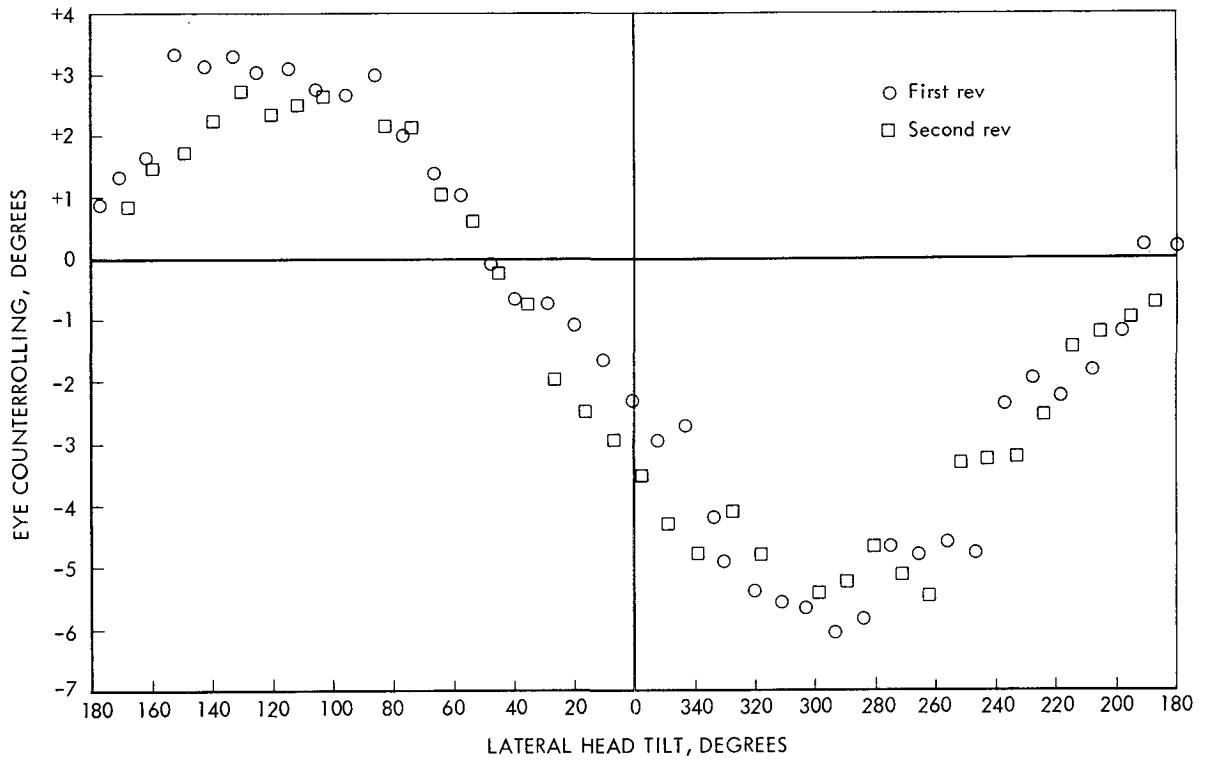


Figure 6.—*Dynamic counterrolling curves for normal subjects: clockwise rotation.*

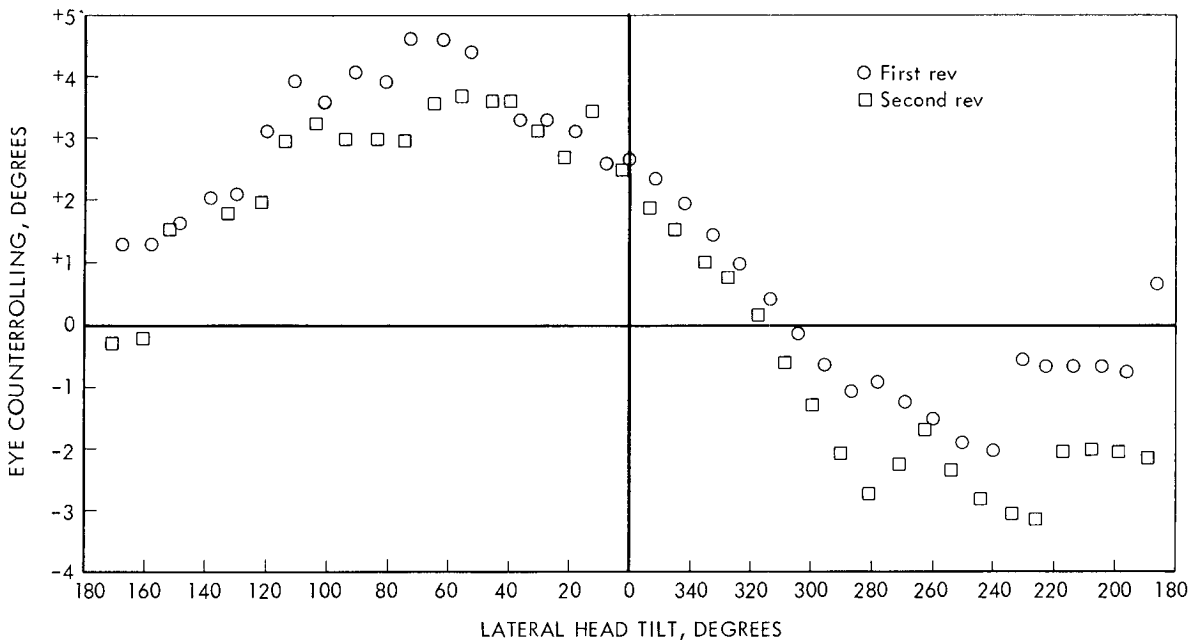


Figure 7.—Dynamic counterrolling curves for normal subjects: counterclockwise rotation.

subjects, taken at 15 rpm. Figure 6 is taken in the clockwise direction and figure 7 in the counterclockwise direction (reference direction is as the experimenter faces rotating seat). These two curves, both taken at 15 rpm, illustrate the trend of counterrolling in the dynamic state. During clockwise rotation (fig. 6) there is a phase shift to the left and a downward shift toward negative counterrolling values. In the case of counterclockwise rotation (fig. 7) just the opposite response takes place—a phase shift to the right and an upward shift toward positive counterrolling values. In both clockwise and counterclockwise rotation, there is a decrease in amplitude of both the positive and negative limbs of the curves.

4. "Normal" Subject at 15 rpm.—Figure 8 shows a very unusual response on the part of subject H. This subject showed a normal static counterrolling response, peaking around  $8^\circ$  negative and positive. However, when subjected to the dynamic state, the curve spread out to a maximum  $+8^\circ$  to  $-18^\circ$ . The phase shift along both axes remained consistent with the other normal data, but the spread of  $26^\circ$  counterroll suggests some

specific otolith sensitivity in the case of subject H.

5. Normal Subjects Composite Average.—Figure 9 is a plot of the mean values on 3 subjects for 4 rotation conditions in the counterclockwise direction. The phase shifts here are seen very clearly. Using the data from these curves, a computer study was made to produce the Fourier coefficients illustrated in figure 10. The Fourier series equation derived from rotation rates of 0, 5, 10 and 15 rpm vs. counterroll is:  $\phi = A_0 + A_1 \sin \theta + A_2 \cos \theta + A_3 \sin 2\theta + A_4 \cos 2\theta$

where the angle  $\phi$  is the angular position of the eyes with respect to the upright position and angle  $\theta$  is the angular position of the head with respect to gravity. Using this equation, theoretical average curves are presented in figure 11.

#### CONCLUDING REMARKS

Although the findings presented here are not complete at all rotation rates in both directions and although the  $N$  is relatively small, certain trends seem clear:

(1) Phase shifts are dependent upon

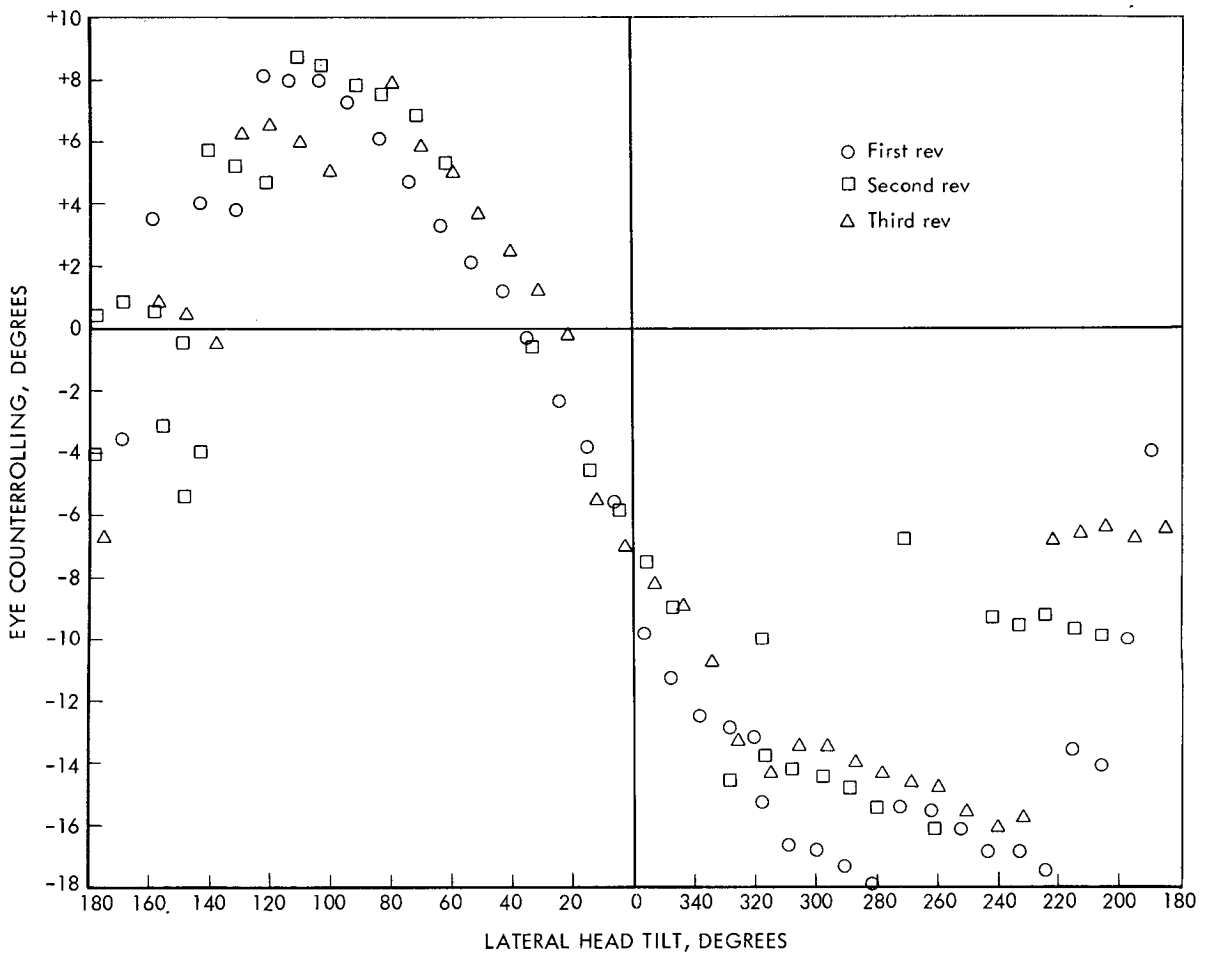


Figure 8.—*Dynamic counterrolling in subject H.*

rate and direction of rotation and follow the pattern described. This may be the result of lag time in otolith response.

- (2) The amplitude of the curves is inversely related to the rotation rates. This may be explained in terms of the

zero-G tumbling effect, which would tend toward less stimulation of the otolith with increasing rates of rotation.

- (3) L-D subjects do not show counterrolling responses either in the static or dynamic condition.

#### BIBLIOGRAPHY

GRAYBIEL, A.; AND MILLER, E. F., II: A Comparison of Ocular Counterrolling Movements Between Normal Persons and Deaf Subjects with Bilateral Labyrinthine Defects, BuMed Project MR005.13-6001, Subtask 1, Report No. 68, and NASA Order No. R-47. Naval School of Aviation Medicine, 1962.

GRAYBIEL, A.; AND WOELLNER, R. C.: A New and Objective Method for Measuring Ocular Torsion. American Journal of Ophthalmology, 47, 1959, pp. 349-352.

GRAYBIEL, A.; AND WOELLNER, R. C.: Counterrolling of the Eyes and Its Dependence on the Magnitude of Gravitational or Inertial Force Acting Laterally on the Body. Journal of Applied Physiology, 14, 1959, pp. 632-634.

MILLER, E. F., II: Counterrolling of the Human Eyes Produced by Head Tilt with Respect to Gravity. BuMed Project MR005.13-6001, Subtask 1, Report No. 75, Naval School of Aviation Medicine, 1962.

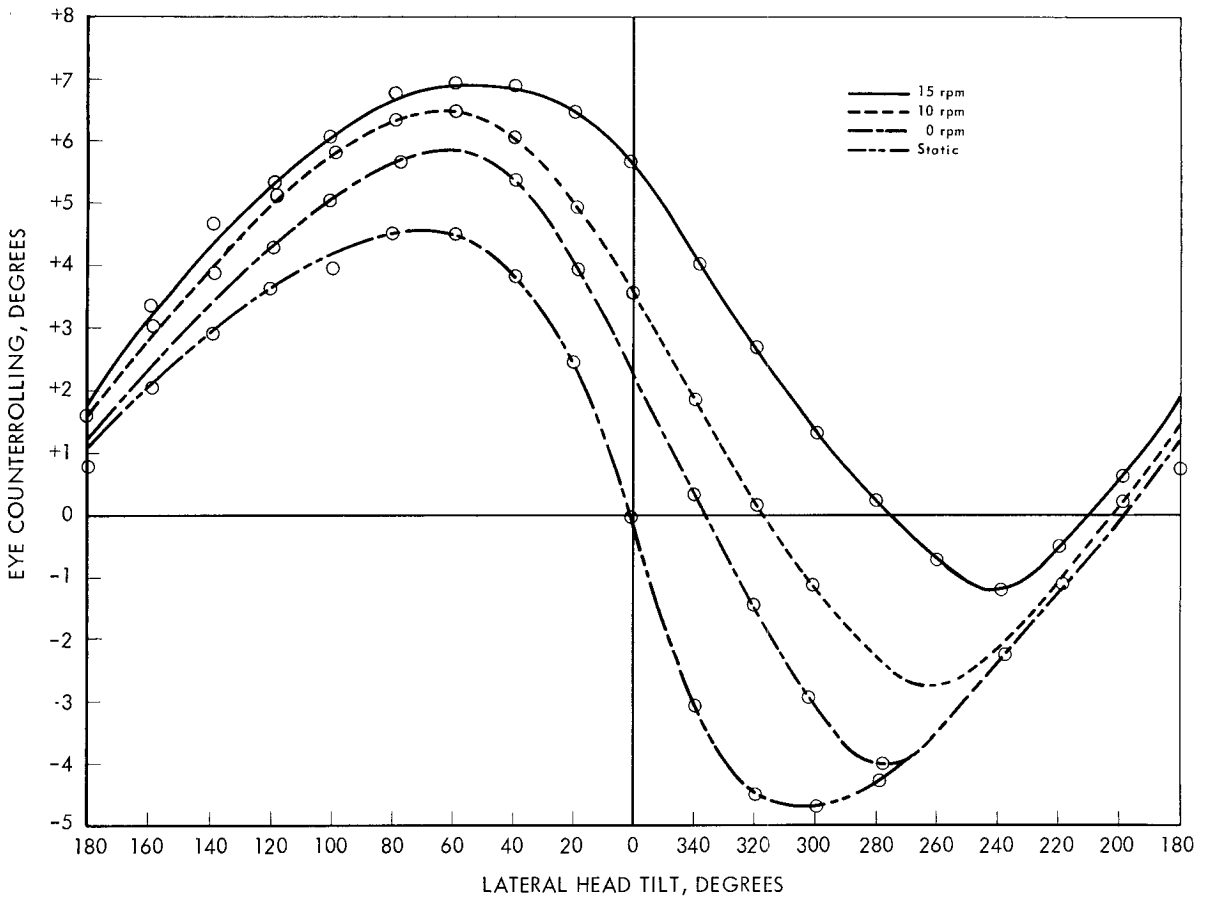


Figure 9.—Plot of mean values on three subjects for four counterclockwise rotations.

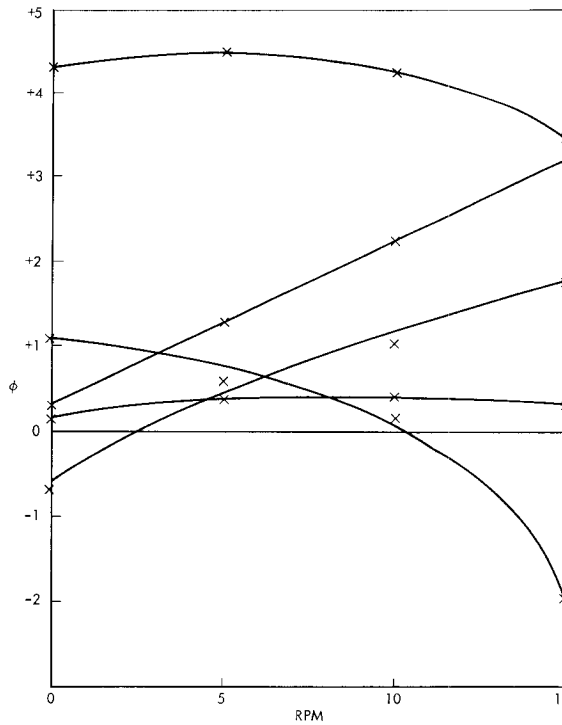


Figure 10.—Fourier coefficients versus rpm counterclockwise rotation.



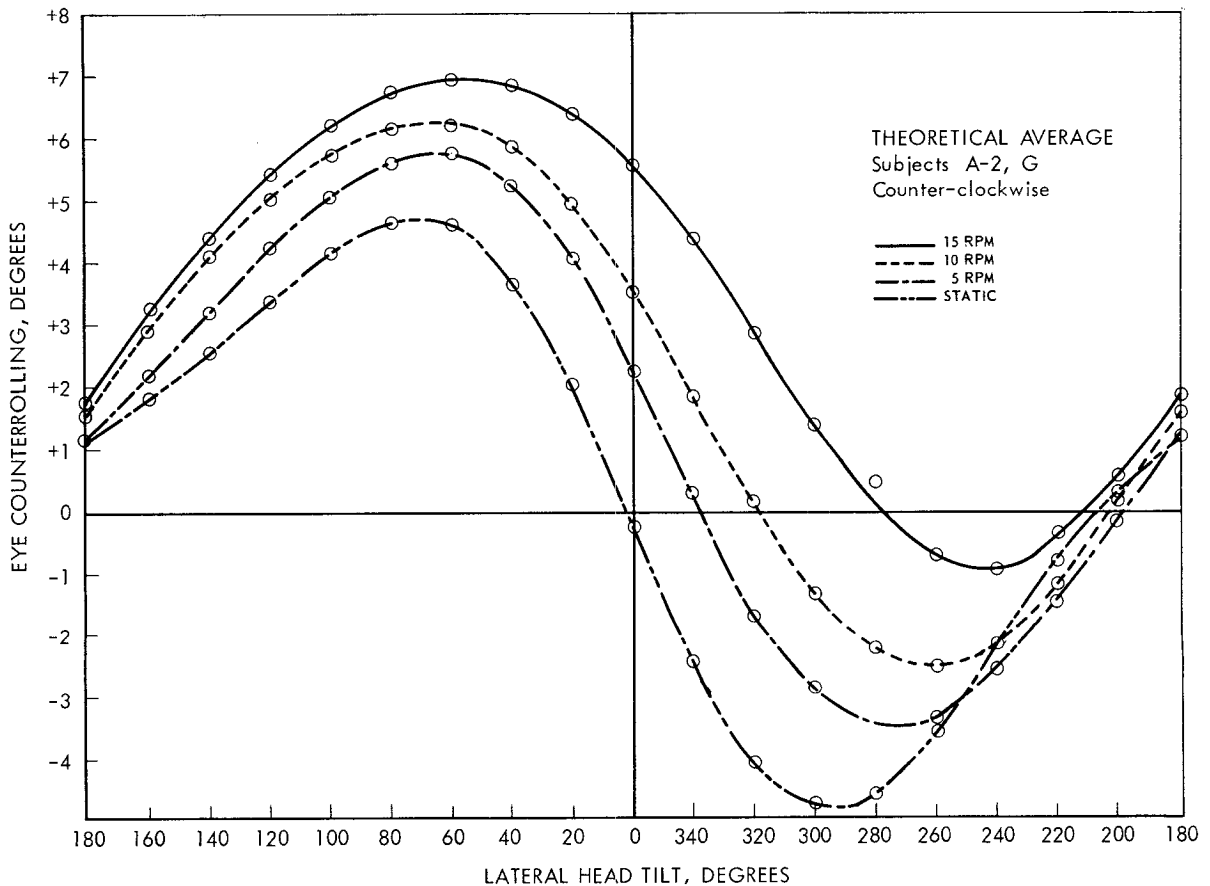


Figure 11.—Theoretical average curves.

#### DISCUSSION

**BILLINGHAM:** A couple of quick questions. What medical difficulties did you run into at 40 rpm?

**KELLOGG:** Well, I don't think they were real medical difficulties with respect to acceleration. We did have difficulties with motion sickness in several cases.

**BILLINGHAM:** The second question is if you go on increasing the number of rpm's, I presume ultimately this curve becomes completely blank and this happens at 50 rpm?

**KELLOGG:** This is a point that I should have made and didn't, and that is that the amplitude of the curve continues to decrease with increasing rates of rotation. You couldn't see it completely on these curves, but the decrease was by about 2 or 3 degrees; but it was constant through all the data. The highest rate of rotation so far used has been 30 rpm, and this was not sufficient to flatten the curve to zero. However, I would agree with your assumption that at a high enough rate of rotation the curve would approach zero.

**BILLINGHAM:** The other question was with increasing rates of rotation you should have an additional semicircular canal stimulus so I presume you do have a time period for stabilization of the canals?

**KELLOGG:** Yes, we do have a stabilization period for the first several revolutions, although in tests, we found that the canals stabilized very rapidly.

**W. SMITH:** What would you say would be the response of the individual undergoing this experiment with the addition of earphones in which interference sound levels would be used to overload his auditory channel? Would there be any displacement?

**KELLOGG:** I don't know.

**MONEY:** I'd like to inquire whether you have any data on how the lights on or darkness affects counter-rolling. Is there more or less in one situation than any other?

**KELLOGG:** We don't have any data on that. One point I might make is that when we first started out with the measurements we felt that the high intensity of light would stimulate the iris enough to close it. We found that in rotating around, that even with the high intensity of light the iris was fluctuating as much as 40%, so we had to resort to the use of 1% Pilocarpine. We keep it black, darkened out the room, during all these runs.

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## Clinical Problems Associated With Prolonged Exposure to Weightlessness

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From the inception of our national program to put man in space for prolonged periods of time, there has been a great deal of concern about the role of clinical disorders involving the vestibular system. Thus far, it has been shown that weightlessness alone does not necessarily produce any disorientation or clinical disorder of vestibular function when the individual is exposed to this state for a period of 24 hours.

In the Mercury Program there was no vertigo or other sensation of equilibratory disturbance reported by any of the astronauts, either under conditions of zero gravity or following reentry. It was reported, however, after the flight of Cosmonaut Titov that he had experienced some vertiginous sensation during the actual flight. There has been speculation that this was due to changing attitude of the capsule, possibly associated with "tumbling." In the early Soviet flights, including that of Titov, there were no means of manual control during the period while the vehicle was on the dark side of the Earth. In the U.S. flights this was accomplished by use of infrared "horizon sensors." It has been assumed that the forces created by "tumbling" of the vehicle could have produced stimuli to the vestibular apparatus, thereby causing symptoms, even though the Cosmonaut was not being influenced by the Earth's gravitational forces.

A great deal has been said and written in the public press about the disorder of equi-

librium suffered by Col. John Glenn between February and November 1964, and insinuations made relating this to his activities in the Mercury Program. It has even been said that the disturbance was a direct result of the space flight in which he took part. Once and for all, I would like to dispel any idea that these events are in any way related.

I had an opportunity to be with Col. Glenn for nearly 3 hours on a commercial airline flight 2½ weeks prior to his unfortunate accident, and saw him again as a patient in Wilford Hall Air Force Hospital 5 weeks after the accident. Col. Glenn had fallen in a bathroom in his apartment in Columbus, Ohio, while attempting to hang a large heavy mirror. The mirror began to slide from his hands, and as he tried to prevent it from falling he slipped on a mat on the bathroom floor. This resulted in a fall toward the left side in which he struck his head on the bathtub at a point just above the pinna of the left ear. He was momentarily stunned, but was not certain whether he actually lost consciousness. Someone in the next room heard the crash and immediately came to his assistance. He was lying on the floor and had numerous small cuts from the flying glass.

Almost immediately following the accident he began to experience extreme vertigo, nausea, and vomiting. This became worse when he was moved to a stretcher and carried to the ambulance, and it continued

throughout the trip to the hospital. He also quickly developed a large subgaleal hematoma. The severe vertigo, nausea, and vomiting continued for approximately 2½ days, and then gradually subsided during the next 3 weeks to the point where he had virtually no disorientation as long as his head remained in one position. Any attempt by him to turn his head, whether suddenly or slowly, produced a sensation of rotational vertigo associated with a wave of nausea. He also noted constant high-pitched tinnitus which he was unable to lateralize. There was a gradual improvement in his condition, although when examined 5 weeks later he still walked with a wide-based gait and had a tendency to veer toward the right. There was horizontal nystagmus on extreme lateral gaze in both directions, more marked toward the right. Rotation of the head to either side produced some vertiginous sensation, although not to the extent originally encountered. Extension and flexion of the neck was possible without producing any ill effects if performed slowly.

At the time of this examination, Col. Glenn was attempting to conduct a campaign for election as Senator from Ohio directly from his hospital room. There was no doubt that the tension and anxiety created by these activities were tending to aggravate rather than to alleviate the situation created by the injury. Col. Glenn was therefore advised to withdraw from his political campaign.

There was steady improvement in his condition during the ensuing 5 months, while he was restricted to a program of gradually increasing activity. By the middle of November he had recovered virtually completely and requested permission to return to flying status. His gait disturbance was no longer apparent, and he was able to turn his head without experiencing any abnormal sensation. The tinnitus had already disappeared several months previously. It has been my opinion and the opinion of other doctors who have attended him that his symptoms were all related to trauma to

the temporal region on the left side. There was never any abnormality noted in either the auditory or the vestibular tests which were performed on several occasions, and consequently it was impossible on this basis to lateralize the lesion. In spite of the fact that X-rays failed to reveal any fracture, it was suspected that there was a "hairline fracture" through the petrous portion of the temporal bone on the left side affecting the vestibular apparatus directly. There was at no time any consideration of etiological factors other than trauma, nor any reason to suspect that space flight had increased his susceptibility to vestibular dysfunction.

No information is available regarding the effects of prolonged periods of weightlessness, such as from 2 weeks to 6 months, on the vestibular apparatus. Except for some recent and incomplete data from the Soviet three-man flight of October 12, 1964, information is also lacking on the effects of weightlessness on other parts of the human organism under conditions of complete mobility.

The Soviet Cosmonauts on this mission were able to move freely about without the restriction of space suits. This should not imply that U.S. space scientists do not have confidence in the integrity of their capsules, but rather that they plan extravehicular maneuvers and recognize also that space suits are essential in a lunar landing.

A physician was included in the Soviet space team and assigned specific tasks for the purpose of obtaining biomedical information. Whether his presence in the ship offers advantages over sophisticated telemetry is subject to question. It must be remembered, however, that he could move freely about the cabin without being hampered by a space suit. I quote from the official Soviet report as follows:

Dr. Yegorov takes blood samples for analysis and records our blood pressures from time to time. He makes vestibular tests on our ear canals to determine how the organs of balance in our inner ears are reacting. He records bioelectric waves of the brain and the electrical potentials associated with volun-

tary and involuntary movements of the eyes; he determines the coordination of movements from our handwriting; he collects data on muscular efficiency by having us perform rhythmical movements of the wrist.

Our space ship is almost continually changing its position by rotation in order to present first one side and then the other to the sun so that the side heated by the sun is periodically cooled in the space ship's own shadow. Since the team is in a state of weightlessness this rotation is not perceptible to us. The only indication of it is the angular motion of the ship relative to the stars, the sun and the earth. However, at any time the commander of the ship, by using a manual control, can reorient his craft as the circumstances dictate.

One would surmise that the rotation described would have to occur only once every 30 to 60 minutes and would therefore be of no importance in stimulating the vestibular apparatus.

The Soviets say further, "It is very easy to move about the cabin in a state of weightlessness and carry on one's work. However, weightlessness is not a state like *nirvana*, but is actually a strain on the entire organism even though a man expends less energy in muscular effort."

Although this is a symposium on the vestibular organs and some of us working in the field become seduced by a kind of "target fascination," it is very difficult to discuss the problem of weightlessness as it relates to the vestibular apparatus only. Undoubtedly vestibular function is also associated in several ways with proper functioning of other systems. Circulation is known to be profoundly affected by prolonged periods of immobilization and is assumed to be similarly affected by weightlessness. Under both circumstances there is a prolongation of mechanical systole and one would surmise that this produces some alteration in mean blood pressure and perhaps in pulsatile flow in peripheral arteries. Consequently, remote effects of systemic circulatory change must be expected. Under conditions of zero gravity, one occasionally encounters a preponderance of vagal influence which results in bradycardia and gastrointestinal disturbance. This may produce nausea and a sense

of uneasiness which could readily be mistaken for the autonomic manifestations associated with vestibular sickness; yet it is completely unrelated to the vestibule.

Not only is it clear that it takes a considerable adjustment of the heart and the remainder of the vascular system to adapt to the weightless state, but there is in addition the problem of rapid adjustment upon return to the Earth's gravitational forces following reentry.

I have recently on several occasions described the mechanisms by which vestibular function may be disturbed by sudden alteration in circulation to the structures within the posterior fossa of the cranial cavity. One of the more important considerations is the relationship of head and neck position to the trunk and extremities. It is well known that flow through the cervical vessels is impeded by hyperextension and extreme rotation of the head and neck. When one of these postures is assumed, even more profound changes in flow and arterial pressure may occur when the entire body is tilted either to or from the horizontal plane. These changes may produce orthostatic hypotension, but in young individuals with elastic arteries other mechanisms must be involved as well. All of our experience in this field has been in acute situations, and consequently we have no knowledge of the effects of prolonged alteration in systemic circulation on the vestibular apparatus. It is reasonable to expect that satisfactory adaptation can occur if the circulatory changes are sufficiently gradual, but it is doubtful that adaptation to rapid change can take place without some symptoms, transient or otherwise. Certainly adaptation is a well recognized factor in both motion sickness produced by spontaneous changes in environmental forces and with those produced by creating artificial environmental forces. When there is no horizon present, adaptation occurs much more rapidly.

According to Graybiel, normal subjects exposed to a constantly rotating environment adapt quite regularly to rates below 3 rpm

and some trained individuals have virtually no symptoms at higher rates. It appears doubtful from his experiments that even the most hardy individuals who perform well at lower rates of rotation can remain free of symptoms at 8 to 10 rpm. There are, however, several observations which are important in respect to the adaptation process. Symptoms are minimized by covering the subject's eyes or by keeping him alert and active. Adaptation occurs more rapidly under these circumstances. It is also important to note that the more complete the adaptation, the more severe the symptoms following cessation of rotation. Furthermore there is considerable individual variation in speed of adaptation and severity of subjective symptoms. With this in mind, it becomes important to do a thorough pre-flight evaluation of each astronaut candidate.

Once the individual is removed from the forces of acceleration to which he has become adapted and a horizon or other new images are introduced, he may suddenly become spatially disoriented and experience ataxia, vertigo, and even autonomic disturbances characteristically associated with vestibular sickness.

Vestibular sickness after the termination of a space flight may be somewhat analogous to the post-rotation effects observed in subjects after removal from the Slow Rotation Room. The individual variation in post-rotation effects is considerable. There is no doubt that the severity of the stress as well as the length of time during which the subject is exposed are both important factors. However, some persons exposed to severe stress for short periods will have greater post-rotation symptoms than those exposed to a lesser stress for a more prolonged period. There is also considerable individual variation in both symptomatic response and general adaptation to forces of varying acceleration and velocity.

When one speculates on the possibility of vestibular disorders occurring during prolonged space flight, it seems highly unlikely that these would be of serious consequence during orbital flight, but there could conceivably be problems in a manned space station. There are, however, other clinical problems of far greater significance which need to be anticipated and overcome both during the flight and upon reentry. The likelihood of experiencing vertigo upon reentry after a long flight is quite good, and one would assume that this is related to insufficient time for proper readaptation and to the disturbances associated with alterations in systemic circulation which produce severe orthostatic hypotension and syncope.

It has been demonstrated that high intensity sound levels at specific frequencies may not only damage the cochlea, but may also produce serious ill effects to the vestibular apparatus. The Nova rocket, which is the planned launch vehicle for future manned space flights, produces high intensity sound in the range below 60 cycles. This is a serious problem even for persons on the ground closer than 2 miles from the point of lift-off. It is obviously of greater concern to the men inside the capsule to be launched. This is a problem which must be solved before astronauts can be launched as part of a payload considerably greater than that which is possible with present launch vehicles.

It is evident from these brief remarks that anything said about this problem is largely speculative. No one has been subjected to zero gravity conditions for a period longer than 4 or 5 days, and consequently there is no body of data upon which to rely. It is obvious to all persons concerned with these problems that one cannot simulate on Earth the conditions encountered in space. The effects on the vestibular mechanism under these conditions is only one of many unsolved problems.

*DISCUSSION*

**BILLINGHAM:** We're grateful to you for laying to rest this bogey about the connection between the space flights of specific astronauts and vestibular after-effects. I would like to endorse your statement that effects of serious consequence cannot at this moment be predicted for manned space flight.

The noise problem we have under investigation in some detail and this is a very interesting and important area. The Apollo vehicle itself is producing noise levels and frequencies where the intensities are very high and the frequencies are low; we have conducted in combination with the people at Baylor University, College of Medicine, a study at the Manned Spacecraft Center in which we exposed subjects in a box and just pumped air in and out of the

box (this is low frequency noise). We got up to 144 decibels in the range of 2 to 14 cycles per second, and the only problem we had was a temporary threshold shift for test frequencies of about 3000 cycles per second. This was our own small effort to satisfy ourselves that the Apollo launch noise environment would not, in fact, be detrimental to the crew during that fairly crucial phase of the mission. Now, going on to the larger vehicles, people exposed outside the vehicles, but particularly in our case the people exposed inside the vehicles, we are turning to Dr. Von Gierke, who has an extensive program underway to establish tolerance data in the complete frequency spectrum right down to 2, 5, 10 cycles per second and at higher intensities.

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# Space-Based Centrifuge

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## SUMMARY

Engineering and biomedical studies are in progress to define the research, therapeutic and training potential of a space-based centrifuge. A brief review of these studies indicates: (a) the short-radius centrifuge is the only practical method of producing a valid acceleration in space; (b) that a steep head-to-foot acceleration gradient neither precludes the measurement of tolerance to positive acceleration nor do the high rates of rotation produce motion sickness problems for the well-trained individual; (c) cardiovascular deterioration produced by recumbency is largely prevented by periodic centrifugation.

## INTRODUCTION

How dependent is man on the force of gravity for his life and health? Gravity is such a ubiquitous force in normal physiological function that very little is known about the biological effects of its removal. This is one question that can be answered satisfactorily only in an orbital research laboratory. Moreover, an answer is requisite for the planning of more ambitious flights—a trip to Mars, for example, or a stay of many months in space. Cogent research requires the production of a physiologically valid acceleration, and as yet, the centrifuge is the only practical method of producing a valid acceleration environment in space. The feasibility of including a short-radius centrifuge in different sizes of Earth-orbiting laboratories is under study both from engineering and biomedical points of view.

## ENGINEERING STUDIES

A system comparison and selection study conducted for NASA's Langley Research Center includes an orbital space station designed to accommodate a centrifuge with a radius of 104 inches. The internal centrifuge is installed in the midsection of the station (fig. 1). The centrifuge rotates about a

stationary center hub. Torque for turning the structure is applied through an idler wheel and friction drive. A counter-momentum wheel is provided too, in order to minimize stabilization and control system coupling, and to null the net torque about the laboratory's roll axis. Each cab incorporates a couch, adjustable for each crew member from the sitting to supine position. Structure and drive are sized to simulate the acceleration profile of Gemini during

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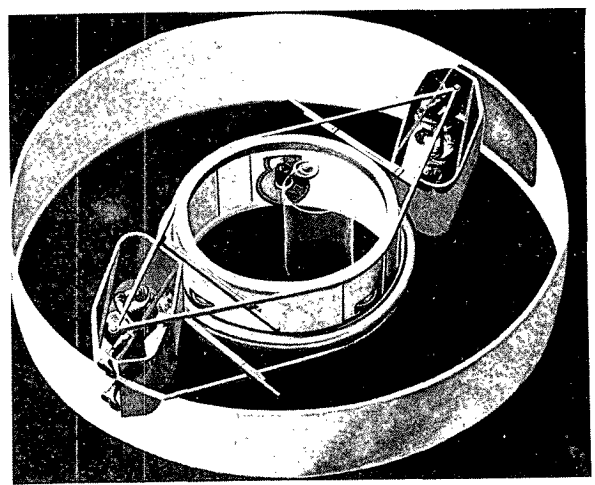


Figure 1.—Internal centrifuge.

reentry. The weight of the centrifuge structure and drive mechanism is estimated to be between 200 and 300 pounds.

For a larger orbital laboratory, in design for NASA's Manned Spacecraft Center, an eight-man counter-rotating centrifuge is proposed (fig. 2). The eight cabs are designed to accommodate an astronaut, space suit and backpack. Task simulation and control panels are included either for research or training purposes. Estimated weight of this design is 1000 pounds; requires 4.5 kW of peak power; produces a minimum of 6G, applied at a rate of 0.07 G/sec.

As a part of Douglas' man-in-space studies, centrifuges with radii of the order of 30 inches are currently under investigation as part of our Biodynamic Program. Biomedical experimentation parallels these engineering analyses. During the past year, over 2000 experimental runs have been made on the human centrifuge using specially selected and trained student "centrinauts."

#### BIOMEDICAL STUDIES

Clearly, studies are needed to determine the effectiveness of the short-radius centrifuge for studying gravity-influenced physiological systems, or the deleterious effects, if any, on man of exposures to large heart-to-foot acceleration gradients.

A major physiological consideration in the design of a centrifuge is arriving at an acceptable trade-off between radius of rotation and angular velocity. The former determines the acceleration gradient across the man (and his work area), while the latter defines the Coriolis force produced by a movement of the arm or leg (fig. 3). Lengthening the radius reduces the Coriolis force produced by a movement of the arm (velocity of arm movement 10 ft/sec) asymptotically. Shortening the radius and maintaining the same level of acceleration increases Coriolis acceleration. For example, at a steady-state of acceleration of 2.5 G the Coriolis would be 2.1 G at a radius of 7 feet and 0.9 G at a 40 foot radius. For the short-radius example, Coriolis would represent 84% of the force environment produced by centripetal acceleration, and, for the longer radius centrifuge, 36% of the force.

A very steep acceleration gradient exists if man's body is a significant length with respect to the radius of the centrifuge. If, for example, the heart were 15 inches from the center of rotation and the feet 54 inches, a 225% heart-to-foot gradient would exist (fig. 4). Lengthening the radius to 15 feet would produce a heart-to-foot gradient of 30%. The combination of gradient and

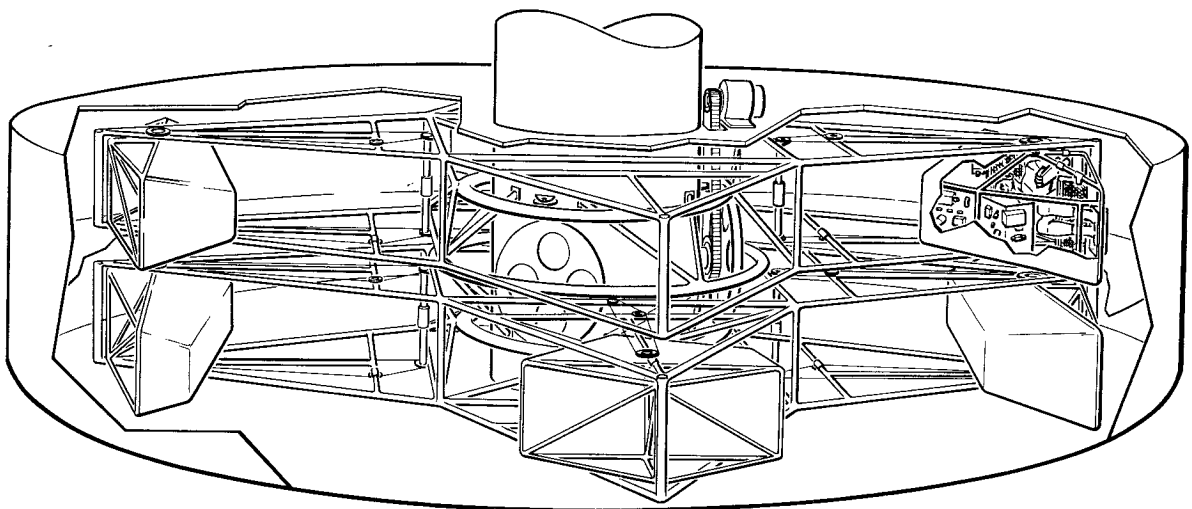


Figure 2.—Centrifuge.



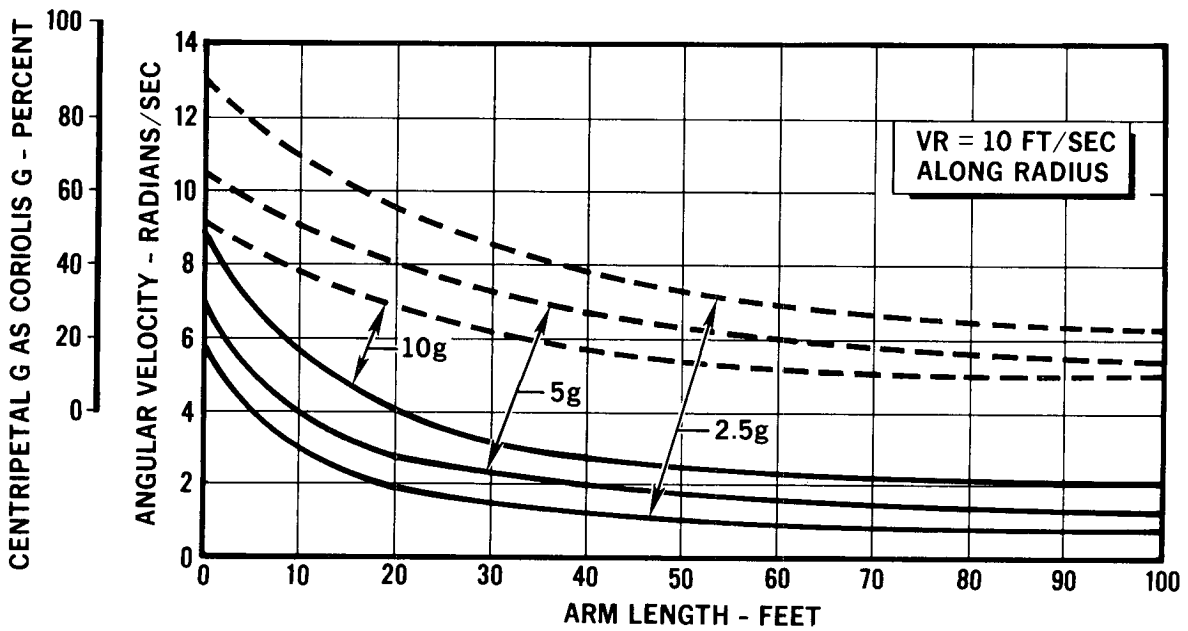


Figure 3.—Arm length, angular velocity and Coriolis G for four levels of constant G.

Coriolis acceleration produces a force environment in which an astronaut would feel a constant side force superimposed upon a varying accelerative force as he reached

for switches and operated controls. The resultant would continually change in both magnitude and direction as his limbs are moved. The consequences of these two fac-

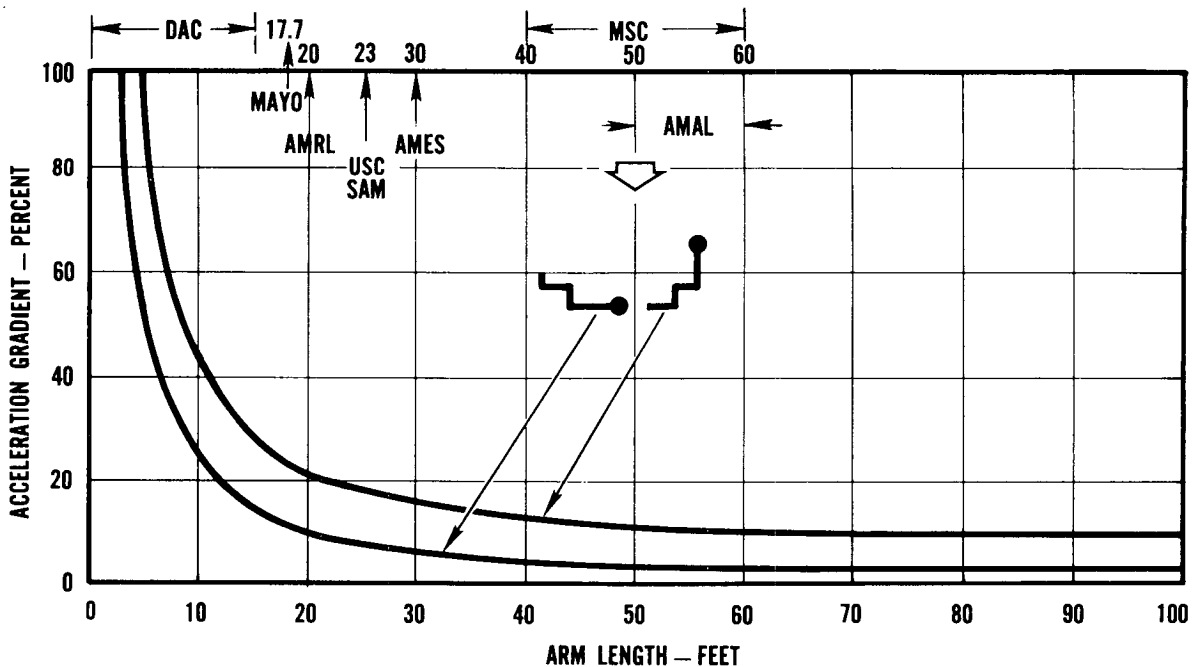


Figure 4.—The acceleration gradient across body for different arm lengths and body positions.

tors for performance and physiology are currently under study.

Tolerance to positive acceleration ( $+G_z$ ) is being studied at radii corresponding to gradients of 30% to 250% (fig. 5). Blackout is that level of acceleration (referenced to the heart) at which a central light disappears. At all radii except 30 inches, the average level of acceleration at blackout corresponds with data obtained in conventional centrifuges. At 30 inches, where the gradient is 250%, determination of tolerance was not possible because of discomfort in the feet and legs. At 3.6 G, the average discomfort level, acceleration at the feet is about 6.5 G. Reduction in the intensity of the light was used to measure blackout at a lower level of acceleration than was used with the conventional high intensity central light. Thus, it is possible to measure tolerance to positive acceleration on a short-radius centrifuge.

It is prudent at this time, in view of the cardiovascular deconditioning seen in both American and Russian astronauts during

post-flight medical examinations, to consider the use of an onboard centrifuge in controlling physiological adaptative changes resulting from space flight. In a series of studies, bedrest was used to produce the symptoms of deconditioning. The protocol of these experiments called for 20 days of recumbency (deconditioning) followed by 21 days of bed rest with periodic rides on a short-radius centrifuge (reconditioning). At the end of 20 days, the subjects showed a deterioration in the mechanism essential for adequate circulation in the erect position as evidenced by syncope and declines in plasma volumes. From the 21st to the 41st day, the subjects rode the centrifuge four times each day; the duration of each ride was 7.5 min. The level of acceleration was  $+1 G_z$  for one group and  $+4 G_z$  for the other. The acceleration gradient was 225%. The deterioration produced by recumbency in the ability of the subjects to maintain the upright position was largely prevented by centrifugation. Subjects exposed to  $+4 G_z$  four times each day showed less lability in blood pressure

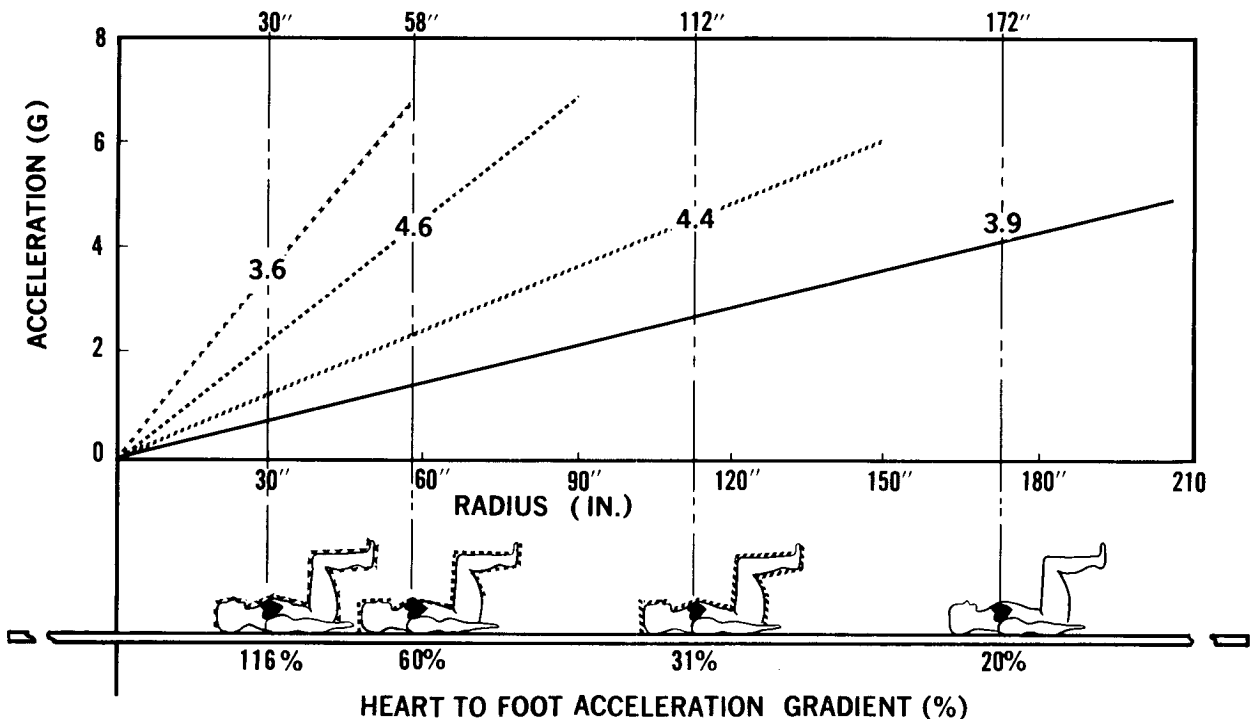


Figure 5.—Force environment at different centrifuge radii.

and heart rate during tilt tests than did the subjects exposed to  $+1 G_z$ . In other words, for 0.5 to 2.0 G hours, a deterioration of cardiovascular system can be reversed.

Centrifuge data also indicate that a steep head-to-foot gradient does not preclude movement of the head, arms and legs; and that motion sickness is not a problem for the well-trained individual when exposed to high rates of rotation and modest head or limb movements. The onboard centrifuge appears to be an attractive alternative to space station rotation in maintaining cardiovascular tone.

#### DISCUSSION

**YORK:** Under the protective measures, under drugs, you mentioned a "G" pill. What is a "G" pill?

**WHITE:** A "G" pill is an idea that never dies. When I was at Wright Field in the Aeromedical Laboratory, people would send in various kinds of pills with the claim that they increased "G" tolerance. It's sort of a fiction. It's just a good way, I guess, of speaking of drugs. We're doing no work on this, but some consideration is given by others to the possibility of drugs being used in this situation.

**KNOBLOCK:** Two questions regarding the chemistry studies. First, did you have an adequate control of your dietary regimen to really assess the loss or utilization of calcium during the 41-day period in terms of bedrest? And, second, what parameters did you measure in terms of blood volume and including perhaps the hemoconcentration problems?

**WHITE:** In answer to your first question: This was not a metabolic study by any stretch of the imagination. We did measure urinary calcium and serum calcium during the course of the experiment. Dur-

The research potential of the onboard centrifuge is also being reckoned. Its merits as a device for measuring tolerance to acceleration, especially reentry G, for stimulating the otolith organs in space, and for determining orthostatic tolerance are apparent. A "two radius" method for measuring body mass is being examined experimentally. Accuracy of the method is placed at  $\pm 1\%$  for a 180 lb man. With this method, the distance from the center of rotation to the center of mass of the man, a value difficult if not impossible to determine, is not required.

ing the 41-day period, we saw very little increase in calcium in either of these media. One subject did show a little bit more calcium output than the rest, but right now we are sort of treating this as a tentative finding. Concerning your second question, in this particular experiment we used Evans blue for plasma volume determinations and used the hematocrit for calculating the total blood volume. In the current study we are using  $I^{131}$  for plasma determinations and tritium for total body water and  $S^{35}$  for extracellular fluids.

**C. CLARK:** In getting up to the 50 rpm, how long did you take? Were there vestibular complaints?

**WHITE:** No, there were no vestibular complaints per se. I think here again this is the difference in the well-seasoned and experienced centrifuge rider. We went up at a rate of about two-tenths of a G per second; so, it was fairly slow. From the time the centrifuge started to when we reached peak G, they always had bioassay lights coming on and off. It took about a minute or less to reach 50 rpm.

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## Some Vestibular Responses Pertaining to Space Travel

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AND

ASHTON GRAYBIEL

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It is well known that appropriate stimulation of the non-auditory labyrinth can result in motion sickness and spatial disorientation, and it is almost unnecessary to point out that these may reduce the efficiency and indeed jeopardize the very survival of astronauts. A complete understanding of both the characteristics of vestibular stimulation and the resulting physiologic responses, as applicable to space flight, is essential to insure, as fully as possible, efficient performance by the space traveler. It is the object of this presentation to describe briefly some experiments that we are currently carrying out in attempts to determine the importance of the labyrinth in spatial orientation during parabolic flight.

### PROCEDURES

The importance of the labyrinth during weightlessness is being studied by comparing the reactions of normal monkeys with those which have been bilaterally or unilaterally labyrinthectomized. In order to determine the significance of the separate sensory receptors, however, the technique developed by Money (ref. 1) was used, which permits occlusion and inactivation of individual semicircular canals without trauma to, or

interference with, the function of the remaining semicircular canals or the otoliths. The application of this procedure to the squirrel monkey (*Saimiri sciureus*) has provided fundamental information as to the function of the separate sensory vestibular receptors in higher animals and will be presented in more detail in a separate report in the near future.

Reactions of these animals and of humans to weightlessness were studied during parabolic flight in a USAF Boeing 707 aircraft. A maximum of 40 seconds of virtual weightlessness was produced by a flight pattern involving a "full parabola." Starting at 36 000 feet the aircraft dove to 26 000 feet obtaining the necessary momentum to enable completion of the zero-G parabola. This maneuver resulted in a 2.5 G force when pulling up from the dive at 26 000 feet and prior to weightlessness. The increase in G force prior to weightlessness was avoided, however, in another flight pattern ("half-parabola") which involved entering the zero-G condition from straight and level flight. This "push-over" technique reduced the zero-G period to a maximum of 15 seconds per maneuver but had the advantage of absence of increased G prior to weightlessness.

Since reflex eye movements constitute objective signs of vestibular stimulation, it was decided to examine several normal human subjects during the various stages of parabolic flight for such signs. Two methods were used, namely, direct visual observation with the aid of a 20 diopter lens ("Frenze Goggles") and moving picture photography. In the latter procedure we used the head-mounted eye camera, as developed by Melvill Jones (ref. 2).

### RESULTS

#### Some Reactions of Monkeys to Weightlessness

Whole body orientation of various monkey preparations exposed to weightlessness was recorded photographically. The results can be summarized as follows:

*Normal animals.*—Two animals continuously ran around in circles (mostly vertically, but sometimes horizontally and diagonally), being held against sides of container (4 feet  $\times$  3 feet  $\times$  3 feet) by centrifugal force. This was also characteristic of these monkeys in a normal 1 G environment. The third animal "floated slowly" within the confines of the container, often changing direction but with a certain degree of stability. There was a decided tendency to spread the limbs as if attempting to execute a four-point landing on any of the six sides of the container, it being apparent that the animal had no special preference as to where the landing was to be made. The head was oriented so that the animal's face was directed toward the nearest landing surface.

That these animals were susceptible to laboratory-induced motion sickness had been established by prior exposure to appropriate acceleration in a rotating room (ref. 3). None of them, however, showed any evidence of sickness during these parabolic flights.

*Bilaterally labyrinthectomized animals.*—These monkeys all exhibited a noticeable attempt to remain attached to any surface. Once detached, they floated around in an unstable fashion, tumbling or looping in a head-over-heels fashion, frequently landing on their backs. Sometimes, while free floating, they rotated slowly around the long axis

of the body, or floated upside down, while at other times remained vertical either in a head-upward or head-downward position.

*All six canals inactivated but otoliths normal.*—One animal resembled the normal monkeys in that it would continually run around in 360° circles (horizontally, vertically or diagonally), apparently also maintaining orientation by centrifugal force thereby produced. Limb movements were well coordinated, and the animal was under good control if it continued the circling movements.

Another monkey performed at times as the animal above, but also floated around with all four limbs extended and with determined attempts at orientation on the nearest surface of the container. This animal, however, differed from the normal in that sudden turns of its body would result in loss of control and the animal would then be flipped over unintentionally; this was never seen in the normal animal.

*Unilaterally labyrinthectomized animals.*—The most noticeable reaction of these animals to zero-G was the almost continuous and rapid spinning around the long axis of the body, the direction of rotation being always toward the operated side, i.e., toward the animal's left in those animals with a left-sided labyrinthectomy, while the rotation was toward the animal's right when the right labyrinth had been removed. Such spinning was never observed in the other animals, including the normal monkeys, and was probably due to imbalance of the afferent discharges of the two eighth cranial nerves, although the animals underwent the unilateral labyrinthectomy more than 10 months previously.

#### Vestibular Responses of Human Subjects During Parabolic Flight

In the human participants the only evidence of stimulation of the organ of balance at any time during parabolic flight consisted of eye deviations developed during the pre- and post-zero G periods, and such stimulation was undoubtedly the result of the centrifugal forces occurring at these times. These eye movements were strikingly simi-

lar to those recorded by the same head-mounted camera in laboratory induced otolithic stimulation. This latter procedure has only recently been described (ref. 4) and involves exposing human subjects to changing direction of centrifugal force by a device which revolves the subject around an axis in such a way that he faces continuously in the same direction. The motion of this device does not cause semicircular canal stimulation, as evidenced by study of a model of the semicircular canals and by lack of nystagmus and subjective vertigo in human subjects.

#### DISCUSSION AND CONCLUSIONS

It has previously been pointed out (ref. 5) that weightlessness alone is unlikely to cause motion sickness. These more recent findings, with both monkeys and human beings, confirm this opinion.

All the monkeys used in these experiments were originally susceptible to laboratory-induced motion sickness and, as previously reported (ref. 3), bilateral labyrinthectomy renders them immune to this malady. Similar evidence as to the importance of the organ of balance in the susceptibility of humans to motion sickness has been obtained using subjects with bilateral labyrinthine defects (ref. 6). As to the relative effectiveness of the semicircular canals and of the otolithic receptors in causing motion sickness, there is ample evidence (ref. 5) to show that semicircular canal stimulation is of prime importance. This has just been confirmed for the squirrel monkey (ref. 7), in which it was found that animals susceptible

preoperatively no longer exhibited any signs of motion sickness after the canals had been inactivated, although normal otolith function was retained.

It is considered that any motion sickness which has occurred in parabolic flight involving aircraft or orbital flight involving space capsules (e.g., the Russian Cosmonaut, Titov (ref. 8)) has apparently been due to appropriate multiplanar angular accelerations occurring as the result of concomitant head movement superimposed on aircraft or space capsule rotation or as the result of the space vehicle itself undergoing appropriate angular accelerations—conditions most likely to result in the appropriate type of semicircular canal stimulation. Another matter for concern to the space physiologist is the problem of spatial orientation which will confront the astronaut during weightlessness. The monkey experiments described in this presentation indicate serious disability in this regard when the animals were either unilaterally or bilaterally labyrinthectomized. Consequently, normally functioning labyrinths should be of value to the space traveller insofar as spatial orientation is concerned. If it is attempted to create an artificial gravity by rotating the space station, however, the organ of balance might be so stimulated as to produce incapacity due to disorientation and motion sickness. Furthermore, there is evidence to indicate that severe canal stimulation might well be accompanied by other disturbances such as antidiuresis (ref. 9), cardiovascular effects (ref. 10), hyperventilation, and possibly other functional changes still to be determined.

#### REFERENCES

1. MONEY, K. E.; AND SCOTT, J. W.: Functions of Separate Sensory Receptors of Nonauditory Labyrinth of the Cat. *Am. J. Physiol.*, 202, 1962, p. 1211.
2. JONES, G. MELVILL: Ocular Nystagmus Recorded Simultaneously in Three Orthogonal Planes. *Acta Otolaryng.*, 56, 1963, p. 619.
3. JOHNSON, W. H.; MEEK, J. C.; AND GRAYBIEL, A.: Effects of Labyrinthectomy on Canal Sickness in Squirrel Monkey. *Ann. Otol. Rhinol. and Laryng.*, 71, 1962, p. 289.
4. JOHNSON, W. H.: The Importance of the Otoliths in Disorientation. *Aerospace Med.*, 35, 1964, p. 874.
5. JOHNSON, W. H.: Some Vestibular Problems in Space Flight. *Ann. Otol. Rhinol. and Laryng.*, 70, 1961, p. 777.
6. GRAYBIEL, ASHTON: Vestibular Sickness and Some of Its Implications for Space Flight. In: *Neural Aspects of Auditory and Vestibular Disorders* (Fields and Alfred, eds.). Ch. XI. Chas. C Thomas Co., 1964.

7. NEWBERRY, P. D.; JOHNSON, W. H.; AND SMILEY, J. R.: The Effect of Hypoxic Hypoxia on Nystagmus Induced by Angular Acceleration. *Aerospace Med.*, 1965.
8. SOVIET ENVIRONMENTAL PHYSIOLOGY AND TOXICOLOGY: A Survey of the Published Literature, 1958-1964. U.S. Dept. of Commerce, Joint Publications Research Service, TT: 64-51901, Dec. 10, 1964, p. 19.
9. TAYLOR, N. B. G.; HUNTER, J.; AND JOHNSON, W. H.: Antidiuresis as a Measurement of Laboratory Induced Motion Sickness. *Can. J. Biochem. Physiol.*, 35, 1957, p. 1017.
10. SUNAHARA, F. A.; JOHNSON, W. H.; AND TAYLOR, N. B. G.: Vestibular Stimulation and Forearm Blood Flow. *Can. J. Physiol. Pharmacol.*, 42, 1964, p. 199.

### DISCUSSION

GILLINGHAM: Dr. Johnson, I am interested in your model. I wonder whether you ever observed any evidence for a Purkinje effect after the stopping of rotation. My understanding is that this effect is caused by the inertia of the endolymph, and I'm wondering whether that is a plausible explanation, i.e., whether you did observe any phenomena in your model that would support that explanation.

JOHNSON: Nodding the head forward after cessation of rotation in the normal upright or sitting position will produce a disorientation sensation of rolling sideways and downward. This is called the Purkinje effect. It is the result of an actual vestibular stimulus resulting from physical flow of endolymph which can be expressed vectorially. This effect can be seen in the model although one should remember that in life the capillary size of the membranous canals and the viscosity of the endolymph constitute a system quite different from the glass model. In the living system, very little endolymph flow occurs to enable stimulation by cupular deflection.

GUALTIEROTTI: You made the statement that the zero gravity condition is comparable to cutting the nerve, although it isn't, as it appears from the results themselves. In fact, at zero gravity you still have spontaneous activity coming from the organ and of course you have responses to movements. I just want to point out that you can't make a comparison between the section of the nerve or destruction of the labyrinth and the condition of weightlessness.

JOHNSON: As you point out, sectioning the vestibular nerves would not produce the same condition physiologically as simply removing an environmental stimulus which activates them. However, exposure to weightlessness gives valuable knowledge as to the origin of the resting discharge, and ability to maintain body orientation under such a condition merits further investigation in this regard. It was interesting to note how some of these monkeys managed more effectively in zero-G apparently by creating an artificial gravity.

THACH: Perhaps I can shed a little light on the monkeys' adjustment to weightlessness. The normal and bilaterally labyrinthectomized groups, aside from free-floating in the 48×48×54 inch Plexiglas box and hanging on in wire home cages, were confined in a smaller 10 inch square chamber where they had been thoroughly trained to press a lever for a squirt

of milk reward. The lever presses "paid off" only intermittently according to a variable interval schedule averaging one per minute. This schedule produces a reliable, steady rate of ongoing objectively measurable behavior against which to compare the 10- to 20-second periods of weightlessness.

Upon initial exposure to zero-G the normal monkeys were suppressed to just 6% of their normal response rate while the labyrinthectomized only dropped to 73% of normal. Adaptation individually and as groups proceeded irregularly across the 120 parabolas in 4 days, with the normals recovering to a final 78% of normal and the labyrinthectomized suppressing further to 52%. The labyrinthectomized monkeys were given much less exposure in the work situation and may have developed a conditioned emotional response. Incidentally, the monkeys were not very disturbed by the 2 G periods entering and exiting from the maneuver, responding with rates nearly comparable to 1 G control periods.

M. JONES: It was interesting to see that the monkey with unilateral labyrinthectomy spun round and round in the zero-G environment, when presumably he must have adapted, or rather habituated, to the one-sided removal. I imagine this animal would not show directional preponderance with conventional tests after having been left for a month with one labyrinth destroyed. And yet in the zero-G environment there seems to be perfectly clear evidence for a marked residual directional preponderance. Have you any views on this apparent discrepancy?

JOHNSON: Yes, it surprised us also in that regard. However, this behavior of running around the cage and thereby maintaining orientation was characteristic of this particular animal prior to surgery. Post-operative tests indicated successful surgery.

MONEY: I am sure, in the case of that monkey, it was at least 3 months since labyrinthectomy that the film was taken and probably closer to 6 months. I take this as evidence that adaptation, in fact, is not complete at that time. In rotary tests, in one of these animals, done at 3 months post-labyrinthectomy, there was, in fact, still evidence of preponderance; namely, a horizontal nystagmus away from the operated ear.

KELLOGG: Just one minor point. We only got about 25 seconds of zero gravity. That's when we were getting a good maneuver.

**FIELDS:** Were these single trials or were these monkeys exposed to repeated trials of this? Were some of them run more than one time?

**JOHNSON:** Yes.

**FIELDS:** The reason I ask the question is this—I wonder if there isn't some attempt by the monkey to get close to the wall during the weightless period because the moment it is subjected to G effect, it is going to go "bang" against the side of the cage. If it is close to the side at that moment, its fall will be much less severe.

**THACH:** The monkeys definitely did modify their behavior as a function of repeated exposures to zero G, however not in quite the fashion suggested by Dr. Fields. We are just beginning analysis of the films and have no quantitative data yet, but as Dr. Johnson has observed the initial reaction was to scramble for a toehold. The direction of adjustment was towards more calm, coordinated movements, postures, and floating and attending to visual cues. Also, reentry to 1 G was quite easy and gradual.

In connection with Dr. Jones' wonderment of the unilaterally operated monkeys' response, Dr. Pitt's albino rats with similar operations performed in a similar fashion, except that the unilateral rats spun faster—at better than 60 rpm—along their longitudinal axis and often beginning rotation before all their feet were clear of the floor, so that their tail would swing around and slap the floor several times.

The easy explanation of this surprising behavior is just that, using one of Dr. Jones' favorite concepts, the entire organism is responding. A sensorily damaged animal merely learns to follow the most consistent patterns of sensory input that enable him to get along in various stimulus complexes. Remove his eyes and he relies more on audition; deafen him and he relies more on olfaction; remove, or partially remove, his semicircular canals and he relies more on vision and proprioception, under normal, regular, constant 1 G conditions. He cannot adapt to zero G without being exposed, and apparently the loss of proprioception under zero G was enough of a change in stimulus input to regress the monkey to a pre-operative or early postoperative stage.

**BILLINGHAM:** We have under consideration some of the interactions which you were talking about between the vestibular system and the cardiovascular system. Considering syncope (acute peripheral circulatory failure) in the standing position in a 1 G environment, there are many possible causes for this, and you are suggesting that violent vestibular disturbances may be one, and, of course, it is. There is a further comment if we are talking about a weightless environment, where we have a very different situation from a 1 G environment; it is very difficult I would predict, to make people faint in a weightless environment, just as it is difficult to make people faint in bed or in water.



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# Spontaneous Firing and Responses to Linear Acceleration of Single Otolith Units of the Frog During Short Periods of Weightlessness During Parabolic Flight

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Tests performed in man and animals during short periods of weightlessness in airplanes and in spacecraft have proven indirectly that alteration of the vestibular function may be observed under these conditions.

Fiorica et al. (ref. 1), recording from the vestibular nerve of a cat with micro-electrodes during 1.2 seconds of free fall, reported a large increase both of the frequency and of the amplitude of the vestibular discharge. It is felt, however, that only a comparison of records from a single vestibular unit yielding spontaneous and evoked activity in weightlessness and at 1 G, respectively, might give accurate quantitative information on the effect of zero gravity on the vestibular responses.

## TECHNIQUE

*Chronic micro-electrode recording.*—Special micro-electrodes have been designed to assure recordings from the same single nerve fiber over a long period of time and during the movements, vibrations and accelerations that are to be expected during flight. The basic principles of this technique are, first, that a body in an environment of the same density is not displaced by an

acceleration applied to the system. Secondly, if the micro-electrode is not restrained at one end, it will not vibrate at the tip, and therefore recording externally from a single nerve fiber will not be impaired by continuous micro-injury at the tip end. The electrode consists of a tungsten micro-electrode about  $\frac{1}{8}$ -inch long which has been sharpened and coated by the Hubel technique (ref. 2) and attached to the top of a long holder by means of a drop of paraffin. The electrode has been balanced against a Ringer solution for equal density by coating it with a low density material until the electrode comes into equilibrium with the agar. The electrode is connected with the main output wire (which is fixed to the bone) by a 1-mil enameled platinum wire of such a length and flexibility that it does not exert an appreciable force on the micro-electrode. The micro-electrode assembly is placed stereotaxically in the appropriate site of the vestibular nerve of the frog; then the nerve, micro-electrode and a part of the platinum wire are imbedded in agar. The output wire and the reference electrode are fixed to the bone with metallic stitches and are conducted outwards through two lateral openings in the jaw.

The tungsten micro-electrode is then re-

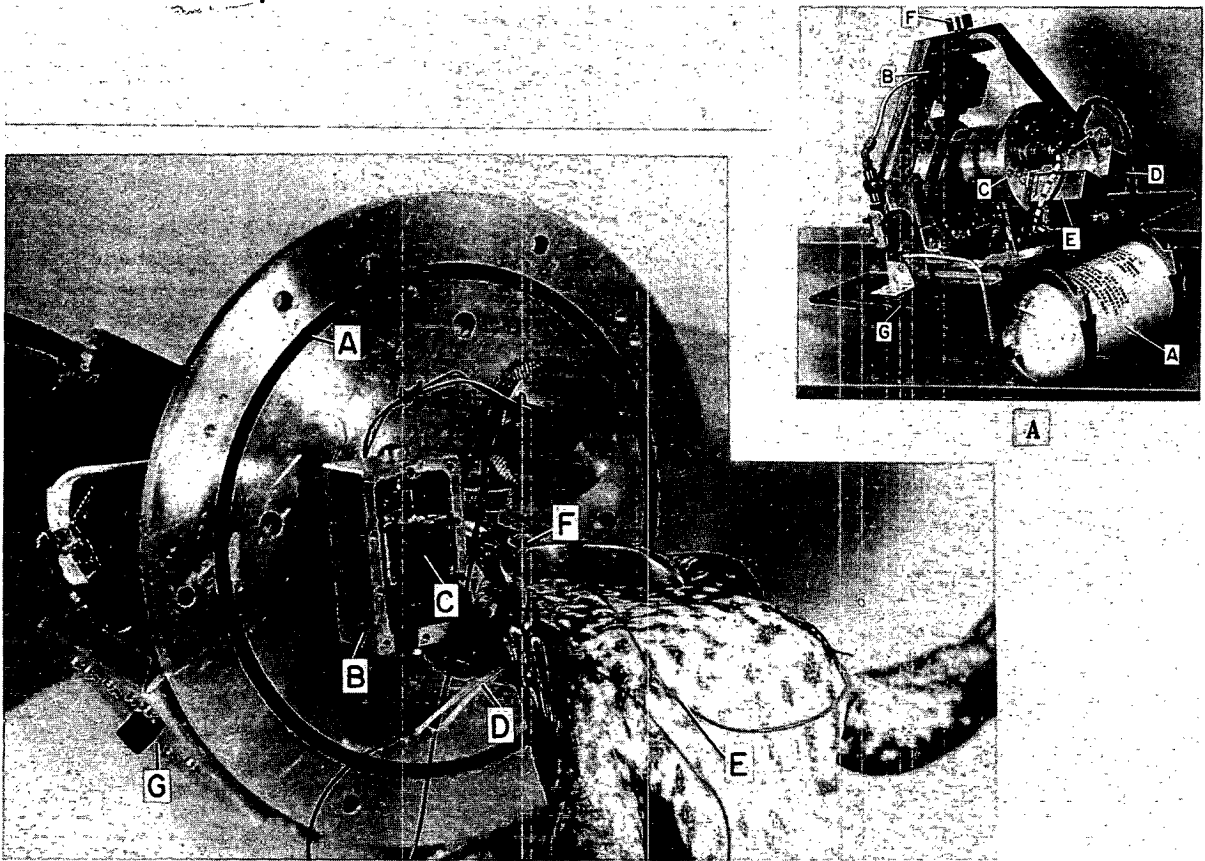


Figure 1.—Life supporting system for plane flight of curarized frog. (A) Complete biopackage ready for mounting aboard T-33A jet plane. A—oxygen supply; B—flow regulator; C—head plate; D—dual bioamplifier; E—power supply; F—exhaust filter; G—mounting plate. (B) Inside of head plate after its removal from biopackage. A—watertight gasket; B—pre-amplifier housing with its watertight cover removed; C—pre-amplifier; D—leads conducting nerve impulses to pre-amplifier; E—EKG leads; F—head support; G—dual bioamplifier.

leased and allowed to float in the agar by heating the electrode holder and thus melting the paraffin junction.

To select the proper fiber in the vestibular nerve, the animal is tested on a tilting table which can be rotated  $360^\circ$ . The animal is then placed in a cylindrical aluminum container (fig. 1) and is completely immersed in water in order to provide a buffering against vibrations, etc. Enough oxygen is bubbled through the water to assure proper oxygenation and the washing off of the  $\text{CO}_2$ . The container also provides adequate shielding against electrical noise. A two-channel amplifier fixed to the container is used for monitoring EKG and the vestibular unit

pulses; the latter are recorded through a miniaturized high impedance—low capacity preamplifier which is fixed near the head of the frog. All the information is recorded on magnetic tape for later analysis.

During the entire period the frog is curarized (Intocostrin  $10\mu/\text{kg}$  bodyweight).

During the flight the pilot is guided by three accelerometers on his instrument panel. The plane follows a Keplerian path in order to obtain zero acceleration in all directions. The stimulus is obtained by accelerating in one direction only ( $A \rightarrow X$ ). The frog is placed in such a position as to have the otolith unit being recorded respond to acceleration in that direction. To this pur-

pose the container may be placed in any position in the plane.

### RESULTS

Only the results related to vestibular units which respond to appropriate tilting with a change in the rate of firing are presented here.

Under laboratory conditions the characteristic vestibular response may be summarized as follows. The single otolith units:

- (a) fired spontaneously at a rate of 0.5–20 per second in the absence of any stimulation (fig. 2(a))
- (b) responded to appropriate stimulation (tilting) by increasing their frequency of firing following a logarithmic function according to the

Weber-Fechner law (fig. 2(b) and fig. 3)

- (c) showed a slow accommodation, but at a steady state the frequency of discharge was significantly different for the various amounts of gravity applied (fig. 3 and fig. 4)
- (d) responded to a decrease of stimulation differently than to an increase, showing a sudden suppression of firing for 2 or 3 seconds (figs. 2(c) and (d) and fig. 3)
- (e) responded with a much higher frequency of discharge when the gravitational stimulus was applied very rapidly.

An overshooting of a frequency of 150 to 200 per second appeared for a speed of tilt-

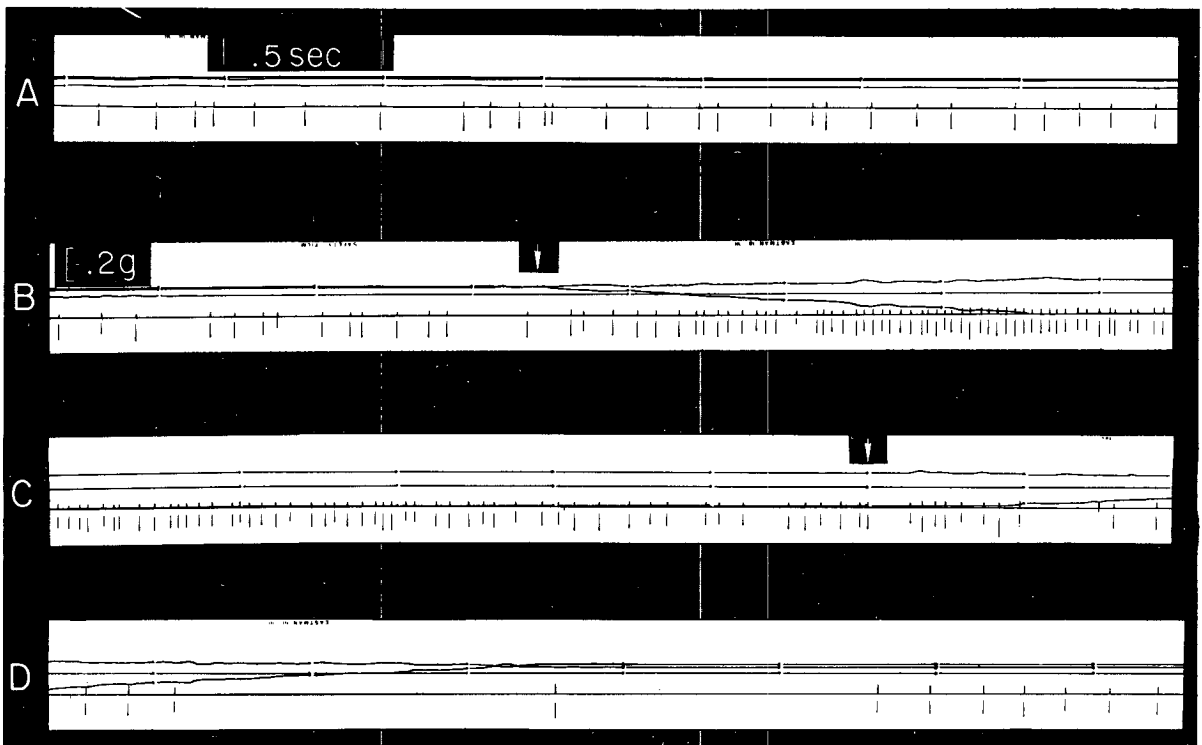


Figure 2.—Frequency changes during gravity variations. Chronically implanted microelectrode. Curarized frog. Recording from a single vestibular nerve fiber during tilting. Upper three tracings—accelerations in the three directions of space. Lower tracing—vestibular impulses. A—spontaneous discharge; B—increase in frequency during progressive tilting downwards. Horizontal accelerometer shows an increasing gravitational force (moving downwards) while frequency of vestibular pulses increases. C—animal is kept tilted. A spontaneous decrease of frequency during steady stimulus indicates accommodation. At “arrow” the tilting is decreased progressively to the horizontal. The “off” response is shown by an immediate interruption of firing (D) which is followed by a spontaneous discharge of same characteristics as in A.

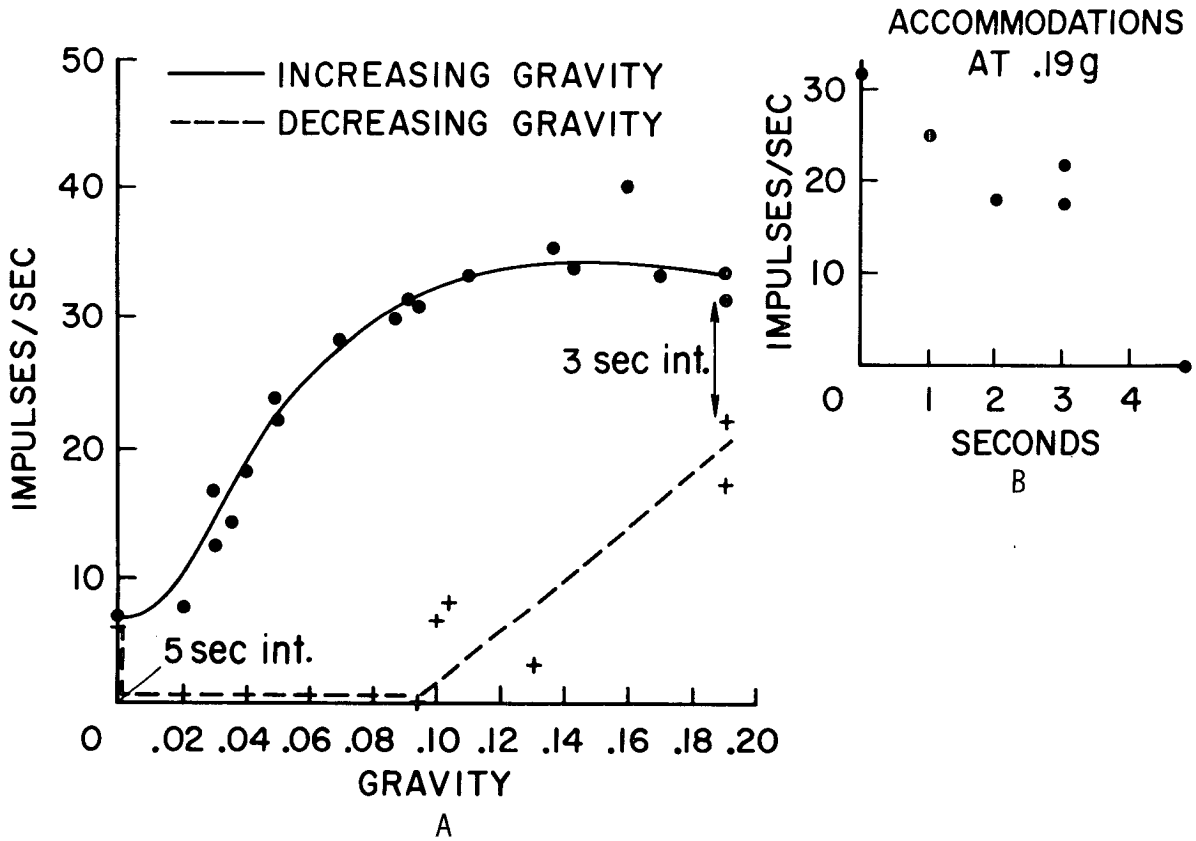


Figure 3.—Frequency changes during gravity variations. (A) Increase in rate of firing of a single vestibular fiber during tilting. On abscissa gravity values. Zero G is not absolute absence of gravity, but conventionally it corresponds to horizontal position of tilting table. Increasing value of gravitational components is indicated by horizontal accelerometer when table is tilted downwards towards horizontal. Obviously 1 G would correspond to vertical position of table. On ordinate rate of firing in reciprocal of interval per second (instantaneous frequency). Curve shown established a logarithmic ratio between rate of firing and rate of stimulus. The "off" response appears as a sudden blocking of firing when the gravitational stimulation is decreased. (B) Abscissa and ordinats as in A. Table is kept tilted in such a way that 0.19 G is applied to vestibular receptors. Accommodation is shown as a gradual decrease of frequency in time at a steady stimulation. However, after 3 seconds a steady frequency of firing is reached which is significantly different from values during spontaneous firing with no stimulus.

ing of approximately 0.2 radian/sec, whereas the same fiber showed a maximum response of 50 per second with a lower speed of tilting.

These results agree with the findings of other authors: Ross (ref. 3); Adrian (ref. 4); Löwenstein (ref. 5); Rupert et al. (ref. 6).

During level flight at 1 G both spontaneous activity and responses to acceleration are similar to the ones obtained on ground. During and after the period of near weight-

lessness the following changes are observed: (a) a burstlike increase of the rate of spontaneous firing at the beginning of 0 G followed by a higher frequency response to  $A \rightarrow X$  acceleration; (b) a sudden block of response during  $A \rightarrow X$  stimulation after approximately 10 seconds of 0 G; (c) normalization of response after going back to 1 G.

In a series of short-interval parabolic flights the most striking effect observed was a large increase in the overall nerve activity. This increase was such that the micro-

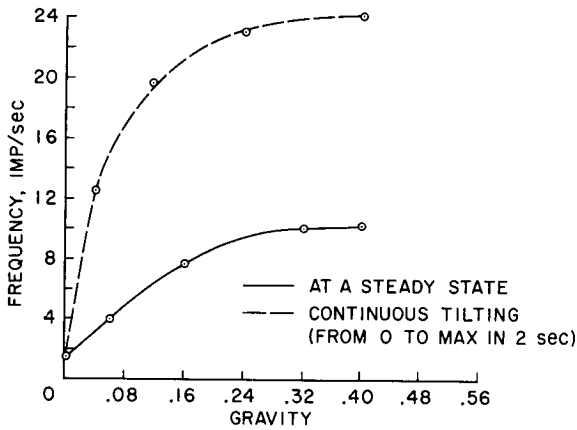


Figure 4.—Diagram showing change in rate of firing of a single otolith unit during continuous tilting and at a steady state. Logarithmic stimulus/response relationship is present in both cases, although overall frequency of discharge is much lower at a steady state.

electrode was picking up the discharges of additional fibers. But the general aspect of the response, and even the suppressory effect, were still evident. Such phenomena disappeared if the series of parabolic flights were discontinued for some minutes. Figure 5 shows the actual recording of the vestibular unit firing and of the accelerations in the three directions of space. It has to be noted that the vertical accelerometer is saturated near the 1 G value and therefore no "high G" reading during the first and last part of the parabolic flight is shown. Figure 6 indicates diagrammatically the relationship between the A→X acceleration and the rate of firing before, during and after the changes in the A→Z (vertical) acceleration from 1 G to nearly 0 G. A→Y acceleration is not shown as it remains approximately constant during this entire period. Figure 7 gives, directly, the comparison between the responses of the otolith unit to the same amount of A→X acceleration at 1 G and at approximately 0 G before suppression started. The difference of sensitivity is very evident. Figure 8 shows the recruiting effect after a number of short interval parabolic flights.

These preliminary results correspond to five flights of which only two were totally

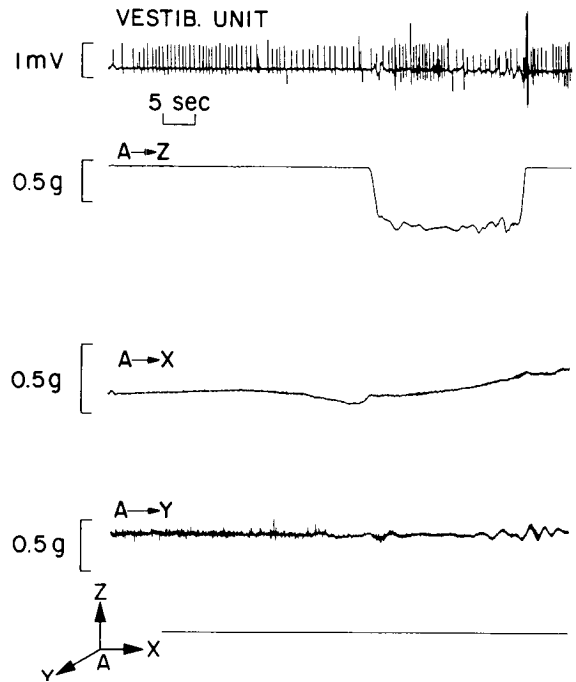


Figure 5.—Otolith unit response to linear acceleration in a Keplerian path. Chronically implanted electrode. Curarized frog. Recording from a single vestibular nerve fiber during parabolic flight. First recording (top)—vestibular impulses. Second, third, and fourth tracings—the vertical, longitudinal, and lateral accelerations, respectively. A period of 25 seconds of weightlessness is shown by downward deflection of the A→Z recording (vertical acceleration). Appropriate stimulation for vestibular unit consists of a positive acceleration in A→X direction only. As shown, stimulation has been applied twice during level flight at 1 G and during the 25 seconds of weightlessness. Frequency response of unit appears to be normal during level flight and is greatly increased during first 10 seconds of weightlessness. After this period a sudden reduction of the rate of firing is observed. As soon as level flight is resumed, normal response is again shown by unit. Spontaneous firing shows a much higher frequency during weightlessness than at 1 G.

successful. Each flight lasted approximately half an hour and included 12–13 parabolic paths.

#### DISCUSSION

Four main changes were observed in the single otolith-unit activity as a consequence of the parabolic flight:

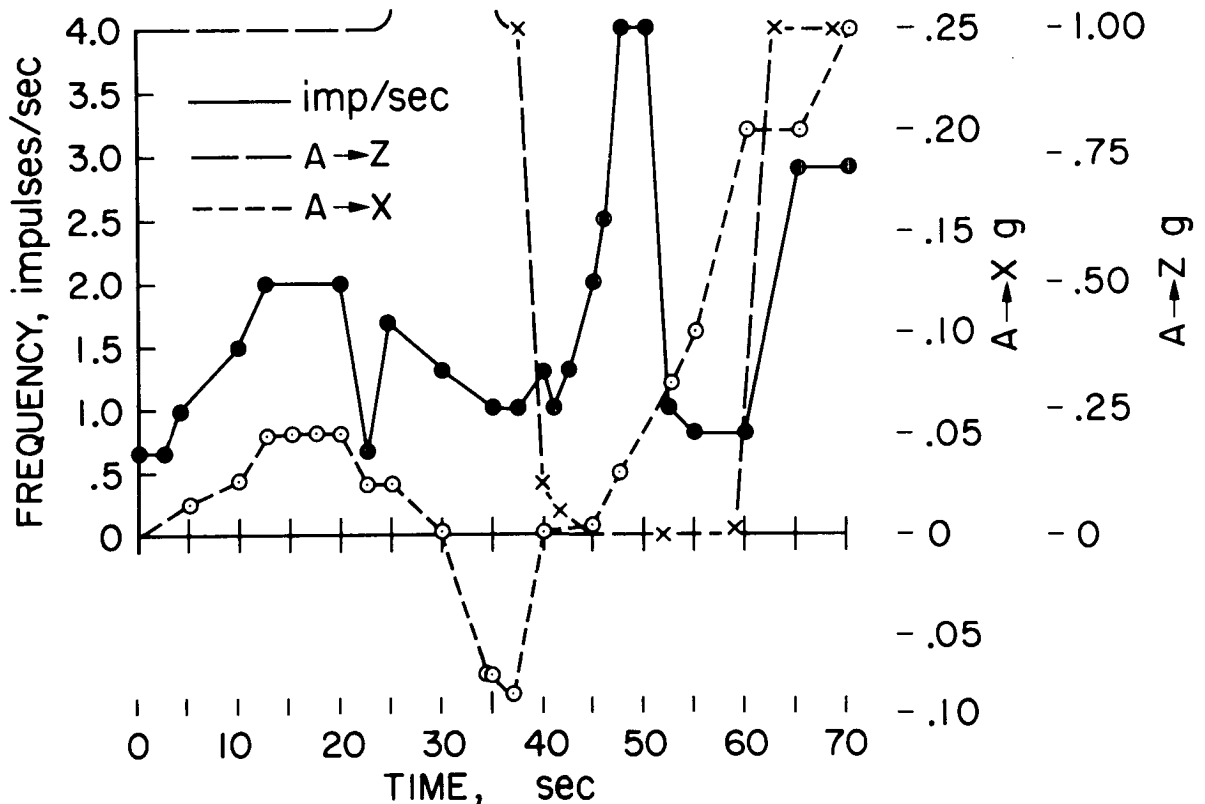


Figure 6.—Curarized frog. Response of a single otolith unit to linear acceleration at 1 G and during 25 sec of weightlessness. Diagram shows changes of rate of firing of a single otolith unit during parabolic flight. Conditions are same as described in figure 5. On abscissa time in seconds. On ordinate, on left rate of firing of vestibular unit and on right acceleration along A→X and A→Z axis. For latter, note that values above 1 G are not recorded as accelerometer was saturated at this value. Frequency response of unit follows closely changes of acceleration along the A→X axis, except for a short period of suppression at onset of acceleration. During weightlessness rate of spontaneous firing increases and response to A→X acceleration is much larger. Suppressory effect described in figure 5 is also shown.

- (a) a sudden increase of spontaneous firing at the beginning of weightlessness preceded by a high G period;
- (b) an initial larger response to acceleration;
- (c) a sudden suppression of response, restored to normal by going back to 1 G;
- (d) a large increase in the overall spontaneous activity of the nerve after a number of short-interval parabola.

It is doubtful whether such changes may be ascribed to the state of weightlessness only, as this state is preceded by a high acceleration (up to 2–3 G) during the onset of the parabolic flight. The sudden increase

of spontaneous firing, however, seems to correspond very well to the already mentioned results of Fiorica et al. (ref. 1), showing the increase of the overall vestibular activity during free fall. These effects might also be due to an actual stimulation of the gravitoceptors during the passage from high acceleration to near weightlessness (Gerathewohl and Stallings, (ref. 7); but this interpretation seems unlikely in this case since the change of gravity is not in the right direction for the units involved (vertical instead of longitudinal). Moreover, the change does not start immediately at the transient but somewhat later (fig. 5). It seems therefore that there might be a true

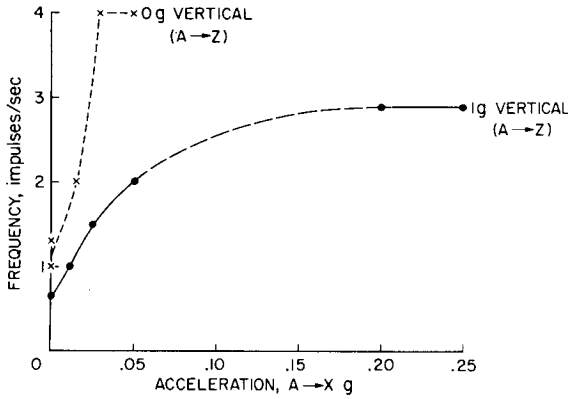


Figure 7.—Responses to linear acceleration of single otolith unit are compared at 1 G with responses of the first 10 seconds at zero G. Latter response is strikingly larger as would be expected (Weber-Fechner law). Note that the response at 1 G before and after period of zero G follows expected logarithmic function.

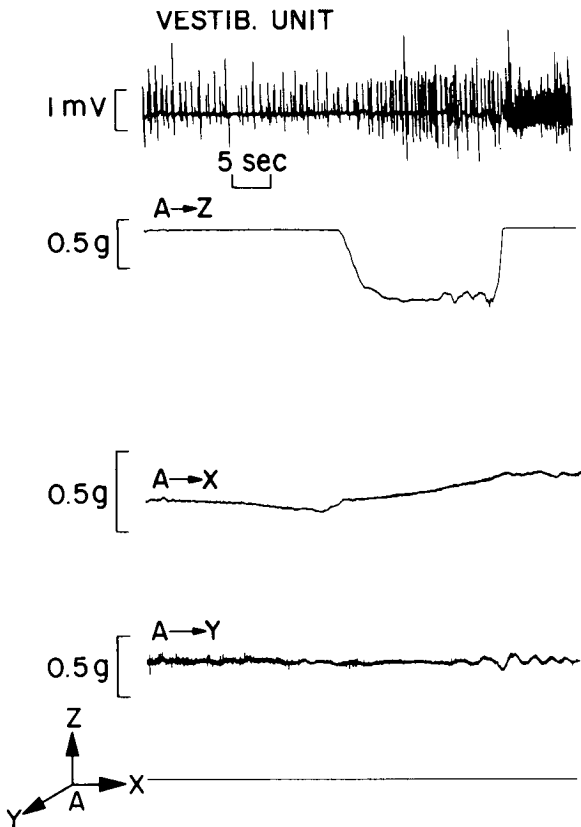


Figure 8.—Otolith unit response to linear acceleration in 13th Keplerian path. Last three at an interval of less than 1 minute from each other. Note large recruiting of new vestibular units.

effect of weightlessness as a result of lack of stimulation of the entire organ—something analogous to what is observed on the retina in absolute darkness.

The larger response to acceleration in weightlessness is a phenomenon in accord with the Weber-Fechner law. This experiment and the results in the laboratory (fig. 3) demonstrate that these vestibular gravitoceptors strictly follow this law, as was first theoretically proposed by Haber and Gerathewohl in 1951 (ref. 8). The observed suppressory phenomenon, however, seems to limit the extreme consequences of this finding. In fact, such an inhibition acts as a safety device against an excessive central inflow.

The suppressory effect is shown also every time the stimulation is decreased. A similar effect is described by Rupert et al. (ref. 6) on the cat; i.e., the firing of a gravitoceptor at maximum tilting is immediately stopped as soon as the movement starts in the opposite direction. Ross (ref. 3) also reported that in the frog the resting discharge can be momentarily suspended by a movement in the direction opposite to that which stimulates the end organ. The mechanism of this suppression might be analogous to the lateral inhibition of *limulus* (horseshoe crab), as described by Hartline and Ratliff (ref. 9). As is known, in the *limulus* retina, collaterals have been shown coming from the afferent fibers of each ommatidium and making synaptic contact with another afferent. When recording from one unit, the illumination of other ommatidia produces no discharge in the eccentric cell axons of the test ommatidium, but its frequency of firing is decreased. This shows that this interaction is purely inhibitory. Collaterals and synaptic knots on the afferent fibers have been shown also in the vestibular receptors (Iurato and Taidelli, ref. 10).

If this analogy applies, an inhibitory mechanism is provided limiting the frequency response of the organ as a function of the number of receptors actually stimulated. Something equivalent has been dem-

onstrated for the recurrent inhibition of the spinal cord. This mechanism might act as a delayed effect of the high G stimulation preceding the zero G condition, blocking the response for some seconds.

This is only a hypothesis, but it shows that parabolic flight does not give complete

information on the effect of weightlessness on the vestibule. Two further experiments are required and are both under consideration: (a) to produce short periods of weightlessness, in a plane, not preceded by high G, and (b) to perform tests during orbital flights.

#### REFERENCES

1. FIORICA, V.; SEMBA, T.; AND STEGGERDA, F. R.: Vestibular Responses of the Unanesthetized Cat Recorded During Free-Fall. *Aerospace Med.*, 33, 1962, pp. 475-481.
2. HUBEL, D. H.: Tungsten Microelectrode for Recording From Single Units. *Science*, 125, 1957, pp. 549-550.
3. ROSS, D. A.: Electrical Studies on the Frog's Labyrinth. *J. Physiol.*, 86, 1936, pp. 117-146.
4. ADRIAN, E. D.: Discharges From Vestibular Receptors in the Cat. *J. Physiol.*, 101, 1943, pp. 389-407.
5. LÖWENSTEIN, O.: Comparative Physiology of the Otolith Organs. *Brit. Med. Bull.*, 12, 1956, pp. 110-114.
6. RUPERT, A.; MOUSHEGIAN G.; AND GALAMBOS, R.: Microelectrode Studies of Primary Vestibular Neurons in Cat. *Exp. Neurol.*, 5, 1962, pp. 100-109.
7. GERATHEWOHL, S. J.; AND STALLINGS, H. D.: The Labyrinthine Posture Reflex in the Cat During Weightlessness. *J. Aviation Med.*, 28, 1957, pp. 345-355.
8. HABER, H.; AND GERATHEWOHL, S. J.: Physics and Psychophysics of Weightlessness. *J. Aviation Med.*, 22, 1951, pp. 180-189.
9. HARTLINE, H. K.; AND RATLIFF, F.: Inhibitory Interaction of Receptor Units in the Eye of *Limulus*. *J. Gen. Physiol.*, 40, 1956, pp. 357-376.
10. IURATO, S.; AND TAIDELLI, G.: Relationships and Structure of the So-Called Much Granulated Nerve Endings in the Crista Ampullaris, Congress of Electronmicroscopy (Prague), Aug. 1964.

#### DISCUSSION

SPOENDLIN: Have you found specific directions in the sensitivity of one unit? You already spoke about increasing activity. Didn't you find a decreasing activity in the opposite tilting direction?

GUALTIEROTTI: Not exactly. In the opposite tilting the activity of the unit decreases but not according to the same logarithmic function by which it increases during tilting in the right direction. What does take place is an immediate suppression of the firing for some seconds as soon as the back movement starts. However, opposite tilting in regard of one unit might be the right tilting for a different one. Suppose you have two units oriented in the opposite way against the vertical. Then if you tilt the entire preparation, one would approach the vertical and the other would go away from it. The first one would be subjected to increased stimulation and the other to decreased stimulation. At steady state, different degrees of opposite tilting will result in rates of firing typical of the excitation due to the corresponding gravitatory components. This happens if each tilting position is maintained long enough to overcome the already described suppressory period resulting from initiating the backtilting. Of course, I can't swear that there aren't units responding differently.

SPOENDLIN: So this is not in agreement with the findings of Löwenstein's group, i.e., tilting in one

direction there is an increase and in the opposite direction there is always a decrease in activity.

GUALTIEROTTI: As I said, you have a decreasing activity at the static state if you wait long enough.

KELOGG: I am trying to reconcile this with some of the findings that Dr. Miller found at 0 G with counterrolling, i.e., a decrease or suppression of response. This would indicate a very marked decrease in the amount of firing during the 0 G period.

GUALTIEROTTI: Yes, it is shown by the decrease of the firing rate which is observed during the suppressory period. After about 10 seconds of 0 G, a suppressory effect is shown. In terms of influence in the central nervous system there is less activity reaching the central nervous system because these units are blocked. The central nervous system doesn't know that this blocking is due to the fact that units are suppressed, or why they don't respond to the stimulation. But if you consider that many of these units would be subjected to suppressory effect, the overall inflow to the central nervous system after a brief period of zero gravity would actually be equivalent to a decrease of firing.

FIELDS: You indicated your concern about the vibration effects in the parabolic flight tests. Yet, I wonder, how do you reconcile this problem with sudden rapid tilt? Don't you anticipate that you are going to have vibration at the end of the arc of the



tilt? Isn't that a factor and how do you account for this in your single unit measure? Does this produce an effect?

**GUALTIEROTTI:** It would certainly produce an effect if the units were responding. As I have shown, during the suppressory effect, the unit does not respond to the stimulation. I don't know how long the suppressory effect would last because I can't have a longer period of zero gravity on this kind of plane. Probably it would wear off eventually. Vibrations would then provoke responses on this unit. But we ran some tests on a vibratory table, both on otolith units and even on the semicircular canal unit. If you have a high frequency vibration—suppose 20 to 30 per second—then the otolith unit will not respond to each stimulus with a burst of high frequency firing, but only intermittently, to some of the stimuli. If you increase the frequency of the vibration, the otolith unit reaches a state in which it doesn't respond to the vibrations at all. This response corresponds to some results of Loewenstein and others; this unit can be distinguished by units corresponding electively to vibration. A special result that I can mention gives some more light, for instance, on the semicircular canal unit activity: We found that the semicircular canal unit would not normally respond to vibration. It would not respond, say to 10 per second vibration. But there is a certain range of frequency at which it starts responding. For instance, one semicircular canal unit started responding at a frequency of 30 per second with intermittent firing. Increasing further the frequency of the shaker, it showed a continuous firing at a very high rate, some 70 per second or more. That seems to be the limit of the firing frequency in this particular condition. Increasing still the frequency of vibration of the shaker, at first an intermittent firing was recorded and then no firing at all. We wonder if in this particular condition we might not have an excitatory effect on the semicircular canal receptor by some sort of microphonic or vibratory effect on the walls of the semicircular canal itself. Because, obviously, it could not be due to the displacement of the endolymph, owing to the large inertia that you have there.

**BILLINGHAM:** I have three comments you might be interested in. First, there are plans to fly the package you saw on the screen aboard one of the long term Apollo mission flights. The second thing I would like to bring out is, has anybody investigated the possibility of simulating weightlessness in a much more

complete way on the entire body system on the ground? It seems to me there are three elements involved: one is water immersion which deals with most of the body; but then you still have the otolith and you have the lungs. These days you can remain alive breathing water with the correct oxygen partial pressure in it and that might solve the lung problem. Then we are left with the otoliths. You now have to get the specific gravity of the otolith and surrounding fluid the same. Either you can reduce the specific gravity of the otolith, which is, I think, quite difficult, or you could increase the specific gravity of the endolymph. Has it occurred to anybody, or has anyone tried, increasing the specific gravity of the endolymph to try and simulate what would happen to an otolith organ under weightless conditions?

**GUALTIEROTTI:** By increasing the specific gravity of the endolymph, you still do not simulate the effect of zero gravity, because you damp the movement of the otolith bodies. In zero gravity you have no weight, on the otolith, but you have still inertia, and no damping effect on the movement of the otolith bodies.

**BILLINGHAM:** What I meant to suggest was replacement of the heavy fluid with normal endolymph before reimposing the test acceleration.

Looking at this from the point of view of manned space flight, one has to ask oneself some extremely crucial and important questions. One question is this: realizing that one has a great deal of knowledge already about the otolith, its peripheral connections and central connections, and reflex mechanisms, if you expose the otolith to 30, 50, 60 days of weightlessness and periodically throughout this time you impose a given linear acceleration, shall we say 10 seconds of 1 G in a specific axis, and measure a specific response, whether this be oculogravic response or counterrolling (specific otolith responses), what would happen in terms of the sensitivity of the otolith reflex mechanism? Does it stay the same throughout? Does it rise and then return to its original level? Does it rise to a plateau? Does it go on rising indefinitely? Does it fall off? Now, from the point of view of manned space flights, the shapes of these curves are very important. Let me give you an example—If you have someone in an extravehicular situation and he has a propulsion unit on his back and has been weightless for a long period of time and he exposes himself suddenly to a test acceleration, is he going to get an oculogravic illusion which is much greater than the same acceleration would have produced in a 1 G environment?

**SESSION IV**

**Chairman: HENNING E. VON GIERKE**  
**Aerospace Medical Research Laboratories**  
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**Cochairman: KLAUS CAPPEL**  
**Franklin Institute Laboratories**

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# Physiological Design Criteria for Artificial Gravity Environments in Manned Space Systems

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Of concern to those planning the exploration of space through extended manned space missions are the subtle effects on the crew which may result from long exposure to the weightless state. Based on rational analysis and trend information given by the relatively short manned orbital flights to date, as well as bed rest and water immersion tests, it appears highly probable that problems will arise due to the great adaptability of the human to his environment. The body makes compensatory adjustments to adapt to changed environments, and these adjustments are not immediate in most cases, but are gradual over a prolonged period of time. Such vegetative adaptive reactions are almost certain to occur from prolonged exposure to removal of the gravity stress unless effective preventive measures are taken. It is quite probable that these changes will not reduce performance in the weightless state but will be of major concern when the gravity overstresses are applied upon return to the earth environment. This may be attributed to the time factor required for physiological readjustment to an increased gravity stress.

Several orbital flights have produced preliminary information indicating that the cardiovascular system will be affected by prolonged exposure to weightlessness. On the third United States orbital flight (9 hr 13 min) the postflight physical examination of Astronaut Schirra disclosed that heart rate averaged 70 beats per minute when

supine and immediately jumped to 100 or more when he stood erect. In addition, immediately after flight all dependent leg veins were engorged, the legs and feet assuming a reddish-purple color on standing (ref. 1). On the fourth United States orbital flight (34 hr 18 min), even though Astronaut Cooper used the stimulant Dexedrine immediately prior to his manually controlled reentry, upon standing upright on the deck of the recovery ship after egress from the capsule his heart rate rose to 188 beats per minute. He began to look pale, swayed slightly, and reported symptoms of impending loss of consciousness including light-headedness, dimming of vision, and tingling of his feet and legs (ref. 2). During the second Russian orbital flight (24 hr) there was evidence of "grey-out" by Cosmonaut Titov during reentry, indicating a possible reduced tolerance to the acceleration stress after a period of weightlessness (ref. 3). On the third and fourth Russian orbital flights Cosmonaut Nikolayev (95 hr) and Cosmonaut Popovich (71 hr) recorded increases in pulse rate of 23 and 72 percent, respectively, over that immediately prior to reentry, while during boost to orbit the increases were only 7 and 3 percent, respectively (ref. 4). Even from these relatively short exposures it appears that the cardiovascular system is adapting itself to the reduced work load of the environment, and atrophy of the circulatory system will result in inability to adapt immediately to the

hydrostatic pressure induced by return of gravitational acceleration after weeks or months of weightlessness.

Based upon examination of subjects exposed to prolonged bed rest or water immersion, where there is no longitudinal load on the long bones of the body, it has been concluded by several investigators that there is a tendency for the bones to adjust to a reduced mineral balance. As these minerals primarily leave the body through the urine, an increase in its mineral and calcium content combined with the reduction in water available for drinking on extended space flights favors the precipitation of mineral salts and the formation of kidney stones (refs. 5 and 6). Also, in studies based on 4 weeks of absolute bed rest it was learned that there was an average 6% loss of red blood cells in 28 days of inactivity followed by an acute 25% decrease in red blood cells within 2 weeks during ambulation (ref. 7). On very long flights this reduced mineral balance and change in red blood cell balance could require an extended period for vegetative readaptation upon return to earth.

Another possible effect of long-term weightlessness, although the evidence is not conclusive that weightlessness alone is responsible, has been indicated by Russian biological experiments (ref. 8). In mice, guinea pigs, and dogs exposed to space flight for up to 5 days, there was an increase in stabs (to 30 percent), increase in the quantity of calcium, and a change in the ratio of protein fractions. Two days after the flight a "sticking-together of the chromosomes" was observed which lasted 30 days. However, most experimenters believe these histological and morphological effects are the result not only of weightlessness but other space flight factors as well.

These preliminary indications of adverse effects from an extended weightless environment require investigation to determine practical methods to counteract undesirable environmental adaptations in order not to compromise future manned space vehicle designs. Note that the long-term effects of

ionizing radiation were not known for many years, and tolerance limits have been continually revised downward. As more knowledge is gained of weightlessness effects, similar tolerance revisions may be necessary.

To counteract these effects experiments have been conducted to find practical techniques for exercise of the affected systems to prevent atrophy. For exercise of the cardiovascular system a method was devised whereby interconnected tourniquets were applied about the four extremities and connected to an air source (ref. 9). In 6-hour tests in a water immersion facility the tourniquets were compressed every 2 minutes, held for a minute, and then released. For all subjects orthostatic tolerance as determined by tilt table was normal or better than for the 1 G controls. However, such encumbrances and the power required to cycle them are undesired by astronaut and engineer alike. Prevention of muscular atrophy and reduction of decalcification of the bony skeleton will require as a minimum a systematic calisthenics program and a carefully controlled diet. It is probable that such a program will not completely offset the effects but should at least prolong their onset and reduce their severity. Problems at the cellular level, if such problems should be confirmed, can be alleviated (in the light of present knowledge) only by artificial gravity.

A practical method of achieving artificial gravity is to rotate the space vehicle so that the resulting centrifugal force provides an apparent gravity vector of sufficient magnitude to permit satisfactory operations, both motor and vegetative. One significant drawback to this technique is the Coriolis acceleration or force that is generated every time a linear motion is attempted perpendicularly to the axis of rotation. This acceleration is the result of a fundamental law relating the time rate of change of a vector, as measured by an observer in a reference inertial space, to the time rate of change of the vector measured by an observer in a space rotating with respect to the reference space (ref. 10)

This relationship is expressed mathematically by the vector equation:

$$\left(\frac{d\vec{V}}{dt}\right)_r = \left(\frac{d\vec{V}}{dt}\right)_m + (\vec{\omega}_{rm} \times \vec{V})$$

where

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

It is illustrated schematically in figure 1. To a human 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 with, or in, the direction of rotation and subtracts from the apparent weight when moving against the direction of rotation. Second, when a body moves toward the center of rotation, the 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 to the direction of rotation. A motion parallel to the axis of rotation will generate no Coriolis acceleration. The value of Coriolis acceleration for a body moving perpendicularly to the

axis of rotation in a spinning system may be determined by:

$$A_{\text{Coriolis}} = 2 V \sqrt{W^2 (A_{\text{Centrifugal}})/g} r$$

where

- $V$  = velocity of body relative to rotating vehicle in ft/sec
  - $W$  = weight of body in pounds
  - $A_{\text{Centrifugal}}$  = centrifugal acceleration in earth G's
  - $g$  = acceleration of gravity (32.2 ft/sec<sup>2</sup>)
  - $r$  = radius of rotation in feet
- and

$$A_{\text{Centrifugal}} = .000341 N^2 r$$

where

- $N$  = revolutions per minute

If the weight is considered unity, the value of Coriolis acceleration will be in Earth G units.

With attempted linear movement in any plane not parallel to the axis of rotation in a rotating system the Coriolis force combines with the centrifugal force to produce a different apparent gravity vector in magnitude alone or in both magnitude and direction. It is desirable to define limits to this force, such that under normal translation velocities the apparent gravity vector will not result in an apparent floor slope which exceeds the angle of sliding friction for leather on metal, or about a 20° slope. As normal translation velocity for walking or climbing stairs is approximately 3 ft/sec, it is reasonable to limit the Coriolis force to 20% of man's apparent weight (as given by the centrifugal force in the rotating system) when translating at this velocity. This allows a contingency factor such that if a 5 ft/sec movement is made, the maximum apparent floor slope would be 19.4°.

Another significant restriction in the use of rotation of a vehicle to provide artificial gravity is the stimulation of the semicircular canals when angular motion is attempted outside the plane of rotation. Under certain conditions of rotation and head movement a debilitating condition, termed "canal sickness," can be generated (ref. 11). Nausea

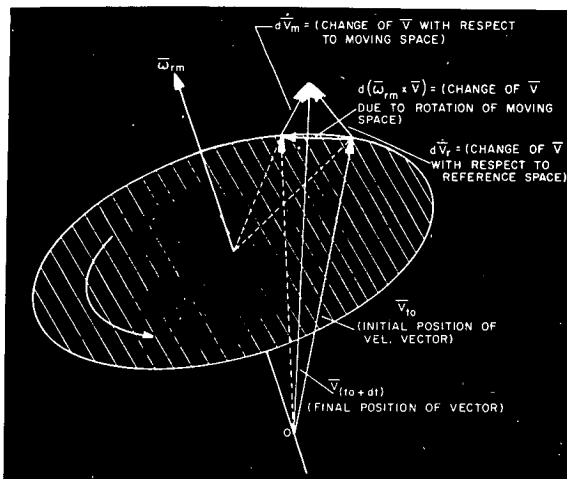


Figure 1.—Time rate of change of a vector in rotating space stations.

and vomiting may result after a few head movements if the rotating system has sufficient angular velocity and the head movements are sufficiently rapid.

As each semicircular canal in a set is mutually orthogonal to the others and is essentially a torus filled with endolymph having a specific gravity near unity, each set of semicircular canals may be considered a gyroscopic element when the head is rotated in a spinning vehicle. The semicircular canal lying in the plane of vehicle rotation is like a gyroscopic rotor spinning about its axis of symmetry. If it receives an angular velocity input about an axis normal to the spin axis, a torque is generated about an output axis that is mutually perpendicular to both the input axis and the spin axis (ref. 12). This three-dimensional space situation is not describable in terms of scalar quantities, but requires the use of vector concepts. A derivation from Newton's dynamical law of rotation results in:

$$\bar{T}_{out} = \bar{H} \times \bar{\omega}_{IA}$$

where

$\bar{T}_{out}$  = torque applied by gyroscopic element to external constraints

$\bar{H}$  = vector angular momentum of spinning body

$\bar{\omega}_{IA}$  = angular velocity of spinning body at right angle to spin axis

and:

$$\bar{H} = I_r \bar{\omega}_r$$

where:

$I_r$  = moment of inertia of rotating body about spin axis

$\bar{\omega}_r$  = vector angular velocity of rotating body about spin axis

or:

$$\bar{T}_{out} = I_r (\bar{\omega}_r \times \bar{\omega}_{IA})$$

This relationship is shown schematically in figure 2. The gyro torque or couple so generated acts upon the endolymph in the third semicircular canal and attempts to set it in rotation. This fluid pressure, or perhaps the torque itself, acts upon the cupula such that it senses an apparent angular velocity about the third orthogonal axis.

To determine the scalar value of this

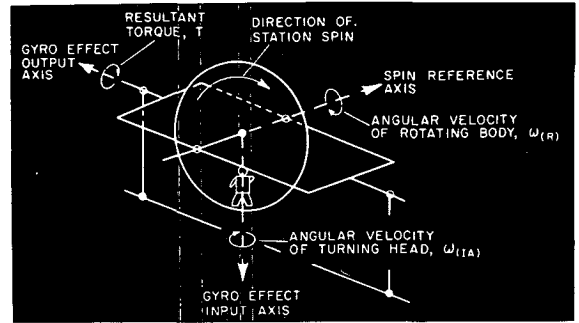


Figure 2.—Schematic diagram illustrating gyro-scope torque effect on semicircular canals when turning head in rotating space station. Resultant torque,  $T = I\omega_r\omega_{IA}$ ; where  $I$  = moment of inertia of endolymph in semicircular canal.

torque on the endolymph the following relationship may be used:

$$\text{TORQUE} = I \omega_r \omega_{IA}$$

where

$I$  = moment of inertia about center axis of the endolymph in a single semicircular canal

$\omega_r$  = angular velocity of spinning vehicle

$\omega_{IA}$  = average angular velocity of turning or nodding head

Based on research accomplished at NASA's Langley Research Center, where subjects were rotated horizontally with feet outboard on a 15 ft radius simulator and head velocities recorded (ref. 13), the torque value in foot-pounds on the endolymph, which resulted in the onset of malaise in all subjects, is calculated to be  $4.35 \times 10^{-10}$  ft lb. Such a low value would be very difficult to measure in the semicircular canal and is indicative of the extreme sensitivity of this organ to angular acceleration. A plot of the rate of head movement versus vehicle rotational rate for two torque values, tolerable of  $3.03 \times 10^{-10}$  ft lb and onset of intolerability without preadaptation of  $4.35 \times 10^{-10}$  ft lb, is presented in figure 3. Based on this analysis a rate of rotation of the vehicle of 6 rpm. would be tolerable for nominal head movements of trained crews, i.e., a torque value of  $3.73 \times 10^{-10}$  ft lb on the endolymph with a  $225^\circ/\text{sec}$  head rotation. Tests in the Slow Rotation Room at the United States Naval

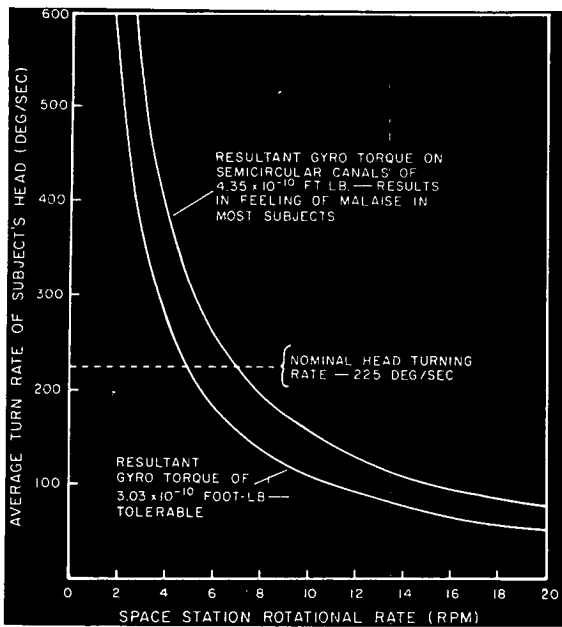


Figure 3.—Nominal resultant gyro torque limits on cross-coupled rotations of semicircular canal system in rotating space stations.

Aviation Medical Center have shown that the semicircular canal tolerance to this gyro torque can be increased by adaptation in incremental steps up to a rate of rotation of approximately 10 rpm; however, readaptation to the nonrotating environment is equally severe.

The boundaries to satisfactory human performance in rotating systems as determined by tolerance to Coriolis acceleration and gyroscopic torque canal stimulation are more severe than such other criteria as gravity gradient or difference in head to foot G, rim or floor velocity of the vehicle, etc., that have been proposed by some investigators. Hence, the design criteria for radius and rate of rotation of space vehicles providing artificial gravity should be based on these two overriding effects.

A reasonable lower limit for artificial gravity has been determined by experimenters both in Russia and the United States as approximately 0.28 G, the lower limit for efficient locomotion. Yuganov of the USSR experimented with rotation of mice and rats in parabolic flight (ref. 14). With accelerations of 0.28 G and above the behavior of the animals during the experiment was the same as under laboratory conditions. United States experimenter E. Sharp conducted human walking experiments in aircraft flying a modified Keplerian trajectory to arrive at a similar value of between 0.2 and 0.3 G (ref. 15). As an upper limit it appears there is no advantage in exceeding 1 Earth gravity. An envelope defining these parameters is shown in figure 4. Little is known about the long-term physiological effects of the various levels of reduced gravity (0.28 Earth G to 1 Earth G), but it is assumed that the 0.28 G is sufficient to prolong or eliminate adverse effects for nominal crew tours of duty in space systems. From figure 4 the lightest acceptable system for providing artificial gravity would be one having a radius of rotation of 48 ft, rotating at 6 rpm, and providing 0.58 of Earth gravity.

Finally, there are indications that prolonged weightlessness will affect the cardiovascular system, mineral balance, and possibly the cellular functioning of man in an adverse manner, due primarily to the adaptability of the human body to its environment, thus reducing tolerance to rapidly applied gravity overstresses of reentry. Exercise and diet will help alleviate these effects but probably will not be a complete solution. Artificial gravity, if properly applied, is a practical solution for manned systems such as space stations even though adaptation to a rotating environment may be required.

#### REFERENCES

1. BERRY, C. A., ET AL.: Aeromedical Analysis. Results of the Third U. S. Manned Orbital Space Flight, October 3, 1962, NASA SP-12, 1962.
2. CATTERSON, A. D., ET AL.: Aeromedical Observations. Project Mercury Summary Including Results of the Fourth Manned Orbital Flight, May 15-16, 1963, NASA SP-45, 1963.
3. GAZENKO, O. G., ET AL.: Biological and Physiological Investigations During Flights on Rockets and Artificial Earth Satellites. IAF/IAA Symposium on Basic Environmental Problems of Man in Space (Paris, France), Oct. 29-Nov. 2, 1962.

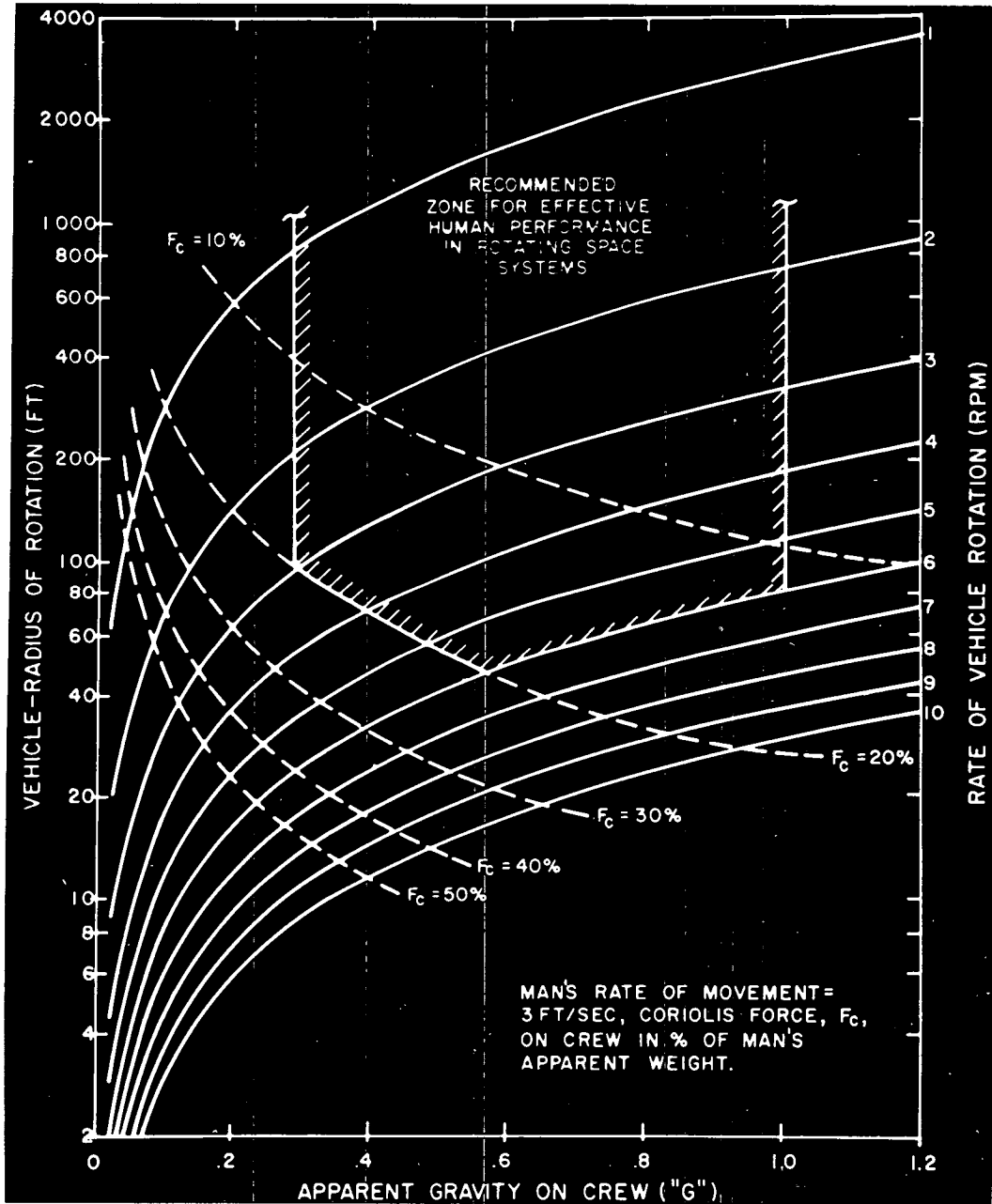


Figure 4.—Design criteria for effective human performance in rotating space systems.

4. YAZDOVSKIY, V. I., ET AL.: Basic Problems in the Study of Weightlessness. Problems in Space Biology, Volume III (Moscow, USSR), 1964.
5. GAUME, J. G., AND KUEHNEGGER, W.: Effects of Chronic Lunar Gravity on Human Physiology. ARS Lunar Missions Meeting (Cleveland, Ohio), July 17-19, 1962.
6. CROCKETT, A. T. K., ET AL.: Hypodynamic Urolithiasis: A Potential Hazard During Prolonged Weightlessness in Space Travel. Review 2-62, School of Aerospace Medicine, USAF Aerospace Medical Center, Brooks AFB, Texas, Dec. 1961.
7. LAMB, L. E.: Circulatory Aspects of Manned Space Flight, Third International Symposium on Bioastronautics and the Exploration of Space (San Antonio, Tex.), Nov. 16-18, 1964.



8. ARSEN'YEVA, M. A., ET AL.: Cytological and Histological Changes in the Hematopoietic Organs of Mice under the Influence of Space Flight on Spaceships. Problems in Space Biology, Vol. II (Moscow, USSR), 1962.
9. GRAVELINE, D. E.: Maintenance of Cardiovascular Adaptability During Prolonged Weightlessness. ASD Tech. Rept. 61-707, Wright-Patterson AFB, Ohio, Dec. 1961.
10. WEEMS, W. R.: An Introduction to the Study of Gyroscopic Instruments. M.I.T., Dept. of Aeronautical Engineering, Instrument Section, Jan. 1948.
11. GRAYBIEL, A., ET AL.: Observations on Human Subjects Living in a 'Slow Rotation Room' for Periods of Two Days. A.M.A. Archives of Neurology, vol. 3, July 1960, pp. 55-73.
12. DRAPER, C. S.; MCKAY, W.; AND LEES, S.: Instrument Engineering. Vol. I. McGraw-Hill Book Co., Inc., 1952.
13. STONE, R. W., JR.; AND LETKO, W.: Tolerance to Vehicle Rotation of Subjects using Turning and Nodding Motion of the Head while Performing Simple Tasks. AIAA Paper No. 64-218, 1st AIAA Annual Meeting (Washington, D. C.), June 29-July 2, 1964.
14. YUGANOV, YE. M.; AND AFANAS'YEV, D. V.: The Vestibular Analyzer and Artificial Gravity in Animals. Problems in Space Biology, Vol. III (Moscow, USSR), 1964.
15. LORET, B. J.: Optimization of Manned Orbital Satellite Vehicle Design with Respect to Artificial Gravity. ASD Tech. Rept. 61-688, Wright-Patterson AFB, Ohio, Dec. 1961.

### DISCUSSION

LANSBERG: The gyroscopic effect that you were discussing is not quite clear to me. Assuming a subject makes a full 180° turn, what would be the direction of the effect and in what direction would you, according to the gyroscopic theory, think the man would turn?

THOMPSON: You would have, as you turn, two effects. As you went past the maximum moment of inertia from one canal to the other one, the two vertical canals which sit at 45° angles off transverse centerline, you would have the sensation of toppling sideways and then pitching, and then sideways and then pitching as you went around. You would change from one to the other as you went around in a complete circle, based on pure gyroscopic theory.

LANSBERG: That's right. Whereas on physiological principles if I made a quick 180° turn, the effect would only be in the plane of rotation of the space station.

THOMPSON: This may be true in the 1 G environment, but until we get under the pure zero G, and we are rotating without the other artifact of gravity, I don't know what the magnitude of the physiological sensation would be. I can only go by what mathematics says will happen. It works that way with gyroscopes and this is a gyroscopic system.

LANSBERG: During the quick 180° turn, the vector will correspond to gyroscopic direction, but immediately after you have made the quick 180° turn things will be different.

THOMPSON: The canal that initially has been rotating is the one that is overriding, because the other one has not had a chance to achieve effective rotation as you make the turn. You are rotating one about its major axis and this is the one that is giving you the initial effect as you rotate it out of the plane. It will take awhile for the other one to become effective.

LANSBERG: That is what I was getting at. I wonder whether the time constant of the system is correct.

GUEDRY: I'm not sure I understand you either, but I don't believe I agree with you. Let's take two canals and look at them on end. Let's consider a plane of rotation, with the axis of rotation called Y. If we tilt these canals, we'll tilt both of them together. They're connected, they are orthogonal to one another. Let's tilt it about the X axis (an axis lying in the plane of rotation). As you tilt both of these canals together, the canal lying in the plane of rotation at the commencement of movement about the X-axis is stimulated almost not at all. The canal which is vertical is stimulated almost maximally. Then as the horizontal canal comes up toward vertical position, it is stimulated more and more, and the other canal less and less. So I think that was a point of confusion that Dr. Lansberg was bringing up.

THOMPSON: What happens is that, as the canal rotating about its major axis is rotated up, it is generating a torque in the others. Now, as it continues to rotate up, the others are becoming effective and generating torques. So there is a transition from one canal to the other which gives you the bizarre stimulation of your canals that Dr. Graybiel has shown in his experiments.

M. JONES: It seems to me that confusion can arise in this matter due to failure to appreciate the importance of distinguishing between the "natural" modes of canal operation, i.e., within the frequency range of about 0.1 to 5.0 cps, and the unnatural ones, i.e., usually less than 0.1 cps. To me, interpretation of the physiological response in the latter case is much simplified by considering the canal system as a 3-D sensing unit feeding vectors of angular velocity to the CNS, determined to a first approximation by the various angles of cupular deflection actually achieved at every instant. The resulting vector has both magnitude and direction (represented along the axis of turn, or apparent axis of turn) and can be imagined as growing and decaying and changing

direction, according to summation of the separate vectors attached to each canal.

Thus when I turn about a vertical axis, a vertical vector will "grow" as I accelerate and then decay again in accordance with the effective time constant of cupular restoration for that particular axis. If I then suddenly stop turning, the vector will shoot out in the reverse direction (still vertical) and again withdraw or decay in accordance with the appropriate time constant. The reversed direction of the vector and its subsequent decay represent the well-known post rotational response.

If immediately after stopping, I turn my head through 90°, for example onto my left shoulder, then I carry the "illusory" vector with me, and it now points out horizontally and again withdraws as before. This then corresponds to an illusion of rotation about a horizontal axis which, as is well known, can cause a violent incorrect postural response (about this axis).

If I turn my head through 90° *while still turning the body about a vertical axis*, then just before turning the head there is no vector owing to complete cupular restoration. But now immediately after turning the head onto the left shoulder, there is a horizontal "illusory" vector, signaling rotation about a horizontal axis, summing with a new but correctly placed vertical vector due to the real rotation of the body about the vertical axis. The resultant apparent vector will, then, be placed at 45° between these two, having magnitude two times either one of the separate vectors. The apparent sensation of turn is correspondingly tilted, and becomes very confusing. Moreover, as before, both vectors then decay (the body is still turning at a constant speed), but in this case the truly vertical vector decays faster than the truly horizontal one so that the direction of the resultant, and hence the apparent axis of turn, progressively changes in the sense that it tends to drift towards the truly horizontal one. Finally, on turning the head suddenly back to the true vertical (again while still turning) a reversed series of sensations is experienced.

Looked at in this way, I think it unnecessary to argue whether there is a gyroscopic effect, or when this effect becomes significant. The complete story can be made to emerge in an easily "visualized" way from a simple consideration of the known dynamic response characteristics of the canals.

**THOMPSON:** In the physical world the gyroscopic couple happens almost immediately. With the time constant of the cupula system, I'm sure that the effects so noted are true and I think with the rotations that we will be getting in space stations, we probably will limit ourselves, just from our physical feeling, to stay within our limitations of rotation. You just automatically do this and I'm sure that the astronauts will adapt to it.

**GRAYBIEL:** Two comments: First, the canalicular effects resulting from rotation of the head about more

than one axis simultaneously differ either qualitatively or quantitatively from the effects of stimulating the horizontal pair of canals. This suggests the need for caution in applying knowledge of the response characteristics of the horizontal canals to the more complex stimulus-response conditions. Eventually, we must go back to the physical stimulus as our point of departure, although agreement has not been reached over the analysis of the force field. Dr. Hermann J. Schaefer has pointed out that the gyroscopic force or torque is also a linear force and that it may not constitute an effective stimulus inasmuch as it acts at right angles to the diameter of the canal. The effective stimulus is an acceleration of unique origin when a canal, say, coplanar with the rotation of the room, is rotated out of the axis of the room's rotation. The uniqueness arises out of the fact that the radial distance changes between the center of rotation of the room and different parts of the canal resulting in corresponding changes of the velocity of the endolymph which in turn produces an effective stimulus to the cupula. Until the matter of these physical forces and the manner in which they act as a stimulus is generally agreed upon, one might refer to such complex forces (when subjects in a rotating environment move their head out of the axis of the room's rotation) as multiplanar angular accelerative forces (MAF) and the responses as MAF effects.

Second, there is much to be gained in terms of clarity in describing the effects of exposure in a rotating environment, if we make a clear distinction between the linear forces which affect to a significant degree the gravito-inertial upright and the angular accelerations which bring about their effects by stimulating the semicircular canals. Therefore, I suggest we reserve the term Coriolis force for that component of linear force which affects the vector sum representing the gravito-inertial upright and that it not be used to indicate a stimulus to the semicircular canals.

Could Cochairman Klaus Cappel comment briefly on these points?

**CAPPEL:** Since Dr. Jones and I have so far failed to agree on this point, I have no doubt that we will not be able to come to a conclusion now. What I would like to say is that I think both points of view will have to be considered. I think Mr. Thompson has the correct approach to the problem and I think it's borne out by some of the work we have done. We have actually demonstrated the reversal that was just demonstrated to Dr. Lansberg. On the other hand, I think that we also realize the truth of Dr. Jones' statement that the semicircular canals are not perfect mechanical transducers with zero lag. I do think, though, that before one can consider the physics of the situation it is necessary to take the approach that Mr. Thompson has taken, to tie down the forces and torques once and for all.

**MAYNE:** I would like to make two comments. In the first place, I don't think the gyroscope concept is

necessary to the proper visualization or analysis of Coriolis phenomena. It is not a question as to whether the concept is right or wrong, it is only a question of whether or not it provides the simplest way to visualize and analyze the phenomenon. The canal sees a velocity which is proportional to the major velocity vector through the canal times the cosine of the angle between the vector and the plane of the canal. Then the acceleration that it experiences is a derivative of this function and can be expressed very simply. Now, the fact that this law is true requires a small demonstration which involves the Coriolis principle. Having proved this, we can, I think, dispense with gyroscope and Coriolis formulations. The second point I would like to make is that the time constant of the canals cannot be dismissed as a secondary phenomena. The wonderful presenta-

tion by Dr. Jones made it clear that the time constant must be included in the formulation of the phenomena as a major part of the canal behavior. This cannot be dismissed by the statement that the semi-circular canals are not perfect transducers.

THOMPSON: Before we get into the complications of inserting the time constants in the equation, we should set down the basic method of operation of these forces. Then you can modify your equations later on as you determine what the time constants are and will be. You will end up with some order of differential equations with appropriate time constants. But right now, let's just get the pure physical mechanics of the operation squared away, get that straight, and then modify the relationships later when we know more about the subject.

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## Comparison of Vestibular Effects in Several Rotating Environments

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There are basic differences in vestibular stimulation in different rotation environments, and consideration of these differences is relevant to anticipation of conditions in a rotating space station. Reactions in these situations can be estimated from analyses given by Bornschein and Schubert (ref. 1), Meda (ref. 2), and others (refs. 3 to 6).

In considering these situations, it is important to remember that the particular pattern of canal stimulation at any one moment during a given head movement relative to the body is controlled by the position of the head relative to the plane of rotation. For example, in the four situations illustrated in figure 1, a head tilt of 30 or 40 deg toward the left shoulder will produce primarily vertical nystagmus in I and II (up and right in I; up and left in II) and primarily horizontal nystagmus left in III and IV. Note in each case that the plane of the nystagmus relative to the plane of rotation would be about the same; e.g., vertical nystagmus relative to the skull in I is in a plane at right angles to the plane of rotation as is horizontal nystagmus relative to the skull in IV. This is because the relative orientation of the head-movement axis and the axis of vehicle rotation, which determine what may be conceptualized as the axis of stimulation, are the same in each of these situations, and the plane of nystagmus follows the plane of stimulation fairly closely. However, in IV this particular head movement would induce a sensation of rotation about a vertical axis

whereas in I, it induces apparent tumbling about a horizontal axis.

Another noteworthy difference between these situations is the direction and magnitude of gravity relative to the plane of rotation. In situation IV, the rotating space station, the G vector (centrifugal force) is oriented radially; it lies in the plane of rotation, and this condition is not reproduced in any of the first three situations. Moreover, it cannot be simulated on Earth without so greatly increasing the G vector as to make the simulation meaningless. In I and II, during upright posture, the G vector is alined with the body as it will be in the space station, but the canal stimulation is not the same for particular head movements in I and II, and neither of these is like the canal stimulation in IV. Moreover, the 1.4 G resultant in II exceeds magnitudes anticipated for rotating space stations, and recent evidence (refs. 7 and 8) including some presented in a preceding paper by Lansberg indicates that a resultant force of this magnitude modifies vestibular nystagmus. Situation III is theoretically identical to IV in regard to patterns of canal stimulation induced by head movements, but concomitant cues from gravity sensitive structures differ in III and IV. We will be concerned herein primarily with situation III and how it relates to situations I and IV.

Lansberg (refs. 4 and 9) and others (refs. 10 and 18) have pointed out that the vestibular stimulation encountered in space stations

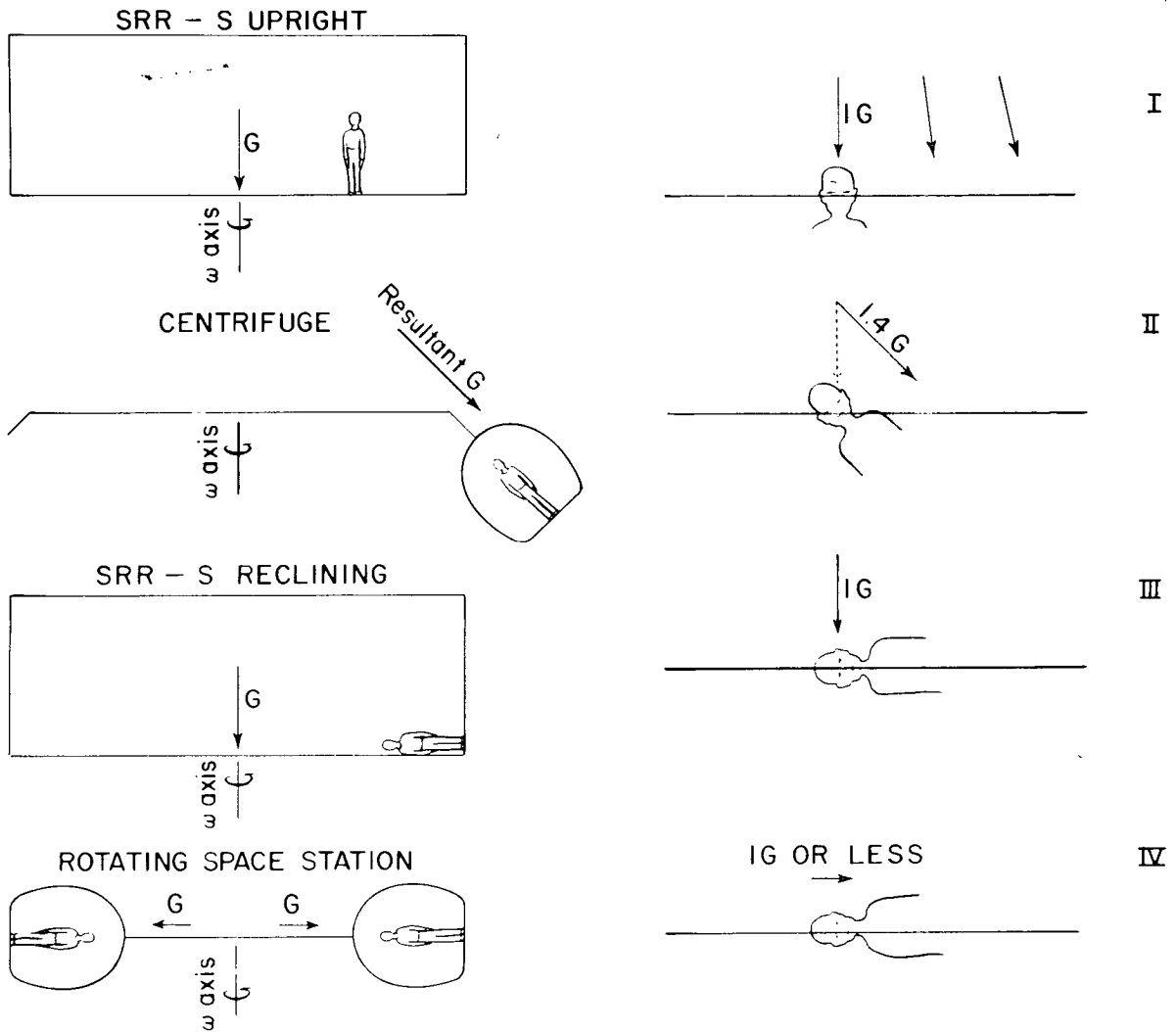


Figure 1.—Schematic representation of several rotation environments. Figures to right illustrate position of man relative to resultant "G-vector" and relative to plane of rotation. In I, arrows designate direction of resultant G-vectors in several positions in the SRR assuming a maximum radius of 7 ft and an angular velocity of 10 rpm.

and in rotating rooms on Earth will have certain similarities and certain differences. Considering an individual in upright posture in both situations, the similarity amounts to the fact that in both situations most head movements will produce "Coriolis vestibular reactions" as defined by Schubert (ref. 5) and by Bornschein and Schubert (ref. 1); the differences lie principally in the fact that particular head movements relative to the body would give different Coriolis vestibular reactions in the SRR and in a rotating space station. More-

over, in the SRR with the subject erect, a given head tilt relative to the body gives the same reaction irrespective of the direction the subject faces relative to the center of rotation, but in the space station the reaction should vary with each new heading of the subject.<sup>1</sup>

<sup>1</sup> Walking is possibly more difficult due to Coriolis acceleration in the SRR than it will be in a space station. Walking in a concentric path in a space station probably would reduce distortion from Coriolis acceleration. However, the change in weight from changes in centripetal acceleration during walking would be a problem in a space station.

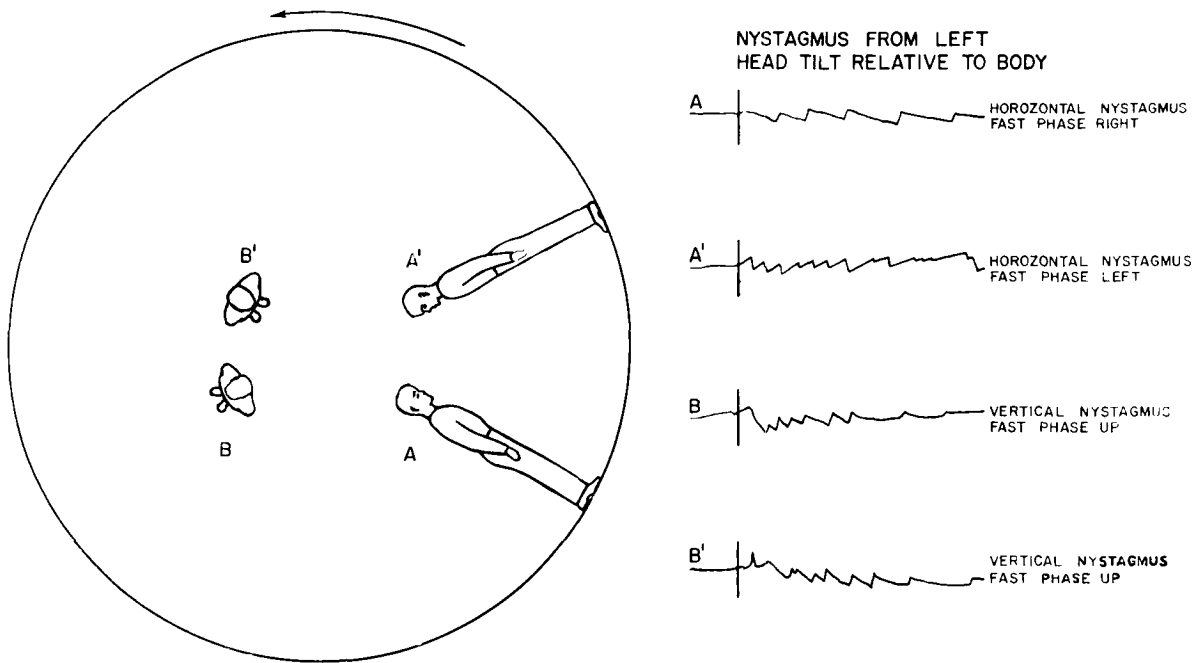


Figure 2.—Nystagmus produced by same head movement relative to body when body was in different orientations during 10 rpm, counterclockwise rotation of the SRR. (Variation of fig. in ref. 17.)

Both of these points are illustrated in figure 2. The same head movement relative to the body is made in all four positions, viz., head tilt toward the left shoulder—yet the reactions in A and A' are horizontal nystagmus, while the reactions in positions B and B' are vertical nystagmus. In the B and B' positions with the subject upright, the reactions are the same, but B and B' differ from reactions in A and A' and A and A' yield reactions which differ from one another.

In order to further check predicted reactions under circumstances which most nearly approximate the canal stimulation in the space station, we had subjects, in various positions on the deck of the SRR, make a number of common head movements as shown in figure 3. Twelve subjects made head movements in the positions shown in figure 3. Nystagmus was recorded by the corneoretinal potential technique with vision excluded by an eye mask. Vertical and horizontal components of eye movements were re-

corded to permit vectorial resolution of the actual direction of eye movement relative

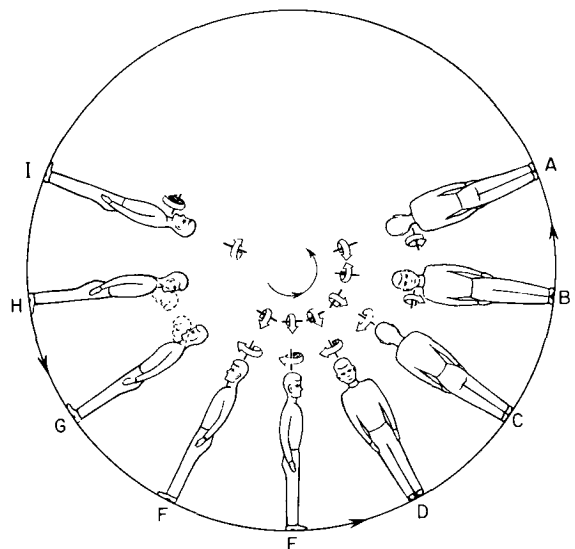


Figure 3.—Various positions and head movements tested in SRR during 10 rpm counterclockwise rotation. Arrows nearest head show axis and plane of head rotation. Arrows nearest center show axis and plane of stimulation.

to the skull. Rate of rotation was 10 rpm in a counterclockwise direction.

The arrow nearest the head illustrates the plane and direction of the head rotation while the arrow nearest the center of rotation is one way to visualize the stimulus, from Coriolis accelerations, which influences the canals. The stimulus may be conceptualized as a plane having an axis at right angles to the head-rotation axis and at right angles to the room-rotation axis. As the plane of one canal is moved toward coincidence with the stimulus plane, its stimulation would increase while stimulation to orthogonal canals would diminish. Since the plane of nystagmus tends to coincide with the stimulus plane, the plane of nystagmus relative to the skull can be estimated in each of the situations.

Note that the head movements in C, D, E, and F are all the same relative to the body, i.e., a rotation of the head toward the right shoulder, and the stimulus plane is the same relative to the rotary structure, but in each case the head is in a different position relative to the stimulus plane. These particular head movements, i.e., turning left and right, produce little or no effect when a person is standing upright in the SRR; but with a radial orientation of the body, they produce a variety of effects as will be seen from the results.

In conditions A, B, G, and H, head movements relative to the body are identical but the relationship of the head-movement axis relative to the axis of rotation varies. We should anticipate primarily horizontal nystagmus with fast phase left in A and horizontal nystagmus with fast phase right in B. In G and H, the head-rotation axis is parallel to the room-rotation axis, and hence we should have no nystagmus from the Coriolis canal stimulus in either G or H. In the case of I, a different head movement relative to the body is made, namely, a lateral tilt toward the right shoulder, but the axis and direction of head rotation relative to the rotating structures is the same as in A. Since the horizontal canals would be approximately

in the stimulus plane in both A and I, nystagmus would be about the same in A and I.

Results for positions C, D, E, and F are shown in figure 4. Here it is apparent that the same head rotation relative to the body has produced different reactions. The arrows showing the recorded reactions illustrate the direction and magnitude of the nystagmus. Magnitude is represented by amount of displacement of the arrow above the X-axis. Direction of nystagmus relative to the skull is represented by direction of the arrow relative to the viewer—for example, an arrow directed upward and to the viewer's right designates diagonal nystagmus up and to the right relative to the subject's skull. These directions and magnitudes were obtained by resolving horizontal and vertical components of the eye movement recordings and represent averages from 12 subjects.

To the right of the figure are shown nystagmus predictions based on the estimated stimulus plane relative to the skull. The small arrow under *Predicted Nystagmus* shows the reaction to be expected from angular acceleration of the head around the head-tilt axis. This is independent of room rotation and would occur even without room rotation. The effect of this stimulus would not persist beyond termination of the head movements which were completed in 2 or 3 seconds. Hence, the first one or two plotted points should be influenced by this effect but the residual effect after the head movement was completed would come from the Coriolis canal reaction represented by the larger arrow on the right. The drawings to the left show the top of the head as viewed from the center of rotation. In C, for example, the man turns his head toward his right shoulder while lying face down in the room which is rotating in a counterclockwise direction, i.e., to the viewer's left. The arrow indicating plane of room rotation represents the far edge of the rotation plane; the axis of room rotation should be visualized as perpendicular to this arrow and behind the viewer. Although the same head movement relative to the body is made in each case, the nystagmus

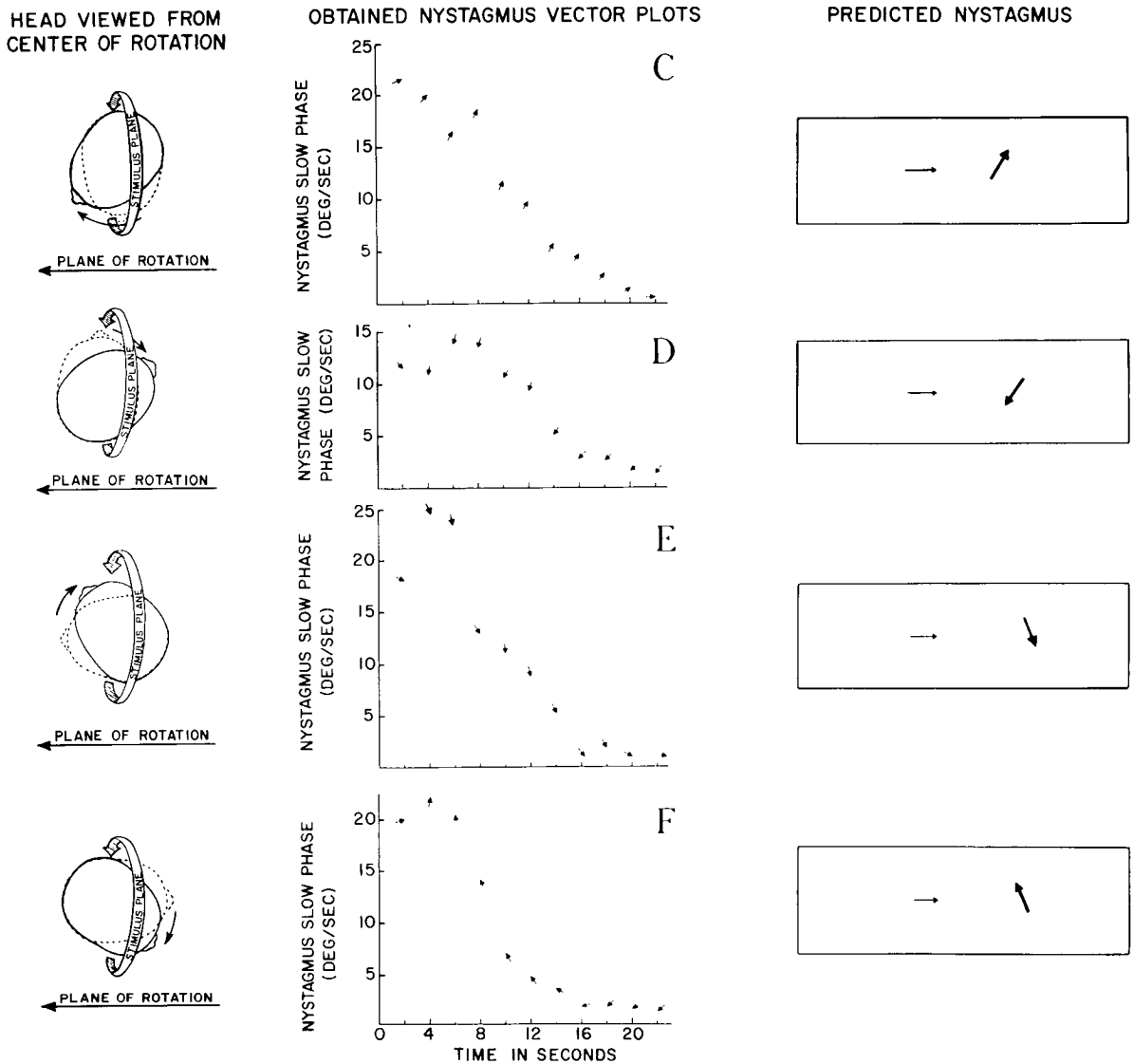


Figure 4.—Results obtained from 12 subjects in Conditions C, D, E, and F.

differs in direction and plane from one condition to the next, as is predictable from the mechanics of concomitant rotation about two axes.

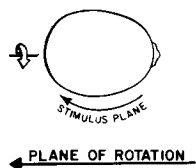
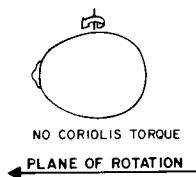
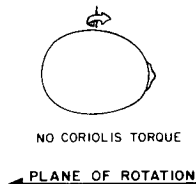
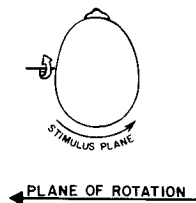
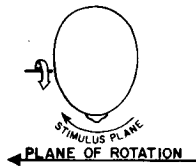
Results for positions A, B, G, H, and I are shown in figure 5. Head movements relative to the body are the same in A, B, G, and H, although the relationship between the head velocity vector and the room velocity vector is changed from one condition to the next. In A and B we merely reverse directions of head rotation relative to the room; hence,

the principal effect is to reverse directions of nystagmus. In G and H, the axis of head rotation and the axis of room rotation are parallel to one another and hence no Coriolis canal stimulus should develop. As can be seen, there is very little response to G or to H except that to be expected from the angular acceleration about the head-movement axis.

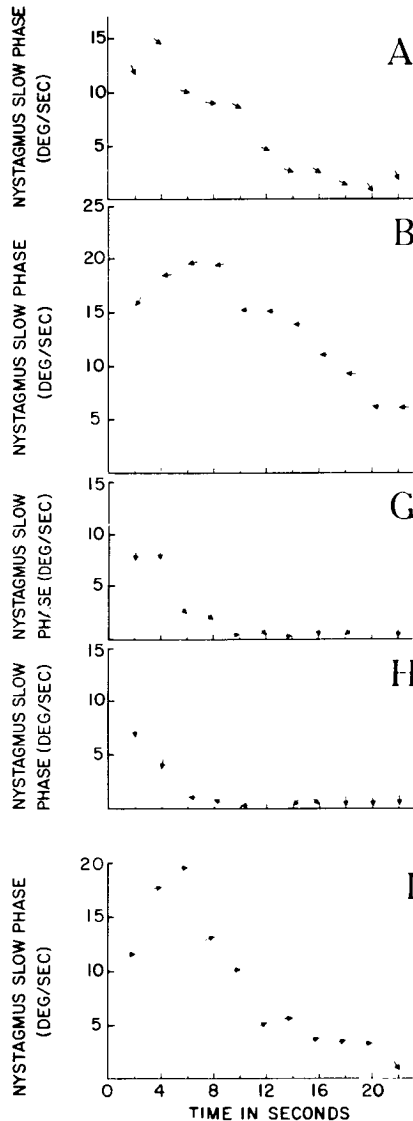
In I we expect little recordable nystagmus from the angular acceleration about the head-movement axis because this nystagmus would



HEAD VIEWED FROM CENTER OF ROTATION



OBTAINED NYSTAGMUS VECTOR PLOTS



PREDICTED NYSTAGMUS

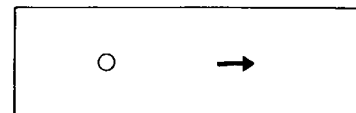
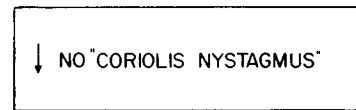
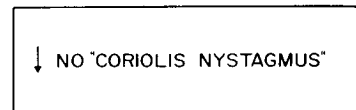
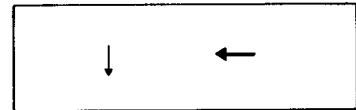
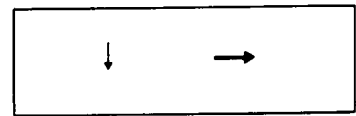


Figure 5.—Results obtained from 12 subjects in Conditions A, B, G, H, and I.

be in the frontal plane, i.e., about the corneoretinal axis, and we get none. However, after the head movement, the residual effect from the Coriolis canal stimulus should be almost the same in A and I in regard to plane and direction of nystagmus, even though A and I are different head movements relative to the body. As can be seen, A and I yielded similar results in regard to plane and direction of response.

These results illustrate clearly that with the man oriented radially as he will be in a space station, the same head movement relative to the body may elicit a variety of reactions depending upon the man's orientation relative to the direction of rotation of the vehicle. The reactions follow predictions from the mechanics of rotating systems fairly closely. The question now arises as to whether the gravity vector at right angles

to the plane of rotation in the SRR would alter the responses to be anticipated in the space station in which the "gravity vector" is oriented radially. (Reference to conditions III and IV of figure 1 will clarify the two situations in question.) Since the Coriolis canal stimulus would be of the same magnitude, plane, and direction in both situations, it seems unlikely that the nystagmus responses would differ in plane or direction for these two situations, especially since nystagmus in the SRR followed the stimulus plane irrespective of the direction of gravity relative to the skull. However, there is evidence from another line of experimentation that some responses recorded in the SRR with the subject in the plane of rotation may have been reduced in intensity as compared with responses to be anticipated in a space station, assuming the same rate of rotation for the SRR and the space station.

This evidence comes from experiments (refs. 11 and 12) in which subjects have been rotated around a horizontal axis of rotation as shown in figure 6. Note that in the two situations illustrated in this slide, the horizontal canals would have the same

orientation relative to the axis of rotation for either vertical-axis or horizontal-axis rotation, and hence, a given angular acceleration about either axis should deliver the same stimulus to the canals. Hence the response in these two situations should be the same. However, with rotation about the vertical axis, gravity sensors would not supplement or deny the rotational sensation during or after rotation. But with rotation about the horizontal axis, gravity sensors would supplement the response during rotation and would deny the rotary sensation after rotation.

Figure 7 shows that nystagmus during constant angular velocity about a horizontal axis is exceptionally long in duration, and, more important for our discussion today, nystagmus after rotation is very short in duration. The same is true for the sensation of rotation; there was a continuous sensation of spinning during rotation and little or none of the expected sensation of counter-rotation from the deceleration. Apparently the continual reorientation relative to gravity of the otolith system and other gravity sensors supplemented semicircular canal stimulation and maintained nystagmus during rotation about the horizontal axis, but when this rotation was stopped, the fixed position of these same sensory systems (including pressure cues) was sufficient, in this body orientation, to reduce nystagmus and almost completely suppress the sensation of rotation which was expected from the deceleration. This reduced reaction on stopping is significant because this particular orientation relative to gravity is the same as that used in the experiments in the SRR with subjects lying in the plane of rotation. It is reasonable to suspect that certain reactions experienced in the SRR following head movements were suppressed by the same cues from gravity and that these reactions would be more intense in a rotating space station for those conditions where there would be no conflict between the canal response and cues from the G vector, e.g., A, B, and I, in figure 3.

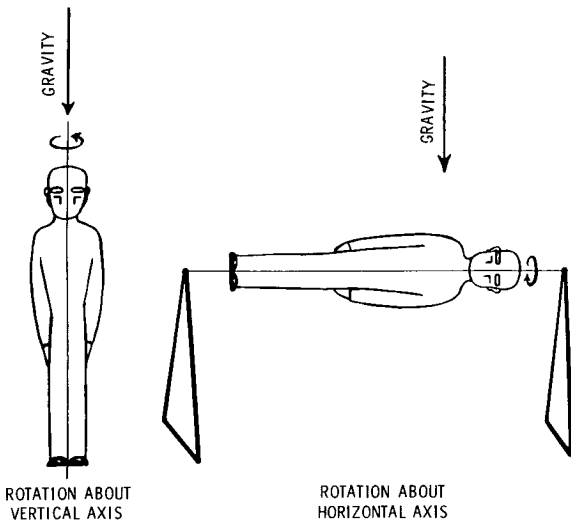


Figure 6.—Two axes of rotation. Horizontal semicircular canals were in plane of rotation in both situations but only in situation to right would gravity sensors supplement canal information during rotation and counteract canal information after rotation. (From ref. 12.)

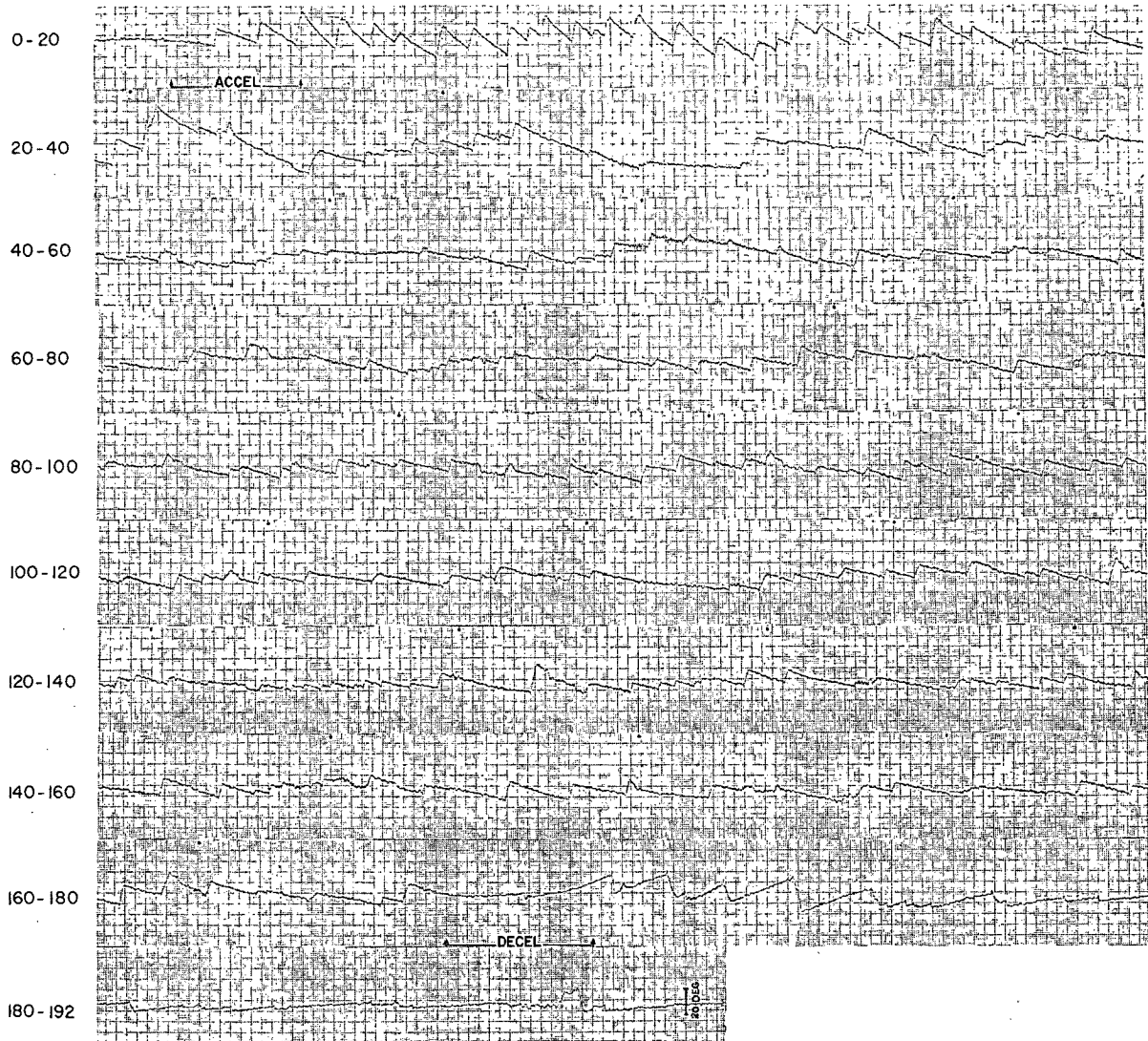


Figure 7.—Prolonged response during rotation about a horizontal axis and short response following rotation about this axis. The short "after response" in this body orientation relative to gravity suggests that reactions to coriolis canal stimuli in similar body orientations may be suppressed by the "G-vector." (From ref. 12.)

Moreover, there is another factor which is apt to influence sensations of apparent motion and that is the apparent stability of the vehicle producing the stimulation. A person sitting in an office seldom feels that the entire building is beginning to rotate. However, people waiting for the beginning of rotation in the SRR frequently feel that rotation has commenced before it actually does. When adequate vestibular stimuli are received while a person is in a vehicle of

questionable stability, such as a space station, a definite sensation of tumbling of the vehicle as a whole is likely to occur. Occupants of rotating space stations should be forewarned of this possibility, and placement of instruments in sufficient number of locations to permit checks of stability would probably be helpful, particularly if rates of rotation exceed 4 or 5 rpm.

Let us now consider briefly the differences in the space station and the SRR in regard

to adaptation. Upright posture, either standing or sitting, is the posture from which most active, purposeful movements are initiated, and there is evidence that active movements which involve what Von Holst (ref. 13) has conceptualized as "reafference" are important in the adjustment which takes place in sensory rearrangement experiments (ref. 14). If we assume that active movements are a primary factor in the adjustment that occurs in a space station, then we are faced with the fact that during upright posture in a space station, similar head movements relative to the body will produce different reactions as the person faces in different directions. This suggests that adjustment in the space station may be more difficult than it is in the SRR, but there are several reasons for optimism: (1) The consistency of the stimulation received in the SRR probably facilitates habituation, but it also facilitates development of specific compensatory reactions (refs. 15 and 16) set off by each head movement. These reactions would be undesirable effects as the man leaves the space station, and they might not develop in a space station where active movements would produce varying responses for each new heading of the person. Hence, a person leaving the rotating station might not

have these compensatory effects. (2) Subjects in our prolonged rotation experiments (refs. 16 and 10) spent many resting hours in the plane of rotation and seemed as well adjusted in this orientation as in upright posture. Although gravity still served as a factor which permitted discrimination of the direction of the subject's head movements relative to the direction of rotation, this situation did produce a variety of vestibular reactions for specific head movements relative to the body, and adaptation did occur. Moreover, in a space station, visual cues can be arranged to permit discrimination of the direction of head movements relative to the direction of rotation. (3) The results from several of our experiments in which individuals have been rotated for 12 days at 10 rpm in one direction indicated that the initial highly specific compensatory reactions which are manifest when a person (in upright posture) first leaves the SRR later give way to a general suppression which carries over even to the opposite direction of rotation. This is illustrated in figure 8. This indicates the possibility that habituation to complex vestibular stimulation in one kind of rotation environment may have a beneficial influence in reducing undesirable effects in other rotation environments.

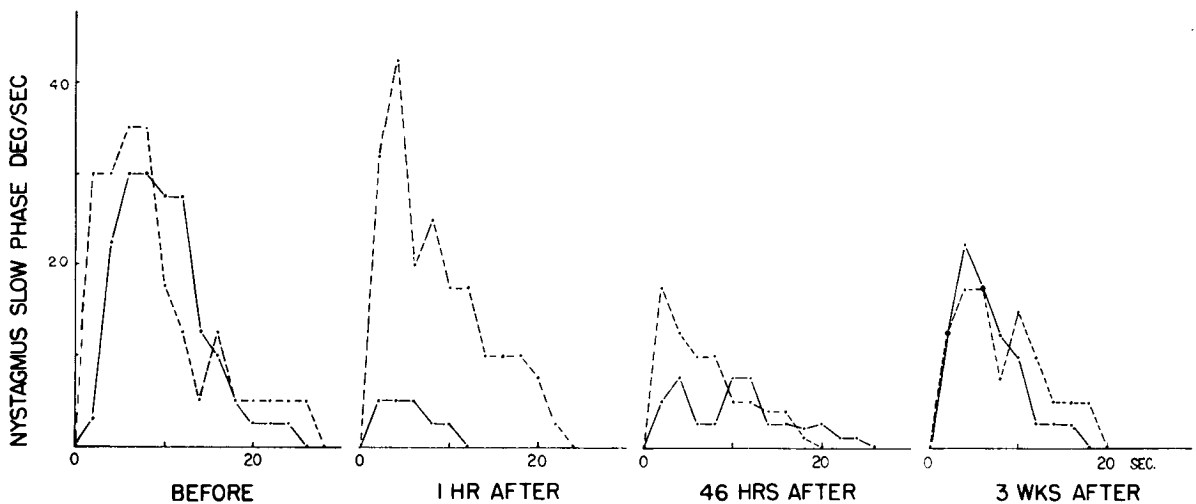


Figure 8.—Nystagmus responses to head movements during 10 rpm rotation before and at several intervals after a period of 12 days of rotation at 10 rpm in a counterclockwise (CCW) direction. Responses during CCW and CW rotation are indicated by solid and dotted lines, respectively.

To increase our confidence that habituation will occur in a rotating space station and also that habituation here will afford some protection in space, we can pursue several lines of research, some of which are already in progress: (1) The study of habituation of individuals who are aligned radially in the plane of rotation in the SRR, and who also are provided with apparatus permitting active head and body movements while in this situation. (2) The study of vestibular habituation with subjects upright in the SRR but with the direction of rotation of the SRR frequently reversed. Both of these situations will yield variations in reactions for each head movement relative to the body

and, assuming that satisfactory adaptation occurs, they will provide added confidence of ability to adapt to rotating space stations. (3) Study of transfer of vestibular habituation. It is apparent that there are differences in the various rotation environments considered, and that the exact conditions of a rotating space station cannot be produced on earth. Hence, continued study of transfer of habituation between various rotation environments on earth is an important direction of research for insuring that we can provide some protection through habituation for the eventual occupants of rotating space stations.

#### REFERENCES

1. BORNSCHEIN, H.; AND SCHUBERT, G.: Die Richtung des Vestibulären Coriolis-Effektes. *Zeitschr. für Biologie*, 113, 1962, pp. 145-160.
2. MEDA, E.: A Research on the Threshold of the Coriolis and Purkinje Phenomena of Excitation of the Semicircular Canals. *Arch. Fisiol.*, 52, 1952, pp. 116-134. (Translated by E. R. Hope. Defense Research Report T 17 1, Defense Scientific Information Service, DRB Canada, 1954.)
3. GUEDRY, F. E.; AND MONTAGUE, E. K.: Quantitative Evaluation of the Vestibular Coriolis Reaction. *Aerospace Med.*, 32, 1961, pp. 487-500.
4. LANSBERG, M. P.: *A Primer of Space Medicine*. Elsevier Publishing Co. (New York), 1960.
5. SCHUBERT, G.: Die Physiologischen Auswirkungen der Coriolisbeschleunigungen bei Flugzeugsteuerung. *Zeitschr. für Biologie*, 30, 1932, pp. 595-604.
6. WEAVER, R. S.: Theoretical Aspects of the Role of Angular Acceleration on Vestibular Stimulation. DRML Research Paper 565, DRB Canada, 1964.
7. BENSON, A. J.; AND WHITESIDE, T. C. D.: The Effect of Linear Acceleration on the Response to Angular Acceleration in Man. *J. Physiol.*, 156, 1961, pp. 6-7.
8. LANSBERG, M. P.; GUEDRY, F. E.; AND GRAYBIEL, A.: The Effect of Changing the Resultant Linear Acceleration Relative to the Subject on Nystagmus Generated by Angular Acceleration. *Aerospace Med.*, 36, 1965, pp. 457-460.
9. LANSBERG, M. P.: The Physiologic Accelerometers. *Aeromed. Acta*, 8, 1961-62, pp. 37-43.
10. GUEDRY, F. E.; KENNEDY, R. S.; HARRIS, C. S.; AND GRAYBIEL, A.: Human Performance During Two Weeks in a Room Rotating at Three RPM. *Aerospace Med.*, 35, 1964, pp. 1071-1082.
11. CORREIA, M. J.; AND GUEDRY, F. E.: Influence of Labyrinth Orientation Relative to Gravity on Responses Elicited by Stimulation of the Horizontal Semicircular Canals. NSAM-905. NASA Order R-93, Naval School of Aviation Medicine, 1964.
12. GUEDRY, F. E.: Orientation of the Rotation-Axis Relative to Gravity: Its Influence on Nystagmus and the Sensation of Rotation. *Acta Otolaryng.*, 60, 1965, pp. 30-48.
13. VON HOLST, E.; AND MITTELSTAEDT, H.: Das Refferenzprinzip. *Z. Naturwiss.*, 37, 1950, pp. 464.
14. HELD, R.: Exposure History as a Factor in Maintaining Stability of Perception and Coordination. *J. Nerv. and Ment. Dis.*, 132, 1961, pp. 26-32.
15. GUEDRY, F. E.: Visual Control of Habituation to Complex Vestibular Stimulation in Man. *Acta Otolaryng.* (Stockh.), 58, 1964, pp. 377-389.
16. GUEDRY, F. E.; AND GRAYBIEL, A.: Compensatory Nystagmus Conditioned During Adaptation to Living in a Rotating Room. *J. Appl. Physiol.*, 17, 1962, pp. 398-404.
17. GUEDRY, F. E.: Vestibular System. *Bioastronautics Data Book*, NASA SP-3006, 1964, pp. 363-381.
18. GUEDRY, F. E.: Habituation to Complex Vestibular Stimulation in Man: Transfer and Retention of Effects from Twelve Days of Rotation at 10 RPM. *Percept. and Motor Skills*, 1965.

## DISCUSSION

COHEN: I was quite pleased to see some of Held's research brought into this because I think that the active-movement factor and its relation to sensory feedback is important. One thing about the space station is particularly interesting. You said that there might be no compensation because of the different sensory contingencies of the head movements, namely that for a given movement, the sensory feedback would be different, depending upon which way the person is facing. If, for example, I turn my head to the right while facing in one direction, one thing would happen, but if I turn my head to the right while facing in the opposite direction, something different might happen.

On Earth we're used to moving around freely in two dimensions, but somehow or other, we're not too disappointed that, to get from here to a distant point on the globe, even if we wanted to, we can't go in a straight line; we have to travel in an arc. I was wondering if anyone had considered the possibility of having "one-way streets" in a space station so that to get from a point here to a point that is, say, two feet behind me, I would have to go around the space station. This type of setup would provide consistent sensory feedback for a given direction of movement. It would eliminate the problem because you would have constant contingencies established between your head movements and the resultant sensory feedback.

GUEDRY: Well, I wouldn't believe it would be feasible to require people always to face the same direction in a space station, which is more or less what you're suggesting. Moreover, I don't believe, at reasonable rates of rotation, that it will be necessary.

COHEN: The suggestion is not really that people would always be required to *face* in the same direction, but rather, that they would always be required to *move actively* in the same direction so that consistent relations between active movement and sensory feedback would be provided. In this way, theoretically at least, adaptation could take place.

Another possibility concerns the color-coding of the space station. I was wondering if you're familiar with some of the recent work of Ivo Kohler. The types of *conditional* adaptations he describes may be of some value here.

GUEDRY: I'm familiar with Kohler's work, and we believe that his research is pertinent to the "sensory rearrangement" experiments in the SRR.

BEAUCHAMP: I believe, in figures 3 and 4 where the man is lying radially and he nods his head, you stated there would not be any Coriolis reaction. I think this is wrong because he's actually changing his spin radius. In going from an upright head position to a chin down, there is a change in radius. And the second comment is that if he's rotating about a horizontal axis on the floor of a rotating space station, I think he's going to have some horrible

reactions which are compounded by alining him in either one of two directions. If he's alined in the plane of the space station with his longitudinal axis normal to the space station spin axis and actually rolls in this local horizontal plane, then the Coriolis component alternates between codirection and contra-direction with respect to the space station rotation.

GUEDRY: In answer to the first comment, only in situations G and H did I say that there would be no "Coriolis canal stimulus." There would be a "Coriolis canal stimulus" in all other situations illustrated. In G and H of figure 3, the axis of rotation of the head is parallel to the axis of rotation of the SRR and from the mechanics of rotation we predicted that this would not produce a "Coriolis canal stimulus" and the results presented in figure 5 substantiated this prediction. It is true that Coriolis acceleration would be present in this situation but Coriolis acceleration per se does not always constitute a stimulus to the canals, presuming that the canals act mechanically like angular accelerometers. For example, linear movement of a set of angular accelerometers in the plane of rotation of the SRR involves Coriolis acceleration, but it does not actuate the angular accelerometers.

In answer to your second comment, I didn't get into that situation at all. In all situations in figure 3 subjects were alined radially, which would be the same thing as standing upright in the space station. In figure 6, I was not referring to a rotating room or a rotating space station; the man was spinning about his own long axis.

BEAUCHAMP: The comments in connection with figure 6 are based on the supposition that if he was in the space station and rotated like this, he would run into some very disturbing effects.

GUEDRY: Yes, but why would he do a thing like that? As a matter of fact, this particular situation (rotating about an Earth-horizontal axis) produces nausea in more than 50% of cadets tested right here on Earth after only a few minutes of stimulation.

VON GIERKE: I do not think you said what rotational speeds you employed in this configuration.

GUEDRY: In the situations illustrated in figure 6, the rate of rotation was 10 rpm. The rate of rotation in the Slow Rotation Room experiments (figs. 3, 4, and 5) was also 10 rpm. The head movements were of a duration of approximately 2 to 3 seconds in the SRR experiments.

VON GIERKE: The particular experiment involving rotation about a horizontal axis has been proposed for producing average zero gravity on Earth on the otoliths. I think you have to go quite a bit higher with your rotation rate to expect theoretically that you will approach such a situation.

GUEDRY: Well, we were not trying to do that here. However, we did spin people up to 30 rpm and that is the subject of a completely different paper. But

at 30 rpm, reactions do break down; we get a reversing nystagmus at 30 rpm in normal subjects, which is like responses of people without inner ear function at 10 rpm. This suggests that we are starting to exceed the rate of response of the otolith. This idea was suggested by Levine (Lockheed, Marietta) and others some time ago. We are not as yet confident that this influence comes solely from the otoliths. I like to think that it does.

LAWTON: You referred to the problems of stability in a space station and I wonder if you'd care to comment further. The engineers are surely going to ask for the tolerable rates of wobble in a rotating space station, in association with head motion, the tolerable rates of change in rate of rotation, and the interactions of these various factors. This is one of the significant areas where the Slow Rotation Room deviates from a real space station. The plane of rotation does not deviate and the rotation rate is always constant. In an operating space station, with people moving within it, these conditions will not pertain.

GUEDRY: It's my guess and I would like to think this out further, that minor wobble in a space station would not produce semicircular canal stimulation comparable to that produced by tilting the head relative to the rotating vehicle. Let me go on to the other point that I can say a little more about. It appears that the disturbance in going to a high speed is reduced by spinning for awhile first at low speed; and, actually, Dr. Bergstedt has done some work on this and will be reporting this later. It looks favorable, though, to start with a lower speed and then to go to a higher speed. As a matter of fact, in some earlier experiments on this, several of us (George Crampton and I) were very much disturbed by head movements during rotation and we felt that head movements during a slow spin first reduced the disturbance during head movements at higher spin rates quite a bit.

LAWTON: I was thinking more of, say, 10 rpm varying  $\pm 1$  rpm.

GUEDRY: You mean a constantly changing angular velocity?

LAWTON: Not necessarily constant, but perhaps random.

GUEDRY: I can't answer that with authority. Adjustment to a random change in speed would probably be more difficult than adjustment to constant speed. Dr. Bergstedt is going to talk on that general subject.

J. MILLER: Inasmuch as we're also concerned with head motions, have you done any work in Pensacola pertaining to physical restraints, such as head or neck braces, so that a person can only move his head in particular planes?

GUEDRY: In our first 10 rpm run we had people fitted with neck braces. This was Dr. Graybiel's idea, and it seemed to help by reducing unnecessary head

motions, but the braces were uncomfortable and the subjects took them off after several hours. Of course, as adaptation progresses, there is no need for restricting head movements, and one of our subjects in the first 12-day 10 rpm run seemed able to move freely after only a few hours. He was able to make all necessary motions from the beginning.

VON GIERKE: Did you ever consider having a gadget in the rotating room similar to the Moon gravity simulator at Langley so that the person would hang in slings and walk on the periphery of the room? If you would do this so that he could turn around his longitudinal axis, he could walk forward and backwards in this rotating environment. This might be a good simulator.

GUEDRY: Dr. Graybiel has commenced a project on this, and the equipment is being prepared to permit this same kind of motion within the SRR.

VON GIERKE: In both directions?

GUEDRY: Right. We're not considering the suspension system, but we are considering a system which will permit people to walk freely around the wall.

VON GIERKE: I was very surprised walking on this Langley simulator how realistic the impression is of what one considers "down," i.e., the Earth's surface. After a while you really think the wall, on which you walk, is "down."

M. JONES: When the subject in the space ship is rotating around and then turns his head to the left, I would expect him, by the time he got through 90°, to have a diagonal response.

GUEDRY: The plane of nystagmus changes relative to the skull as you turn.

M. JONES: If I was to move smartly around (90 deg turn), I'd expect the response to be first horizontal and then vertical.

GUEDRY: I'm sorry, I should have indicated that these head movements were about 40°. They were not 90°. If you rotate the head from 45 deg left to 45 deg right, while the room is rotating CCW, the nystagmus is initially vertical with fast phase up and to the right, and as you continue through to right tilt, it shifts on over and goes up and to the left.

M. JONES: I would like to raise a point in connection with your beautiful demonstration that a rotating G vector apparently generates almost as significant a physiological response to rotational movement as does the semicircular canal system. There are many situations, even in conventional atmospheric flight, in which the relation between the rotation signals derived from the canals and from the rotating G vector is discrepant. For example, when rolling into a turn, the canals register correctly (provided of course the roll maneuver is of short duration), while there is no G vector rotation relative to the pilot. Perhaps in this case his "image stabilizing" oculomotor response will suffer accordingly.

Again, during the pushover from a steady climb

I think one can show that the G vector when vectorally combined with the induced radial acceleration will rotate "backwards" with respect to the pilot, tending to give him the impression he is pitching upwards into a loop; while canal information will say he is pitching down. Perhaps if the response to the G vector dominates, a serious illusion could develop which, in view of your experimental results, may be expected to be supported by corresponding retinal image movement due to the resulting "compensatory" oculomotor response; looking inside the cockpit the visual impression would then be of the aircraft rotating upwards.

It occurred to me that this possibility might have relevance in connection with some recent civil aircraft accidents in which the aircraft appears to have nosed over into a steep dive, when, at least in one case I believe, the pilot and copilot thought they were going up and over onto their backs. Would you care to comment on these possibilities?

GUEDRY: I have read recently in Life Magazine about some of these recent accidents in that kind of situation. I never thought about it in this context before, but the rotation of the resultant vector as you describe it could influence both the sensation and nystagmus to give an erroneous sensation of motion.



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## Vestibular Problems in Rotating Spacecraft

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It is now well known that in a rotating environment certain movements of the head cause motion sickness. The sickness under these circumstances is associated with Coriolis accelerations which occur with movements in a rotating environment (ref. 1) and which appear to be peculiarly effective in causing motion sickness. The term "Coriolis acceleration" is often used as a synonym for "semicircular canal stimulation," and the effectiveness of Coriolis accelerations in causing motion sickness is apparently dependent upon the presence of functioning semicircular canals (from "Role of the Semicircular Canals in Motion Sickness and Positional Alcohol Nystagmus in the Squirrel Monkey" by A. Graybiel, W. H. Johnson, and K. E. Money, in preparation), but it is of interest to note that Coriolis acceleration is actually a linear acceleration which could be expected to stimulate the otoliths, depending on their thresholds, whenever it stimulates the semicircular canals and in many circumstances under which the canals are not stimulated.

Another vestibular problem in rotating environments, also associated with Coriolis accelerations, is the instability of body posture while moving. An artificial gravity of 0.2 g will allow man to walk unaided (ref. 2), but even with a radius of 40 feet, the rotation required to produce the artificial gravity (4 rpm) will create locomotion difficulties. The magnitude of the Coriolis acceleration affecting a body in a rotating environ-

ment depends on the magnitude and direction of the body's velocity relative to the rotating environment. In other words, the Coriolis acceleration is different for different speeds and directions of movement. This variability in the incident accelerations is undoubtedly responsible for the instability of the body while walking, and if an astronaut (for example, in an emergency) were walking unusually fast in a rotating spacecraft, he would be unusually likely to fall or bump into things because unusually large Coriolis accelerations would be applied both to the vestibular apparatus and to the limbs and other parts of the body.

One other vestibular problem possible in rotating environments is a decrement in visual acuity during head movements. Astronauts walking in a rotating spacecraft would probably be less able to read small digits than when walking on Earth. In the normal environment on Earth, eye movements (which occur automatically because of vestibular stimulation with the head movements associated with walking) stabilize the retinal image and improve visual acuity. The improvement of visual acuity, while walking or otherwise moving the head, can be considered the basic physiological function of the vestibulo-ocular system. The complaint of people who have recently lost the function of the inner ears that they must stop walking and stabilize the head in order to read street signs (personal communication, W. J. McNally), is readily understood on this basis. In rotating environments, movement of the head results in vestibular stimulation which

causes *inappropriate* eye movements which almost certainly *decrease* visual acuity.

Of these three vestibular problems in rotating environments, the motion sickness problem has been investigated most extensively, because the occurrence of motion sickness in space could cause a catastrophe. On the basis of studies in rotating rooms it has been suggested that the rate of rotation of spacecraft should be kept as low as possible, preferably 3 to 5 rpm (refs. 2 to 4), in order to avoid motion sickness.

#### PROPOSITION AND EVIDENCE

It is proposed here that, from the point of view of vestibular physiology, (1) zero g is acceptable for long term manned space flights, and (2) rotation rates of 3 to 5 rpm are hazardous.

(1) The evidence for concluding that zero g is acceptable for long term space flights, considering only motion sickness, is very straightforward. First, motion sickness has been found to occur in environments in which accelerations of the head are changing but not in environments of constant acceleration, so that it could be predicted that zero g would not cause motion sickness (ref. 5). Second, the several manned space flights already completed under zero g conditions have confirmed directly that zero g does not cause motion sickness. Only in the case of Titov are there available reports of sickness occurring in zero g, and in this case the effective stimulus appears to have been head movement "during rotation of the satellite" (ref. 6) and not zero g. Additional information of value in this regard could be obtained by observing in orbit (without rotation) experimental animals selected for exquisite susceptibility to motion sickness, and for comparison, animals made immune to motion sickness by temporal bone surgery.

(2) According to available evidence, if spacecraft rotating at 3 to 5 rpm are used for manned space flights of long duration, a risk of sickness because of vestibular stimulation will be present even if astronauts are selected for resistance to motion

sickness and ability to adapt, and even if they are given some kind of preflight adaptation. There are several reasons for believing this to be the case:

- (a) Except for extremely susceptible individuals, susceptibility to motion sickness in one situation is not a reliable indication of susceptibility in another situation (ref. 7). When John Glenn made his orbital space flight, for example, he was presumably not susceptible to sickness either in certain laboratory devices or in certain aircraft, but after the flight while floating in the ocean waiting to be picked up, he experienced stomach awareness (ref. 7). Titov also was presumably not susceptible to motion sickness in experiments involving laboratory devices and aircraft, but he was sick in space, apparently as a result of moving his head while his spacecraft was rotating.

Susceptibility to motion sickness, even in a rotating room, is not a reliable indication of susceptibility to motion sickness in a rotating spacecraft. The vestibular stimulus from a given head movement in a rotating room on Earth is not the same as that which would result from the same head movement in a rotating spacecraft. This is so because in a rotating room humans stand with the body's long axis parallel to the axis of rotation, but they would stand in a rotating spacecraft with the body's long axis at right angles to the axis of rotation (and the feet remote from the axis). In a rotating spacecraft, furthermore, as the astronaut turned to face in different directions, the stimulus from the same head movement would vary according to the direction in which he was facing; in rotating rooms, on the other hand, the same head movement normally gives the same stimulus.

This difference between rotating rooms and rotating spacecraft has been carefully explained by at least two authors (refs. 3 and 8) and it is an important difference. The rotating room on Earth and the rotating spacecraft present quite different conditions of stimulation, and it is therefore hazardous to predict susceptibility to motion sickness in either on the basis of experience in the other. It appears that there is no way of knowing definitely in advance whether or not an astronaut will be sick in a spacecraft rotating at 3 to 5 rpm.

- (b) Since the most extensive angular head movements in daily life are made about the spinal axis (as in shaking the head to say "No") and since in the standing or sitting posture this movement does not stimulate the semicircular canals with Coriolis accelerations in the rotating room but does in the rotating spacecraft, the spacecraft can be expected to provide the more intense stimulation, at the same rate of rotation.
- (c) Since the vestibular stimulus in a rotating spacecraft can be expected to be more variable (a) and more intense (b) than the stimulus in a room rotating on Earth at the same speed, if it is decided that the stimulus caused by a rotating room at 3 to 5 rpm is the *maximum* acceptable in space, then rotating spacecraft must be rotated at a speed *less* than 3 to 5 rpm.
- (d) The mechanism of adaptation to the rotating room includes the appearance of compensatory nystagmuses as a conditioned response to specific head movements (ref. 9). It is reasonable to expect that the part of the adaptation mechanism responsible for this phenomenon will be of no benefit whatever to the astronaut in a rotating spacecraft in which for the same

head movement the vestibular stimulus varies according to orientation of the head. Thus it appears that adaptation to the rotating spacecraft will be more difficult than adaptation to the rotating room, although presumably not impossible (ref. 10).

If the astronaut is to be adapted to the rotating spacecraft by experience in a rotating room, the direction of the room's rotation should be reversed frequently, and the astronaut should walk on the walls rather than on the floor of the rotating room in order to increase his chances of achieving an adaptation of real value in the rotating spacecraft. Available evidence indicates that adaptation to one kind of complex vestibular stimulation does not transfer to a different kind of complex vestibular stimulation (ref. 11), but the preflight achievement of adaptation to as similar an environment as possible is probably desirable if rotating spacecraft are to be used.

- (e) Symptoms and signs of motion sickness which have disappeared during prolonged exposure to a rotating room often reappear in the adapted individuals after the rotation has stopped. This is a possibility in rotating spacecraft also, although its occurrence is less likely than in rotating rooms because of the variability of the stimulus in rotating spacecraft.

#### DISCUSSION

It is impossible at the present time to estimate with any accuracy the likelihood of a given astronaut's becoming motion sick in a spacecraft rotating at, for example, 4 rpm, but one study (ref. 12) reveals that of two subjects who had less than average susceptibility to motion sickness both experienced nausea from head movements in a rotating room at 1.71 rpm; of three other subjects tested at 2.21 rpm, one had nausea and vomiting episodes, and two of these three when tested in a later experiment at 5.4 rpm had

nausea and vomiting episodes. Sickness at 3 to 5 rpm cannot therefore be considered unusual, and as indicated above, a rotating spacecraft can be expected to be a more potent stimulator than a room rotating at the same speed. A 1% chance of being sick in space might be considered unacceptable, and the likelihood of sickness occurring in a spacecraft rotating at 3 rpm appears to be considerably greater than 1%. Titov's experience illustrates that unexpected sickness, apparently motion sickness, can happen during space flight.

It seems unwise to accept a considerable risk of motion sickness on the grounds that adaptation will probably occur, because an astronaut may have to deal with a mechanical or other emergency while his well-being, and probably his performance also, are lessened by motion sickness or while he is incapacitated by vomiting, before adaptation occurs or (following adaptation) after the rotation stops. The presence of vomitus inside a space suit might constitute more than just a nuisance. Furthermore, adaptation to the motion of a rotating spacecraft might take two or three times as long as predicted, or adaptation might not occur; an astronaut might become sick and stay sick until he stops moving his head. These possibilities are not inconsistent with what is now known, and they suggest that it would be most unwise (at the current level of knowledge) to put men into a spacecraft which *must* keep rotating and in which men *must* move.

Before rotation of the spacecraft is adopted

in order to solve the cardiovascular problems of weightlessness, it should be demonstrated that the cardiovascular problems at zero g are a greater danger to the mission than the vestibular problems are at the proposed rate of rotation. It should also be demonstrated that the cardiovascular problems will be solved (at the available radius) by the rotation proposed, and that they cannot be solved in some other safer way, such as by periodic inflation of pressure cuffs on the limbs, by special exercises, by administration of appropriate drugs just before re-entry, by using g suits, or by a combination of such techniques. Another possibility is intermittent centrifugation.

#### CONCLUSION

From the point of view of vestibular physiology, and particularly motion sickness, zero g is acceptable for long term space flights, whereas rotation rates of 3 to 5 rpm are hazardous. Motion sickness has been reported in a room rotating at 3 to 5 rpm, and the stimulus in a rotating spacecraft can be expected to be more variable and more intense than the stimulus in a room rotating at the same rate.

If spacecraft must be continuously rotated to solve other problems, rotation should be slow enough to avoid creating vestibular problems which could cause a catastrophe. The speed that is slow enough can be determined by appropriate threshold experiments on Earth, and is probably of the order of 1 rpm (refs. 7 and 13).

#### REFERENCES

1. WEAVER, R. S.: Theoretical Aspects of the Role of Angular Acceleration in Vestibular Stimulation. DRML Research Paper 565, 1965. Defence Research Medical Laboratories, Postal Station "K," Toronto, Canada.
2. LORET, B. J.: Optimization of Manned Orbital Satellite Vehicle Design With Respect to Artificial Gravity. ASD TR 61-688, Dec. 1961, Aerospace Medical Laboratory, Wright-Patterson Air Force Base, Ohio.
3. GUEDRY, FREDERICK E.: Vestibular System. Bioastronautics Data Book, NASA SP 3006, 1964, pp. 363-381.
4. GRAYBIEL, A.: Vestibular Sickness and Some of its Implications for Space Flight. *In*: Neurological Aspects of Auditory and Vestibular Disorders (William S. Fields and Bob R. Alford, eds.), Ch. 11, Charles C Thomas (Springfield), 1964.
5. JOHNSON, W. H.: Some Vestibular Problems in Space Flight. *Ann. Otol. Rhinol. Laryngol.* 70, 1961, pp. 777-84.
6. SISAKYAN, N. M.; AND YAZDOVSKIY, V. I.: First Group Flight Into Outer Space: August 11-15, 1962. Nauka Publishing House, 1964. (English Translation. Joint Publications Research Service, Washington, D.C., 25, 272, TT: 64-31567, June 29, 1964.)

7. GRAYBIEL, A.: Predicting the Susceptibility to Vestibular Sickness Under Conditions of Weightlessness. *In: Proc. of First International Sym. on Basic Environmental Problems of Man in Space* (H. Bjurstedt, ed.), Springer-Verlag (New York), 1964, pp. 327-344.
8. LANSBERG, M. P.: The Physiologic Accelerometers. *Aeromedica Acta VIII*, 1961-62, pp. 37-43.
9. GUEDRY, F. E.; AND GRAYBIEL, A.: The Appearance of Compensatory Nystagmus in Human Subjects as a Conditioned Response During Adaptation to a Continuously Rotating Environment. NSAM-795, NASA order No. R-1. U.S. Naval School of Aviation Medicine, 1961.
10. LANSBERG, M. P.: Canal Sickness—Fact or Fiction. *Rev. Med. Aeronautique* 2, 1961, pp. 173-178.
11. GUEDRY, F. E.; COLLINS, W. E.; AND GRAYBIEL, A.: Vestibular Habituation During Repetitive Complex Stimulation: A Study of Transfer Effects. NSAM-884, NASA order No. R-93. U.S. Naval School of Aviation Medicine, 1964.
12. GRAYBIEL, A.; CLARK, B.; AND ZARRIELLO, J. J.: Observations on Human Subjects Living in a "Slow Rotation Room" for Periods of Two Days. *A.M.A. Archives Neurol.* 3, 1960, pp. 55-73.
13. KENNEDY, R. S.; AND GRAYBIEL, A.: Symptomatology During Prolonged Exposure in a Constantly Rotating Environment at a Velocity of One Revolution Per Minute. *Aerospace Med.* 33, 1962, pp. 817-825.

### DISCUSSION

**FIELDS:** In respect to the cardiovascular problems and how they relate to the vestibular function, there is at least one situation in which there is a subjective response that may be mistaken for a vestibular problem. It is recognized that under conditions of zero  $g$  and also under conditions of  $g$  influence, one may have vagal preponderance which produces bradycardia and gastrointestinal uneasiness. This may be misinterpreted as vestibular sickness. I think one has to be very careful to avoid making this mistake.

**MONEY:** Yes, that is certainly true.

**THOMPSON:** In speaking of Titov's flight, I believe that the Russians knew he was susceptible to motion sickness. I saw a film which they showed at the COSPAR meeting about 2 years ago where they had him in a three degrees of freedom rotational simulator, rotating him at various combined rates of rotation. When he emerged, this was color film, his face was absolutely green. I think they knew this and that this was part of the investigation.

**MONEY:** Yes. Under the circumstances which you described almost anyone would have come out looking green.

**GUEDRY:** I would like to comment on your statements in connection with sickness at 3 rpm. We did a 2-week 3 rpm run. Among the subjects selected, we purposely included one subject who was fairly sensitive and although there was some nausea reported, none of the subjects became sick to the point of vomiting in this run.

**MONEY:** Concerning that experiment at 3 rpm, actually it was 1.71 and 2.21 and again something over 5. That particular experiment was done by Graybiel, Clark, and Zariello, and it was stated that two of the mentioned four subjects who did become sick at lower rpm rates were described as being above average in resistance to motion sickness and, about the others, there was no comment whether they were more or less susceptible than average, and I took them to be average subjects. Although I know

in many studies no one got sick at 3 or 4 rpm, here is a case where four out of five did. Now if you have, say, 20 that didn't and then four that did, your chances of sickness occurring under those circumstances are something of the order of 20%. In space the chances would be even greater and I am pointing out here that it's a greater risk than is commonly sensed.

**GUEDRY:** I'd like to make two additional comments. First, we have reason to believe that we can select people who will be disturbed very little by rotation, particularly at low rpm. Moreover, on our first 12-day 10 rpm run, we were able to retest our subjects in the Slow Rotation Room for several hours 3 weeks after the 12-day run had been completed. Nystagmus and sensations were both suppressed at this time but aside from this, I was impressed by the fact that these people moved around with complete freedom for several hours without nausea, whereas initially only one of the subjects was able to move around freely during the first 2 hours and the others reported severe nausea. So it appears that a beneficial habituation is retained in these situations. Second, the question of transfer of habituation is still with us, and I agree that we should be conservative in our estimates of feasible rates of rotation because the conditions in a rotating space station cannot be exactly reproduced on Earth.

**GUALTIEROTTI:** I would like to object to the statement that a zero gravity condition by itself could not be a reason of motion sickness. And I would like to object to the concept of motion sickness itself. We are being hypnotized by the fact that the Coriolis acceleration is one cause of the kind of syndrome that is called motion sickness. There are many other sources of the same type of symptoms. For instance, a falling motion can cause nausea and all the effects and the feelings of motion sickness. Visual stimulation without any motion can provoke a similar syndrome. I would say you couldn't call it motion

sickness. So we are in a bit of confusion when we talk about motion sickness, and I'd like to point out that the Coriolis acceleration is a very peculiar cause of motion sickness. Another point is that when we talk about vagus excitation being the real cause of nausea, vomiting and so forth, and not being related with vestibular symptoms, we forget the fact that the vestibule causes this kind of syndrome through the vagus. The mechanism by which the vestibular apparatus produces the motion sickness syndrome is just due to its paramount, very strong inference on the vagus. So I would say that we have to be a little more careful when we limit our point of view. I wouldn't say, for instance, that we can now exclude that weightlessness by itself is a cause of, let's not say motion sickness, but equivalent syndromes: nausea, unbalance, muscle uncoordination. Another point is this. Some of the circulatory syndromes, symptoms, might be reflexly produced by the vestibule itself. So, it's a rather more complex situation than just limiting ourselves to the concept of Coriolis movement which is only one particular cause of the feeling of motion sickness.

**MONEY:** Regarding your first point, I agree. There is a chance that zero g can cause motion sickness. This has not been ruled out yet, and in fact, I did suggest that observation of very susceptible animals in space would be a very worthwhile experiment. It would yield worthwhile information. Your second point—I will grant you that in certain conditioned individuals nausea and vomiting can be brought on in various ways. Certain visual distortions can cause nausea and vomiting, severe pain can cause nausea and vomiting, many drugs can cause nausea and vomiting. And the presence of these things can influence susceptibility to motion sickness. But, nevertheless, I feel that in the interest of simple language, motion sickness is simply a sickness caused by motion of the body. There is a sickness which is caused by motion of the body in the complete absence of all these other predisposing factors, and this is motion sickness. In Dr. Graybiel's labyrinthine defective subjects, there was a broad display of visual stimuli in aerobatics in aircraft, there were all sorts of accompanying influences, and none of these subjects had the least bit of motion sickness. So I think that there is a simple sickness caused by vestibular stimulation, by motion of the body, and that this is correctly called motion sickness.

**BILLINGHAM:** I do feel that one can't dismiss too easily the problem of prolonged weightlessness at this stage, particularly with reference to linear accelerations. All your points about rotating vehicles were extremely interesting. Would you attempt to predict what would be the reaction to a given linear acceleration on the otolith mechanism after a prolonged period of weightlessness, because this in itself might cause some disorientation which may be of particular practical importance in different situations in the space vehicle and outside it?

**MONEY:** I really couldn't give an informed opinion on that. If you like, I'd guess there wouldn't be very much change in the vestibular response simply because there doesn't appear to be much change after considerable bedrest at different orientations of the otoliths. There is very little else to go on.

**GUALTIEROTTI:** I would like to add one thing. You get disorientation and vertigo and nausea of vestibular origin when you don't have any vestibule, for instance, or in some injuries of the vestibule, like for instance in the Menière syndrome. Now this is not caused by motion. It is caused possibly by absence of stimulation or by the absence of a particular inflow or alteration of this inflow from the vestibule. So this might be a cause of syndromes equivalent to what is motion sickness without motion. This may be one of the mechanisms by which weightlessness itself can cause a syndrome of motion sickness.

**MONEY:** Yes, I will acknowledge that possibility, but nevertheless I wouldn't call sickness from vestibular injuries motion sickness—just in the interest of simplicity in language.

**FIELDS:** I would hate to leave the impression that what I was trying to say about vagal influences indicated that I didn't think that the vestibule acted through vagal influences to produce autonomic manifestations. All I was trying to say is that this is a final common pathway for visceral motor effects and that one does not have to have the impulse originate in the vestibule. In respect to Dr. Billingham's comment, I presume that the kind of situation that he envisions is one that might occur during extra-vehicular maneuver when an astronaut is giving himself a jet propulsion to get back to the spacecraft. Since he might have been exposed previously to a long period of zero g, this might produce a serious effect on his otolith organ.

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# Some Observations on the Stimulation of the Vestibular System of Man in a Rotating Environment

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Weightlessness remains an enigma of the space age. Can man tolerate it for long space flights, will he adapt to it, will he be degraded by it so as not to tolerate the high accelerations of earth return or just his weight when he has returned? The riddle has one basic solution with numerous approaches to it; the basic solution is, of course, the prevention of degradation.

The use of exercise, special devices such as cuffs, centrifuges, and rotating space vehicles are some of the numerous approaches being diligently pursued. The last of these, rotating vehicles to attain artificial gravity, creates the concern to which this paper is addressed, the undesirable effects of rotation, which can be disquieting if not intolerable. The fundamental physical phenomena that create these disquieting effects occur when the head or body is moved in a rotating environment resulting in cross-coupled angular accelerations which of course are sensed by the semicircular canals shown schematically in figure 1. The problems of man's tolerance to the cross-coupled acceleration have been studied extensively by Ashton Graybiel, et al. (refs. 1 and 2, for example) by the present authors (refs. 3, 4, and 5), and numerous others. When attaining artificial gravity by rotation, the astronauts will be oriented most of the time with their long-body axis perpen-

dicular to the axis of rotation of the vehicle and this is the orientation examined by the present authors. The experiments of references 3, 4, and 5 have indicated a tolerance to 10 rpm while turning the head from side to side (hereinafter called turning) and while nodding the head fore and aft (hereinafter called nodding) although in the latter case there is evidence that fewer people may be tolerant of this head motion. These are rather restricted conditions using only head motions, in view of the random motions and general freedom expected in flight and used in Graybiel's work. Studies are now being initiated at the Langley Research Center using a new rotating vehicle simulator to examine the effects of rotation allowing greater freedom and more randomness than were possible for the experiments of references 3 to 5. This paper reviews the results of references 3 to 5 and presents some initial studies using the new rotating vehicle simulator.

### SYMBOLS

- $a_{G\theta}$  cross-coupled nodding acceleration
- $a_{G\psi}$  cross-coupled turning acceleration
- $a_{G\phi}$  cross-coupled rolling acceleration
- $\omega_{G\theta} = \int a_{G\theta} dt$
- $\omega_{G\psi} = \int a_{G\psi} dt$
- $\omega_{G\phi} = \int a_{G\phi} dt$

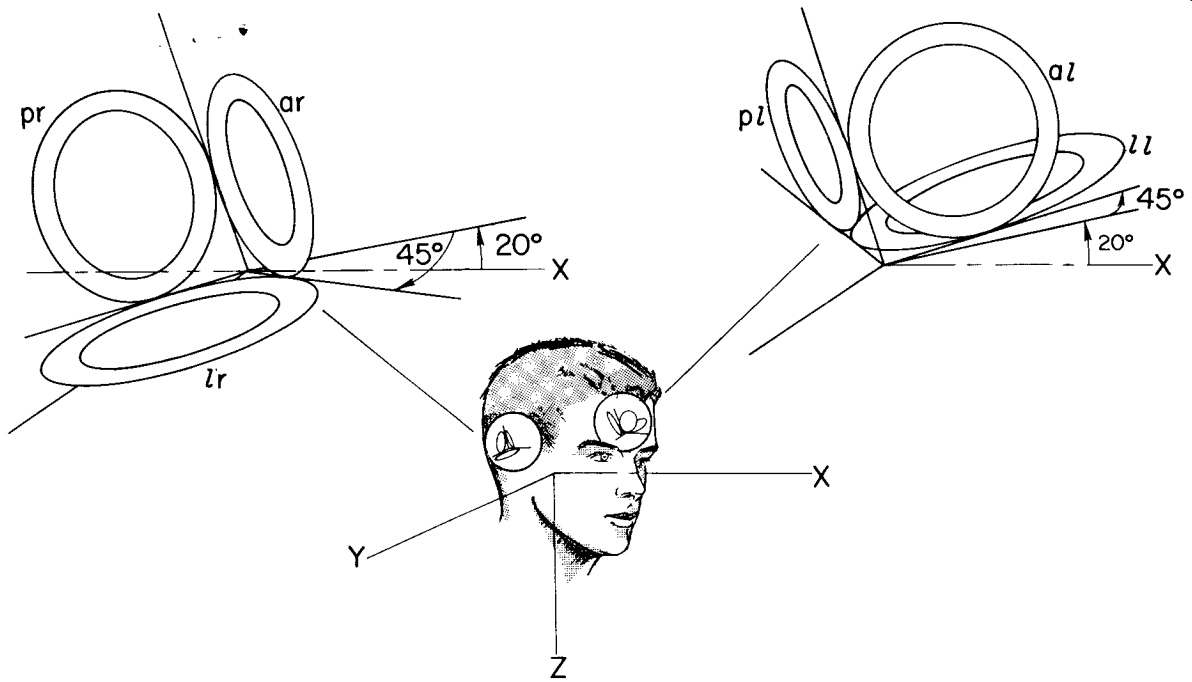


Figure 1.—Semicircular canal system.

- $\omega_{h\theta}$  nodding velocity — a fore and aft motion of the head at the neck or from the whole body
- $\omega_{h\psi}$  turning velocity — a motion about the neck or long-body axis
- $\omega_{h\phi}$  rolling velocity — a sideways motion of the head or from the body
- $\omega_V$  vehicle rotational velocity
- $\omega_{h_x}$  total angular velocity of head about rolling axis
- $\omega_{h_y}$  total angular velocity of head about nodding axis
- $\omega_{h_z}$  total angular velocity of head about turning axis
- $t$  time
- $\theta_G = \iint a_{G\theta} dt^2$
- $\psi_G = \iint a_{G\psi} dt^2$
- $\phi_G = \iint a_{G\phi} dt^2$

These are angular head motions and may be from motions at the neck and shoulders or from body bending, etc.

- $\theta_n$  nodding displacement
- $\psi_n$  turning displacement
- $\phi_n$  rolling displacement
- $\theta_e, \psi_e, \phi_e$  Euler angular displacement using the order of rotation shown in figure 2

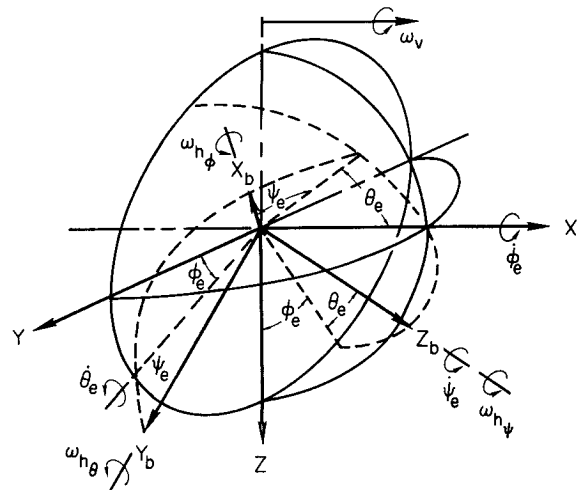


Figure 2.—Vectorial representation of head orientation and angular motions in a rotating space vehicle.



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

$\psi_{sc}$  rotation of semicircular canals from  $X_b Z_b$  plane

$X, Y, Z$  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

A dot over a symbol indicates its first derivative with respect to time.

ANGULAR MOTIONS EXPERIENCED IN ROTATING SPACECRAFT

As has been indicated, the actual angular motions, particularly the angular accelerations experienced by an individual when moving his head in a rotating system, are greatly different from those normally encountered in everyday life for the same head motion (ref. 5). Except after adaptation, if such is possible, these differences, in association with a visual environment limited to the interior of the rotating system, create the disquieting sensations that are of concern when considering artificial gravity. The stimulation of the semicircular canals is, of course, the direct result of the angular accelerations experienced. In a rotating environment these may be calculated as follows.

The total angular velocities experienced by the head are the sum of the various angular velocities acting

$$\left. \begin{aligned} \omega_{h_x} &= \omega_{h_\phi} + \omega_V \cos \theta_c \cos \psi_c \\ \omega_{h_y} &= \omega_{h_\theta} - \omega_V \cos \theta_c \sin \psi_c \\ \omega_{h_z} &= \omega_{h_\psi} + \omega_V \sin \theta_c \end{aligned} \right\} \quad (1)$$

where  $\omega_V$ , the rotational velocity of the vehicle, is assumed to be constant and alined with the inertial  $X$  axis.

The derivatives of equations (1) with time will then give the angular accelerations experienced by the head when moving, where  $\dot{\omega}_{h_x}$ ,  $\dot{\omega}_{h_y}$ , and  $\dot{\omega}_{h_z}$  are the angular accelera-

tions 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.

$$\left. \begin{aligned} \dot{\omega}_{h_x} &= \dot{\omega}_{h_\phi} - \omega_V (\sin \theta_c \cos \psi_c \dot{\theta}_c \\ &\quad + \cos \theta_c \sin \psi_c \dot{\psi}_c) \\ \dot{\omega}_{h_y} &= \dot{\omega}_{h_\theta} - \omega_V (\cos \theta_c \cos \psi_c \dot{\psi}_c \\ &\quad - \sin \theta_c \sin \psi_c \dot{\theta}_c) \\ \dot{\omega}_{h_z} &= \dot{\omega}_{h_\psi} + \omega_V \cos \theta_c \dot{\theta}_c \end{aligned} \right\} \quad (2)$$

Further

$$\left. \begin{aligned} \dot{\phi}_c &= (\omega_{h_\phi} \cos \psi_c \\ &\quad - \omega_{h_\theta} \sin \psi_c \frac{1}{\cos \theta_c}) \\ \dot{\theta}_c &= (\omega_{h_\phi} \sin \psi_c \\ &\quad + \omega_{h_\theta} \cos \psi_c) \\ \dot{\psi}_c &= \omega_{h_\psi} - \tan \theta_c (\omega_{h_\phi} \cos \psi_c \\ &\quad - \omega_{h_\theta} \sin \psi_c) \end{aligned} \right\} \quad (3)$$

A substitution of equations (3) in equations (2) then gives the general expression for the angular accelerations that will be experienced while moving the head in a rotating space vehicle having constant velocity. There results the following expressions:

$$\left. \begin{aligned} \dot{\omega}_{h_x} &= \dot{\omega}_{h_\phi} - \omega_V (\omega_{h_\theta} \sin \theta_c \\ &\quad + \omega_{h_\psi} \cos \theta_c \sin \psi_c) \\ \dot{\omega}_{h_y} &= \dot{\omega}_{h_\theta} - \omega_V (\omega_{h_\psi} \cos \theta_c \cos \psi_c \\ &\quad - \omega_{h_\phi} \sin \theta_c) \\ \dot{\omega}_{h_z} &= \dot{\omega}_{h_\psi} + \omega_V (\omega_{h_\theta} \cos \theta_c \cos \psi_c \\ &\quad + \omega_{h_\phi} \cos \theta_c \sin \psi_c) \end{aligned} \right\} \quad (4)$$

These equations (4) as noted describe 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.

When the vehicle is not rotating ( $\omega_V = 0$ ) we obtain

$$\left. \begin{aligned} \dot{\omega}_{h_x} &= \dot{\omega}_{h_\phi} \\ \dot{\omega}_{h_y} &= \dot{\omega}_{h_\theta} \\ \dot{\omega}_{h_z} &= \dot{\omega}_{h_\psi} \end{aligned} \right\} \quad (5)$$

which are the equations expressing our normal experiences (ignoring the subliminal effects of earth's rotation).

The differences between equations (4) and (5) are thus the angular accelerations caused by the vehicle rotation and are herein called the cross-coupled angular accelerations.

$$\left. \begin{aligned} a_{G\phi} &= \omega_V (\omega_{h\phi} \sin \theta_c + \omega_{h\psi} \cos \theta_c \cos \psi_c) \\ a_{G\theta} &= \omega_V (\omega_{h\phi} \sin \theta_c - \omega_{h\psi} \cos \theta_c \cos \psi_c) \\ a_{G\psi} &= \omega_V (\omega_{h\theta} \cos \theta_c \cos \psi_c + \omega_{h\phi} \cos \theta_c \sin \psi_c) \end{aligned} \right\} \quad (6)$$

These accelerations are those sensed by the semicircular canals and are the cause of the disquieting effects experienced in rotating devices particularly when the vision is restricted to the rotating frame of reference.

STIMULATION OF THE SEMICIRCULAR CANALS

Equations (4) are the accelerations experienced by the head. The stimulation of each semicircular canal may also be of in-

terest. The canal system is approximately orthogonal but is oriented in the head so as not to be aligned with the body axis system. This orientation varies with individuals so that the lateral canals are tilted back from 15° to 30° up in the front (ref. 6) and the anterior and posterior canals are turned somewhere from 35° to 65° about an axis tilted back and about normal to the plane of the lateral canals. The stimulation of the canals is expressed as follows:

$$\left. \begin{aligned} \omega_{sc_{lr}} &= \omega_{hx} \sin \theta_{sc} + \omega_{hz} \cos \theta_{sc} \\ \omega_{sc_{ll}} &= \omega_{hx} \sin \theta_{sc} + \omega_{hz} \cos \theta_{sc} \\ \omega_{sc_{ar}} &= \omega_{hy} \cos \psi_{sc} - \omega_{hx} \cos \theta_{sc} \sin \psi_{sc} + \omega_{hz} \sin \theta_{sc} \sin \psi_{sc} \\ \omega_{sc_{al}} &= -\omega_{hy} \cos \psi_{sc} - \omega_{hx} \cos \theta_{sc} \sin \psi_{sc} + \omega_{hz} \sin \theta_{sc} \sin \psi_{sc} \\ \omega_{sc_{pr}} &= \omega_{hx} \cos \theta_{sc} \cos \psi_{sc} + \omega_{hy} \sin \psi_{sc} - \omega_{hz} \sin \theta_{sc} \cos \psi_{sc} \\ \omega_{sc_{pl}} &= \omega_{hx} \cos \theta_{sc} \cos \psi_{sc} - \omega_{hy} \sin \psi_{sc} - \omega_{hz} \sin \theta_{sc} \cos \psi_{sc} \end{aligned} \right\} \quad (7)$$

Table I.—Canal Stimulation for Various Orientations of the Canals in the Head

[Assume  $\psi_c = \phi_c = \theta_c = 0$  with the head moving steadily through these values for consideration of this table]

Canal acceleration	$\theta_{sc} = 15^\circ$		$\theta_{sc} = 30^\circ$	
	$\psi_{sc}$		$\psi_{sc}$	
	35°	65°	35°	65°
Head nodding				
$\omega_{sc_{lr}}$	$0.9659\omega_V\omega_{h\theta}$	$0.9659\omega_V\omega_{h\theta}$	$0.8660\omega_V\omega_{h\theta}$	$0.8660\omega_V\omega_{h\theta}$
$\omega_{sc_{ar}}$	$0.1480\omega_V\omega_{h\theta}$	$0.2120\omega_V\omega_{h\theta}$	$0.2868\omega_V\omega_{h\theta}$	$0.4531\omega_V\omega_{h\theta}$
$\omega_{sc_{pr}}$	$-0.2345\omega_V\omega_{h\theta}$	$-0.4096\omega_V\omega_{h\theta}$	$0.1092\omega_V\omega_{h\theta}$	$0.2113\omega_V\omega_{h\theta}$
Head turning				
$\omega_{sc_{lr}}$	0	0	0	0
$\omega_{sc_{ar}}$	$-0.8192\omega_V\omega_{h\psi}$	$-0.4226\omega_V\omega_{h\psi}$	$-0.8192\omega_V\omega_{h\psi}$	$-0.4226\omega_V\omega_{h\psi}$
$\omega_{sc_{pr}}$	$-0.2588\omega_V\omega_{h\psi}$	$-0.5000\omega_V\omega_{h\psi}$	$-0.2588\omega_V\omega_{h\psi}$	$-0.5000\omega_V\omega_{h\psi}$

These equations are based on the arbitrary assumption that the angular acceleration vectors of the canals are positive outward from the head and downwards. A substitution of equations (4) into equations (7) leads to expressions for the total angular acceleration experienced by each semicircular canal. As just noted, the lateral canals can be tilted from  $15^\circ$  to  $30^\circ$  back so that  $\theta_{sc}$  will range through these values and similarly  $\psi_{sc}$  will range from  $35^\circ$  to  $65^\circ$  for various people. It is interesting to think that these differences between people may contribute to the different sensitivities to motions that exist among people. Table I shows the angular accelerations that would exist in a rotating space vehicle (or other rotating system) in the separate canals of the right ear. It is evident that in a nodding motion the stimulation of a given canal, particularly the anterior and posterior canals, can vary nearly 3 to 1 among various people. Further, in a turning motion of the head a 2 to 1 variation in canal stimulation is possible among various people. These differences possibly could cause some people to adapt less readily to rotation than others, possibly those with the greater stimulation, i.e., those with canals tilted  $30^\circ$  back and rotated  $65^\circ$ .

#### METHOD FOR SIMULATION OF ROTATING SPACE VEHICLES

In rotating space vehicles the astronauts normally will be oriented when standing or sitting with the long body axis perpendicular to the axis of rotation which is represented by the value of  $\theta_e = 0$ . The head is then moved about that point of reference. The significant force acting is the centrifugal force along the long axis of the body ( $Z_b$ ) and the otoliths are affected by this force only. Because of the presence of gravity on earth it is absolutely impossible to simulate on earth the situation just described. Thus simulation is a compromise of the sundry factors acting. The questions deriving from these factors are: Are the disquieting or even sickening effects of rotation primarily derived from the stimulation of the semicircular canals or is the stimulation of the

otoliths also significant and is it more appropriate to stimulate the semicircular canals properly or to stimulate the otoliths properly or compromise both stimulations? It is generally conceded that the semicircular canals sense angular accelerations and the otoliths sense linear accelerations and that the systems are essentially independent (ref. 7). Recent results from Pensacola (ref. 8) indicate that the semicircular canals are not responsive to linear accelerations. One would thus conclude that the presence of the gravitational vector in a simulation will not affect the stimulation of the semicircular canals and therefore the effect of this stimulation on the subjects. The question is whether the orientation of the total linear acceleration vector in itself has a significant effect on the subject such as the cause or prevention of adaptation to rotation or the instigation or prevention of sickness. We are accustomed to two extremes of orientation of the long body axis to the gravitational vector, either parallel or perpendicular. No difficulties derive from this situation. The subjects of references 3, 4, and 5 were oriented perpendicular to the gravitational vector with the total linear acceleration vector oriented from perpendicular to less than  $45^\circ$  to the long body axis depending on the rate of simulator rotation. These subjects adapted to this orientation without concern to this condition. The subjects of reference 9 when using the Langley lunar walking device are oriented about  $81^\circ$  to the gravitational vector and readily adapt to the situation, recognizing the tilted walking board as the ground. For a short time after dismounting the ground appears tilted to them. Thus the orientation of the linear acceleration vector at some condition other than parallel to long body axis does not seem to have any disquieting effects and seems to be readily adapted to. From this discussion it has been concluded that a correct stimulation of the semicircular canals, that is, with the long body axis perpendicular to the axis of rotation, is the proper method of simulation for rotating vehicles regardless of the orientation of the

Table II.—*Angular Accelerations for Various Orientations of Subjects in a Rotating Space Vehicle*(a)  $\psi_e = \phi_e = 0^\circ$ 

$\theta_e$ (a)	$0^\circ$	$-45^\circ$	$-90^\circ$
$\omega_{h_x} = \omega_h \phi - \omega_v$	0	$0.7071 \omega_{h\theta}$	$-\omega_{h\theta}$
$\omega_{h_y} = \omega_h \theta - \omega_v$	$\omega_{h\psi}$	$0.7071 (\omega_{h\phi} - \omega_{h\psi})$	$\omega_{h\phi}$
$\omega_{h_z} = \omega_h \psi + \omega_v$	$\omega_{h\theta}$	$0.7071 \omega_{h\theta}$	0

(b)  $\psi_e = 90^\circ; \phi_e = 0^\circ$ 

$\theta_e$ (a)	$0^\circ$	$-45^\circ$	$-90^\circ$
$\omega_{h_x} = \omega_h \phi - \omega_v$	$\omega_{h\psi}$	$0.7071 (\omega_{h\psi} - \omega_{h\theta})$	$\omega_{h\theta}$
$\omega_{h_y} = \omega_h \theta - \omega_v$	0	$0.7071 \omega_{h\phi}$	$\omega_{h\phi}$
$\omega_{h_z} = \omega_h \psi + \omega_v$	$\omega_{h\phi}$	$0.7071 \omega_{h\phi}$	0

<sup>a</sup> The total angular accelerations are obtained by multiplying  $\omega_v$  by the specific column of concern and adding the result to  $\omega_{h\phi}$ ,  $\omega_{h\theta}$ , and  $\omega_{h\psi}$  as noted.

linear acceleration vector. In order to more clearly identify the effects of the orientation of the total linear acceleration vector while maintaining the orientation of the long body axis perpendicular to the axis of rotation a new simulator, to be described subsequently, has been built at Langley. On this simulator experiments can be run at two different radii so that a rather wide variation of the inclination of the total linear acceleration vector to the long body axis can be examined.

Table II expresses the angular accelerations experienced in a rotating reference frame for various orientations of the long body axis ( $Z_b$ ) from the vehicle rotational vector. The value of  $\theta_e = 0$  (the long body axis perpendicular to the rotational axis) is the actual orientation that will exist in rotating space vehicles and is the orientation used in references 3, 4, and 5 and for the results presented herein. The value of  $\theta_e = 45^\circ$  is approximately the value used in reference 10. The value of  $\theta_e = 90^\circ$  (the long body axis parallel to the rotational axis) is the value used in references 1 and 2. The results of table II indicate greatly different cross-

coupled angular accelerations for the three situations. An adaptation to one situation may not indicate adaptation to another because of the grossly different stimulations involved. The use of  $\theta_e = 0$  would seem most appropriate for simulation.

The Coriolis force acting in rotating vehicles has also received considerable concern. However in a rotating space station the floor normally used lies in a plane always parallel to the axis of rotation. When moving on this floor the astronaut can move along the floor in a direction parallel to the axis of rotation for which the Coriolis force is zero or he can move along the floor perpendicular to the axis of rotation for which the Coriolis force causes the astronaut to become effectively heavier or lighter as he increases or decreases the centrifugal force. The more dramatic effects of Coriolis force occur when moving in a radial direction which in actual space flights would occur infrequently. When moving on the floor so as to increase or decrease the centrifugal force there is the possibility of degrading performance as one approaches weightlessness when moving

counter to the direction of rotation. It is not felt, however, that any disquieting effects would occur from this situation.

On the basis of these considerations the work at the Langley Research Center has been performed with  $\theta_c = 0$  and the results presented herein are based totally on this condition.

#### TEST EQUIPMENT AND TECHNIQUE

In pursuit of a clearer understanding of the problems outlined so far the Langley Research Center has used a simple rotating-vehicle simulator shown in figure 3. This device was used for the results of references 3 to 5 which are used in part in this paper. In this simulator the subjects lay on their backs enclosed in a small cabin with their feet 15 feet from the center of rotation. The centrifugal force was taken on the soles of the feet as it would in a rotating space vehicle.

The internal features of the rotating simulator are shown in figure 4. The subject's task was to observe either the lights on his left or over his head which were controlled by the experimenter located external to the simulator. The color of the lights was varied by the experimenter, and the subject, upon observing a light of certain color, was required to turn his head to the right or nod his head forward depending on the light observed and place a probe in an appropriate

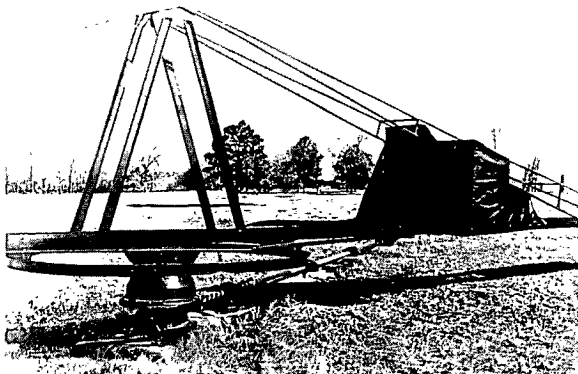


Figure 3.—External view of rotating vehicle simulator.

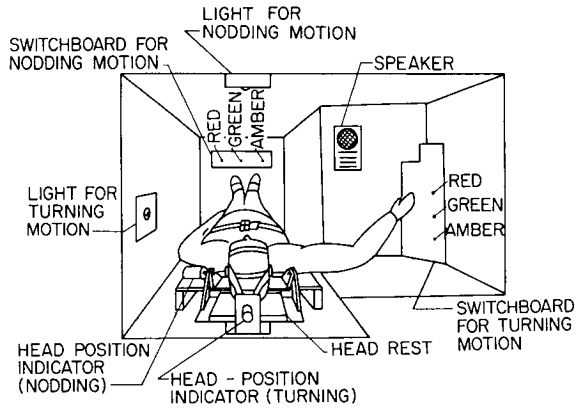


Figure 4.—Internal features of simulator.

hole to extinguish the light. The head position and head rate were measured by head-position indicators which were attached to a harness on the subject's head and to the moving head rest. The moving head rest supported the head by negator springs and allowed nodding motions without the need to hold up the head in the supine position. The time from light activation to light cutoff was also measured.

Subsequent to the performance of the experiments for reference 5, a new rotating

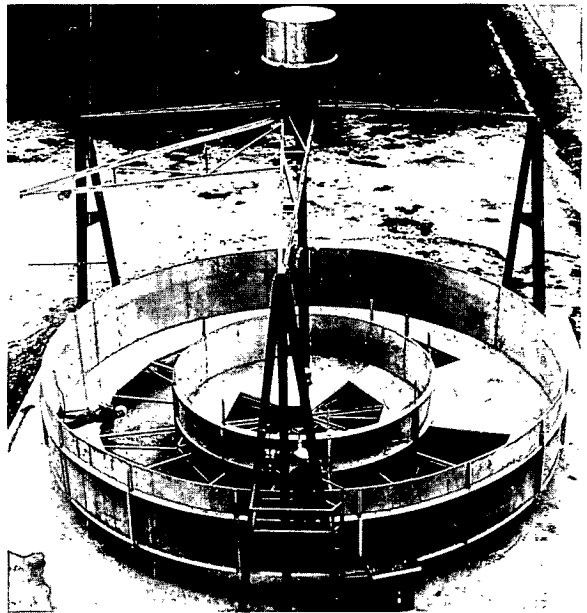


Figure 5.—The new Langley rotating vehicle simulator.

vehicle simulator was constructed at the Langley Research Center. This device is shown in figure 5. There are two concentric rotating cylindrical walls one with 20-foot diameter and one with 40-foot diameter. These walls are used to emulate the floor of a rotating vehicle upon which persons, with the use of a suspension system like that of reference 9, can walk and otherwise manipulate. Tests of this nature have not yet begun; however, two small cabins similar to that shown in figure 4, were installed, one at each radius. The same systems and measuring devices shown in figure 4 and described above are installed in these cabins. Thus some indications of the effects of radius, if any, on man's responses to rotating systems will be obtained. Some initial results with this facility are presented in this paper.

Results summarized herein from references 3 to 5 were made with nodding motions alone, with turning motions alone, and with combination nodding and turning motions.

Twenty-nine subjects were used in turning experiments, nine subjects were used in the nodding experiments, and ten were used in the combined motions. The subjects who participated in the nodding experiments did not participate in any of the previous experiments. However, three subjects who participated in the experiments of references 3 and 4 also participated in the combined motion experiment (ref. 5). The turning experiment lasted 1 hour and rates of rotation of 0, 7, 10, 14, and 17 rpm were used. The nodding experiment also lasted 1 hour and rates of rotation of 0, 2, 4, 6, 8, and 10 rpm were used. The combined nodding and turning experiments were run at 10 rpm and lasted 3 hours. Figure 6 shows typical initial segments of the time history of light activation for both the nodding-alone experiments and the combined nodding and turning experiments. Each line indicates an activation of the light. For the turning and nodding experiments alone the light was activated

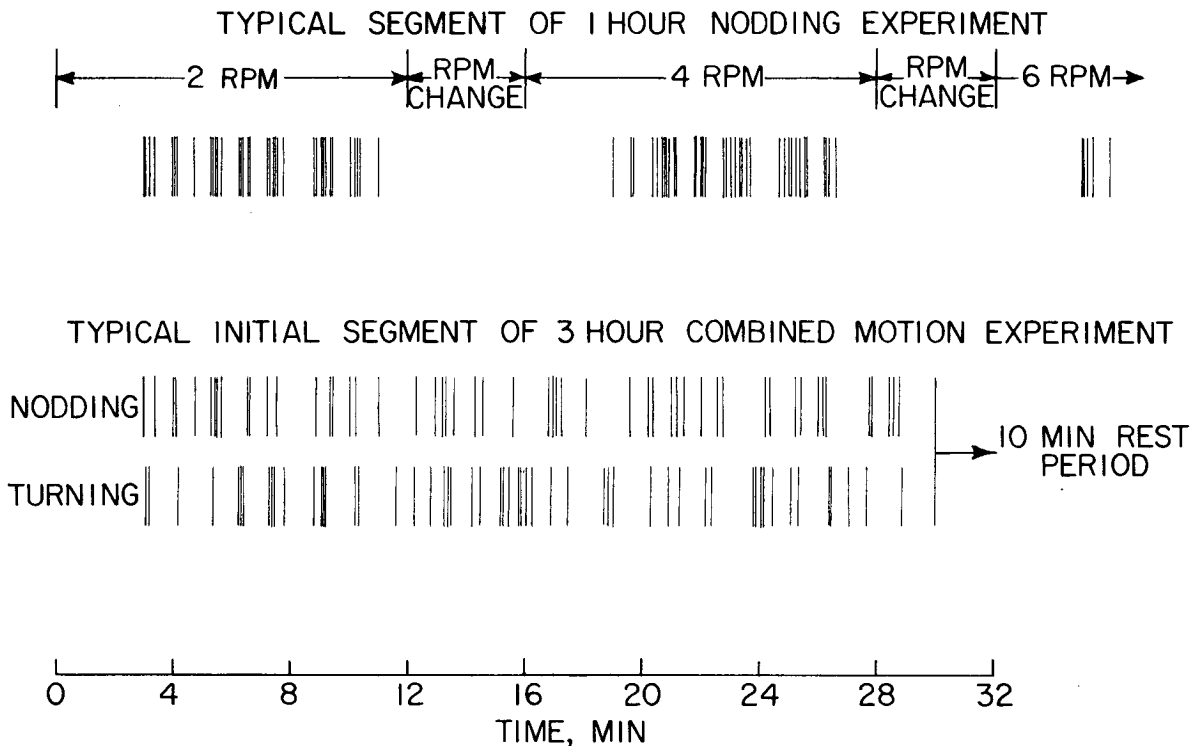
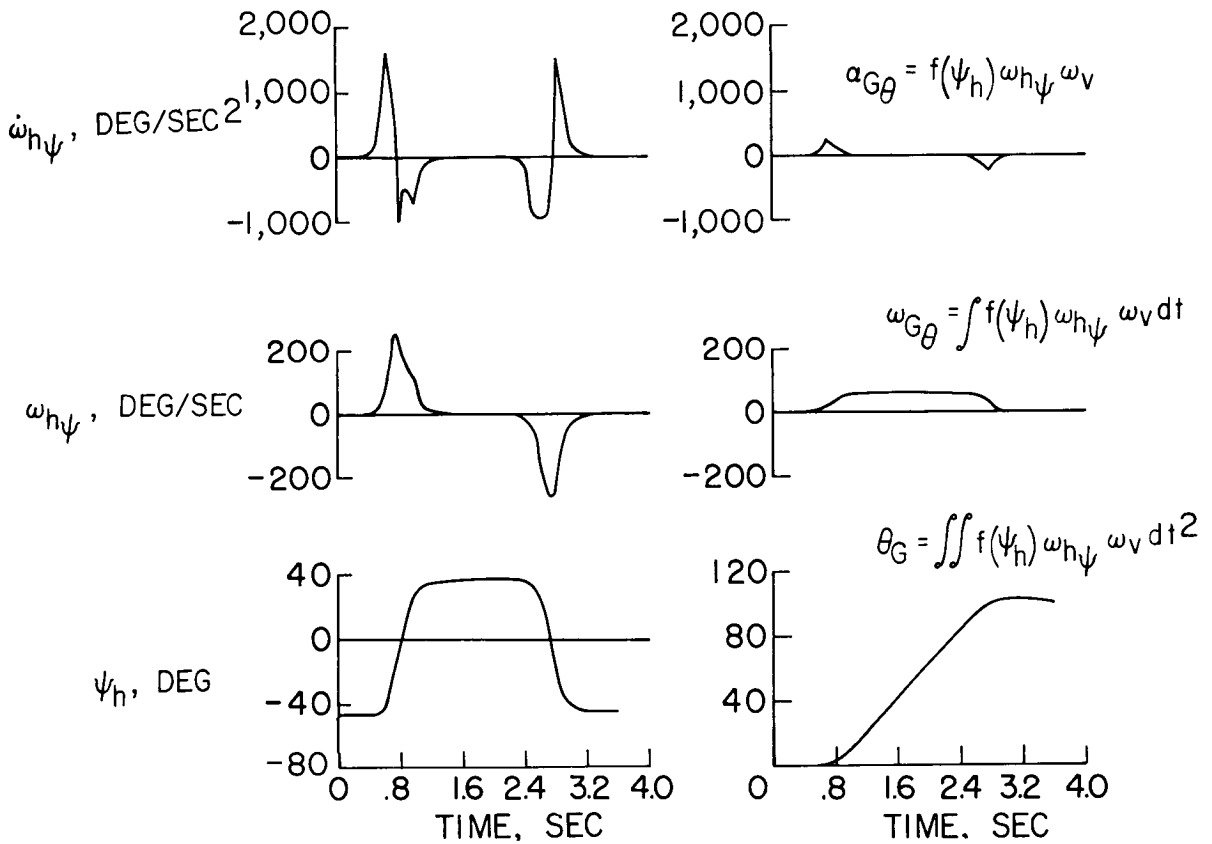


Figure 6.—Time histories of light activations. Each line indicates activation of light.

about 32 times for each vehicle rotational rate. The light was activated 160 times during the 1-hour experiment. During the combined nodding and turning motions 500 light activations were made. These were randomly distributed between the nodding and turning motions as shown in figure 6. Some motion pictures of eye motions were made to determine qualitatively the motion of the eyes under the conditions of the experiments. Experiments on the new rotating-vehicle simulator presented herein were performed with a nodding motion alone with eight subjects. The experiments were run at 9, 12, 14, and 16 rpm and lasted 1 hour. Each subject performed the experiment at the 10-foot radius and at the 20-foot radius of the simulator. The same light activation as discussed previously was also used for these experiments.

RESULTS AND DISCUSSION

In order to more clearly identify the conditions of stimulation that exist when turning one's head in a rotating vehicle, figures 7 and 8 have been taken from reference 5. Figure 7 shows a typical turning head motion on the left of the figure and the resultant effects of cross-coupling when this typical turning motion is performed at 10 rpm on the right. This motion has conditions that represent the average in turning velocity for more than 20 subjects. There are turning accelerations of 1600 deg/sec<sup>2</sup>, turning velocities of 220 deg/sec, and a head displacement of ±45°. The cross-coupled angular acceleration  $a_{G\theta}$  is about 250 deg/sec<sup>2</sup> and unlike the natural turning motion of the head on the left the initial acceleration is not followed immediately by a counter



(a) Real turning head motion.

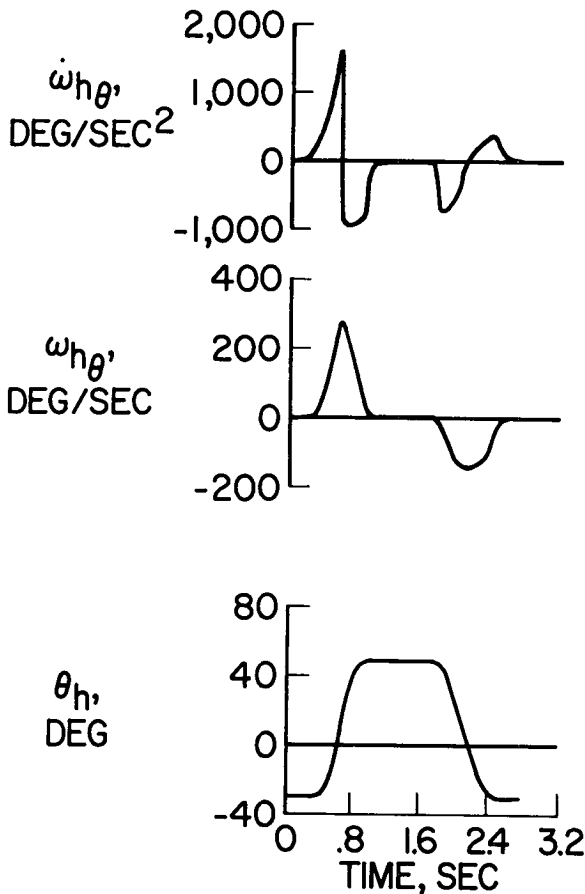
(b) Apparent nodding motion.

Figure 7.—Typical head turning motion and resulting apparent nodding motion. Simulator rate:  $\omega_v = 10$  rpm.

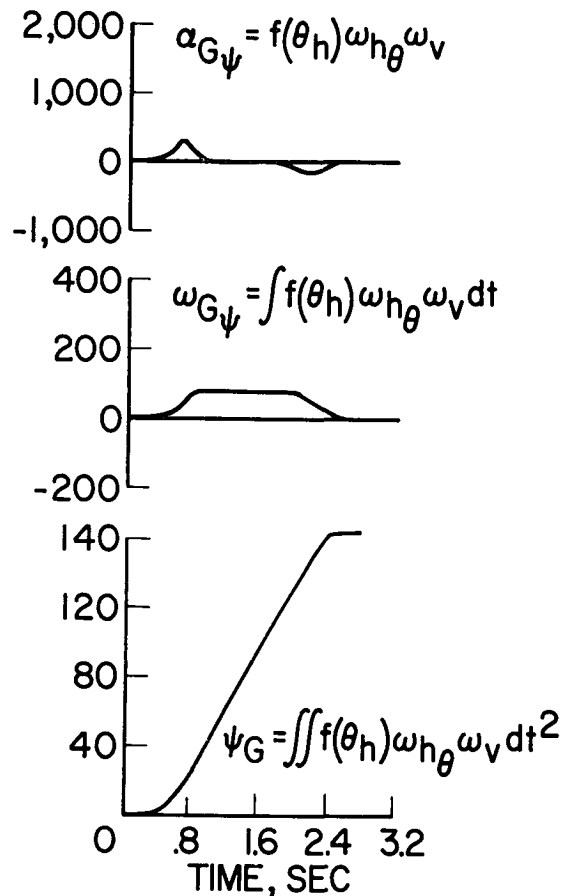
acceleration in the opposite direction. The counter acceleration occurs only when the head is moved back. For these kinds of motions vertical nystagmus and fuzzy vision exist following any single motion. The curves of  $\omega_{G\theta}$  and  $\theta_G$  which are integrations of the cross-coupled angular acceleration ( $a_{G\theta}$ ) are also shown in figure 7. These are termed apparent nodding motions because an illusion of body pitching occurs.

In figure 8 is shown a similar set of data for a typical nodding motion. The results are quite similar to those for the turning motion in figure 7. Again, in the natural head motion on the left, an acceleration in one direction is always followed of course by a counter acceleration required to stop the head once it is started. In a contrary sense, however, the cross-coupled acceleration

( $a_{G\psi}$ ) shown on the right as in figure 7 for  $a_{G\theta}$  is not followed by an acceleration in the opposite sense until the head is moved back toward its original position. In the case of a nodding head motion the cross-coupling causes an apparent turning motion the illusion of which is quite evident until adaptation occurs. The sensation of motion like that experienced on any turntable device would persist until the cupula of the semicircular canals has returned to its neutral position. As was noted previously, a vertical nystagmus and fuzzy vision existed for head turning as shown in figure 7. For head nodding as shown in figure 8, however, the subjects reported no fuzziness of vision and had no apparent lateral nystagmus. As reported in reference 11 the response characteristics of the semicircular canals are somewhat dif-



(a) Real nodding head motion.



(b) Apparent turning motion.

Figure 8.—Typical head nodding motion and resulting apparent turning motion. Simulator rate:  $\omega_v = 10$  rpm.



ferent for different kinds of motion, being quite similar for stimulations in nodding and rolling the head, both of which however have time constants of the order of twice the size of that for head turning. It is possible that these differences have some influence on the effects just reported as a result of the kinds of motions shown in figures 7 and 8.

The results of most of the experiments performed at the Langley Research Center have been reported previously in references 3 to 5. A summary of these results is presented in figures 9 and 10. These figures are plots of head rate vs. the vehicle rate of rotation. The curves shown are hyperbolas along which the product of head rate ( $\omega_{h\theta}$  or  $\omega_{h\psi}$ ) times the vehicle rate of rotation ( $\omega_v$ ) is constant. These curves are, therefore, loci of constant cross-coupled angular acceleration. The sense of these plots is that if the significant element in the disquieting effects of rotation lies principally in the cross-

coupled angular acceleration, then the problem is not simply what the vehicle rotation is but what is the product of the vehicle rotation rate and the head motion rate. Thus, if tolerance to rotation is governed by

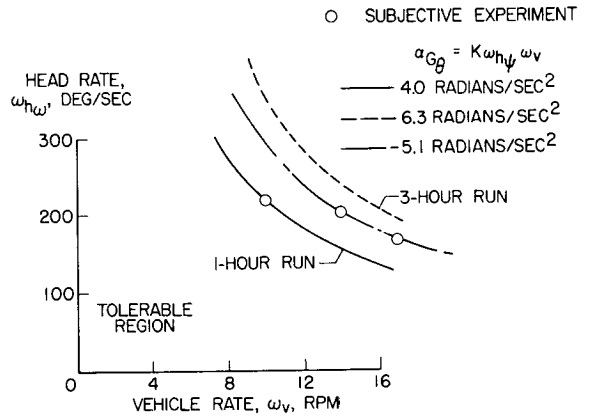


Figure 9.—Tolerance to cross-coupled angular acceleration while turning head.

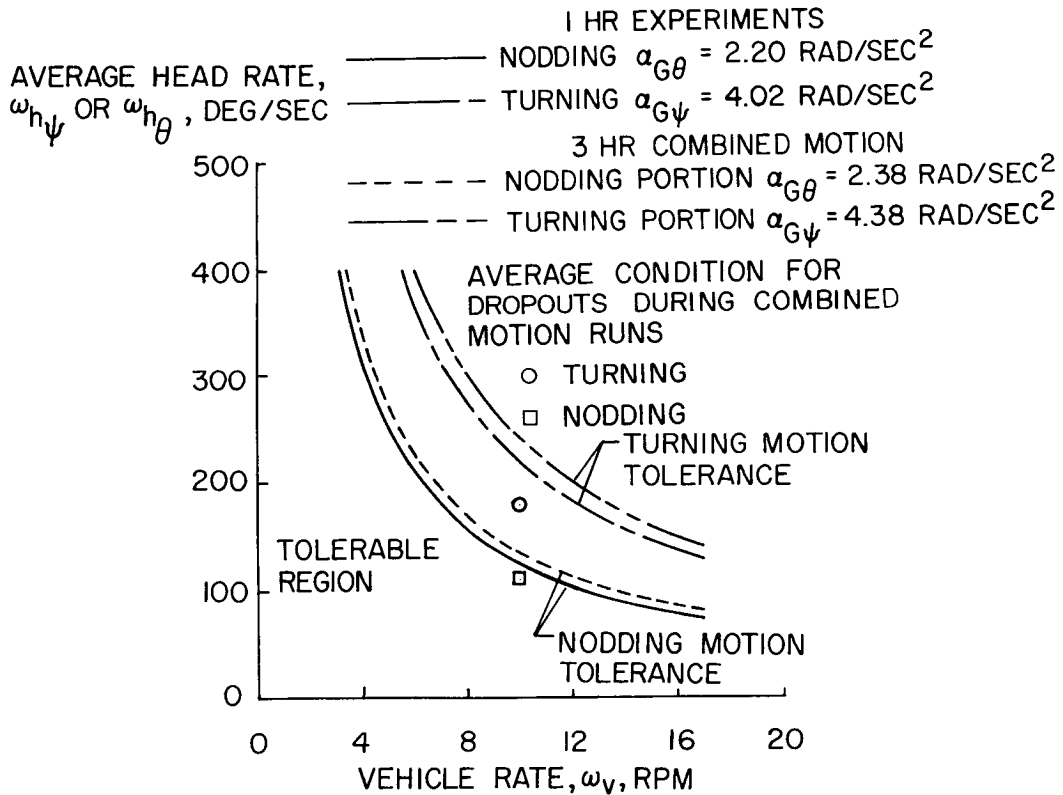


Figure 10.—Tolerance to cross-coupled angular accelerations while turning and nodding head.

tolerance to cross-coupled angular acceleration, constant values of this acceleration form boundaries of tolerance to rotation. This would imply that on a slowly rotating vehicle the subject could use and tolerate head motions with larger rates than he could on a rapidly rotating vehicle.

The results shown in figure 9 are for head turning motions. The solid line results from an hour long experiment wherein the rotation of the vehicle was increased in steps from 0 to 17 rpm. All subjects tolerated the motion to 10 rpm which is the data point upon which the solid curve is based. The cross-coupled angular acceleration for this condition is  $4.0 \text{ rad/sec}^2$ , and is an average for all subjects. The values of cross-coupled angular acceleration used herein are based on the maximum values of head rates of rotation which referenced to figures 7 and 8 are the peaks in the variation of head rate with time. Although there is a wide range of individual experience from this average value all subjects were tolerant of 10 rpm and performed well. The other data points shown beyond this boundary and indicated by the dash-dot curve are average values for those subjects that tolerated rates of rotation greater than 10 rpm. It is interesting and significant to note that as the vehicle rate of rotation was increased to 14 and 17 rpm these subjects decreased their rate of head motion in such a manner as to maintain a cross-coupled angular acceleration of  $5.1 \text{ rad/sec}^2$ . This is indicative of the fact that subjects adjust to a given situation to maintain tolerable conditions. The dotted curve in figure 9 is based on the maximum head rates used during a 3-hour run at a constant vehicle rate of rotation of 10 rpm. As indicated in reference 4, the subjects of this experiment continued to increase their head rates as the test progressed and values used on this plot are those used at the end of the 3 hours. The differences between the solid and dotted curves are an indication of the adaptation that occurred in 3 hours. The fuzziness of vision and the general apprehension that existed in the short experiments disappeared

from the subjects within an hour during these 3-hour runs. The tolerable cross-coupled angular acceleration for the case is more than 50 percent greater than for the shorter experiments.

The results for nodding head motions and for combined nodding and turning head motions from reference 5 are summarized in figure 10. These plots are derived in the same manner as figure 9. The solid curve is for a 1-hour experiment wherein the vehicle rate of rotation was increased stepwise from 0 to 10 rpm. At this last rate of rotation there was one subject who dropped out and four others who became uncomfortable. The test was therefore not extended to higher values of rotational rate. The cross-coupled angular acceleration for this case is based on the peak nodding head motions for those subjects that tolerated 10 rpm and is about  $2.1 \text{ rad/sec}^2$ . This cross-coupled angular acceleration is only one-half of that generally established for turning head motions (fig. 9) just discussed. As noted before, the induced motion sensation that occurs when nodding the head is a turning motion for which the lateral canals are primarily affected. As previously noted, the time constant of the semicircular canals in response to head turning according to reference 11 is about twice as large as the time constant for nodding and rolling motions. It is worthy of note then that cross-coupled angular accelerations which stimulate a turning sensation cause about one-half the tolerance level in a sense of motion where the time constant is twice as large as for other motions. The implication that there is some relation of the acceleration and the time period of cupula motion after an applied acceleration is apparent. It should be pointed out, however, that during the tests discussed herein there were few times when the head was still for 10 to 15 seconds, the time constant for lateral stimulation. An integration of the cross-coupled angular acceleration for the length of the time constant therefore may not be related to the tolerance boundaries shown regardless of the implications.

The other data shown in figure 10 are for an experiment where both nodding and turning motions were used in a combined and random fashion. The dotted curve and the dash-double dotted curve are derived from these data. This experiment was run for 3 hours at a constant vehicular angular velocity of 10 rpm. The two to one ratio in turning to pitching motions is evident. The increase in tolerance for the 3-hour experiment over the 1-hour experiment is indicated by the shift upwards of the curves for nodding alone as well as for turning from the dash-dot curve which is replotted from figure 9. Adaptation to the combined motion appears slower than that to the individual motion of turning where a 50-percent increase occurred (fig. 9). This compares with about 5-percent increase in tolerance shown in figure 10 even for the turning motion portion of the combined motions. Whether this is a result of increased work load where the subjects are concerned with two tasks or whether adaptation occurs more slowly when combined motions are used is a question requiring additional study.

The program at the Langley Research Center based on the results just summarized was continued on the new rotating space vehicle simulator previously discussed and shown in figure 5. The first experiments as previously noted were performed at the two radii available, 10 and 20 feet. These experiments lasted for 1 hour at each radius and the results are shown in figures 11, 12, and 13. These data, as were those of references 3 to 5, are numerical averages of the data from all the subjects participating in the experiment at each radius of rotation. As in reference 5 the subject was requested to look over his head at lights of three colors; when he saw a light he nodded his head forward and placed a probe in the appropriate hole to extinguish the light. Each subject's head position, rate of motion, and response time (the time from when the light was turned on until extinguished) were recorded. As noted, eight subjects were used, one dropped out of the experiment at the inner radius at 14 rpm

and did not perform the experiment at the outer radius.

The average amplitude of head nodding motion at each vehicle rate of rotation is shown by the circle and square symbols in figure 11, for the 10- and 20-foot radii, respectively. The periods of time spent at each rotational rate are designated by the stepped lines. The amplitudes of head motions are between  $50^\circ$  and  $60^\circ$  and nearly the same for the two radii and for all rates of rotation. There are no consistent variations with either radius or speed. It is significant to note that these amplitudes are appreciably less than those reported in reference 5 although the experimental setup was essentially the same. The essential difference was that the current subjects did not tilt their heads as far back as those of reference 5. It should be pointed out that none of the subjects used in the present tests participated in the earlier simple nodding experiments. However, five of the current subjects did participate in the combined head-motion tests of reference 5. There is no evidence to indicate why less amplitude was used although some subjects from the reference 5 experiments noted that putting the head far back was quite disquieting. Five of the nine subjects of reference 5 became disturbed at 10 rpm whereas the only subject who dropped out of the current experiment dropped out at 14 rpm. One subject was slightly dizzy and another somewhat disturbed although

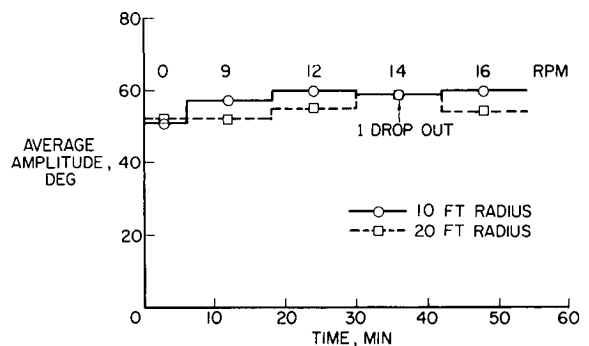


Figure 11.—Amplitude of head nodding at various rates of simulator rotation and two radii.

not ill following the experiment at 16 rpm. One possible explanation for not tilting the head far back is that the cross-coupled turning motion induced by nodding (eqs. (6)) changes direction when the head is nodded back of its normal position and then moved forward. Such a change in direction of the induced stimulation may be more uncomfortable than the mere existence of the stimulation in one direction. The latter was the condition experienced in the current tests whereas the former was experienced in the test of reference 5. It appears that some further examination of this specific problem is warranted.

The average rate of head nodding motion is shown in figure 12. The results are shown by the symbols and the stepped lines indicate the time at each rotational rate. The rates varied from 140 to 165 deg/sec with no apparently significant effects of radius or vehicle rate of rotation. These rates are somewhat larger (about 20 deg/sec) than the results of reference 5. There was no apparent reduction of head rate with vehicle rotation rate as was noted in reference 5 even though the rates of rotation for the current experiments were much greater than for those of reference 5. It is expected only that this result is related to the factors previously discussed relative to head amplitude in that a less disquieting situation may have prompted greater head rates.

The response times are shown in figure 13. There is no marked change in this time with either vehicle rate or radius. The time ranges

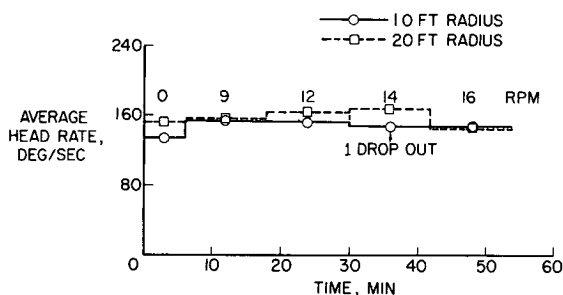


Figure 12.—Rates of head nodding at various rates of simulator rotation and two radii.

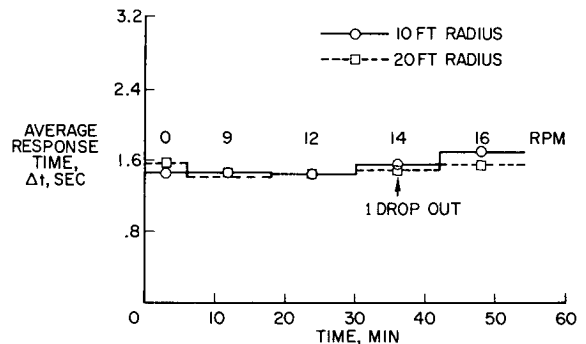


Figure 13.—Response time while nodding at various rates of simulator rotation at two radii.

from about 1.4 seconds to 1.7 seconds becoming somewhat larger as the vehicle rate of rotation increased. These response times are nearly the same as those obtained for reference 5 for comparable vehicle rates.

As noted previously, one subject dropped out of the experiment at 14 rpm. He was nauseous. Another subject felt uncomfortable but not sufficiently so to stop the experiment. Another subject felt slightly dizzy following the experiment for a very few minutes. All subjects were able to assume normal duties immediately following the experiment. This is contrary to the experiment of reference 5 where two subjects were affected for several hours after the experiment. Whether the current subjects were less prone to sickness than those of reference 5 or whether the different amplitudes of head motion used in the two experiments created the difference in the results is not evident. It would appear to be a point of further consideration. Preventing backward movement of the head by a collar of sorts is worthy of consideration if the latter cause of the difference proves true.

Tolerance boundaries similar to those in figures 9 and 10 also were prepared for the current results. These are shown in figure 14. In this figure the average rates of head rotation are plotted for the radii of 10 and 20 feet and, as noted before, show no decrease with vehicle rate of rotation. In that tolerance boundaries have been prepared before on the basis of 10 rpm, such boundaries are

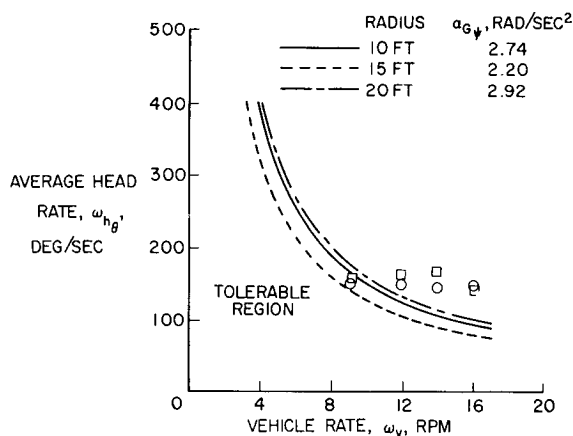


Figure 14.—Tolerance to cross-coupled angular accelerations while nodding head at different radii.

shown in this figure including one for the 15-foot radius from figure 10. There is only a slight difference between the 10- and 20-foot radii, 2.74 rad/sec and 2.92 rad/sec, respectively. Both of these are somewhat larger than the value of 2.20 rad/sec for the 15-foot radius of reference 5. The cause, of course, relates to the previous discussion of head amplitude. Actually at 16 rpm a cross-coupled angular acceleration of 4.52 rad/sec was tolerated in the current tests. These results indicate no significant effect of radius of rotation indicating that performance and tolerance are essentially independent of radius. This conclusion would imply that the semicircular canals alone influence performance and tolerance for the conditions studied at least. Further, it implies the

otoliths do not affect the results for the range of conditions studied, as the centrifugal force felt on the soles of the feet of the subjects ranged from slightly over  $\frac{1}{4}$  g to  $1\frac{3}{4}$  g. The inclination of the total linear-acceleration vector ranges therefore from  $74^\circ$  to  $30^\circ$  from the long body axis, respectively, for these conditions.

#### CONCLUDING REMARKS

Deliberation of the results of this paper indicates that the effects of rotation, as it would be experienced in a rotating space vehicle, are best evaluated by orienting subjects in earth-bound simulators with their long body axis perpendicular to the axis of rotation of the vehicle. The studies indicate a considerable variation in the stimulation of the various semicircular canals possible among people with normal vestibular functions. The data reviewed generally indicate a tolerance to 10 rpm for most subjects. The data presented herein show no essential effect of radius of rotation on the subjects' performance and tolerance to rotation. Finally these data indicate that a movement of the head back from the normal upright position when oriented with the long body axis perpendicular to the axis of rotation may cause some subjects to be less tolerant of nodding motions than if the head was not nodded back. These results are for a limited number of subjects and for only relatively short periods of exposure and should be confirmed by other subjects and experiments.

#### REFERENCES

1. GUEDRY, F. E.; COLLINS, W. E.; AND GRAYBIEL, A.: Vestibular Habituation During Repetitive Complex Simulation: A Study of Transfer Effects. Joint report with NASA. (NASA CR-53123: BuMed-93) January 15, 1964.
2. GUEDRY, F. E.; KENNEDY, R. S.; HARRIS, C. S.; AND GRAYBIEL, A.: Human Performance During Two Weeks in a Room Rotating at Three RPM. BuMed Report 74, NASA CR-55622, August 28, 1964.
3. STONE, RALPH W., JR.; AND LETKO, WILLIAM: The Effects of Angular Motion of Rotating Space Vehicles on the Ability of an Astronaut to Perform Simple Tasks. Institute of Environmental Sciences 1962 Annual Technical Meeting Proceedings, April 11-13, 1962, Chicago, Ill.
4. LANGLEY RESEARCH CENTER STAFF: A Report on the Research and Technological Problems of Manned Rotating Spacecraft. NASA TN D-1504, August 1962, pp. 85-90.
5. STONE, RALPH W., JR.; AND LETKO, WILLIAM: Tolerance to Vehicle Rotation of Subjects Using Turning and Nodding Motion of the Head While Performing Simple Tasks. Presented at the First Annual Meeting of the AIAA, June 28-July 2, 1964, Washington, D.C.

6. SPIEGEL, E. A.; AND SOMMER, I.: Vestibular Mechanisms. Medical Physics., vol. 1, pp. 1638-1652.
7. GUEDRY, F. E.; AND GRAYBIEL, A.: Rotation Devices, Other Than Centrifuges and Motion Simulators. National Academy of Sciences—National Research Council, Publication 902.
8. MCLEOD, M. E.; AND CORREIA, M. J.: Use of Caloric Test in Evaluating the Effects of Gravity on Cupula Displacement. NSAM-889, Naval School of Aviation Medicine, Pensacola, Fla., April 2, 1964.
9. HEWES, DONALD E.; AND SPADY, AMOS A., JR.: Evaluation of a Gravity Simulation Technique for Studies of Man's Self-Locomotion in the Lunar Environment. NASA TN D-2176, 1964.
10. NEWSOM, BERNARD D.: The Significance of the Resultant Vector From Gravity and Centrifugal Accelerations in Defining Human Tolerance to Rotation. AIAA Paper No. 64-219, First AIAA Annual Meeting, June 29-July 2, 1964, Washington, D.C.
11. JONES, G. MELVILL; BARRY, W.; AND KOWALSKY, N.: Dynamics of the Semicircular Canals Compared in Yaw, Pitch, and Roll. Aerospace Medicine, vol. 35, October 1964, pp. 984-989.

#### DISCUSSION

CAPPEL: The approach reported here is going to be required as a first step. I may say that we hope to be able to measure head motions of a freely walking subject some time in the future.

STONE: We have plans and ideas of how we could do this on the present system. We hope to get all the positional angles and be able then to find the position of the head under any given reference system.

CAPPEL: Yes, we have to determine that by other means with respect to the centrifuge. But I feel that we would very much like to look at your records and analyze them in greater detail with respect to the actual accelerations of flight to individual organs which I still feel is going to be the next step.

STONE: We would like very much to have you do so. The equations in this paper express the actual acceleration in any given semicircular canal. If you can sum them up in some fashion, you could perhaps determine which canals are predominantly responsible for the sensations perceived. But, as Dr. Jones says, let's consider it as operating as a whole unit and experiencing angular accelerations expressed by equations.

GUEDRY: I gather that what you're measuring is how fast the subject is willing to move his head?

STONE: That's right, yes.

GUEDRY: Well, then I have a point which undoubtedly you have considered, but I think it bears more consideration. Not only the magnitude, but also the duration of the cross-coupled angular acceleration controls the effect on the semicircular canal system. The effect on the cupula of a stimulus of given magnitude and duration may be equaled by a lower magnitude stimulus applied for a longer time.

STONE: Well, certainly there are wide variations in the numerous subjects. What I've shown are averages of all of them and their reactions are different.

GUEDRY: Another point. I find your device intriguing and it looks like a quite interesting simulator. One thing was mentioned previously which I believe may be significant in your use of this device. I can confirm from my personal observations that, if head movements are made where the subject is permitted to see relative motion between himself and the Earth, at least under the conditions that I observed this, the effects are greatly reduced. As soon as the man is enclosed and he makes the same head movement, then the effects are increased; so, I would suggest the possibility that if your subjects were seeing relative motion, this may account for the reaction.

STONE: No. In the data we show here, they were not. They were enclosed in the cabin.

CAPPEL: May I point out that the product of the head angular velocity and the centrifuge velocity on your tolerance curve was almost a perfect hyperbola. The product added up to about 1600 degrees per second per rpm per minute.

STONE: It is a hyperbola. Of course, people don't move their heads that rapidly; so, if you wanted to really establish this curve, you would probably have to force the man. You would put him on a spit as Dr. Guedry has shown and force him to the high rates which are essential to define the curve completely. I don't think that the subjects could have voluntarily moved much faster than they moved here. They were reasonably motivated and asked to perform as quickly as they could, and the motions represent fairly well the maximum rates which they could attain without considerable discomfort.

C. CLARK: It is appropriate to make a little plea for the international units. You were using degrees per second and rpm and radians per second. Why not radians per second and radians per second squared? Then this hyperbola comes out in radians per second of the head and radians per second of the turning vehicle.

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# Observations on Subjects Exposed to Prolonged Rotation in a Space Station Simulator

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## SUMMARY

Rotating space vehicle design and task analysis require biofunctional guidelines based upon the integrated information of all levels of vestibular physiologic research, with final criteria determined by the measure of crew performance as a function of controlled environmental changes in a realistic space vehicle simulator. The Manned Revolving Space Station Simulator (MRSSS) at GD/Convair is the result of this concept and allows adjustments in radius, angular velocity, force field and stability. As a baseline study for projected investigations of the stability requirements of a rotogravic system, four subjects performed a comprehensive array of psychologic and physiologic tests during five days of continuous rotation at 6 rpm in the MRSSS. Subjects adjusted completely to the environment, required little post-spin readaptation and showed minor performance decrement. Paper discusses importance of stability as a design parameter and the five-day test program and results.

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## INTRODUCTION

A previous speaker referred to this being less a symposium on the role of vestibular physiology in the exploration of space, and more a symposium on the role of space exploration in vestibular physiology. Perhaps we can help restore the original orientation, for as members of a research group created to provide biofunctional guidelines for space vehicle design and task analysis, it is our responsibility to integrate information from all levels of vestibular physiologic research into satisfactory design criteria, expressing these criteria in parameters of vehicle and task design.

Though the response of the otic labyrinth to both weightless and rotogravity environments requires further elucidation, our studies have been directed primarily toward questions concerning man in a rotogravity field. We need to determine the work poten-

tial of man in rotating vehicles of various dimensions and force field characteristics, to guide the engineers in choosing optimal man-machine trade-offs. The guidelines we provide must be realistic not only for the large orbiting vehicles of the future, but—even more acutely—as back-up concepts for today's approved vehicle programs.

Some sizings of the biofunctional envelope for manned rotogravic vehicles have appeared in the literature. Those of Loret (ref. 1) and Dole (ref. 2) have perhaps received the most currency and are in essential agreement. Figure 1 incorporates the design limits (crosshatched area) suggested by Loret. Loret based his angular velocity ceiling of 4 rpm upon observations made by Graybiel and his group in their Pensacola experiments (ref. 3). We designated this area the "accepted design envelope" as it has met substantial approval in the field of bioastronau-

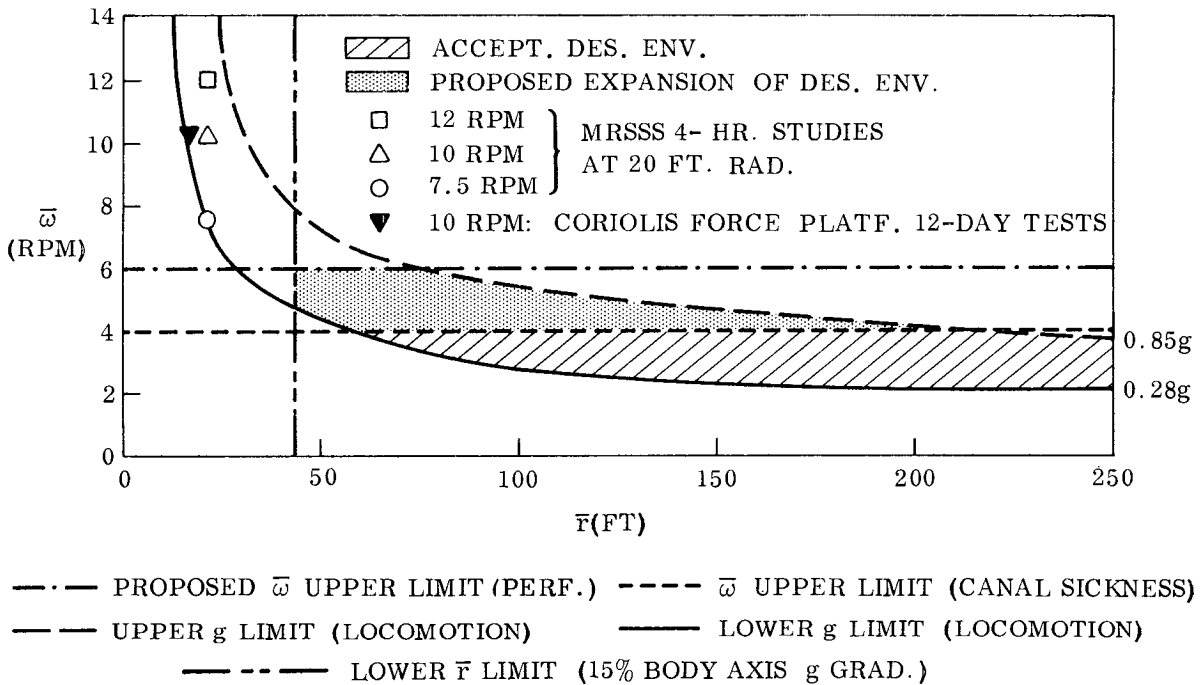


Figure 1.—Biofunctional design limits in manned rotogravic space vehicle. (From *Aerospace Med.*, ref. 1.)

tics. This is not to suggest that there is complete concurrence as to the limits of the envelope; for example, angular velocity ceilings of less than 4 rpm (ref. 4), and even less than 1 rpm (ref. 5) have been suggested.

It is of interest that neither in Loret's or Dole's envelopes is the stability of the vehicle considered a design limitation. It is our contention that, in providing a habitable rotating space vehicle, stability is no less important than angular velocity, radius, g-level and rim velocity. In a rotating space vehicle, vehicle precession as well as head rotation can cause stimulation of the crewman's labyrinth. Vehicle stability, therefore, becomes not only a necessary contingency in manned vehicle design, but also the necessary consideration of the vestibular physiologist concerned about biofunctional guidelines for such design. The bionegative effects of vehicle instability may lower the permitted angular velocity. It would seem reasonable that the stimuli to the labyrinth due to vehicle instability would complement that due to the crewman's active head movements.

Consideration of vehicle precession predicates caution in assigning spin rate ceilings and recommends investigation of the total dynamic environment in the simulated rotating vehicle in relation to habitability and crew performance. Without such ground work, design engineers must work in an arbitrary manner. This could be costly and mission limiting. Workers such as Larson (ref. 6) and Kurzahls (ref. 7) have lent theoretical and empirical consideration to the engineering problem of instability in the manned rotogravic vehicle. Coupled to such efforts must be crew performance tests as a function of instability, as well as of the previously considered parameters.

Disturbances, such as docking impacts and active or passive changes in crew or hardware mass, may cause many combinations of structural and force field oscillations, most of which could be significantly detrimental to crew function.

As stability of a rotating space vehicle is related directly to its total mass, the relatively light state-of-the-art vehicles would



be particularly susceptible to instability from mass disturbances.

Consider a hypothetical discoid or toroidal vehicle rotating about its principal axis Z, with transverse orthogonal axes X and Y. A crewman aligned with one of the transverse axes could be subject not only to the disturbing effects resulting from his active head movements relative to the spin plane, but also to a variety of oscillating forces beyond his active control.

Any impulsive torque applied about either one of the two transverse axes of the rotating vehicle would result in a wobble (defined as an oscillatory curvilinear movement) about both transverse axes, X and Y. The amplitudes of these oscillations would be directly proportional to the angular impulse and inversely proportional to the moment of inertia around the transverse axis normal to the axis of torque. Figure 2, taken directly from Kurzahls' paper (ref. 7), shows a typical wobbling response to a 2 foot movement of a crew member out of the plane of spin of a 30 foot station. The precession amplitude is measured relative to a space-fixed coordinate system. These data were derived by

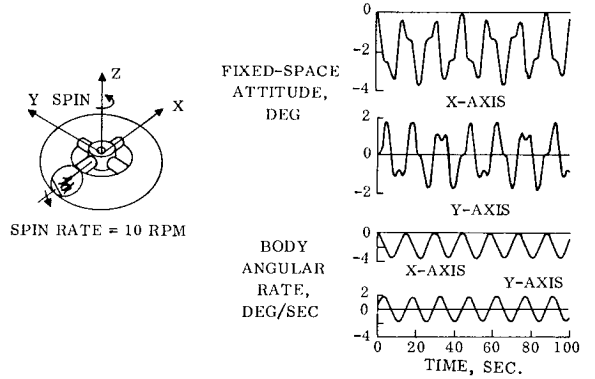


Figure 2.—Motion of uncontrolled station for a typical disturbance.

Kurzahls' using a scale model and programed movements of lumped masses. Figure 3 shows the results of similar experiments using maximum disturbances for entire crews performing maximal excursions at running rates. As can be seen, a reduction in vehicle size causes a dramatic increase in wobble. These figures, it is emphasized, represent an uncontrolled station. Several methods of active and passive dampening can be used to increase stability, though they do entail weight and power penalties. The

TYPE OF DISTURBANCE	DISTURBANCE EFFECTS			
	MAX WOBBLE ANGLE, DEG	APPARENT STA. ROLLING, DEG	MAX WOBBLE ANGLE, DEG	APPARENT STA. ROLLING, DEG
RADIAL CREW MOTIONS	9	0 TO 12	0.7	0 TO 0.8
TRANVERSE CREW MOTIONS	13	0 TO 5	1	0 TO 0.3
CIRCUMFERENTIAL CREW MOTIONS	108	80 TO -80	3	3 TO -3
DOCKING IMPACTS	2	2 TO -2	0.05	0 TO 0.04

Figure 3.—Disturbance effects for two space station configurations.

crewman will perceive wobble as lateral rocking and as oscillating rotation about his longitudinal body axis.

Even in the absence of a mass movement out of the plane of spin, an inertial unbalance produced by an uncompensated mass movement along a transverse axis within the plane of spin will couple with the moment of inertia about the Z axis to produce a vehicle spin about that transverse axis that is directly proportional to the initial vehicle spin rate and the product of the inertias about the two axes. This generated spin coupled with the initial vehicle spin will produce varying angular velocity patterns as shown by the polhode trace on the inertial ellipsoid in figure 4. This diagram by Larson (ref. 6) represents the inertial result of a dynamic mass unbalance along the X axis. A crewman alined with this axis will experience the illusion of complex and ever-varying tilting of the floor as his body perceives the resultant of the linear acceleration oscillating along his longitudinal body axis and the linear acceleration normal to this axis. The linear acceleration normal to his long body axis would trace the vectorial pattern shown in figure 5. Simultaneous dynamic mass unbalances along both transverse axes (the usual real-life situation) would complex the vector pattern and the resulting disturbances.

Therefore, the primary disturbances that may result from vehicle instability are: (1) wobbling, the precession of the space vehicle spin axes relative to space-fixed coordinates,

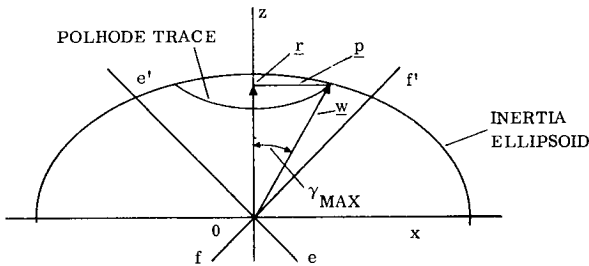


Figure 4.—Polhode trace of gravity vector in the xz plane. (Note: ee' and ff' are planes separating polhode traces about the axis of maximum moment of inertia.) (From ref. 6.)

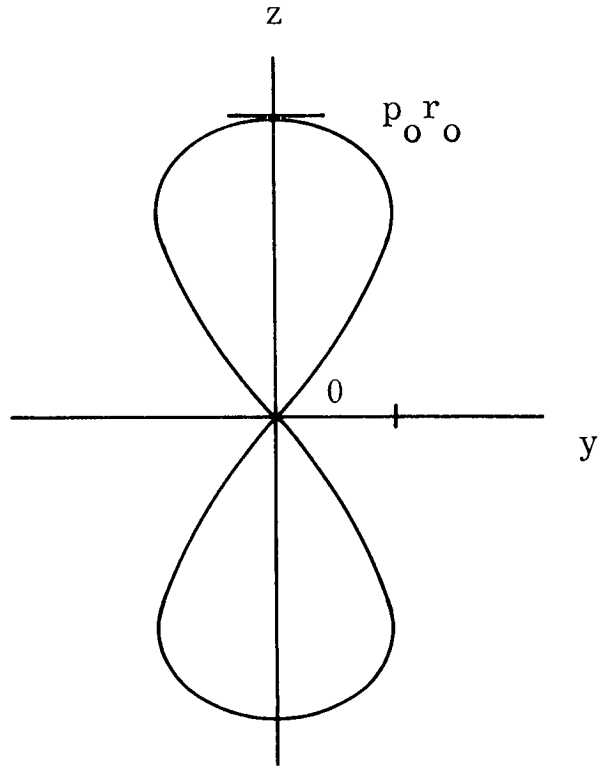


Figure 5.—Gravity vector trace in the yz plane. (Note: Plot for an initial disturbance  $p_0$  about the x axis.)

and (2) rotogravic oscillation, the precession of the vehicle angular momentum vector. These phenomena require that the biofunctional design envelopes be defined inclusive of vehicle stability, as shown in figure 6. What is suggested is that, for given combinations of angular velocity and vehicle radius, the biofunctional envelope be limited by an instability of given precession rate and amplitude. There must be consideration of both spin axis precession (wobble) and spin vector precession (rotogravic oscillation). Wobble may be simulated by oscillating the floor tilt of a revolving simulator during constant spin rate. Rotogravic oscillation may be simulated by oscillating the simulator spin rate with a constant floor tilt.

A secondary disturbance that may result from vehicle instability, especially significant in a small rotating space vehicle, is the variation of rotogravity either through compensated or uncompensated radial mass

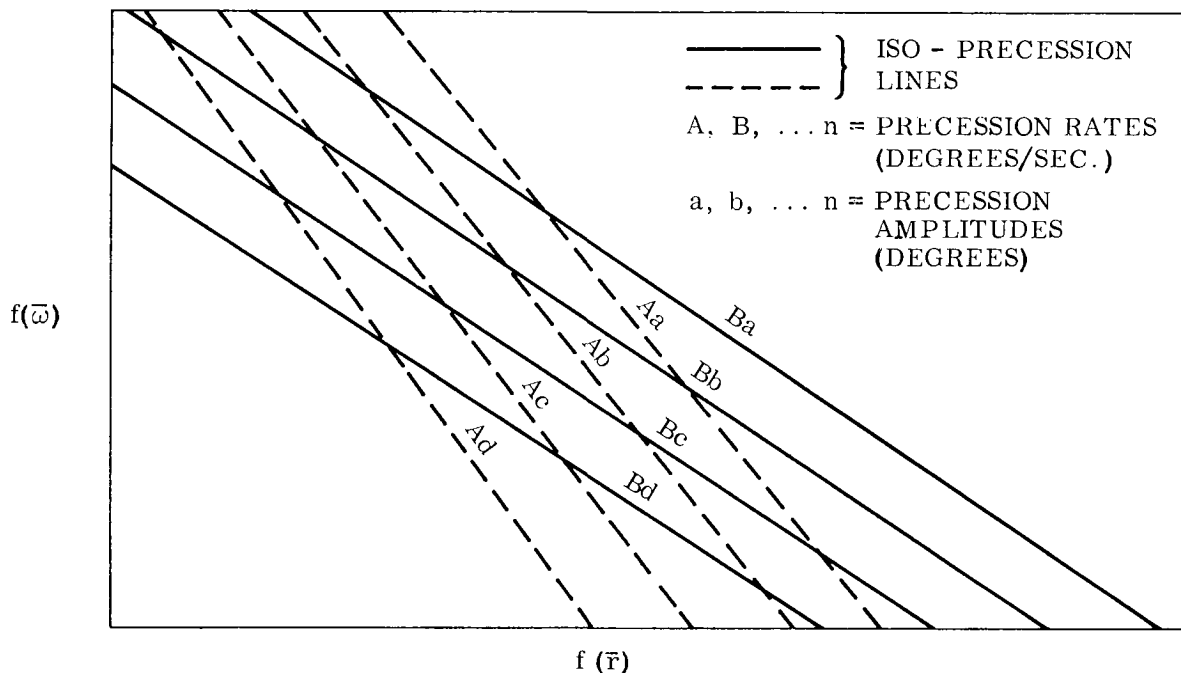


Figure 6.—Stability as a design parameter for a manned rotogravic space vehicle. (From AIAA J., ref. 6.)

movements, or through static mass unbalances. Radial mass movements will vary the angular velocity through a conservation of angular momentum; static mass unbalance will vary it through radial rotogravity differences inherent in a noncoincidence of vehicle and spin axis. These pure variations in rotogravity may be simulated by coordinating angular accelerations with changes in simulator tilt to obtain a continuity of inertial alinement.

All the above stability phenomena may occur singly, sequentially or simultaneously. Stability, therefore, presents a critical and complex biofunctional problem. It is, moreover, a labile quality and can be assured only at the expense of vehicle power and responsiveness.

The design trade-offs required can only be optimal when the moduli of crew response to these dynamic stresses are predictable. Therefore, simulation difficulties and unavoidable artifacts should not preclude the inclusion of stability in defining the vehicle biofunctional design envelope. Even ball-

park guidelines are better than arbitrary design choices.

To expedite the definition of these guidelines, we have made use of the engineers' "black box" approach in preference to a purely analytic investigation.

For this type of study a significant number of subjects are required to perform appropriate tasks within a simulator that is a reasonable facsimile of a rotating space station. By varying the dynamic characteristics of the environment (angular velocity, stability, force field) we obtain guidelines in terms of crew performance. In this way we can estimate the design requirements for stability systems and enable the engineer to go to work on this important aspect of the program.

The first requirement of the program was to design and build the Manned Revolving Space Station Simulator (MRSSS). Figures 7 and 8 are a photograph and diagram, respectively, of the MRSSS. Described in greater detail in another paper (ref. 8), it consists of an 8 ft × 14 ft × 7 ft cabin trunnioned to supporting I-beams 18 ft from the spin axis of a 220 000 g-pound centrifuge.

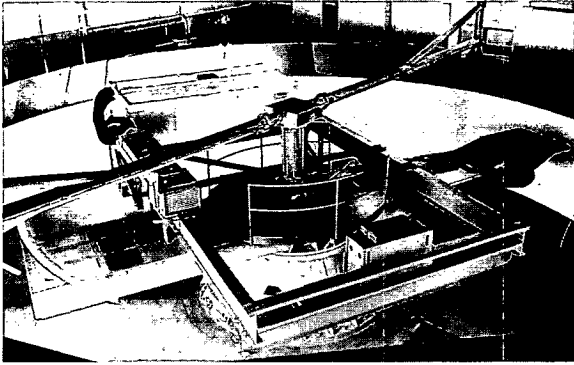


Figure 7.—MRSSS testing complex.

The cabin is divided into two separate rooms, one containing the sleeping and toilet facilities, and the other food preparation and refrigeration facilities, and space for recreation, testing and study. Rotary couplings and slip rings provide running water, sewage disposal and data transmission during rotation. Additional communication is provided by voice, television and FM telemetry. A loading port in the outboard bulkhead of the MRSSS permits in-rotation transfer of parcels (food, mail, physiologic specimens, etc.).

Figure 9 shows the room inclined from the vertical on its trunnions, increasing the effective cabin radius (approximately 20 feet at 10 rpm) as the angular velocity increases. The inclination of the room can be varied pneumatically according to program for the

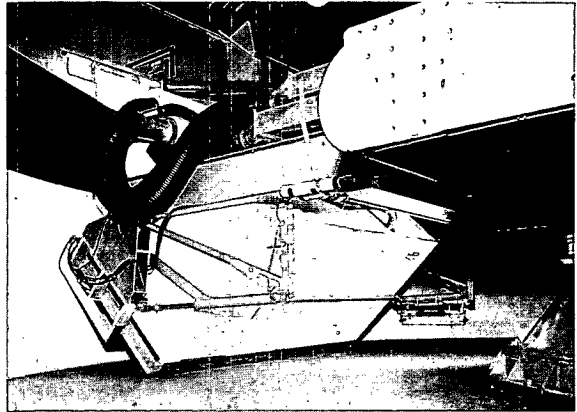


Figure 9.—MRSSS inclined on trunnions.

stability studies described above. The inclinability of the MRSSS and its peripheral location at a relatively long radius from the spin axis eliminate three important artifacts inherent in carousel-type simulators:

1. The inclinability permits alinement of the MRSSS with the inertial resultant of the rotogravity and geogravity vectors. This permits standing normal to the floor as in a space station.

2. The long-radius peripheral location (and room length tangential to spin circle) minimizes the amount of radial translation during a series of random movements and reduces the relative linear velocity changes involved in such translations. The former would approach the space station situation as a limit; the latter may be a performance desideratum, if a Weber-Fechner dependency is applicable.

3. The inclination and long radius combine to cause identical head rotations about the Y or X cranial axes to produce different labyrinthine stimulation, depending upon the orientation of the subject. This is due to the different angles produced between the plane of head turn and the spin plane of the vehicle. This again approaches the space station situation and is of particular importance as a consideration in the phenomenon of habituation<sup>1</sup> to the rotating environment.

<sup>1</sup> *Habituation* is used here as the state of adjustment of the organism resulting from the functional change called *adaptation*.

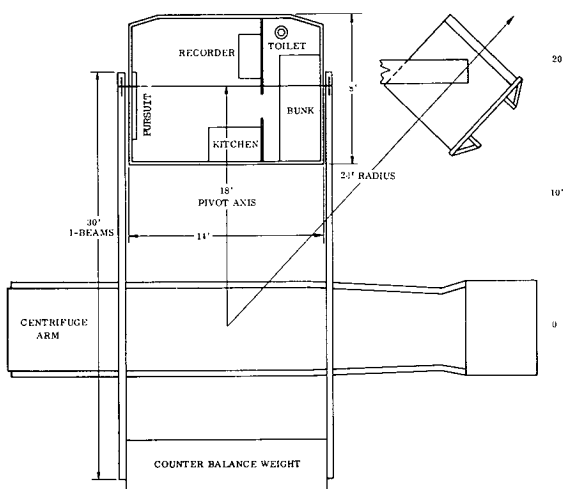


Figure 8.—MRSSS testing complex (diagrammatic).

Since its activation 2 years ago, the MRSSS has been used for several thousand man-hours of testing, the subjects being either GD/C engineers or Navy Pilots from North Island and Miramar in San Diego.

Tests were run to determine what tasks were particularly sensitive to a rotating environment and what physiologic problems could result from rotational testing. This information was required prior to attempting extended time exposures. As seen in figure 1, acute exposures ran to four hours at angular velocities as high as 12 rpm. Even at this high rate of rpm, normal subjects could spin up directly and perform complex tasks with minimal malaise, little decrement in performance, and rapid adaptation (ref. 8). At 12 rpm and 20 feet from the spin axis, the inertial resultant is approaching  $1\frac{1}{2}$  g, which was felt to constitute an artifact prohibiting exploration of any higher angular velocities at that radius.

The next step has been to increase the time function. This resulted in a four-man, 5-day test at 6 rpm, which we recently completed. The remainder of this paper will discuss this 5-day test and its function as a baseline study for projected stability investigations.

The angular velocity of 6 rpm was chosen for several reasons: (1) with a 19 ft working radius, the g artifact would still be only 0.05; (2) this would be a realistic angular velocity for the small radius state-of-the-art station, providing 0.5 g at 40 feet, and Loret's (ref. 1) lower limit of 0.28 g at 24 feet; and (3) Clark and Graybiel's (ref. 9) published work had shown that subjects could tolerate extended exposures to rpm's as high as 5.4. If this proved to be a good baseline performance study, we could then proceed to programming instability profiles into additional exposures at this rpm.

#### METHODS

The test plan included:

(1) Subjects: Four subjects divided into two teams of two subjects each (one psychologist and one physiologist). Subjects' ages ranged from 26 years to 37 years. They

were required to pass Class II flight physicals.

(2) Dynamic Profile: Three 1-hour exposures to 6 rpm, two during test week minus two, and one during test week minus one. Three of the four subjects were completely naive regarding the MRSSS, and it was felt that these brief exposures would allay anxiety that might favor kinetosis during the test. For the test, subjects entered the MRSSS 48 hours prior to spin-up, then were rotated for 120 continuous hours (in sequence: 4 hours at 2 rpm, 4 hours at 4 rpm, 104 hours at 6 rpm, 4 hours at 4 rpm, terminating with 4 hours at 2 rpm). Forty-eight hours of postrotatory testing were scheduled, but an unpredicted ease of readaptation shortened this to 8 hours. The step-wise spin-up and spin-down were chosen to increase the assurance that neither the rotatory nor postrotatory testing would have to be aborted due to severe physiologic reactions on part of any of the subjects. Such a form of graduated transition is entirely realistic for a rotating space vehicle. The length of the step (4 hours) was not entirely arbitrary, as a test array required 3 hours to run.

(3) Schedule of Testing. During the two weeks preceding the test, subjects were tested to approach an asymptotic performance plateau, and monitored physiologically for a functional baseline. During the test, teams observed an alternating four-hour work-rest cycle, one team testing while the other slept, ate or relaxed. This schedule allowed repetition of all tests every 16 hours, some every 8 hours.

(4) Monitoring and Testing: *Medical Monitoring* consisted of onboard blood pressures and oral temperatures, and remote monitoring through televising and polygraph traces of EKG's and respiration rates. During rotation, blood and urine samples were taken for subsequent biochemical analysis. Shifts in blood volume were monitored using radio-albumin tracers. The cardiovascular reserve was monitored utilizing a modified Flack test, requiring the maintenance of a 40 mm Hg Valsalva for 50

seconds. *Psycho-physiologic Testing* consisted of six categories of function which previous studies suggested might be sensitive to the test environment: (a) Vision: both Perimetric and Orthoscopic Tests; (b) Audition: an Interrupted Audiometric Test; (c) Vestibulo-ocular Reflex: Caloric, Oculogyral Illusion, and Ballistic Aiming Tests; (d) Brachial Proprioception: Ballistic Aiming, Digital Proprioception and Steadiness Tests; (e) Walking and Standing: Visual Tandem Walking and Blind Tandem Walking and Standing Tests; (f) Mentation: Logical Inference, Response Analysis, Time Estimation, and Mathematics Tests. The description of each test: *Orthoscopic Test*: Using Keystone Orthoscope, tests are for lateral and vertical phorias, convergence and divergence, macular suppression, and stereopsis. *Perimetric Test*: Using an American Optical screening perimeter, visual fields are plotted. *Audiometric Test*: Using a Maico MA-6 Audiometer, % normal auditory acuity (in decibels) is determined for frequencies 500, 1000, 2000, and 4000 cps. *Caloric Test*: In darkened room, 50 cc of 20°C water are syringed into the ear at 1 cc/second. Response is measured by duration of resulting oculogyral illusion. *Oculogyral Illusion Test*: Subjects, constrained by a bite-bar, perform four head rotations about the X cranial axis in interrupted sequence (to and from 45° to the right, and to and from 70° to the left). Response is measured from subject's audible description of direction, magnitude and duration of displacement of illuminated target in darkened room. The same target is used for the caloric test. *Ballistic Aiming*: Facing a 4-foot diameter vertically-mounted test table, subject attempts to strike two 1-inch diameter targets in sequence (one appearing at 6 o'clock, the second immediately thereafter at 12 o'clock). Postural constraints and exposure time of targets require that the subject perform a 90° rotation of his upper torso (and head) about the body Y axis in 0.3 to 0.5 second every 10 seconds during a three-minute test run. The striking stylus and targets connect electrically through a counter

for scoring. *Digital Proprioception*: A quantitative past-pointing test, it requires subject to keep his eyes closed while striking in a radial direction at the center of a vertical grid, bending to touch his shoe, and returning to strike at the grid center a second time. The bending and return are done to the 1-second beat of a metronome. Scorer records miss distances. *Steadiness*: Subject is required to hold a hand probe in one of four holes ranging from 0.14 in. to 0.54 in. in diameter for ten seconds with a minimum number of touches. Scored on number of touches. *Blind Tandem Walking*: Heel-to-toe radial walking on floor grid, forward five steps toward spin axis then backward five steps away from axis. Arms must be folded against the chest. T-connected stethoscope ear pieces transmit sounds symmetrically to both ears, eliminating this cause of tropic unbalance. Subject is scored on steps completed and grid coordinates of unbalance point. *Visual Tandem Walking*: Same test as preceding, but with eyes open. *Blind Tandem Standing*: Heel-to-toe standing, facing spin axis. Same postural constraints as in walking tests. Subject scored on number of seconds in balance, up to 60. *Logical Interference Tester (LOGIT)*: An instrument designed by French (ref. 10) of our laboratory to assess higher mental processes, including reasoning, memory and decision skills, LOGIT requires that the subject learn in a minimum number of moves an unknown 20-light sequence. A patch panel allows programming the instrument with one of 5000 sequences for each test run. Subject is scored on number of moves required. *Response Analysis Tester (RATER)*: Also designed by French (ref. 10), RATER gages sensory-motor efficiency by requiring the subject to press the correct one of four buttons in response to the appearance of one of four symbols (cross, triangle, square, and circle). Each test run consists of 20 minutes' responding, 5 minutes to each of four different button-symbol connection patterns. The four connection patterns remained fixed throughout the 5-day test, but were presented in random

permutations. Subjects are scored on total responses, total errors and total correct responses. *Time Estimation*: Subjects are asked to estimate five time intervals (2, 3, 6, 8, and 11 seconds), presented in a random order, by pressing the time clock actuation button. Scored on accuracy. *Math Test*: Designed to assess the efficiency of problem solving, test presents a large number of arithmetic problems, each requiring addition, subtraction, multiplication and division, for solution in a 15-minute time period. Scoring is on number correct.

(5) *Miscellaneous Demand Activity*: Subjects were required to fill out routine psychosocial ratings on themselves and other subjects, keep diaries, and fill out daily 50-item medical histories.

(6) *Dress*: Subjects wore nylon flight suits and tennis shoes.

(7) *Food*: Subjects' primary diet consisted of freeze-dry space food provided by the Pillsbury Company. The diet was found quite acceptable and the quality of this food proved to be excellent. If other ingestibles were desired—milk, fruit, ice cream, sandwiches, pastries, etc.—they were transferred aboard.

#### RESULTS AND DISCUSSION

In the interest of economy, only those tests which appear to have been significantly affected by the test environment will be discussed. The remaining tests can be considered to be unaffected.

Figure 10 shows the subjective habituation of the subject as a function of exposure time. By definition, the subject's feelings of habituation—based upon his diary and onboard medical history—measure his total feeling of adjustment to the environment. This factor goes beyond mere ability to perform certain tasks adequately. For example, a subject might show little or no decrement in a complex task such as a LOGIT problem, yet estimate he feels only 75% as "good" as he normally would. The graph indicates that by the end of 3½ to 4 days of rotation, three of the four subjects felt as good as they would under conventional circumstances. The subjects are listed alphabetically in decreasing ability for overall adaptation. The letters A, B, C and D designate the same subjects in subsequent graphs.

*Auditory Acuity Test*.—Figure 11 illustrates a rather surprising result. Averaging the % auditory norms for the four fre-

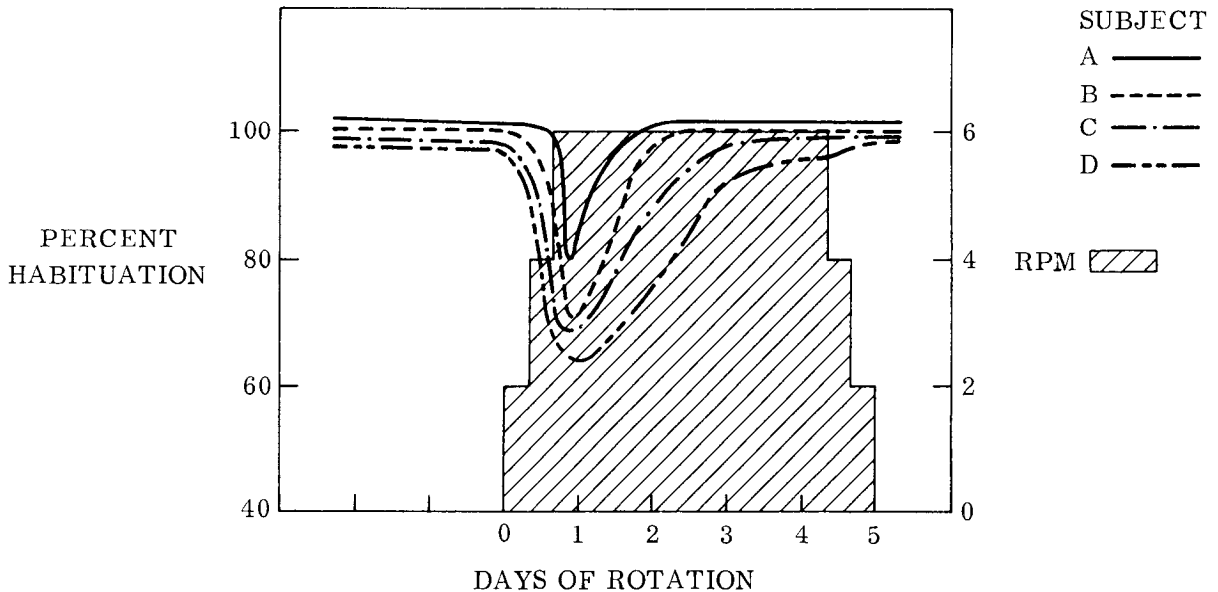


Figure 10.—Subjective habituation vs. days of rotation. Based on subject's evaluation of his well being relative to static baseline.

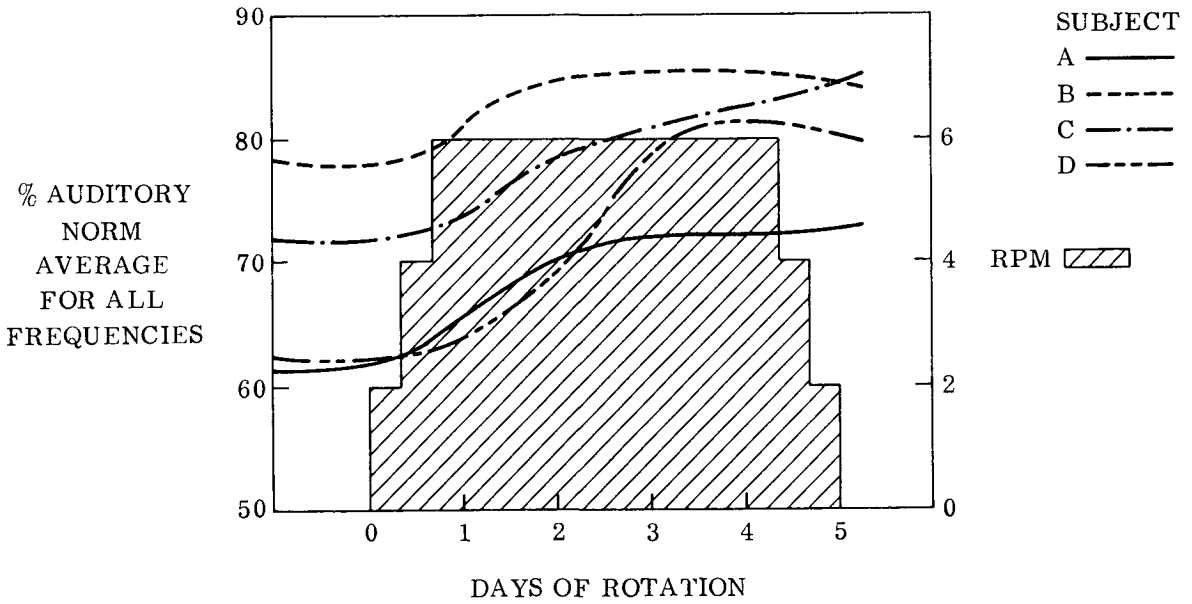


Figure 11.—Auditory acuity vs. days of rotation.

quencies, all subjects showed increases in auditory acuity during rotation. One must hesitate to explain such a change until additional studies verify that it is not a learning artifact. However, the consistency of effect and the discipline of the audience caused us to bring it to your attention.

*Oculogyral Illusion Test.*—Figure 12 suggests that little correlation can be drawn between the OGI response and a subject's ability to tolerate a rotating environment. The decrement in OGI response with time is consistent with the observations of other workers (refs. 11 and 12). It is of signifi-

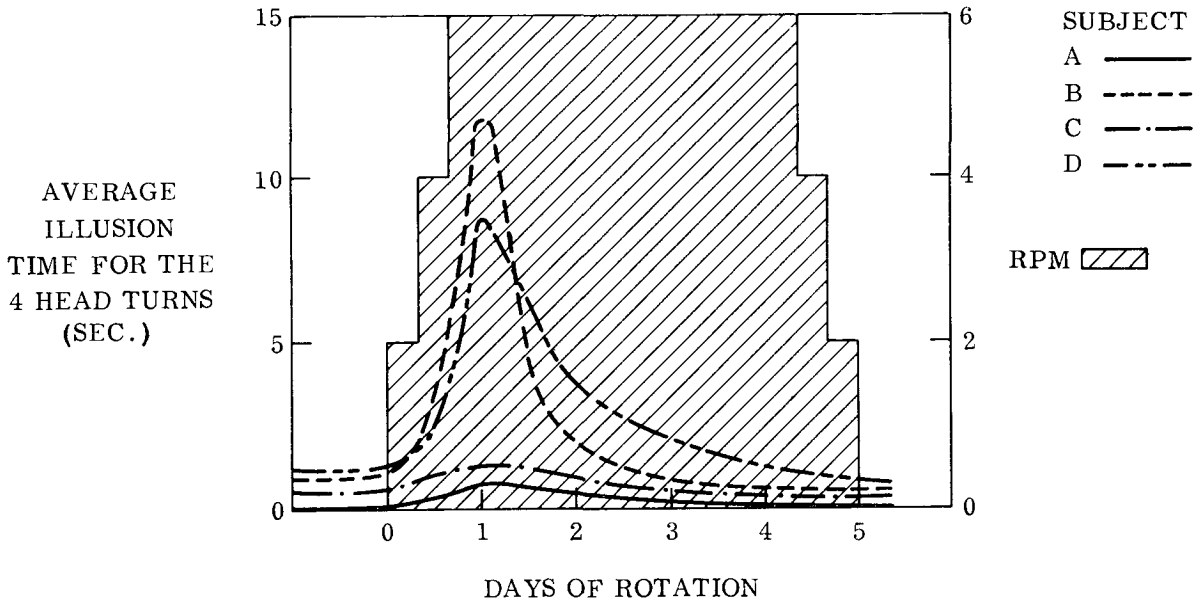


Figure 12.—Oculogyral illusion vs. days of rotation.



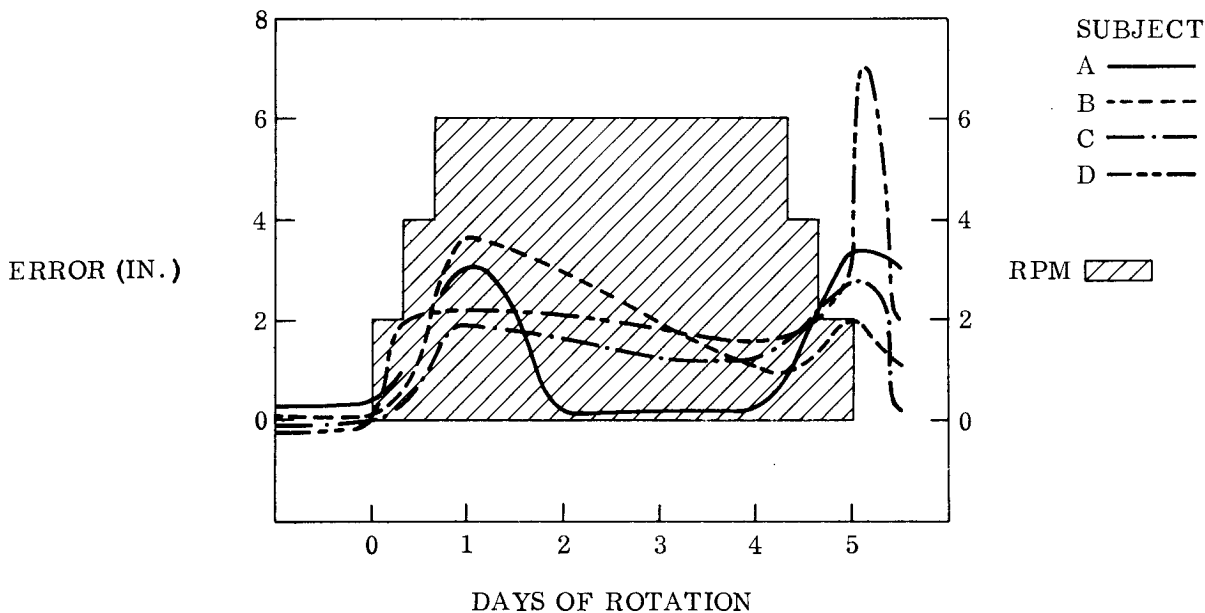


Figure 13.—Digital proprioception vs. days of rotation.

cance that the caloric responses of subjects did not show decrement, and that even on the 4th and 5th days of rotation, intense caloric responses lasting 3 and 4 minutes were reported. Assuming an endolymph-mediated caloric response, the decrement in OGI suggests that it is principally a central phenomenon, from which the caloric response is qualitatively excluded.

*Digital Proprioception.*—Figure 13 graphs the miss distance as a function of exposure time. To simplify the illustration, misses were considered only as scalar quantities; however, with the start of spindown, the direction of misses became 180° to that of preceding test trials. Digital proprioception was one of two tests that showed any post-rotation decrement. It is interesting that this decrement occurred when the subjects felt “completely” readapted to the normal environment. For comparison of subjects, this test correlated very well with their ability to adapt.

*Tandem Walking With Eyes Open.*—Figure 14 indicates that with vision the subjects showed a rapid adaptation in precision locomotion tests. The adaptation in this test also shows some correlation with overall habituation.

*Tandem Walking With Eyes Closed.*—Figure 15 shows the marked reduction in ability to adapt to precise locomotions in a rotating environment when vision is not available. With the exception of Subject B (an Air Force Reserve Pilot), all subjects showed decrement in this test throughout rotation.

*Tandem Standing With Eyes Closed.*—Figure 16 indicates that the subjects were unable to perform this test with any facility at 6 rpm, and that no improvement occurred with time. Only this test and the digital proprioception test showed post-rotation decrement, and when post-rotation testing was concluded at the end of 8 hours, the ability to stand tandemly with eyes closed was still less than normal. Both tests are performed with the subjects standing in one spot with their eyes closed. The deletion of vision and kinematic stimulus to the deep proprioceptors may account for the sensitivity of these tests to the inertial change.

Other than those two tests, the subjects performed effectively. Coupled to their performance capability were the surprising phenomena of “complete” habituation and no apparent need for static readaptation with the step-wise spindown. Only one vomiting

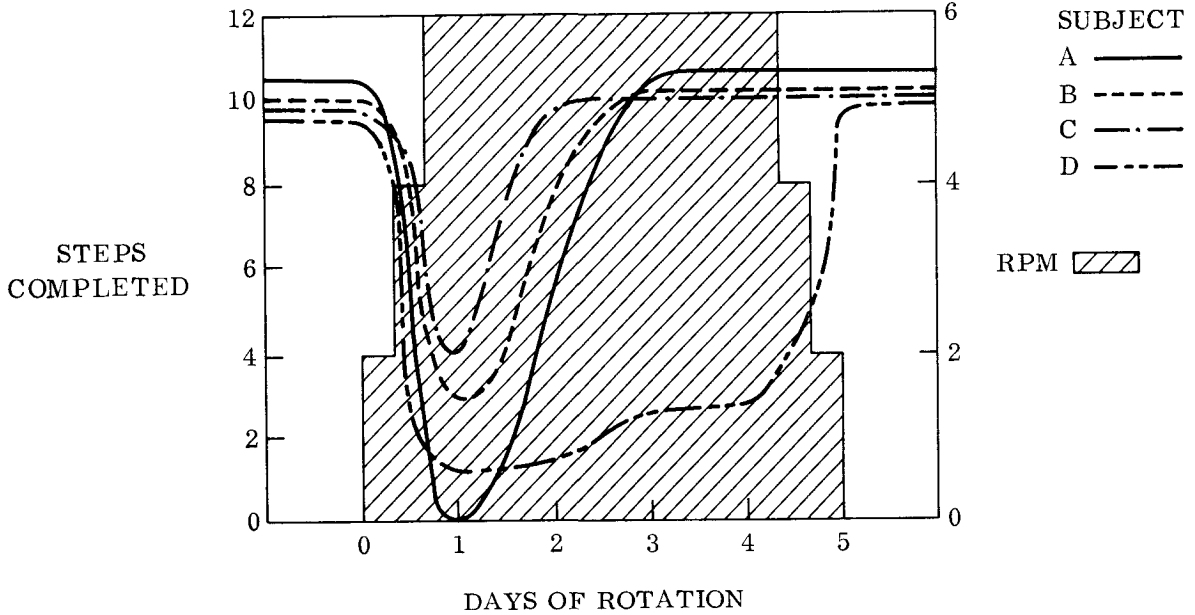


Figure 14.—Visual tandem walking vs. days of rotation.

episode occurred. During the first 24 hours at 6 rpm, Subject C had just taken a large drink of cold water when the engineer operating the centrifuge actuated the MRSSS positioning system to correct for an error in room inclination. The action resulted in a few sec-

onds of severe oscillation. Less than 12 hours later, this same subject spent 30 minutes manually freeing the toilet of soiled paper that was blocking drainage. This task—which could be expected to elicit nausea—caused him no additional trouble.

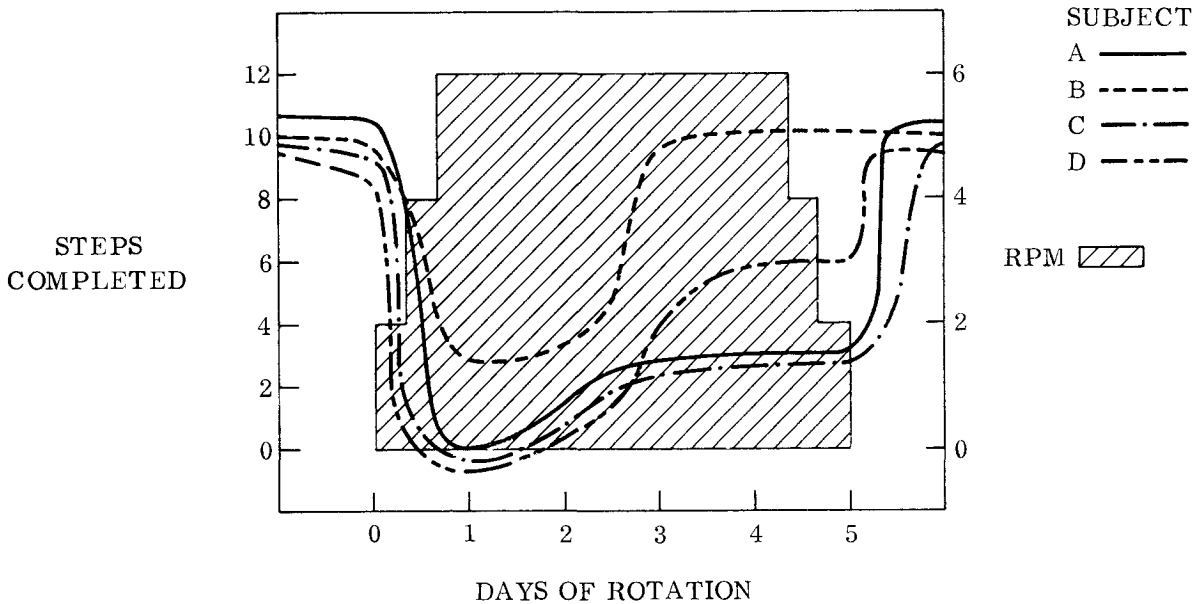


Figure 15.—Blind tandem walking vs. days of rotation.

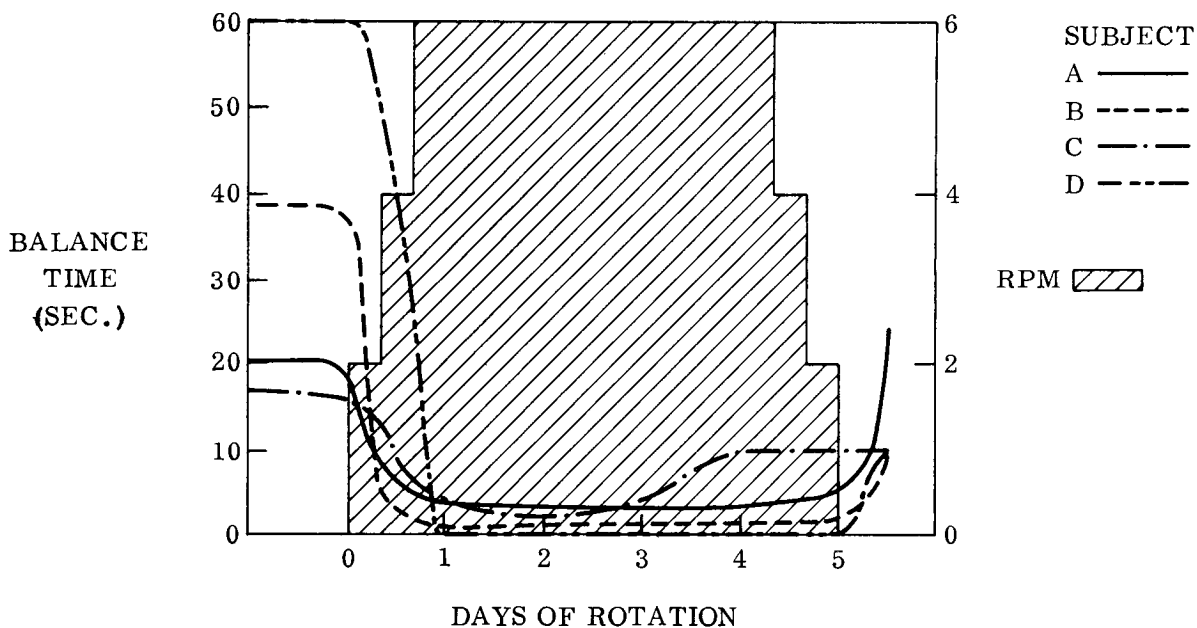


Figure 16.—Blind tandem standing vs. days of rotation.

By the second day all subjects reported normal appetites and the entire crew began the "space food" diet. For the supper just prior to spindown, a feast of charcoal-broiled steaks, baked potatoes, tossed salad, and garlic bread was transferred aboard. There was little decrement in crew appetite.

Most of the onboard photographs, slides and movies were taken by Subject D. His functional ability remained at a highly satis-

factory level although his subjective habituation was the lowest of the entire crew.

In conclusion (referring to fig. 1), the observations made during this test would indicate that the angular velocity ceiling for stable rotating spacecraft design may be raised to 6 rpm. It is at this rpm that we plan to begin our studies to determine the stability dimensions of the biofunctional envelope of a rotating space vehicle.

REFERENCES

1. LORET, B. J.: Optimization of Space Vehicle Design With Respect to Artificial Gravity. *Aerospace Med.*, 34, 1963, pp. 430-441.
2. DOLE, S. H.: Design Criteria for Rotating Space Vehicles. Research Memo 2668, Rand Corporation, October 18, 1960.
3. GRAYBIEL, A.; CLARK, B.; AND ZARRIELLO, J. J.: Observations on Human Subjects Living in a "Slow Rotation Room" for Periods of Two Days. *A.M.A. Archives of Neurology*, 3, 1960, p. 55.
4. MAYO, A. F.; AND NOLAN, J. P., JR.: Bioengineering and Bioinstrumentation. *In: Bioastronautics* (Schaefer, K. E., ed.). The Macmillan Company, 1964, p. 253.
5. CLARK, C. C.; AND HARDY, J.: Gravity Problems in Manned Space Stations. *In Proceedings of the Manned Space Stations Symposium. Inst. of Aeronaut. Sciences, Apr. 22, 1960.*
6. LARSON, C. A.: Space Station Design Parameter Effects on Artificial g Field. *AIAA Journal*, Aug. 1964, pp. 1454-55.
7. KURZHALS, P. R.; ET AL: Space-station Dynamics and Control. *In: A Report on the Research and Technological Problems of Manned Rotating Spacecraft. NASA TN D-1504, August 1962.*
8. BRADY, J. F.; AND NEWSOM, B. D.: Large Excursion Rotary Tracking of Target and Target Light in a Space Station Simulator Revolving at 7.5, 10.0 and 12.0 RPM. Presented at the Aerospace Medical Association Meeting in Bal Harbour, Florida, May 11, 1964.
9. CLARK, B.; AND GRAYBIEL, A.: Human Performance During Adaptation to Stress in the Pensacola Slow Rotation Room. *Aerospace Med.*, 32, 1961, p. 93.
10. FRENCH, R. S.; AND PIATT, J. L.: RATER and

- LOGIT: Devices for Assessing Crew Efficiency Under Stress. ERR-AN-408, General Dynamics/Astronautics, San Diego, Dec. 1, 1963.
11. GRAYBIEL, A.; GUEDRY, F.; JOHNSON, W.; AND KENNEDY, R. S.: Adaptation to Bizarre Stimulation of the Semicircular Canals as Indicated by the Oculogyral Illusion. *Aerospace Med.*, 32, 1961, p. 321.
  12. GUEDRY, F. E.; GRAYBIEL, A.; AND COLLINS, W. E.: Reduction of Nystagmus and Disorientation in Human Subjects. *Aerospace Med.*, 33, 1962, p. 1356.

#### DISCUSSION

MONEY: I would like to emphasize that when it is decided what rpm produces the maximum acceptable stimulus in a rotating room, that the maximum acceptable rpm for a rotating spacecraft is something less than this because the rotating spacecraft

has a stimulus which is different, more variable, more intense, and harder to adapt to.

NEWSOM: I would have to agree that this might be the case. I will reserve judgment until I see some data.

**SESSION V**

***Chairman:* ERIC OGDEN  
Ames Research Center, NASA**

***Cochairman:* JORGE HUERTAS  
Ames Research Center, NASA**

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# The Effects of Exposure to a Rotating Environment (10 rpm) on Four Aviators for a Period of 12 Days

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The chief purpose of this investigation was to measure the effects of prolonged exposure in a rotating room under environmental and working conditions which simulated in many respects those which might obtain in a rotating orbiting spacecraft. The rate of rotation selected, 10 rpm, was considered to be near the upper limits of angular velocity to which man might adapt without impractical side effects. The duration of the run, 12 days, seemed sufficient for studying adaptive changes noted in previous studies of a similar nature (refs. 1 and 2). Because it was recognized that the angular velocity was near the upper feasible limits, participants were carefully selected from officers in the Navy flight training program. The primary objectives in the study were to determine: (1) whether these selected individuals would remain on board for the planned duration without the need for medical treatment; (2) whether any of a wide variety of physiological and biochemical tests would reflect any adverse effects of prolonged rotation; and (3) whether the

performance of tasks designed to test different abilities would be adversely affected. The findings are discussed both from their theoretical and practical aspects.

In previous reports from this laboratory the widespread symptomatology which normal persons have manifested in the Pensacola Slow Rotation Room (SRR) has been designated "canal sickness" to distinguish this etiologic type of motion sickness. The symptoms are not experienced by individuals who have lost vestibular function and are reduced or even absent in those with a partial loss of function (from "Effects of Partial Suppression of Vestibular Function Ten Years After Treatment of Ménière's Disease With Streptomycin Sulfate" by A. Graybiel, H. F. Schuknecht, M. E. McLeod, E. F. Miller II, and A. R. Fregly, in preparation). The symptomatology is dependent not only on unnatural stimulation of the canals per se, but also on many other factors, e.g., conflict among normally synergic inputs, lack of fitness, and past conditioning. Symptoms range in severity from mild malaise to prostration. They include dizziness, headache,

apathy, drowsiness, and fatigue. Objective signs include pallor, cold sweating, vomiting, inactivity, and ataxia. First order effects apparently give rise to secondary effects of still greater diversity which are declared by biochemical changes and other objective signs, e.g., compensatory nystagmus (ref. 3). The time-course for changes in one symptom may reveal little of the overall complexity of the adjustment; e.g., nausea may persist after nystagmus has declined. If perrotation adaptation has taken place, a return of symptoms may be expected following cessation of rotation. There is evidence of habituation with repeated exposure (ref. 4), and countermeasures including drug therapy are demonstrably beneficial.

Prior experiments involving prolonged exposure of man to continuous rotation at 10 rpm are relevant to the present report. Of three subjects who lived in the Pensacola Slow Rotation Room for two days at 10 rpm, two were normal subjects adjudged to be of different sensitivities to canal sickness, and the third was a control subject who had lost vestibular function (refs. 1 and 2). The control subject did not report sickness and exhibited only slight difficulty in walking heel-to-toe, a difficulty to be anticipated as a result of conflicting visual and proprioceptive clues in this unusual environment. Of the two normal subjects, neither appeared to have motivation to continue beyond the 2-day run, despite the fact that there was evidence of some adaptation in both subjects. The less susceptible "normal subject," who had had previous experience in the room, appeared less affected than the other "normal subject."

A series of short runs conducted primarily to evaluate susceptibility to canal sickness has involved angular velocities higher than 10 rpm. Systematization of stimulation was accomplished by means of an experimenter-paced Dial Test which necessitated different head and trunk movements in setting the needle of five dials arranged around a chair (ref. 1). It was found that 7.5 rpm was a convenient velocity for screening tests but

that in some cases velocities up to and including 20 rpm were required to produce symptoms of sickness. Occasionally, subjects on repeated testing showed little evidence of malaise at this velocity for periods up to an hour (ref. 5).

Similar results were obtained by Stone and Letko (ref. 6) whose subjects were placed in a supine position, feet outboard, and supported by a "floor" 15 feet from the center of rotation, thus simulating the orientation in a rotating spacecraft. Short periods of rotation were used, and maximum angular velocities were 17 rpm. The authors encountered malaise in all subjects at 10 rpm and above, and rightly concluded that more simulation studies should be conducted.

The present experiment was undertaken in the light of this background, with subjects purposely selected to be resistant to motion sickness and instructed to avoid unnecessary head movements until adaptation ensued. Effort was made to ensure motivation of the subjects by (1) explaining the importance of their participation to the space effort; (2) selecting two Marine and two Navy officers in the hopes of engendering a competitive spirit; (3) arranging for publicity in the local news media; and (4) impressing upon the subjects that the objective was to succeed in all tests without getting sick, if possible.

#### PROCEDURE

##### Subjects

Two Navy and two Marine officers, who had completed the aerobic stage of flight training, were chosen as subjects. Additional factors influencing selection were their youth, high motivation, good general fitness, good mental discipline, and a history of less than average susceptibility to motion sickness.

The general clinical findings of the subjects are summarized in table I. The systolic murmur in the case of LI was of doubtful pathological significance; the varicocele in the case of MO was not troublesome; the ballistocardiogram in the case of SH revealed

Table I.—General Clinical Findings on Experimental Subjects

Subject	Age	Ht., in.	Wt., lbs.	Past history	General fitness	Phy. exam., B.P.	Chest X-ray	Work ECG, <sup>a</sup> ht. rate <sup>b</sup>	BCG	Blood morph., Hematocrit, PBI	Urinalysis, Micro.
LI	24	70.5	162	Pilonidal cyst	Excellent	Sys. mur. G II base 126/54	N-L	N-L 58/76	N-L	N 40	N N
MO	23	72	153	Not sig.	Very good	Left varicocele 120/64	N-L	N-L 77/109	N-L	N 45	N N
SH	22	74	190	Not sig.	Excellent	N-L <sup>c</sup> 120/64	N-L	N-L 52/88	Grade 1 Abnorm.	N 42	N N
WI	21	76	191	Low T waves ECG	Excellent	N-L 134/78	N-L	N-L <sup>d</sup> 68/84	Too tall for bed	N 47 4.2	N N

<sup>a</sup> 20 inch step up; 20 X per min for 3 min.<sup>b</sup> N = normal; N-L = normal limits.<sup>c</sup> Ventricular rates before and immediately after exercise.<sup>d</sup> Basal ECG normal; resting ECG diphasic. T<sub>2</sub>, V<sub>5</sub>, V<sub>6</sub>, inverted T<sub>3</sub>, fasting-normal.



normal and abnormal complexes, the latter predominating but their pathological significance was doubtful in the absence of any other cardiovascular abnormality; the T wave alterations in the case of WI led to a thorough cardiovascular evaluation on two different occasions, and it was concluded that there was small likelihood that they had pathological significance.

The special findings, mainly referable to the sensory organs of the inner ear, are summarized in table II. The "threshold caloric tests" (ref. 7) were conducted with the subject's head so positioned that a line between the tragus and outer canthus was vertical. The ear was irrigated for 40 seconds with 100 cc of water at an outlet-controlled temperature of 36.4° C. Nystagmograms were recorded while the eyes were observed through Frenzel lenses. In the absence of any response the temperature of the water was lowered in 0.2° C steps until nystagmus appeared on the trace. McLeod and Meek (ref. 7) found that 95 percent of 104 normal subjects manifested nystagmus when the irrigating temperature was 35.4° C or higher; hence, the value of 34.6° C for the right ear in the case of MO represents, probably, abnormally low sensitivity.

The subjects were evaluated with regard to susceptibility to motion sickness from different approaches, and the results are summarized in table III. The first approach was

based on responses to the Rorschach test but scored in an unusual way (refs. 8 and 9) for specific dimensions, viz., anxiety, dependency, drive, hostility, and rigidity. The second was based on a lengthy interview emphasizing the "social history" and the reaction to initial exposure to an unusual force environment. Two provocative tests were employed. The first consisted of a brief exposure to "Coriolis vestibular stimulation" (ref. 10), and the second test was exposure to a standardized pattern of acrobatics designed to induce motion sickness (ref. 5).

It was concluded that all four subjects were less susceptible than the average person to motion sickness. Only two had a history of motion sickness, and this was limited to a single instance. All were unsusceptible to airsickness, but significant differences were revealed in the vestibular test.

#### Apparatus and Methods

The Pensacola Slow Rotation Room (SRR), described in an earlier report (ref. 1), was used in this experiment. It is a multisided windowless room about 15 feet in diameter and 7 feet high with a nearly square (15¾ in. × 12½ in.) center post. The motive power is supplied by a gas burning engine geared to a rubber-tired wheel in contact with the driving band of a flywheel to which the superstructure (SRR) could be clutched or unclutched. Slip rings provided the

Table II.—*Special Clinical Findings*

Subj.	History of deafness, otitis, vertiginous attacks	Otosopic exam		Hearing		Threshold caloric test, °C		Parallel swing <sup>a</sup>
		R	L	R	L	R	L	
LI	No	N	N	N	N	36.2	36.4	Normal response
MO	No	N	N	N	N	34.6	35.8	Normal response
SH	No	N	N	N	N	36.2	36.4	Normal response
WI	No.	N	N	N	N	36.2	36.4	Normal response

<sup>a</sup> This is a measure of otolith function.

Table III.—*Evaluation of Susceptibility to Motion Sickness in Experimental Subjects*

Tests	Subject LI		Subject MO		Subject SH		Subject WI	
Rorschach	<i>Rank</i>	<i>Score</i>	<i>Rank</i>	<i>Score</i>	<i>Rank</i>	<i>Score</i>	<i>Rank</i>	<i>Score</i>
Anxiety-----	2.5	+2	1.0	+3	2.5	+2	4.5	+1
Dependency-----	4.5	0	4.5	0	2.0	+3	3.0	+2
Drive-----	3.0	0	3.0	0	3.0	0	3.0	0
Hostility-----	5.0	-2	1.5	+3	1.5	+3	3.0	+1
Rigidity-----	1.5	+2	4.0	+1	4.0	+1	4.0	+1
Overall-----	5.0	+2	2.0	+7	1.0	+9	4.0	+3
Interview								
Age at reaction-----	4 or 5		8 or 9		-----	-----	-----	-----
Type of reaction-----	Sick		Nausea		None	-----	None	-----
Place of reaction-----	Car		Ocean		-----	-----	-----	-----
Overall susc. rank order-----	2		1 <sup>a</sup>		3	-----	4	-----
Vestibular Test								
Overall score-----	46		58		53	-----	36	-----
Rank order-----	3		1 <sup>a</sup>		2	-----	4	-----
Acrobatics								
Motion sickness-----	Nil		Nil		Nil	-----	Nil	-----

<sup>a</sup> Most susceptible.

means of transmitting power and electrical signals. The room contained laboratory equipment and living facilities. Despite the crowded conditions there was room to provide for relatively comfortable bunking, recreation (including television), and exercise. Particular attention was given to creature comforts, including the preparation of foods to suit individual tastes.

At the angular velocity of 10 rpm the centripetal force generated at different radii and the deviation of the gravito-inertial up-

right from the visual upright (direction of gravity) are given in table IV. This was the force environment to which the subject was exposed when motionless. Movements of the subject resulted in changes in the force environment caused in part by the generation of inertial forces and in part by variations in the level of centripetal force with changing distance from the center of rotation. Linear motions generated Coriolis forces, and rotary motions gyroscopic forces. The former which would affect the direction and

Table IV.—*Certain Parameters of the Force Environment in the Slow Rotation Room with an Angular Velocity of 10 RPM*

Radius, ft	Centripetal force, G units	Gravito-inertial force, G units	Gravito-inertial upright angle $\phi$	Coriolis force in % of centripetal force <sup>a</sup> with man walking at:	
				1 ft/sec	2 ft/sec
2	0.0682	1.0021	3°54"	95.4	190.9
3	.1023	1.0050	5°50"	63.6	127.2
4	.1364	1.0092	7°46"	47.7	95.4
5	.1705	1.0144	9°41"	38.1	76.2
5.5	.1876	1.0179	10°37"	34.7	69.4
6	.2046	1.0207	11°34"	31.8	63.6
7	.2387	1.0281	13°26"	27.3	54.5

<sup>a</sup> This would be "in % of apparent weight" in an orbiting rotating spacecraft.

magnitude of the resultant linear force vector (gravitoinertial upright) were not large, and a few "instantaneous" values are given in table IV. Gyroscopic forces generated by simultaneous rotations about two axes were extremely small and are significant only when discussing rotary motions of the head and stimulation of the semicircular canals.

The changes in the total force environment caused by the motions of the subject were incredibly complex. They were not measured, but an estimate of their principal effects was obtained in terms of the subject's movements. It was convenient to distinguish between two types of motions of the subject even though they were not always separate, namely, rotary movements of the head out of the plane of rotation of the room (head movements) and movements of the body which affected the linear force vector (body movements).

The head movements resulted in an unusual or bizarre stimulation of the semicircular canals and were the essential factor in causing canal sickness although additional etiological factors also were operating. The number of these head movements depended on voluntary restriction, imposed by the subject, and activities with which they were inescapably associated. Each subject was fitted with a modified orthopedic collar which, when worn, greatly minimized head tilt with reference to the thorax.

Body movements were associated with changes in the linear force vectors, and the disposition of the laboratory equipment and living facilities was such that the subjects carried out most of their activities in a zone varying in radius from 2.0 to 5.5 feet (table IV). These changes increased the difficulty in maintaining postural equilibrium, thereby adding to the muscular work which was also augmented by the increased gravitoinertial force. In the absence of head movements this was not an important factor in causing canal sickness.

Although the lack of precise measurements of the force environment was a handicap,

nevertheless past experience revealed that the movements of subjects, unless restricted, tended to be sufficiently constant so that the severity of symptoms was directly related to the velocity of rotation (refs. 1 and 2). Thus if deviations from the usual pattern of activities are noted, a rough measure of the change in stressful stimuli is afforded. Nearly always this was estimated in terms of restriction in head or body movements.

The methods used in studying the time-course of responses to stress are conveniently though somewhat arbitrarily grouped under three headings: clinical symptoms and signs, clinical laboratory findings, and psychophysiological measurements. Here, only a general description will be given; the necessary details will be added below when discussing specific tests.

A record was kept of body weight, oral temperature, pulse rate, and blood pressure. Each subject kept his own log in which he described the changing subjective symptomatology. The onboard experimenter kept a record of the significant manifestations of canal sickness in all subjects and their extrinsic behavior in terms of daily activities and interpersonal relations. Moreover, he recorded his own experiences which were of particular interest in that he was active during the brief periods when the room had to be stopped for experimenters to go aboard or leave; in other words, he was intermittently adapting to a stationary and rotating environment. Electrocardiograms were obtained and tilt tests conducted throughout the entire experiment. In measuring adaptation during rotation much reliance was placed on the Coriolis oculogyral illusion (ref. 4) and the "walking test" (ref. 11). Pre- and post-rotation tests included the Graybiel-Fregly ataxia test battery described in a preceding paper and the nystagmic response to head movements while the subjects were rotated clockwise and counterclockwise at 10 rpm, utilizing the Stille-Werner chair. Three visual tests were included: critical flicker fusion, visual fields, and ocular imbalance.

Biochemical measurements were designed

to study water balance, acid-base balance, intestinal absorption, excretion of electrolytes, release of stress hormones, glucose metabolism, and changes in certain serum enzymes. The basic composition of the diet was about the same for each man for each day with fluids recorded as taken. The conditions of the experiment precluded weighing of individual portions; however, these were estimated as closely as possible. All excreta including feces, urine, and vomitus were collected. The period of collection extended from 0800 one day through 0800 the following. There were four urine collection periods: 0800-1300, 1300-1800, 1800-2300, and 2300-0800. Collections began 3 entire days prior to rotation. Fasting blood specimens were collected at 0900 each day of sampling to avoid diurnal variations, and all of the chemical measurements were made on the plasma.

Performance measures included hand dynamometry, spoke test (eye-hand coordination), tracking test, speed of tapping, reaction time, time estimation, digit span, complex counting task, reading and mathematics tests, and four "vigilance" tests.

#### Routine

The experiment fell naturally into three periods, namely, prerotation, perrotation, and postrotation. The "day" extended from 0900 to 0900. When necessary to avoid confusion, three designations are used. For example, from 0900 to 0900 is termed the "entire day," from 0900 to 2400, "Day 1," and from 2400 to 0900 "end of Day 1." The prerotation period was approximately 4 days and numbered backward minus four (-4) through minus one (-1). This was a period for practice and for obtaining baseline measurements. The latter were obtained mainly on Days -2 and -1 when the four subjects and one on-board experimenter remained in the SRR. The perrotation period was 12 days, numbered 1-12. At 0900 on the morning of Day 1, after the final prerotation tests were completed, the room was set in motion at 10 rpm and, except for three short stops daily, continued to rotate for the 12 days. During these stops the subjects sat with their heads fixed in

order not to lose their adaptation. The daily schedule of events was as follows: 0745 awakened; 0800 brief stop to take experimenters aboard; 0900 stop, experimenters offboarded; 0905 light breakfast; 0910 morning testing program; 1130 brunch and relaxation; 1230 afternoon testing program; 1600 begin "free" period; 1820 stop for evening specimens; 1830 dinner; 2030 begin night watch.

The postrotation period began at 0900 on Day +1. On cessation of rotation certain tests were conducted outside the SRR after which the subjects returned and remained in the room for 2 days.

## RESULTS

### Clinical Symptoms and Signs

With the sudden onset of rotation all the subjects immediately experienced difficulty in walking and in carrying out tasks involving bodily movements. The full impact was not felt at once, typical symptoms of canal sickness appearing only after a delay. LI experienced only mild symptoms referable to the nausea syndrome, probably due in large part to the great restriction of head movements which he imposed from the early minutes of the run. He did not vomit but lost two pounds during Day 1 because of nausea and consequent food and fluid restriction. He slept at every opportunity during Days 1 and 2 and fell asleep during his "watch" on end of Day 1. He regained his appetite on Day 3.

MO was the first to become sick and in retrospect he wished he had restricted his head movements earlier than he did. The first of his eight vomiting episodes occurred within 35 minutes and the last during the evening of Day 2. He carried out the performance tests but minimized all other activities. There was a weight loss of six pounds during Days 1 and 2 which was regained by Day 8. He discarded the head brace on Day 3 and that evening his appetite had returned. Thereafter he did not restrict his head movements but continued to restrict body movements.

SH experienced typical symptoms of canal

sickness within the first hour and vomited once the afternoon of Day 1. He wore the brace on Day 1, restricted head movements through Day 2, and appetite returned by the evening of Day 3. There was a weight loss of 3 pounds which was regained by Day 4.

WI experienced slight nausea, and this was limited to the first 3 hours. He did not wear the brace but restricted head movements through Day 2. He was the only one of the four subjects to gain weight during the early perrotation period; he gained 2 pounds during Days 1 and 2 but lost 1 pound during Days 3 and 4.

Even after symptoms of nausea and anorexia disappeared and no further head restrictions were enforced, all of the subjects continued to experience drowsiness and fatigue and to restrict their physical activity which in turn minimized their head movements. LI had his "first real desire" to work on Day 5. During the remainder of the run he continued to nap occasionally during leisure periods and complained in the morning of excessive drowsiness and fatigue, even after 8 or 9 hours of sleep. The feelings of drowsiness and fatigue in MO, although less prominent after Day 4, persisted throughout the run. On Day 6 he "exercised a little" for the first time, but his comment on Day 7 was "tired again, feel normal though." Leisure time was occupied either with activities that required little mental effort or by resting or sleeping. SH had "no desire to work" on Day 4 and slept during the late afternoon and evening of Day 5. On Day 6 he was in "good spirits," and his first desire for exercise was satisfied with "a few push-ups." With the exception of Day 8, his log contained references to fatigue, such as "very tired at end of the day" (Day 11) and "very tired despite more sleep" on the morning of Day 12. WI found it "difficult to stay awake" on Day 2; was "tired and sleepy" on Day 3; had a "good day" although "no desire to work" on Day 4. He complained of fatigue on Days 5 and 6 and on Day 10 stated "typical day, the tests are becoming tiresome and fatigue is a big problem."

None of the subjects had fully adapted to the experimental conditions by the end of Day 12. The common complaint was "fatigue," and although they were carrying out all of their assignments, the employment of their free time was directed toward rest and relaxation rather than toward things which required mental alertness or physical work. MO complained most and WI least, but the differences between the subjects were not pronounced.

Cessation of rotation created an impact but far less than at the start. The immediate effect was on neuromuscular coordination and was declared by ataxia which rapidly diminished during the first hour or two. Additional symptoms were mainly a mixture of excitement and pleasure on stopping and residual perrotation effects. None complained of nausea although SH experienced "stomach awareness" which usually precedes frank nausea. SH also complained of slight lightheadedness and was "very tired" in the evening of Day +1. MO was "impressed" with the effects on coordination but adaptation was "quicker than expected." Additional comments were: "not at all tired;" "we made the news." WI reported "no ill effects" and "after about one hour I could walk straight without difficulty."

#### *Cardiovascular Measurements*

The values for pulse rate and blood pressure obtained while the subjects were recumbent did not change significantly throughout the entire experiment. The maximum changes in these values on actively assuming a position of tilt 15 degrees from the upright are given in table V. With regard to pulse rate every subject manifested a significant increase ( $> 10$  beats) on every test throughout the entire experimental period. In only a few instances, however, were the increases significantly greater during the per- or post-rotation periods when compared with the maximal increase on either Day -2 or Day -1: SH on Days 11 and 12, and WI on Day +1.

Systolic blood pressure dropped in the single test carried out on each subject before



rotation, and in three of the four it was significant, i.e., greater than 10 mm Hg. During rotation, increases as well as decreases were observed. All the increases were meaningful; they occurred on Day 1 (LI); Days 7 and 8 (MO); and Days 2 and 3 (WI). Most of the decreases were salient ones but in only six instances were they significantly greater during than before rotation and all occurred on or after Day 5.

Diastolic blood pressure increased in the single test carried out before rotation and the increase was important in three of the four subjects; similar changes were observed on Day +1 in the three subjects on whom measurements were made. During rotation both increases and decreases in diastolic pressure were noted. Most of the significant increases occurred early and the decreases late in the perrotation period, but there were enough exceptions to minimize the significance of this trend. During perrotation, when comparison is made with prerotation periods, there were only five instances in which there was either a concomitant significant rise in pulse rate and significant fall in systolic or diastolic pressure or a concomitant fall in both systolic and diastolic pressures, all occurring on or after Day 5.

A standard 12-lead electrocardiogram was taken each day during the experiment, and there were no significant changes when com-

pared with the control tracings. Electrocardiograms (Lead 2) obtained supine and during tilt-up revealed only minor changes in addition to variations in heart rate. All four subjects demonstrated an increase in P wave on assuming the upright position, and this was present on all tracings before, during, and after the rotation. The maximum T wave changes are shown in table VI. Subjects LI, MO, and SH demonstrated a decrease in T wave amplitude of 1.5 mm in Lead 2 during the tilt test. This finding was present during the control test as well as during the entire perrotation period. Subject WI showed the most striking changes during the tilt test, his ECG having S-T segment depression of 0.5 to 1.0 mm and negative T waves, but these changes were present before rotation and did not change perrotation. The R wave amplitude was included to evaluate relative as well as absolute changes in the T wave.

#### *Tests of Postural Equilibrium and Ataxia*

These fell into two categories: the regular test battery used in comparative measurements before and after rotation and a modified version for use under the restrictions imposed in the SRR.

Two tests were carried out daily in the perrotation period. In the first test the subject was required to stand heel-to-toe with eyes closed and arms folded in front of him

Table VI.—*Maximum T Wave Changes*

Subject	Position	Amplitude of T, mm												
		Day -2	Day -1	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 11	Day 12	Day +1
LI	Supine	4.5	5	4.5	3	4	4	4.5	4	4	3.5	4	3.5	4
	Tilt	3	3	3	2	2.5	2	2	2	2	2	2	2	2
MO	Supine	6	5			5	3.5	4	4	2	4	4	4	4
	Tilt	4	2.5			1.5	2	2	2.5	2	1.5	1	1.5	2
SH	Supine	4	4.5		4	3.5	3	3.5	3	3	3	3	3	3
	Tilt	2.5	3		3	2	1	3	2	2	2	2	2	2
WI	Supine	.5	1.5	2	1	.5	1	1	1.5	.5	1	.5	.5	1
	Tilt	-2	-1.5	-1.5	-2	-2.5	-2	-2	-1.5	-2	-1.5	-1.5	-1.5	-1.5

for 60 seconds, for two trials. If the 60-second criterion was not met, a third trial was given, and the score was the best two of three (with a maximum of 120). In the second trial the subject was required to walk five steps heel-to-toe (without deviating from line on the floor) from the wall to the center column with arms folded for two trials. If he did not complete five steps on each trial (score of 10), a third trial was given, and the score was the best two of three.

The scores for "walking" are summarized in figure 1. All of the subjects were able to achieve a maximum score prior to rotation. On Day 1 there was a decrement in performance, but the subjects improved on Day 2 and Day 3 and all were able to achieve a maximum score by the fourth day of rotation. Performance on the walking test was unchanged in two (LI and WI) and improved in two; the Stand Eyes Open Test performance was improved in all

The Graybiel-Fregly postural equilibrium tests administered on Day -7 and Day +3 were: (1) walking with eyes open on a  $\frac{3}{4}$  in. wide rail (8 ft long); (2) standing with eyes open on a  $\frac{3}{4}$  in. wide rail; and (3) standing with eyes closed on a  $2\frac{1}{4}$  in. wide rail (30 in. long). The subject was required to stand erect, or nearly erect with shoes on, arms folded against the chest, and feet in heel-to-toe position. The dynamic (walking) test was scored in terms of the number of correct steps, and the static (stand) tests were scored in terms of number of seconds without "falling" off the rail. The results of the tests are shown in table VII. Performance on the walking test was unchanged in two (LI and WI) and improved in two; the Stand Eyes Open Test performance was improved in all

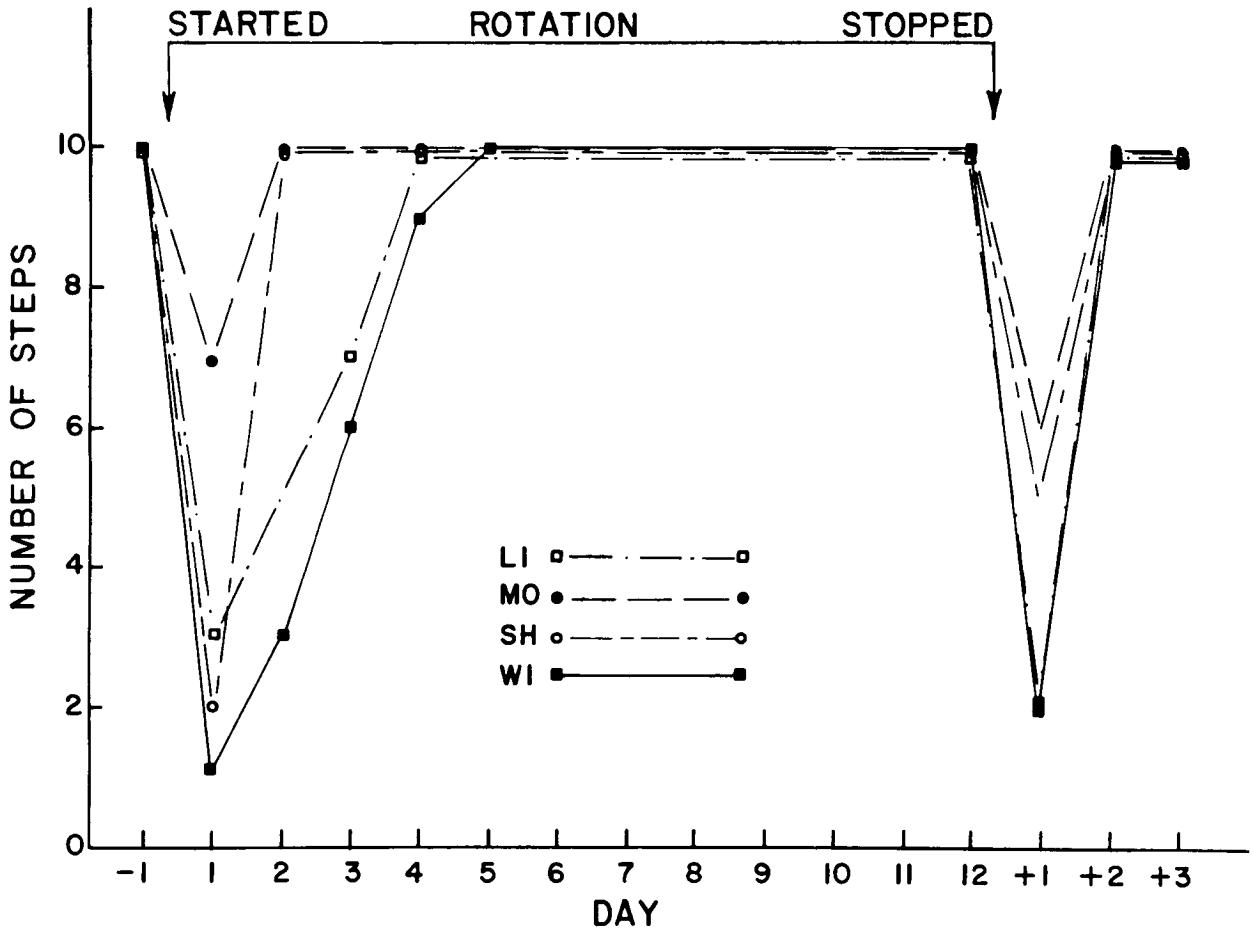


Figure 1.—Heel-to-toe walking scores for four subjects.



Table VII.—Results of Graybiel-Fregly Postural Equilibrium Test Before and After Rotation

Subject	Before rotation						After rotation						Rotation effects			
	Walking test	Per cent <sup>a</sup>	Stand eyes open test	Per cent	Stand eyes closed test	Per cent	Walking test	Per cent	Stand eyes open test	Per cent	Stand eyes closed test	Per cent	Walk	Stand open	Stand closed	
																Per cent
LJ	11	31	18	27	129	61	11	31	21	36	40	26	0	+	-	
MO	8	11	21	36	82	46	13	56	38	68	52	32	+	+	-	
SH	10	26	16	20	33	17	13	56	23	45	23	12	+	+	-	
WI	8	11	9	3	14	6	8	11	10	4	14	6	0	+	0	

<sup>a</sup> Percentile rank in a distribution of scores on several hundred normal males.

four subjects; the Stand Eyes Closed Test performance was unchanged in one subject (WI) but decreased slightly to markedly in the other three. It should be noted that initially the performance of all these subjects was at low average or below average on the two visually influenced tests (Walking and Stand Eyes Open) and that two of the subjects scored below average on the nonvisual influenced test (Stand Eyes Closed). The postrotation Stand Eyes Closed Test findings suggest that the subjects had not fully recovered from the effects of exposure in the SRR.

#### *Coriolis Oculogyral Illusion*

This was elicited by requiring the subject to tilt his head quickly (1.0–1.25 sec) toward the shoulder, and, after a delay, to return to the upright while fixating the dimly illuminated outline of a box in the dark. The scoring consisted of the subjective perception of apparent movement estimated in inches, and the direction of the movement. The results are summarized in table VIII.

Despite the fact that this illusion is difficult to quantify and that, with continued elicitation it appears to diminish even in one who is not well adapted to a rotational environment, it remains a good indicator of a certain kind of vestibular adaptation (ref. 4). As may be seen in the table, on Day 1 the illusions experienced by LJ and WI were of relatively large magnitude and those of MO and SH, relatively small. At the next test period (Day 3) the illusion had disappeared for MO and SH, was negligible for WI, but prominent for LJ. On Day 6 and Day 10 the illusion was absent for all men.

When the test was repeated after cessation of rotation, the stimulus to the canals was angular acceleration in the frontal plane, a normal stimulus, and perception of the illusion was evidence of a conditioned response. This was perceived by all subjects except SH. MO reported almost a perfect reversal both in terms of magnitude and direction, implying that the perrotation adaptation was in the nature of a compensatory phenomenon (refs. 3 and 4). LJ and WI also reported ap-

Table VIII.—*Magnitude of the Coriolis Oculogyral Illusion*

Day	Subject	Head left	Return	Head right	Return
1	LI	7 ↑	7 ↓	9 ↓	0
	MO	2 ↑	3 ↓	2 ↓	2
	SH	3 ↑	3 ↓	3 ↓	3
	WI	18 ↑	18 ↓	12 ↓	12
3	LI	12 ↑	18 ↓	12 ↓	0
	MO	0	0	0	0
	SH	0	0	0	0
	WI	0	0	1 ↓	1 ↑
6	LI	0	0	0	0
	MO	0	0	0	0
	SH	0	0	0	0
	WI	0	0	0	0
10	LI	0	0	0	0
	MO	0	0	0	0
	SH	0	0	0	0
	WI	0	0	0	0
+1	LI	4 ↑	6 ↓	3 ←	5 ↓
	MO	2 ↓	4 ↑	3 ↑	4 ↓
	SH	0	"0 →	"0 ←	0
	WI	6 ↓	6 ↑	0	2 ↑

<sup>a</sup> No apparent displacement—just velocity.

parent movement, indicating conditioned responses but not always in the opposite sense.

#### *Nystagmography*

Rotation tests, with subjects seated upright in a Stille-Werner Chair, were conducted just before and after the 12-day run. Nystagmus elicited by head movements was recorded during clockwise (CW) and counterclockwise (CCW) rotation of the chair at 10 rpm. Subjects were instructed to carry out silent arithmetic computation throughout each rotation in an effort to induce artificial arousal. The tests after the 12-day run were given at three different intervals:

- (1) Post-Test I: Within the first 2 hours after the 12-day run, the men exhibited a greatly reduced nystagmus and no subjective reaction to recorded head movements during rotation in the accustomed direction of rotation (CCW); in the unaccustomed direc-

tion of rotation (CW), the average nystagmus and subjective reactions elicited by head movements were of about the same intensity as they had been prior to the 12-day period of rotation. In some cases, responses during clockwise rotation were greater than their prehabitation counterparts. This was probably attributable to a conditioned compensatory nystagmus which had been detected by static tests given 5 to 30 minutes after the prolonged rotation.

- (2) Post-Test II: Two days after the 12-day rotation, subjects were again given 10 rpm tests on the Stille-Werner chair. In the interim period (about 46 hours) they were not exposed to rotation but participated in other tests which required them to remain active throughout each normal work day. In the unaccustomed rotation direction, nystagmus elicited by head movements during Post-Test II generally was less than responses to equivalent stimuli in the prehabitation tests, and it was also less than that elicited during Post-Test I. In the accustomed rotation direction, nystagmus had recovered, although not to its initial level. Reports of sensation indicated a similar course. Hence, the accustomed rotation direction still yielded the weaker responses, nystagmus and subjective, but the responses to head movements with the two directions of rotation had begun to equalize.
- (3) Post-Test III: Three weeks after the habituation run nystagmus output elicited by head movements was nearly equal for the two directions of rotation and was still less than the prehabitation level. Subjects indicated that the subjective effects had returned although comparisons with initial response intensities are obviously difficult due to the length of the intervening time.

Nystagmus produced by the acceleration and deceleration of the Stille-Werner chair showed little or no decline as a result of the 12-day run. Hence the habituation appeared to be specific to the bizarre vestibular stimulation of the concomitant rotation about two axes.

The amount of nystagmus produced during either the head tilt tests or the passive angular acceleration did not serve as a good indicator of the amount of sickness and apparent discomfort experienced by the four subjects. MO yielded the least nystagmus, yet showed the greatest neurovegetative

symptoms. However, the sample of only four subjects is not sufficient to test for the possibility of a low positive correlation between nystagmus output during initial testing and susceptibility to motion sickness.

*Visual Tests*

*Critical Flicker Frequency.*—Monocular (right eye) flicker thresholds were determined with a standard CFF measuring device (Ivy-Krasno) in which the test field, viewed against a dark background, subtended a visual angle of approximately 11 minutes of arc. With the use of a method of limits, three

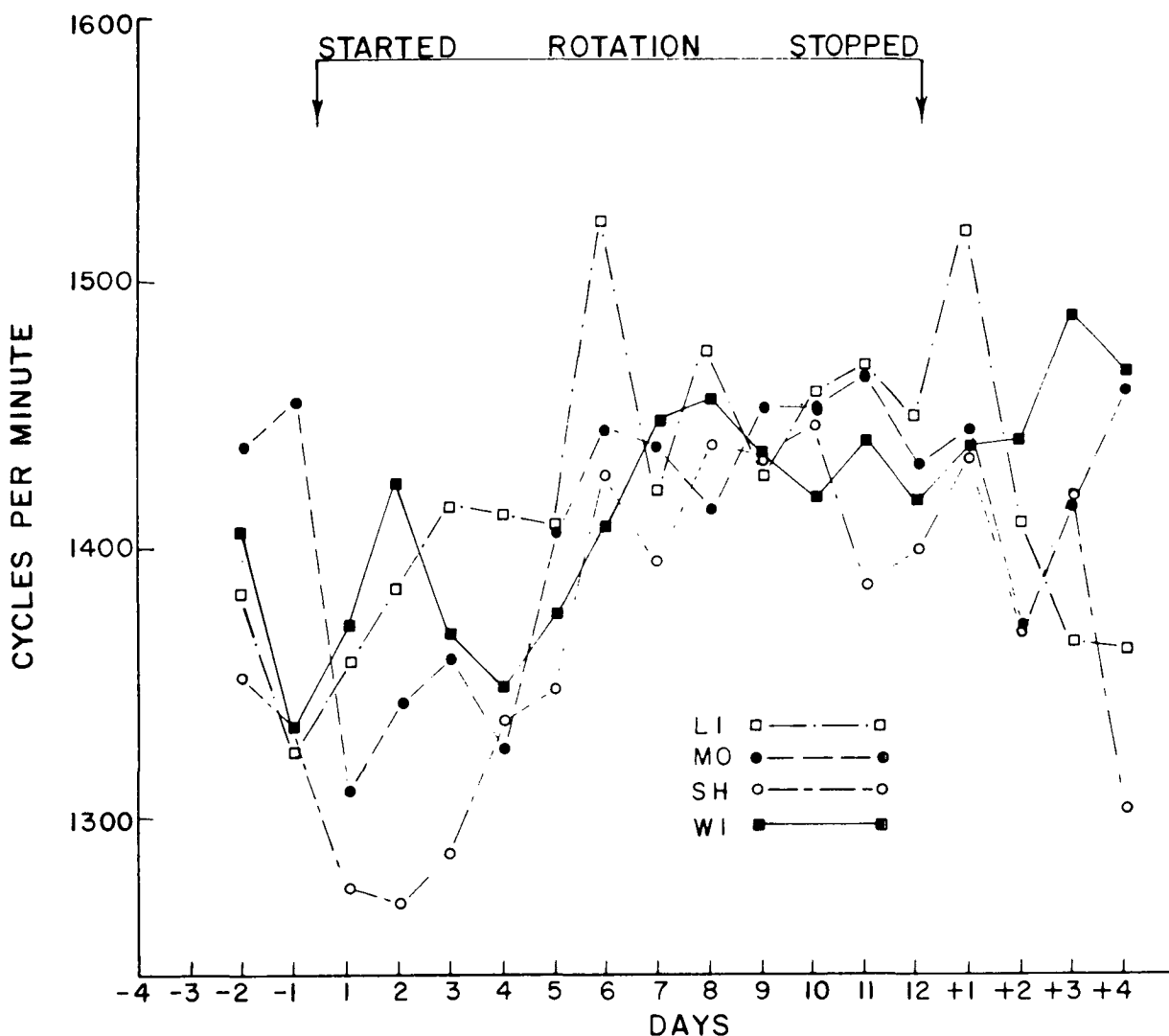


Figure 2.—Critical flicker frequency for four men.

ascending and three descending thresholds of fusion and flicker, respectively, were determined daily for each subject. The average of these six values served as the indicator of CFF for each day tested (fig. 2). Two of the subjects (MO, SH) revealed a significant decrease in performance relative to initial control levels during the early perrotation period. This apparent decrement in visual function lasted for about 3 to 4 days, after which time a gradual yet complete recovery took place. The other two subjects (LI, WI), on the other hand, who manifested fewer symptoms, had an apparent increase in CFF during the rotational phase of the experiment. Following rotation the CFF level decreased to its basic, prerotational level in subject LI but not in subject WI.

*Visual Fields.*—The visual field of the right eye of each subject was explored daily in the

classical way, using a meter tangent screen. Detection of a red (2/1000) and blue (2/1000) test-object was recorded in each of four meridians (vertical, horizontal, and the two intermediate). The data were plotted on standardized forms, and the areas contained within the isopter lines for red and blue were measured with a polar planimeter. The resultant relative areal size served to indicate overall visual field sensitivity for a given test period.

The results obtained with the red (2/1000) and blue (2/1000) target correlated satisfactorily, indicating that there were no selective visual field changes in the regions tested. In general, the average overall size of the visual field (fig. 3) remained essentially at the same level. Variability on this test appeared to be directly related to the symptomatology of motion sickness; i.e., the subjects who ex-

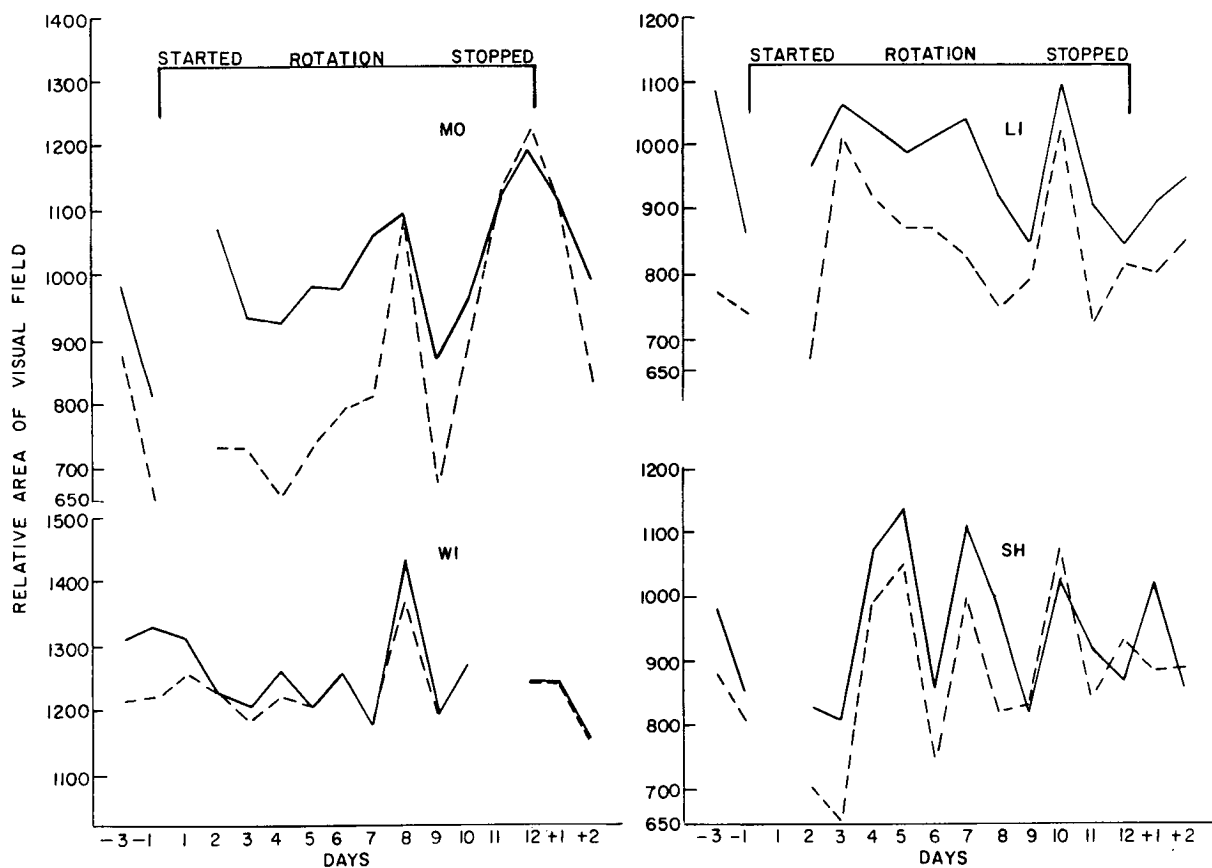


Figure 3.—Peripheral fields for four men.

hibited the most symptoms (viz., 8 and 1 vomiting episode, respectively) had the greatest fluctuations.

*Eye Muscle Imbalance.*—The near (40 cm) heterophoria of each subject was determined daily by a modified Von Graefe technique. The stimulus to accommodation was controlled by requiring the subject to resolve a vertical row of 20/20 Snellen letters while he alined vertically the double image of this target. The surroundings of the target were free from fusional cues. Fixation of the head using a dental bite device prevented any movement. As revealed in figure 4, there were slight day-to-day fluctuations in the latent deviation manifested by each subject. These variations were not unexpected; the important general finding is that there were only small changes in magnitude (and therefore direction) of the

habitual muscle imbalance as a result of prolonged rotational stimulation.

*Discussion of Clinical Findings*

Three major periods were distinguishable in terms of the manifestations of stress: (1) the early perrotation period characterized by ataxia and the nausea syndrome, (2) the late perrotation period characterized by the fatigue syndrome, and (3) the post-rotation period characterized by ataxia. In addition, it is sometimes convenient to distinguish the periods immediately associated with the onset and cessation of rotation.

The sudden transition from the stationary to the rotating environment had an immediate and maximal impact on bodily activities involving neuromuscular coordination. This was emphasized by the initial period of free-

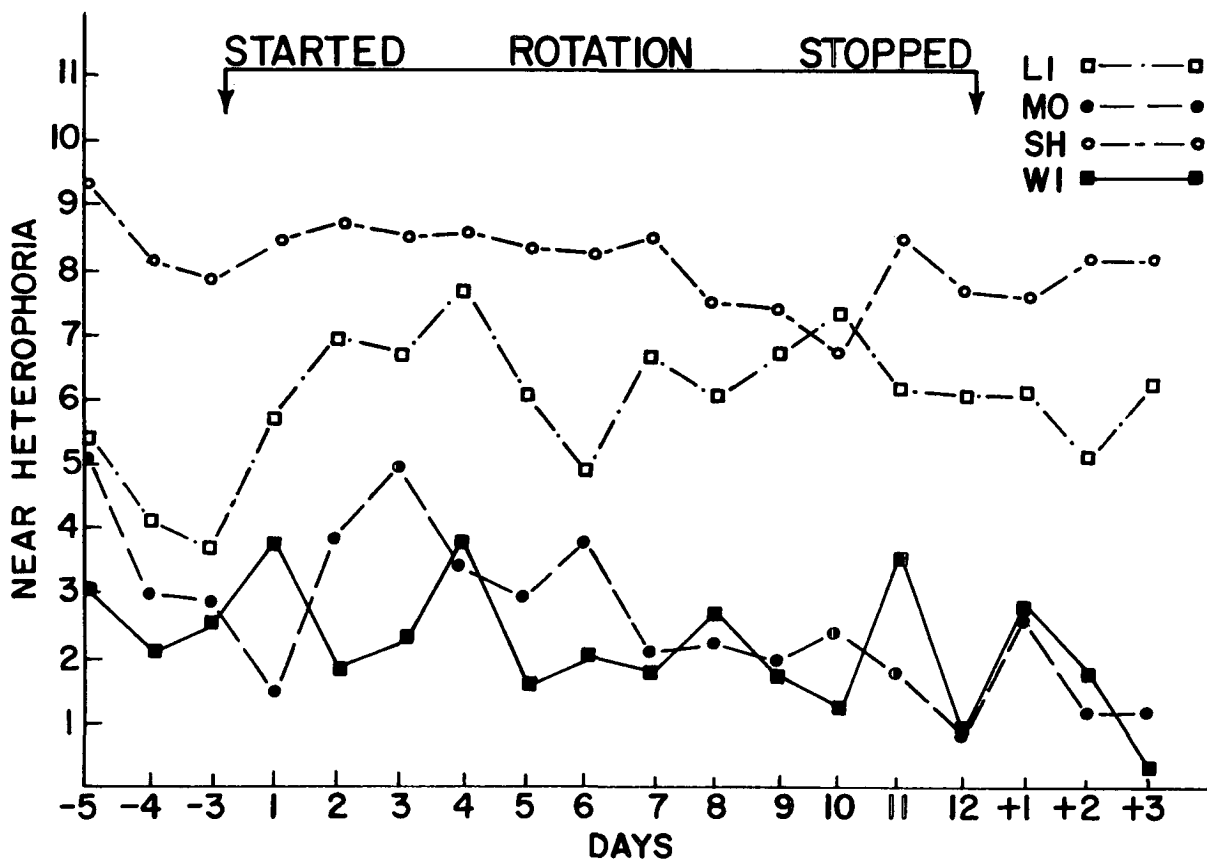


Figure 4.—Muscle imbalance for four men.

dom from canal sickness which encouraged all except LI to move about until voluntary restrictions were imposed. At that point all of the subjects were handicapped in varying degrees, either because of canal sickness or the self-imposed restrictions in bodily movements, or both. The cardinal symptoms were nausea and drowsiness, although additional complaints included dizziness, headache, sweating, anorexia, and general discomfort and fatigue. The time-course of summation and adaptation effects with regard to the nausea syndrome differed for each subject. It should be pointed out that while "subject-paced" stimulation is advantageous in terms of preventing symptoms, the "feed back control" is not perfect in the case of nausea which is characterized by a buildup that involves second order effects.

The subjects' rankings in terms of increasing severity of symptoms were WI, LI, SH, and MO. Their ranking in terms of susceptibility was complicated by the fact that the number of head movements, the essential stimulus, differed. MO and WI represented the two extremes in susceptibility and LI and SH fell in between. The vestibular test, which was a brief performance test, had the best predictive value. Acrobatics, something to which they were accustomed, did not discriminate among the four subjects. The interview was not very helpful in that only two had a history of motion sickness and there was only one episode in each case. The anxiety dimension in the Rorschach evaluation was the only item which ranked the subjects satisfactorily. It is worth noting that the slight depression of the right semicircular canal response indicated by the caloric test did not confer any benefit on MO.

With the disappearance of certain symptoms, particularly nausea, the subjects cautiously increased their "head movements," which marked the end of the early phase of adaptation. The symptomatology of motion sickness in general is so often dominated by the nausea syndrome that not only its disappearance but also concern over its re-

appearance seemed an important landmark in adaptation to the rotating environment; consequently, for descriptive purposes this period was given a special designation. It is necessary to point out that, except for a short time immediately after onset of rotation, this period was also characterized by physical inactivity which affected a number of physiological and psychophysiological parameters.

The lifting of restrictions on head movements increased the bizarre stimulation to the semicircular canals. This "increase" involved *number* of stimuli or movements, and, insofar as this increase affected velocity of head movement, it was equivalent to increasing the rpm of the room, i.e., intensity of stimulation. Head movements associated with bodily movements were still reduced because the latter were restricted. The reason for this restriction was the apathy, drowsiness, and fatigue which characterized the subjective symptomatology. These symptoms were present in the early perrotation period but were overshadowed by the nausea syndrome. This suggests that they were not simply the result of confinement and boredom. With increasing length of exposure the total stimulus situation was complicated by the time-dependent cumulative effects of boredom and confinement as well as those of the force environment. In retrospect, it is unfortunate the experiment was not continued until the men either were fully adapted or had demonstrated in what respects they would not fully adapt. Their best day was the last, Day 12, although the anticipation of return to a stationary environment may have been a factor.

Cessation of rotation had a mild but distinct impact characterized mainly by ataxia especially when turning. All were surprised at feeling a little strange in the stationary environment. Other new symptoms were minimal and far less than in many instances when subjects left the SRR after short exposures.

The total clinical picture was complicated by the residual effects both of long confine-

ment and perrotation stress. In general, the symptoms appearing after rotation were neither severe nor long-lived and constituted an elegant demonstration of our habituation to the stationary environment.

The only positive cardiovascular findings were the alterations in pulse rate and blood pressure manifested during the tilt test. It is important to emphasize two shortcomings in carrying out the test in the SRR; the subject was not passively tilted and the tilt period was only 5 minutes. The former shortcoming and the fact that the subjects were relatively inactive for at least 4 days prior to rotation are the only explanations for the unexpectedly large increase in pulse rate and decrease in systolic blood pressure manifested before the onset of rotation. A comparison of these values obtained on Days -1 and +1 reveals surprisingly few significant differences. Allowance must be made for not carrying out the tilt test during rotation when MO (Days 1 and 2) and SH (Day 1) were sick which minimizes the significance of group trends on these days. The most significant change was in diastolic blood pressure although the downward trend was erratic. All the observed instances of mild orthostatic intolerance occurred in the later perrotation period.

The results of the ataxia test carried out in the SRR were clear-cut which was similar to what was found in previous experiments (ref. 11). The uniformity of the subjects' rate of adaptation, all reaching their pre-rotation level on Day 4, contrasts with their differing rates of adaptation to the nausea syndrome and the oculogyral illusion. Unfortunately, the results provide no information regarding either the several etiological factors involved or the mechanisms of adaptation. The findings in the tests carried out before, and 3 days after, rotation are of interest. The improvement in the scores in tests carried out with "eyes open" might be explained by the fact that "learning" was a more important factor than "residual ataxia." With "eyes closed" the residual ataxia was now a more important factor

than learning. Stated differently, the role of vision was very great and only with eyes closed was it possible to elicit the fact that postrotation adaptation, in the case of ataxia, was incomplete.

The Coriolis or gyroscopic oculogyral illusion is a unique, immediately evoked response to which subjects readily adapt (ref. 4). They adapt so readily, in fact, that an "order effect" may be demonstrable with successive tests on a single occasion. It is interesting that LI, who greatly minimized head movements, was the only subject who had not adapted by Day 3. The close relationship of the illusion to the nystagmus response is shown in the similarity of the mechanisms underlying adaptation (ref. 3). In both cases in this experiment there was evidence of perrotation adaptation and a conditioned compensatory response postrotation.

With regard to the visual tests, the only significant changes observed involved the flicker fusion test. In the early perrotation period MO and SH manifested significant temporary decreases in performance and in the late period all except MO demonstrated an increase. The decreases were consistent with the fact that MO and SH experienced more severe symptoms than the other two subjects, and the question arises whether this was a specific response to stimulation of the semicircular canals or a nonspecific effect. Simonson, Fox, and Enzer (ref. 12) found that thermal stimulation of the semicircular canal using 5 cc of water at a temperature of 70° to 76° F caused a depression in CFF in 16 normal subjects, and they implied at least that it was a specific effect. Our failure to demonstrate a decrease in CFF in two subjects is readily explicable if the subsequent increases in the later perrotation period are regarded as practice effects which were delayed in their appearance by the depressing effect of vestibular stimulation. It is noteworthy that in the later period the only increases in performance occurred despite increases in head movements and symptoms of fatigue.

Whatever the explanation for the changes in CFF observed, they are of interest in demonstrating the readiness with which the measurements can be influenced.

#### Clinical Laboratory Findings

##### *Urine Volume and Electrolytes*

The findings are summarized in table IX and figure 5. There were significant individual differences in patterns of urinary output, although in the early perrotation period all subjects showed a decrease followed by an increase and all except possibly SH showed a second decrease in the later period. No other observations on urine, including specific gravity, pH, and total solids, reflected any variations not accounted for merely by changes in volume per collection period.

The changes in urinary excretion of sodium, potassium, and chloride during each

collection period were essentially parallel for all four subjects as shown in table IX. The changes in excretion of sodium were greatest (fig. 6) and reflect the greater severity of symptoms in MO and SH. Urinary calcium and phosphate excretion varied throughout the experiment with a clear tendency toward a decrease in excretion associated with nausea and vomiting.

##### *Microscopic Examination of Blood*

Hematocrit determinations and blood findings are shown in table X. Hematocrit values varied within a narrow range. There were no significant variations in the WBC counts, although mean value was lowest on Day 4 and a tendency toward a decrease in lymphocytes was manifested in the case of MO.

##### *Total Protein and Acid-Base Values*

Total protein was determined using the refractive index method, and the results are

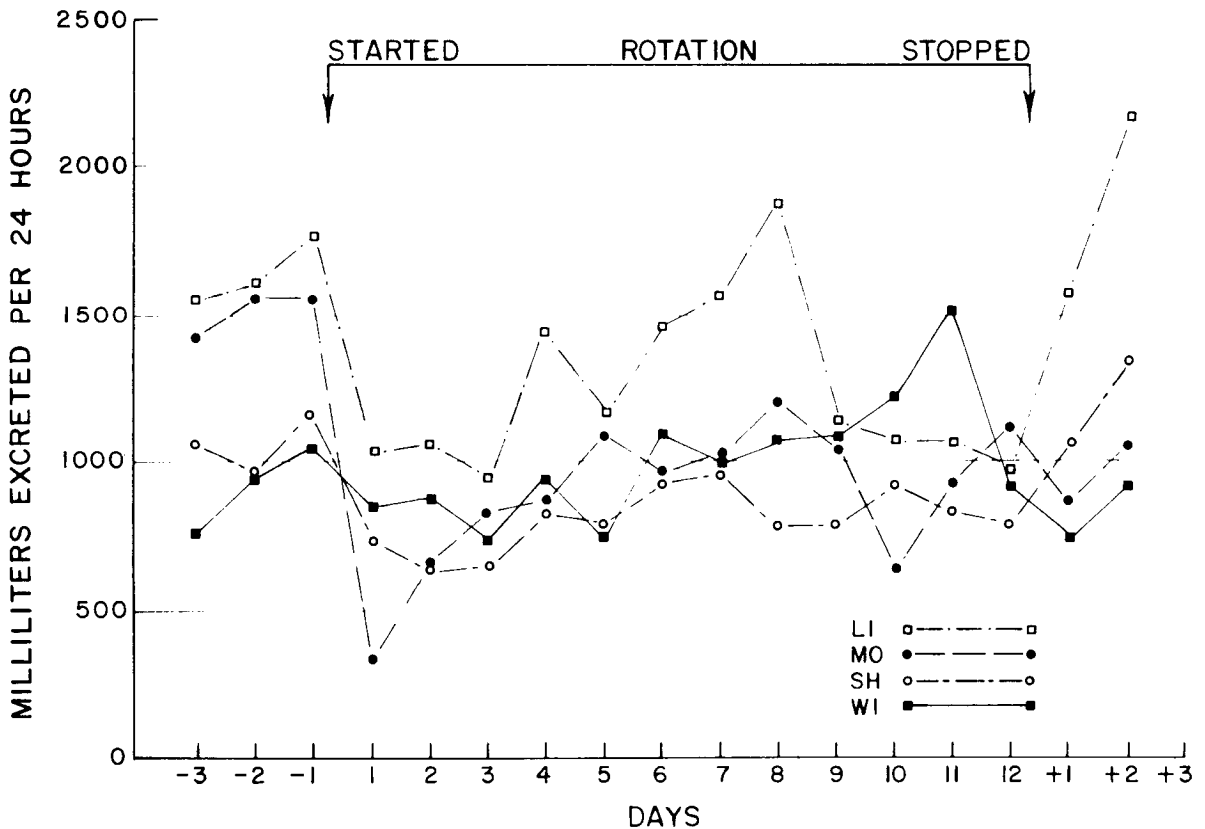


Figure 5.—Urine volume—twenty-four hours—for four men.



Table IX.—Urine—Excretion of Ions

Determination	Sub-ject	Day of experiment																		
		-3	-2	-1	Mean	1	2	3	4	5	6	7	8	9	10	11	12	Mean	+1	+2
Volume, ml	LI	1573	1605	1787	---	1020	1068	957	1443	1178	1485	1565	1895	1137	1075	1071	988	---	1585	2180
	MO	1426	1590	1570	---	325	675	812	865	1100	985	1010	1202	1063	613	945	1110	---	863	1078
	SH	1038	987	1192	---	765	618	633	825	795	937	969	788	797	931	818	796	---	1086	1445
	WI	780	985	1015	---	820	882	720	950	784	1100	1010	1097	1093	1202	1528	907	---	735	910
Sodium, meq/24 hr	LI	239	209	210	219	138	190	113	129	126	158	97	219	---	136	155	173	149	421	227
	MO	166	270	177	204	41	32	61	61	174	112	119	165	140	161	208	201	123	137	149
	SH	147	168	194	170	63	55	75	119	96	171	143	116	131	190	165	163	124	262	155
	WI	115	192	189	165	149	186	120	159	153	189	181	168	168	179	203	120	165	138	153
Potassium, meq/24 hr	LI	82	110	93	95	100	85	65	93	95	87	66	133	---	95	89	99	92	136	83
	MO	44	86	109	80	41	46	61	54	91	80	64	71	73	95	79	94	71	70	60
	SH	60	69	80	70	74	31	47	61	66	83	67	60	68	88	70	79	66	91	79
	WI	53	82	98	78	87	74	63	69	85	71	61	65	69	70	84	77	73	40	64
Chloride, meq/24 hr	LI	207	159	218	195	137	186	130	138	148	140	---	163	---	127	166	152	149	150	305
	MO	155	237	157	183	54	45	74	50	140	120	108	167	129	166	206	169	119	122	---
	SH	174	177	192	181	85	50	74	122	117	162	122	118	125	149	164	125	118	189	197
	WI	139	165	216	173	146	153	125	168	156	183	166	154	166	153	196	129	158	123	145
Calcium, meq/24 hr	LI	5.9	7.6	7.5	7.0	7.8	7.9	7.8	7.6	4.9	6.7	---	10.7	---	9.1	9.5	7.5	8.0	10.6	11.0
	MO	10.1	8.6	8.9	9.2	0.8	12.1	8.3	8.2	6.2	6.4	10.3	10.7	9.1	9.4	8.3	9.3	8.3	12.3	---
	SH	2.2	3.2	3.4	2.9	2.6	6.2	4.3	7.6	3.2	6.8	7.1	6.5	7.7	9.5	9.0	6.5	6.4	7.1	9.6
	WI	7.0	7.8	9.1	8.0	7.8	8.1	9.2	12.4	6.6	11.1	16.2	14.2	8.7	9.1	11.7	7.5	10.2	7.6	9.3
Phosphate, mg/24 hr <sup>a</sup>	LI	105	94	115	105	110	125	116	118	81	103	---	91	---	140	110	95	109	122	173
	MO	102	112	108	107	25	115	63	84	74	75	90	95	106	112	90	117	87	101	---
	SH	113	150	110	124	105	74	102	124	79	123	122	94	125	120	96	86	104	87	193
	WI	103	130	120	118	103	110	105	130	84	108	127	125	116	128	154	104	116	90	112

<sup>a</sup> The "phosphate" excretion must be expressed as phosphorus in mg per day with the last digit left off. Normal excretion is 1000 to 2000 mg per day, mean 1100.



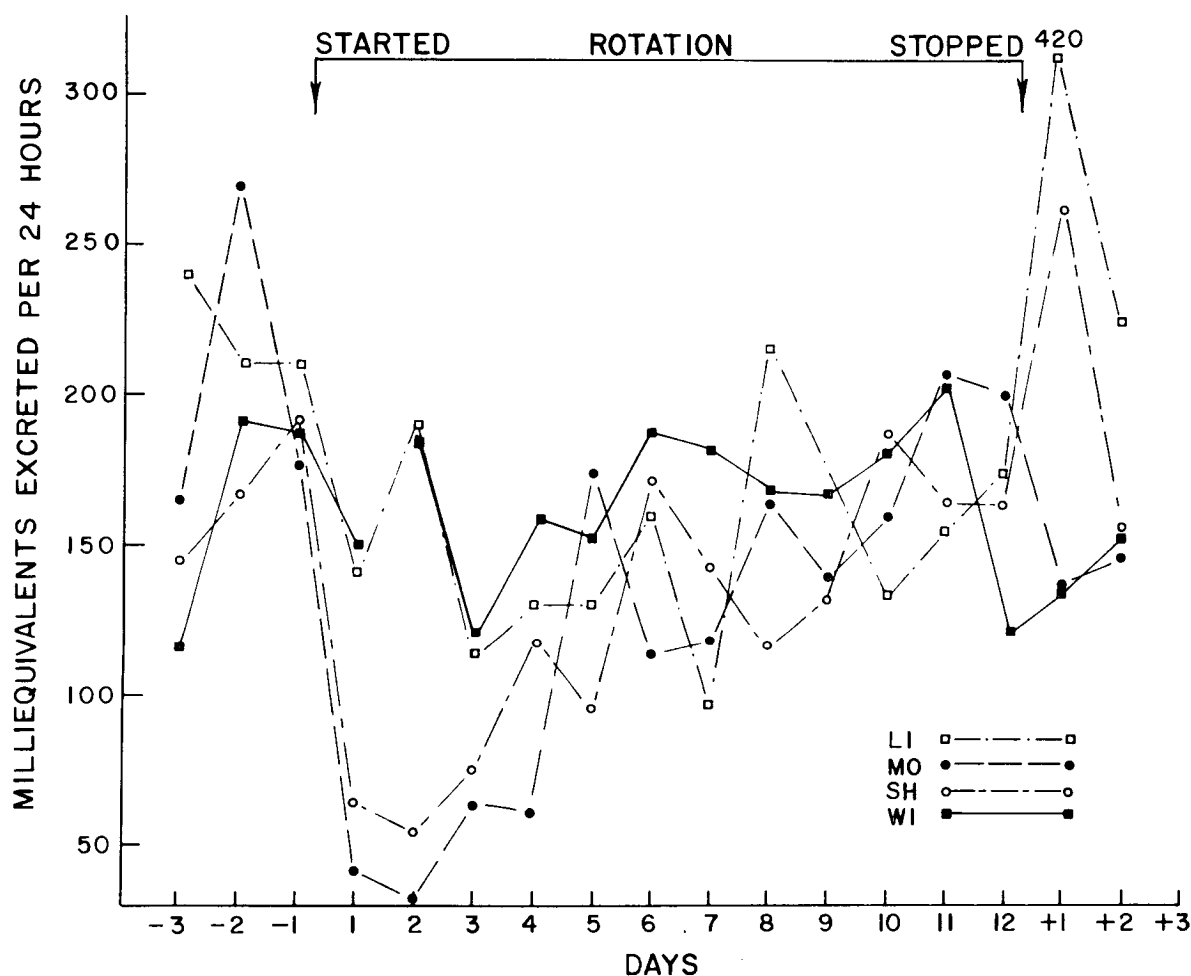


Figure 6.—Urinary excretion of sodium of four subjects.

shown in table XI. None of the variations throughout the experiment were significant.

Serum pH was measured on venous blood using the Beckman pH electrode. Nearly all of the values were consistently in the low normal range (normal 7.36–7.46) or slightly below. The fact that distinct patterns did not emerge suggests technical difficulties.

Serum  $p\text{CO}_2$  was measured using the Clark electrode. Some of the obtained values (table XI) were outside the normal range (47–54), and, in the case of MO, a clear tendency toward alkalosis is seen in the early perrotation period while, in the other subjects, some values indicate slight acidosis.

Serum sodium and potassium were measured using a Technico autoanalyzer flame

photometer with a lithium internal standard; chloride was determined using an ultramicromodification of the method of Schales and Schales (ref. 13). The values obtained in table XI were within the normal range and did not change significantly throughout the experimental period. Serum calcium was measured using an ultramicro-modification of the method of Diehl and Ellingboe (ref. 14) and phosphorus (inorganic) by the method of Fisk and Subbarow (ref. 15). The results are included in table XI. The values for calcium tended toward the lower end of the normal range (4.5–5.7) during Days 2, 3, and 8. Some of the values for inorganic phosphorus were above the normal range (2.4–3.8). There were slight tenden-

Table XI.—Total Protein Acid-Base Values (Serum)

Determination	Subject	Prerotat- ion <sup>a</sup>	Day of rotation										Mean <sup>c</sup>					
			2	3	5	8	12	Mean <sup>b</sup>	+1	+2	+3							
Total protein, gm/100 ml	LI	6.8	6.3	7.4	6.9	6.3	6.0	6.6	6.4	6.4	6.4	6.4	6.4	6.4	6.4	6.4	6.4	
	MO	7.2	7.1	7.0	6.7	6.9	6.9	6.8	6.9	7.0	6.6	6.6	6.6	6.6	6.6	6.6	6.6	6.6
	SH	6.8	7.1	6.9	6.7	6.8	6.8	6.8	6.8	6.8	6.8	6.8	6.8	6.8	6.8	6.8	6.8	6.8
	WI	6.8	7.7	6.9	6.9	7.5	7.5	6.6	7.1	6.8	6.4	6.4	6.4	6.4	6.4	6.4	6.4	6.4
pH	LI	M6.9	7.0	7.0	6.8	6.8	6.5	6.7	M6.7	6.4	6.4	6.7	6.4	6.4	6.4	6.4	6.4	6.4
	MO	7.33	7.33	7.31	7.30	7.34	7.49	7.35	7.36	7.36	7.33	7.35	7.33	7.33	7.35	7.35	7.35	7.35
	SH	7.33	7.35	7.32	7.37	7.32	7.37	7.36	7.35	7.36	7.36	7.35	7.36	7.36	7.36	7.36	7.36	7.36
	WI	7.33	7.35	7.27	7.33	7.31	7.34	7.34	7.32	7.36	7.31	7.32	7.31	7.31	7.31	7.31	7.31	7.31
CO <sub>2</sub> , pCO <sub>2</sub>	LI	M7.32	7.34	7.32	7.34	7.33	7.33	7.39	M7.35	7.33	7.33	7.34	7.33	7.33	7.34	7.34	7.34	7.34
	MO	46.2	49.0	55.5	54.6	49.2	50.2	50.2	51.7	47.8	47.5	47.5	47.5	47.5	47.5	47.5	47.5	47.5
	SH	48.0	52.1	52.8	47.3	49.5	46.8	49.5	43.5	52.8	49.5	49.5	49.5	49.5	49.5	49.5	49.5	49.5
	WI	49.1	48.0	62.7	45.3	56.2	49.1	51.1	54.0	49.7	53.7	57.5	57.5	57.5	57.5	57.5	57.5	57.5
N <sub>a</sub> , meq/L	LI	M48.1	45.7	52.9	49.3	52.5	49.9	49.9	M50.1	51.8	51.8	51.8	51.8	51.8	51.8	51.8	51.8	51.8
	MO	134	138	132	136	135	135	135	141	136	136	136	136	136	136	136	136	136
	SH	135	139	134	136	136	139	137	137	141	138	139	140	140	140	140	140	140
	WI	135	135	135	138	137	141	137	137	138	139	139	139	139	139	139	139	139
K, meq/L	LI	M133	136	133	137	136	138	138	M139	138	138	138	138	138	138	138	138	138
	MO	3.9	3.7	3.3	3.7	4.1	4.1	4.1	3.8	3.7	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5
	SH	4.5	4.2	4.4	4.0	4.2	4.3	4.3	4.0	4.1	3.8	3.8	3.8	3.8	3.8	3.8	3.8	3.8
	WI	4.4	4.2	3.9	3.9	4.2	4.2	4.5	4.1	3.9	3.6	3.9	4.1	4.1	4.0	4.0	4.0	4.0
CL, meq/L	LI	M4.1	4.0	3.9	3.8	4.1	4.3	4.3	M3.9	3.7	3.7	3.7	3.7	3.7	3.7	3.7	3.7	3.7
	MO	99	99	98	97	99	103	99	103	98	100	98	98	98	98	98	98	98
	SH	99	94	96	103	98	98	98	98	96	98	98	98	98	98	98	98	98
	WI	98	98	93	99	95	98	97	97	101	100	100	100	100	100	100	100	100
Ca, meq/L	LI	M9.9	9.7	9.6	9.9	9.6	10.0	9.9	M10.0	9.8	9.8	9.8	9.8	9.8	9.8	9.8	9.8	9.8
	MO	4.8	4.9	4.6	4.9	4.7	4.6	4.6	4.7	4.9	4.6	4.6	4.6	4.6	4.6	4.6	4.6	4.6
	SH	4.9	5.0	4.7	4.6	5.0	4.9	4.8	4.8	5.1	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0
	WI	5.1	4.7	4.8	5.1	4.4	5.1	4.8	4.8	5.1	4.9	4.9	4.9	4.9	4.9	4.9	4.9	4.9
Inorganic P, mg/100 ml	LI	M4.9	4.7	4.7	4.9	4.7	4.9	4.9	M5.0	4.8	4.8	4.8	4.8	4.8	4.8	4.8	4.8	4.8
	MO	3.7	4.0	3.8	3.4	3.5	3.3	3.3	3.6	—	—	—	—	—	—	—	—	—
	SH	3.7	3.6	3.4	3.4	4.0	3.8	3.8	3.6	3.5	3.1	3.1	3.1	3.1	3.1	3.1	3.1	3.1
	WI	4.3	4.1	3.8	3.1	3.9	3.4	3.4	3.7	3.7	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5
		M3.9	3.8	3.6	3.3	3.7	3.7	M3.8	3.8	3.3	3.3	3.3	3.3	3.3	3.3	3.3	3.3	3.3

<sup>a</sup> Mean of values, days -3, -2, -1. <sup>b</sup> Mean of values, 5 experimental days. <sup>c</sup> Mean of values, days +1, +2, +3.

cies toward lower values perrotation and on Day +2.

*Catechol Amines in Blood and Urine*

Measurements of catechol amines in the blood were done by the method of Weil-Malherbe and Bone (ref. 16), and the results are shown in table XII. With regard to adrenalin the single highest value was obtained on MO 3 days prior to rotation; this probably represents a response to drawing blood. There is a pattern evident with the lowest value between Days -2 and 3 and the highest values between Days 8 and +2. There were only minor variations in levels of noradrenalin.

Crout's method (ref. 17) was used to measure the urine catechol amine for each of the four collection periods, and all of the results were graphed. There were no significant changes in release of noradrenalin; the graphs (not reproduced) show much the same saw-tooth curves for each subject. Although the release of adrenalin was probably within normal limits, the individual variance was so characteristic for each that

the curves are depicted in figure 7. Note that the values for the "night" samples are similar for all and that the variance in values for "day" samples reflects differences in lability of response.

*Urine Corticosteroids (Mostly Cortisone, Hydrocortisone, and Tetra Derivatives)*

These corticosteroids were determined in each of the four collection samples by the method of Kornel (ref. 18), and the results graphed. It should be pointed out that there is a "lag" of 2 to 3 hours in excretion of the products of 17-hydroxycorticosteroids released. Figure 8 represents the mean values for each day and shows two trends. The first trend downward in the early perrotation period is not significant, but the second upward trend during the late period is highly significant.

*Glucose Utilization*

Thirty grams of dextrose were injected intravenously after an overnight's fast, and the subsequent decline of excess blood sugar values was determined for 1 hour on Days

Table XII.—*Blood Catechol Determinations*  
[Normal range: Adrenalin 0.2-0.6 µg/L; noradrenalin 2-6 µg/L]

Subject	Day of experiment										
	-3	-2	-1 <sup>a</sup>	2	3	5	8	12 <sup>c</sup>	+1 <sup>c</sup>	+2	+3
Adrenalin, micrograms per liter of serum											
LI	---	(b)	(b)	(b)	(b)	(b)	1.3	0.9	1.2	1.1	(a)
MO	1.8	(b)	(b)	(b)	(b)	0.3	0.4	0.1	0.2	1.1	0.2
SH	0.1	(b)	(b)	(b)	(b)	(b)	(b)	(b)	0.1	0.1	(b)
WI	0.3	(b)	(b)	(b)	(b)	(b)	0.3	(b)	1.1	0.1	(b)
Mean:	0.7	(b)	(b)	(b)	(b)	0.1	0.5	0.25	0.65	0.6	0.1
Noradrenalin, micrograms per liter of serum											
LI	---	6.2	4.3	8.7	5.7	4.0	7.3	5.1	5.7	7.1	3.5
MO	6.5	5.0	5.2	6.4	5.6	3.8	3.6	3.0	4.2	6.4	3.7
SH	2.7	2.8	2.3	2.4	2.9	3.4	4.2	2.6	4.5	4.3	3.5
WI	3.1	3.9	2.9	3.3	4.9	5.3	3.5	2.9	5.4	3.5	2.9
Mean:	4.1	4.5	3.9	5.2	4.8	4.1	4.65	3.4	4.95	5.3	3.4

<sup>a</sup> Run began 0900.  
<sup>b</sup> None detectable.  
<sup>c</sup> Run stopped 0900.



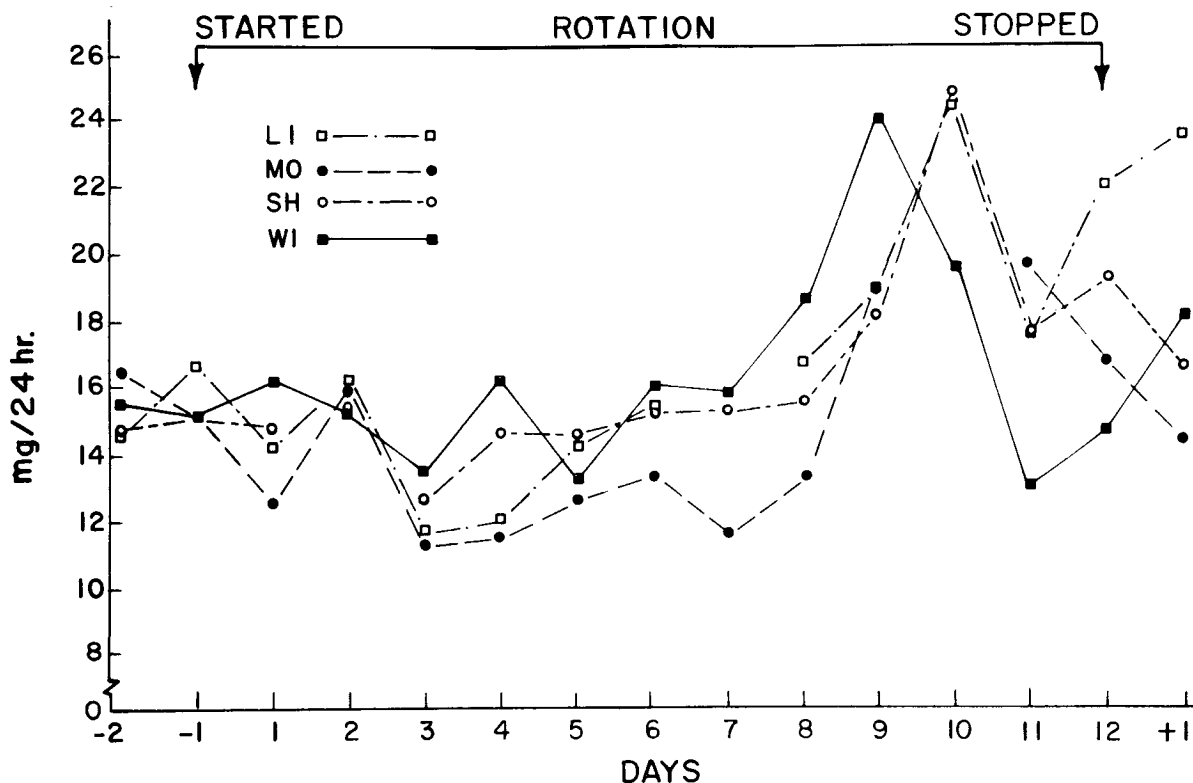


Figure 8.—Urine corticosteroid excretion of four subjects.

4 percent per minute, a four-fold increase over his Day 2 value. On Day +1 a return to normal was found; after the end of rotation, his rate was almost identical to its initial value. In MO, the initial decline of removal rates continued through entire Day 3, but on Day 11 a significant increase to 4 percent per minute was observed. After the end of the experiment the rate returned to below its control value. SH demonstrated the greatest reaction. After the initial decline on Day 2, unusually rapid removal rates (7–10 percent) were observed throughout the experiment. On Day 5, blood glucose levels fell nearly to hypoglycemic levels 1 hour after the glucose load. On Day +1, blood sugar also declined to a point moderately below the fasting level. On Day +2 the utilization rate was again quite close to the initial normal value. In WI the typical initial decrease of utilization was observed. However, a significant increase occurred

only toward the end of the experimental period and was followed by a further increase in the postrotation period.

#### Blood Enzymes

Glutamic-oxaloacetic acid transaminase (SGOT) and alkaline phosphatase and lactic dehydrogenase (LDH) were determined (table XIV). The only significant changes were in LDH which are summarized in figure 10. Two of the four aviators showed a considerable increase in LDH on Day 1, and all of them showed reduced activity during the following 4 days. On Day 5 and again on Day 11 there were significant additional increases. After cessation of rotation the high values on Day +2 were preceded and followed by values near the control level.

#### Food Absorption

Intestinal absorption of food, as reflected by the oral zylose absorption, did not show

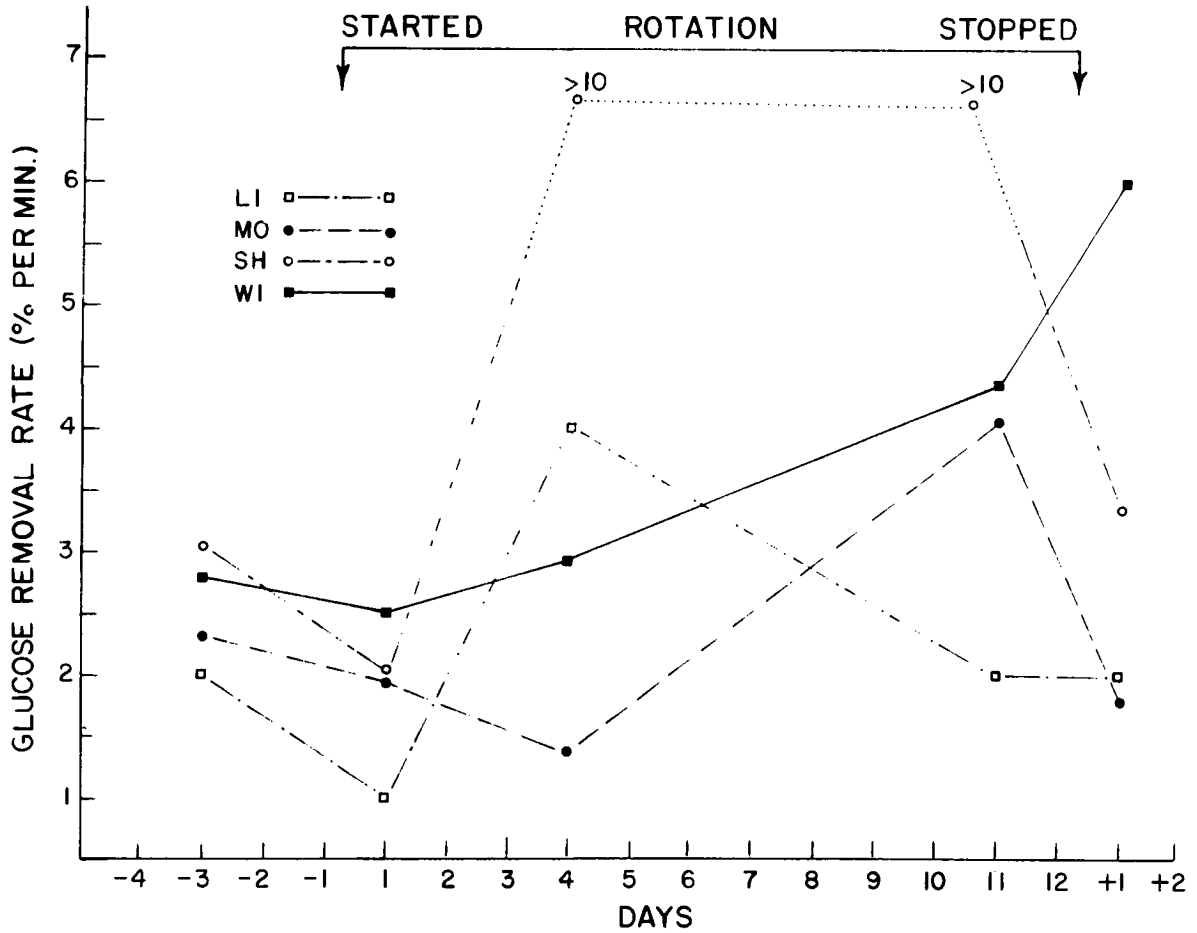


Figure 9.—Glucose removal rate of four subjects.

any significant change during any phase of the experiment. Any variations were considered to be within the experimental error.

#### Discussion of Biochemical Findings

In general, the significant changes in the biochemical and other laboratory findings paralleled the changing symptomatology described in the previous section. In attempting to interpret these changes in terms of the effects of stimulation by the accelerative forces, it is important to emphasize that they might reflect not only specific effects of such stimulation but also nonspecific or complicating effects. Moreover, one is handicapped by the limited quantitation both of the stimulus and its effects and the

lack of previous experimentation to provide background information.

In the early perrotation period the subjects learned by experience that it was necessary to restrict their head movements although prior to this they were, in varying degrees, less cautious, and, as a result, MO vomited on eight occasions and SH once. Moreover, in varying degrees, they reduced their physical activity, and limited their intake of fluid and food. As they adapted to the nausea syndrome they increased their head movements and eventually imposed no restriction. Consequently, in the late perrotation period the level of accelerative stimulus was increased, the food and water intake was guided by hunger and thirst, and physi-



Table XIII.—*Intravenous Glucose Tolerance Tests*

Subject	Day of experiment	Blood fasting	Sugar, 15 min	Concentration in mg			Glucose rem rate, %/min
				30 min	45 min	60 min	
LI	2	82	204	174	144	139	1.78
	5	79	201	180	160	153	1.14
	8	73	190	109	83	90	4.7
	15	67	188	142	131	105	2.41
	17	73	196	154	138	118	2.15
MO	2	88	214	161	155	129	2.30
	5	81	208	167	150	128	2.13
	8	76	222	189	175	153	1.37
	15	85	225	140	130	105	4.03
	17	70	207	181	146	131	1.86
SH	2	75	194	160	122	104	3.2
	5	74	201	176	151	120	2.3
	8	78	118	109	76	40	high
	15	81	184	114	71	60	high
	17	78	186	146	123	100	3.45
WI	2	67	205	164	122	104	3.01
	5	71	200	169	130	110	2.74
	8	76	177	132	100	102	3.27
	15	62	198	134	99	78	4.72
	17	75	178	123	87	80	6.97
Average N = 4	2	78	294	164	135	119	2.51
	5	76	202	173	147	127	2.00
	8	75	176	134	108	96	3.52
	15	73	198	132	107	87	5.19
	17	74	191	151	123	107	2.82

cal activity was increased compared with the early period. The time-course of these trends differed in different subjects, but in general their extrinsic behavior became more uniform in the late perrotation period. One other factor deserves mention: the possible role of "anticipation" as the end of rotation drew near. This is difficult to estimate; in all likelihood it was not a significant factor except possibly on the morning of Day 12. These subjects not only were conditioned to stressful experience but also the end of rotation was not the end of the experiment.

With regard to changes in urine output, the release of antidiuretic hormone, previously shown to be associated with unusual vestibular stimulation (ref. 19), was the probable primary factor; variations in fluid intake and, in the case of MO and SH, vom-

iting, were complicating factors. In the early perrotation period when the nausea syndrome was prominent, WI was the only subject who gained weight, and this occurred while his urine output was falling, suggesting water retention. It is possible that LI and MO had a greater tendency to release the antidiuretic hormone than SH and WI inasmuch as they manifested the greater decreases in urinary output both in the early and late perrotation periods. In the early period LI, who did not vomit, had nearly as great a decrease in output as MO who vomited, and in the late period, not characterized by nausea, the decrease in output may have been associated with increase in head movements (stimulus). If this last assumption is allowed, it demonstrates that they adapted more readily to

Table XIV.—*Blood Enzyme Responses*

[Normal values: Lactic dehydrogenase, 100–350 units; SGOT, 8–40 units;  
Alkaline phosphatase, 0.8–2.3 sigma units per ml]

Subject	Day of experiment									
	–3	1 <sup>a</sup>	2	3	5	8	12	End of run <sup>b</sup>	+1	+2
LDH										
LI	60	30	40	20	120	440	1040	260	460	160
MO	240	720	80	40	100	360	660	360	520	350
SH	260	220	60	60	100	440	480	260	560	180
WI	140	720	100	100	120	160	800	280	620	200
SGOT										
LI	26	30	34	28	24	32	44	25	26	26
MO	28	36	32	28	26	28	32	30	28	22
SH	64	28	28	30	26	26	38	28	26	32
WI	44	30	42	28	24	40	38	18	24	24
Alkaline phosphatase										
LI	1.45	1.45	1.15	1.25	1.7	1.15	1.6	1.4	1.4	0.80
MO	1.05	1.20	1.75	0.6	0.9	0.9	1.0	0.9	0.85	0.70
SH	1.5	1.35	1.80	0.7	1.0	0.9	0.8	0.8	1.1	0.78
WI	0.87	0.85	0.65	0.7	0.9	0.85	0.65	1.6	0.72	0.70

<sup>a</sup> Run began 0900.

<sup>b</sup> Run stopped 0900.

the nausea syndrome than to the factor responsible for the antidiuretic effect. Especially interesting are the similar variations in urine output for SH and WI who nevertheless varied in their susceptibility to nausea.

The changes in urine volume and electrolytes were not reflected in the blood hematocrit, total protein, and serum electrolytes, at least to any significant degree. In other words, the excretory mechanisms constituting our first line of defense in adjustments to stresses imposed were adequate to preserve homeostasis with respect to serum electrolytes. The determinations of serum pH may not have been reliable, but the pCO<sub>2</sub> determinations indicated slight variations from normal, the most significant a tendency toward alkalosis in the subject who vomited eight times.

There was no significant change in release of noradrenalin as indicated from

measurements on blood and urine. Similar measurements on release of adrenalin showed only minor fluctuations and these in LI and MO. These negative findings are nevertheless significant in that they tend to minimize the possibility that emotional stress was an important variable in the experiment.

With regard to the release of catechols and corticoids the increase in urinary corticoids in the late perrotation period alone was significant. If it is assumed that this increase was in response to increasing the head movements, two interesting facts emerge. First, it was not related in time to manifestations of the nausea syndrome, and second, all subjects were affected almost equally but not at the same time. Previously we had assumed that there was a relation between occurrence of nausea and the release of adrenal corticoids, but the present findings suggest that even an association

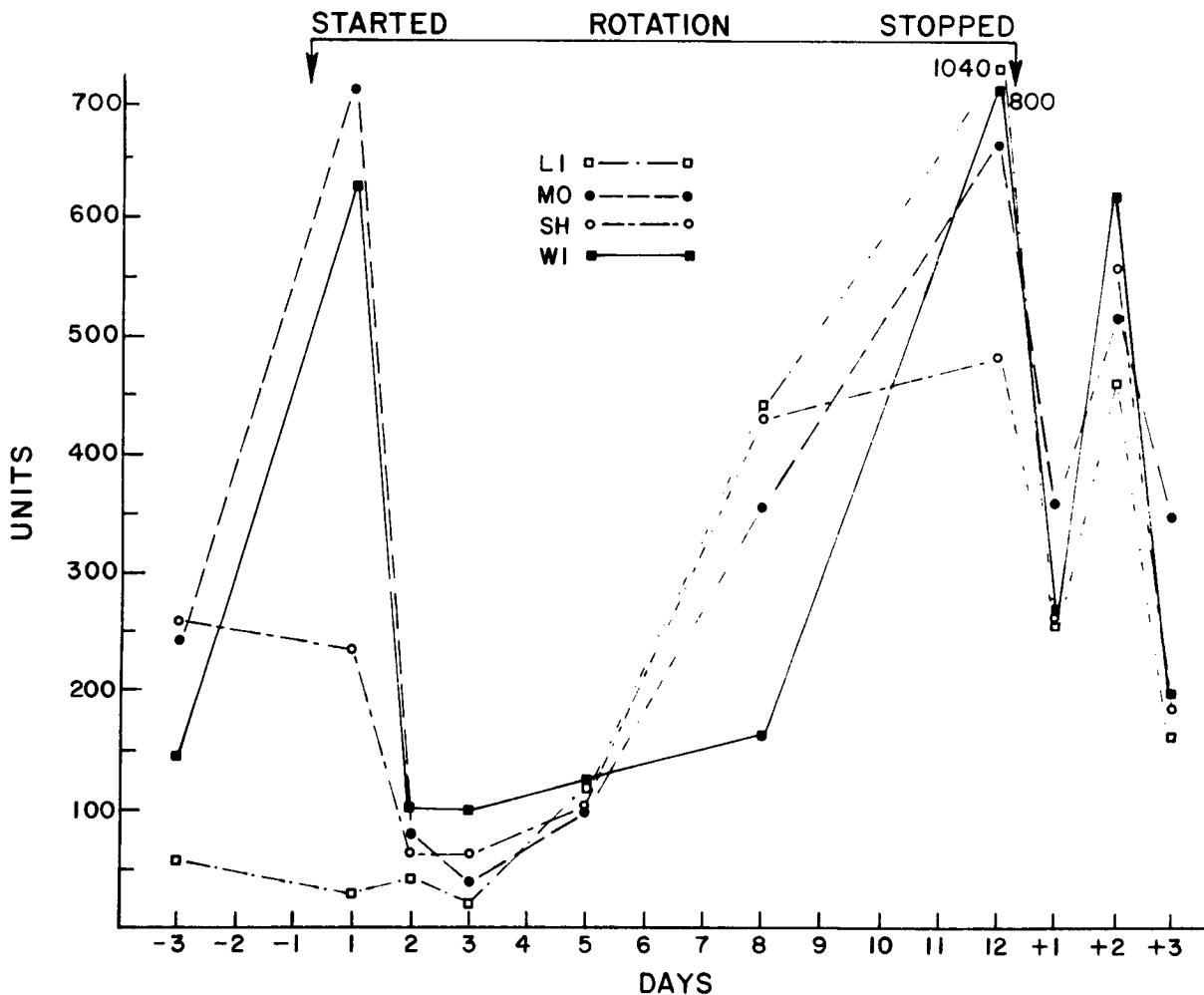


Figure 10.—Lactic dehydrogenase response of four men.

between the two may be absent. Of all the significant positive findings the increases in release of corticoids showed the least individual variance.

The most striking findings requiring explanation were the changes in rate of glucose utilization and in LDH level in the plasma. The group trends showed some similarity, i.e., an initial decrease followed by an increase above prerotation values, but there were exceptions to the group trends, and there was often a lack of parallelism between the glucose and LDH responses for a given subject. An attempt was made to relate both findings to an increase in muscle metabolism, and considera-

tion was given to the difficulty in maintaining equilibrium while standing and walking and the possibility that there was an increase in muscle tonus (ref. 20) due to bizarre stimulation of the semicircular canals. There was a closer parallelism between physical activity and LDH than glucose utilization levels.

The increase of glucose removal rates is highly significant. The qualitative similarity of response in all four men indicates that the exposure to stress had a pronounced effect on carbohydrate metabolism and that the only difference among individuals is the time at which the reaction set in. Two mechanisms could explain the findings:

First, there is the possibility of increased levels of circulating insulin in the blood caused by rotation. No measurements of plasma insulin were made, but the finding that fasting blood glucose levels were little affected by rotation would make the existence of significant hyperinsulinism unlikely. Secondly, there was an increased permeability of peripheral tissue to glucose. It is known that muscular activity causes a great increase of cell permeability to sugars and consequently of glucose metabolism and that this effect is independent of insulin. This increased activity is normally compensated by increased hepatic glucose production, and thus the blood glucose level remains unchanged. The additional increase of permeability caused by the release of insulin during the glucose tolerance test might well lead to greatly increased glucose removal rates and even to hyperglycemia.

The initial decrease of glucose removal rates could then be explained by a diminished physical activity during the first day of rotation, very pronounced in LI and SH, less in MO and WI. It is more difficult to explain the subsequent striking increase inasmuch as none of the subjects became very active.

#### Results of Psychophysiological Tests

##### *Hand Dynamometry*

The dynamometer used in this experiment was a standard model Stoelting hand dynamometer. The subject's daily score was the arithmetic mean for five trials. From the group data in figure 11, it may be seen that on Day 1 there was a sharp drop in performance which by Day 2 returned to the average for the 4 prerotation days. From this point until Day 10 there was a gradual increase in performance where the

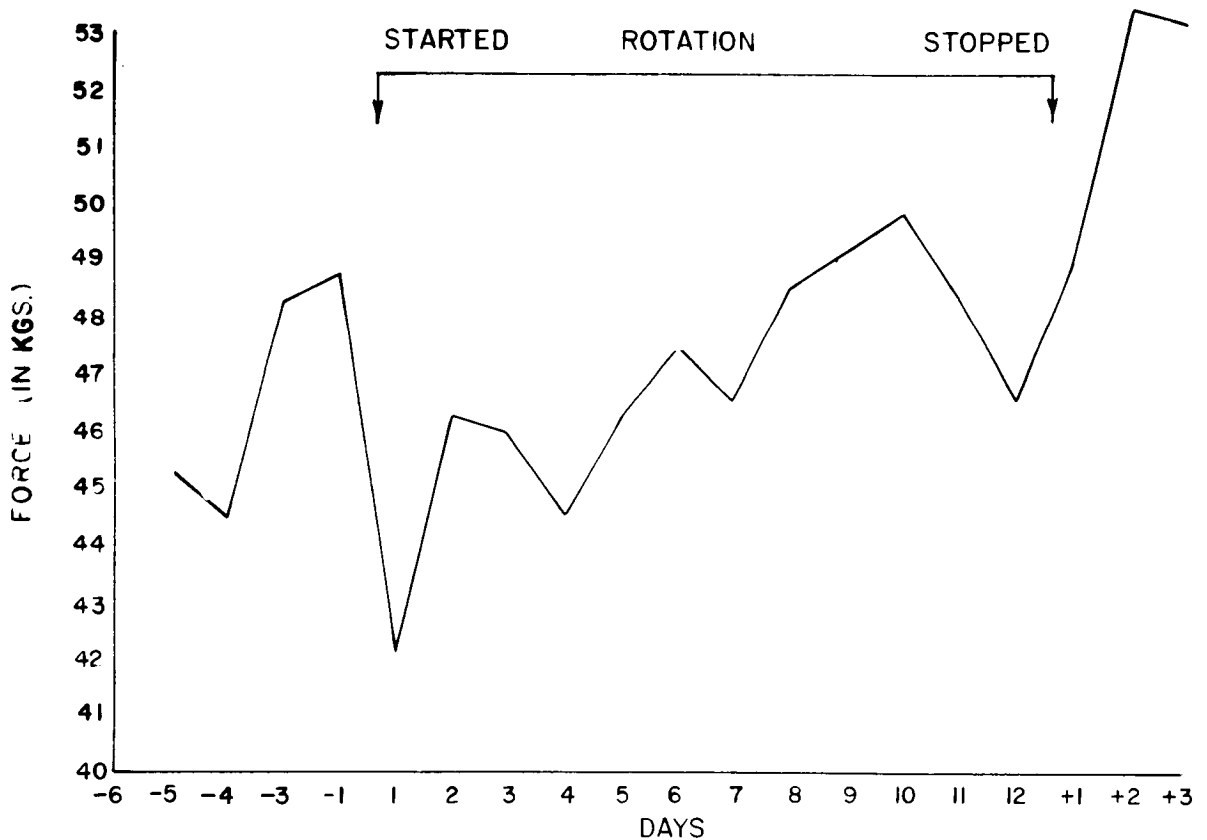


Figure 11.—*Hand dynamometer scores for four men.*

best prerotation score was equaled. The last 2 days of rotation performance showed a pronounced drop, and postrotation there was a sharp rise to values greater than the initial control level.

#### Spoke Test

This is a simple test of eye-hand coordination which requires that the subject point to different compass coordinates and then to the center. In test A the coordinates are randomly numbered, and the subject must point progressively (e.g., 1, 2, 3, . . .). The task is similar in test B; however, the subject must alternate numbers and letters progressively (1, a, 2, b, etc.). The score is in seconds to completion of the task.

The data appear to indicate an overall practice effect. However, the learning curve for the complex task (test B) is interrupted at the onset and cessation of rotation although the change is small. The decrease in performance at the cessation of rotation on test A appears less than an expected day-to-day variability.

#### Simplified Electronic Tracking Apparatus (SETA)

The *tracking test* used in this experiment was similar to the one devised by Fitts (ref. 21), but a knurled knob was substituted for the lever. In this test an irregular motorized cam drives a pointer off zero, and the subject's task is to keep the pointer on the dial at zero. A computer element monitored the pointer and compiled the subject's total error (in microvolts). Each trial lasted 1 minute, and there were five trials in a session. The first data point in figure 12 is a mean of 100+ trials and may be considered stable. The overall impression is of a learning function, and the interruption of this trend on the first day of rotation is therefore probably meaningful. By Day 2 the prerotational level was obtained and improvement continued.

#### Speed of Tapping

This required that the subject alternately depress buttons on two cumulative counters

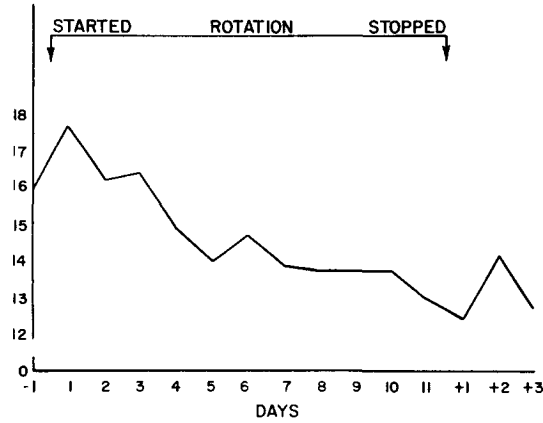


Figure 12.—Simplified electronic tracking apparatus: mean score for four men.

with the index and second finger of his preferred hand. Each trial lasted 30 seconds and there were 10 trials per session. The scores were read directly from the counters, and error was the difference between two totals. Two subjects improved in performance and two remained about the same. The onset and cessation of rotation did not appear to have any effect upon performance. The variability did not change with time nor did the error score vary in any consistent fashion.

#### Reaction Time

This consisted of a simple reaction time to a visual stimulus. There were 50 trials per day for each subject. The data are plotted in figure 13 with the first (practice) session included. The group performance appears to indicate a simple learning function which flattens slightly at the onset of rotation. However, the individual data show that WI (least affected overall) continued to improve until Day 10 and then proceeded to do less well. Subject SH continued to improve slightly throughout. The other two subjects also continued to improve, but their learning appeared to be interrupted the first few days of rotation. Subject WI obtained one of his poorest scores on Day +1.

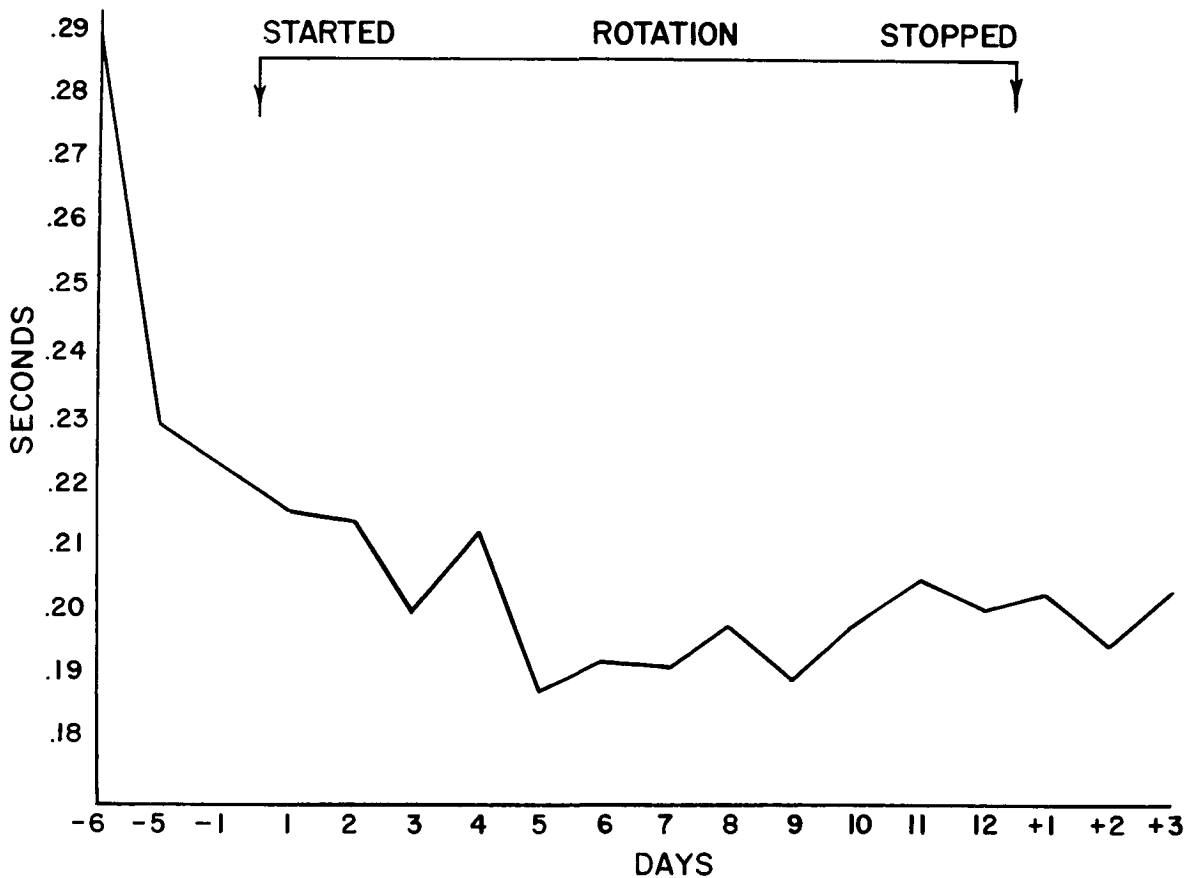


Figure 13.—Reaction time: mean for four men.

#### Time Estimation

This was tested by the "production" method and required the subject to depress a key for a given number of seconds. Ten judgments were made at each of eight intervals (2, 3, 5, 6, 8, 9, 11, 12 secs) on each day of the experimental period. Because these data showed no consistent over- or underestimation within a session or from one session to the next, they were plotted in terms of total absolute error. Average deviations for each interval also were obtained. Figure 14 shows the average deviation for the four men at each interval. The magnitude of the error appears to be directly related to the length of the interval to be estimated and is as expected. Figure 15 shows the mean absolute error for all intervals for the four subjects for the entire experimental period. It may be seen that

the greatest errors occur at the onset and cessation of rotation.

#### Digit Span

The Digit Span Test was administered in a way similar to that devised by Wechsler (ref. 22), but the numbers were presented visually once per second. The results of this

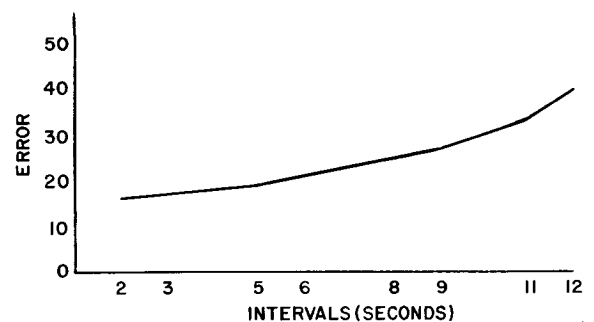


Figure 14.—Time estimation: average error per day for four men.

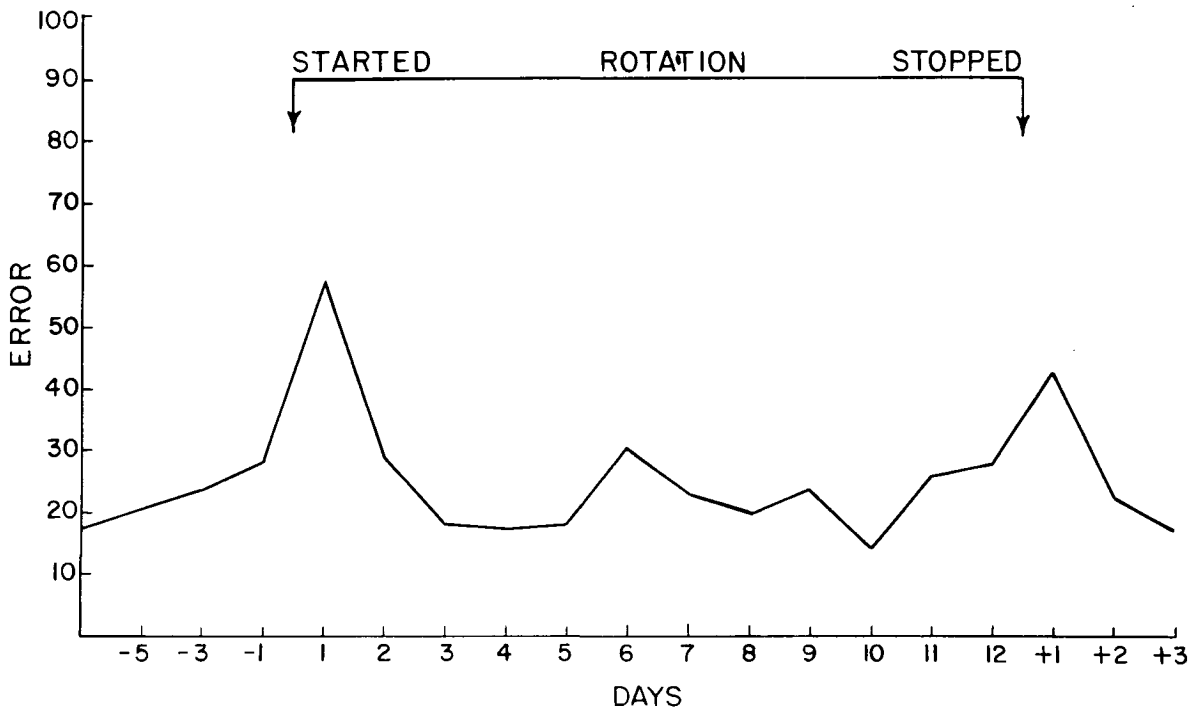


Figure 15.—Time estimation: mean average error per day for four men.

test appear to show a learning effect, which may in part be explained by the fact that only 260 number sequences were used and the subjects saw them more than once. The learning curve is interrupted at the onset of rotation and midway through the run indicating a decrement in performance.

#### *Complex Counting Task*

In the Complex Counting Task (ref. 23) the subject is presented with a panel containing three lights (A, B, C) with a button below each light. The lights were programmed to flash at different frequencies (viz., A—5.4 times per minute; B—8.9 tpm; C—4.6 tpm). The test lasted 5 minutes, and the subject's task was to depress the button below the light after it had flashed four times. Stimuli and responses were recorded on an Easterline Angus event recorder.

Group means indicate a small decrease in performance at the onset and at cessation of rotation which is within the range of day-to-day variability. On Day 8 there was a pronounced drop in performances. The in-

dividual data show WI (the subject least affected by rotation) with an overall increase in performance as the experiment continued. Subject SH remained about the same, and the other two subjects appeared to make some errors as the run progressed. Subject WI obtained one of his poorest scores on the first day after rotation.

#### *Reading Test*

The Reading Test consisted of assignments selected from two workbooks routinely used in the reading course at the U. S. Naval School, Pre-Flight. A range of difficulty level was assigned according to the "Flesch Scores" (ref. 24). The results of the reading test are inconclusive. Intersubject and intrasubject variabilities of speed and comprehension are enormous. The subjects suggested that interest value should have been included in the score for difficulty level.

#### *Mathematics Test*

The Mathematics Test was a simple 5-minute test of addition, subtraction, and

multiplication. For the most part there was a gradual increase in performance which was probably a function of learning (fig. 16). Although many vigilance and fatigue studies indicate a spurt or upsurge in performance just prior to completion of the experiment, here on Day 11 there was a decrease in performance. Motivation was believed not to be a factor.

#### Vigilance Tests

Each subject stood a night watch for 90 minutes at the same time every evening after the crew had secured. The subject was required to monitor flashing lights, an auditory signal, and several gages; however, only four of the tasks were scored:

*Alternating Lights.*—In the center of the console two lights flashed alternately (30

cycles per minute each) to produce one flash per second. At irregular intervals (and less than 10 times in 90 minutes) one light stopped flashing and the other doubled in frequency. The subject was to depress a button when this occurred. Both stimulus and response were read out on an event recorder. Score was delay of response in seconds. A step relay which caused the light to stop flashing produced an auditory cue. However, the light did not change every time the relay advanced. It is felt that if sensory summation made the task less difficult it did so by a small amount.

In the main there was a very small increase in performance which for these men did not appear to be interrupted by the onset or cessation of rotation. The individ-

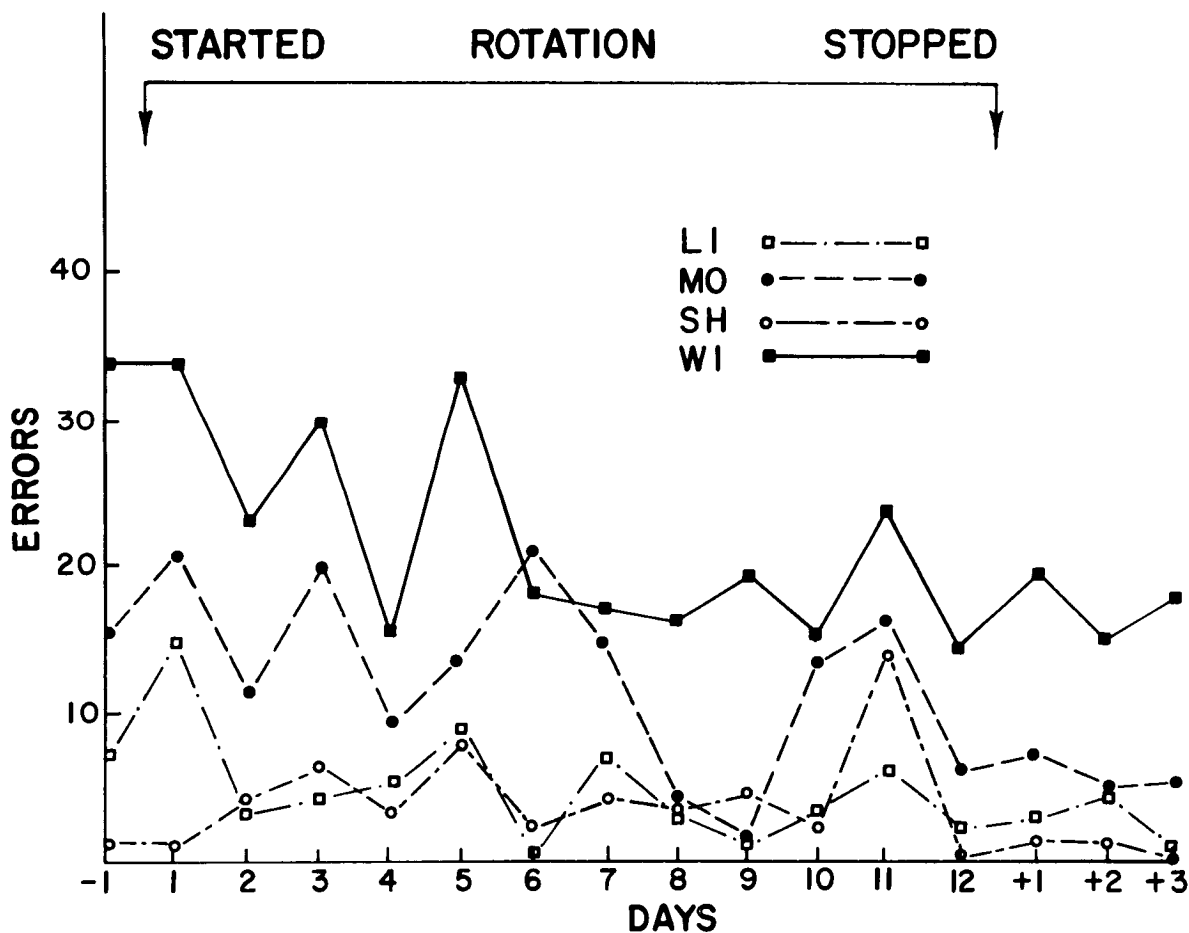


Figure 16.—Math Test scores for four men.



ual data showed one very long response and this by subject LI on Day 1. Actually, the subject fell asleep, and this is particularly meaningful since of the four men LI appeared to be most competitive and least likely to fall asleep on watch.

*Three-Digit Test.*—In the center of the console and below the alternating lights was a nixie tube. The tube lit once a minute for 10 seconds, and numbers were programed randomly. The subject was assigned a code number for the evening, and it was his task to record when his number came up (vigilance). Also he was obliged to remember the number which appeared previously (memory) and the number which occurred next (attention). There were more attention errors than vigilance errors and more memory errors than attention errors. The performance of these men appeared to decrease at the onset of rotation but for WI (least affected) it did not. All did poorer at the cessation of rotation but the change was small (fig. 17).

*Audio Vigilance.*—Along with 84 db broad band noise, an 87 db tone (1175 cps) of 0.25 second duration was presented every 0.95 second, for 90 minutes. Sporadically (but less than 12 times in a session), the interval was stretched from 0.95 to 1.40 seconds. It was the subject's task to note the time at which this occurred. Score was number of intervals missed. From the group data figure 18, there appeared to be a learning effect which did not seem to be interrupted by the onset of rotation. However,

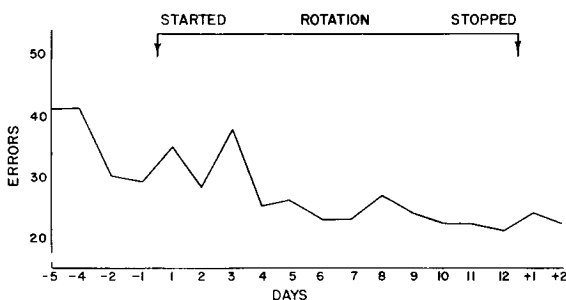


Figure 17.—*Three-Digit Test (Night Watch) for four men.*



Figure 18.—*Audio Vigilance Test (Night Watch) for four men.*

there was a decrease in performance at cessation.

*Fifteen-Minute Test.*—In this test the subject was required to read a continuously varying gage every fifteen minutes. The score was the absolute error in time. The data seem to indicate that subjects were able to perform this task quite well and with very few gross errors. There were only two errors of more than 1 minute (3 min and 6 min) in a total of 56 trials (i.e., 14 for 4 subjects). Most errors are less than 10 seconds.

#### *Discussion of Psychophysiological Tests*

All the subjects carried out all of the tests except on one occasion when LI fell asleep during his watch. After making allowance for practice effects and time-to-time variance, it is obvious that significant changes in performance were either absent or small except in the case of the hand dynamometry test. It is interesting that these changes in performance, aside from those in close relation to the onset or cessation of rotation, were manifested more frequently in the late than in the early perrotation period. Hand dynamometry deserves particular notice inasmuch as the scores seemed to reflect the general fitness of the subjects throughout the entire experimental period. Moreover, the sharp rise in values after cessation of rotation suggests that disturbances in neuro-

muscular coordination were not a factor in carrying out the test.

#### GENERAL DISCUSSION AND SUMMARY

Prolonged exposure in a rotating environment is a unique experience and differs in important respects from exposure to turbulent seas or to turbulent air. One striking difference has its origin in the fact that in the rotating environment the subject's movements are essential to the generation of the bizarre accelerations whereas at sea or in the air, fixation of the head and body might influence the effects but would not abolish the unusual inertial forces to which the traveler is passively subjected. Thus, during rotation in the SRR when the subject is seated with head fixed or asleep the conditions are not far different from those with the room stationary. Movements of the subject resulting in functional disturbances fall mainly into two categories, namely, rotation of the head out of the plane of rotation of the room and whole body movements. The former generate very small inertial forces and their effect, at least when the subject is near the center of the SRR, would be negligible were it not for the semicircular canals. They are so structured that the sensory elements are stimulated by the gyroscopic forces, and the bizarre pattern of the stimulus is the essential factor causing the symptomatology of canal sickness, although other etiologic factors are also involved.

Whole body movements, insofar as they generate Coriolis forces and affect, through change in distance from the center of the SRR, the level of centripetal force, result in a change in the gravito-inertial force vector and, consequently, in the direction of the force upright with respect to the room. Neuromuscular coordination is affected which increases the difficulty in maintaining postural equilibrium, for example, and the amount of work involved in walking. A distinction is made between these neuromuscular effects and canal sickness, inasmuch as the semicircular canals are not

directly implicated unless the head is rotated out of the horizontal plane of the SRR. Such rotations of the head may be unavoidable, and then the bizarre stimulus to the canals contributes to the neuromuscular disturbances.

Past experience has shown that there is a good correlation between the velocity of rotation of the room and the severity of the symptoms manifested by the subjects. The reason is simply that the movements required in meeting essential needs and carrying out prescribed tasks are similar at all velocities, thus making velocity of the room the dependent variable. The subject can, however, by omitting tasks and remaining inactive in his free time, reduce his activities. Hence, not only the velocity of rotation but also the movements of the subject must be taken into account if any attempt is made to estimate the changing levels of exposure to the unusual inertial forces at any given velocity. In this experiment a precise measure of bodily movements was not obtained; the estimates are in terms of "restriction of head movements" and "level of physical activity." In the SRR the subject could anticipate every stressful stimulus, perfectly with respect to occurrence, although imperfectly with respect to disturbing effects characterized by summation and perseveration. With training and experience, however, the subject became adept at avoiding disturbances by regulating the stimulus both quantitatively and qualitatively. Thus, in terms of the stressful inertial forces each subject, consciously at first then automatically to an increasing extent, exerted a modulating influence on the level of stimulation.

A second important difference refers to the stability of the SRR compared with ship or aircraft. With full illumination of the room and while he is engaged in ordinary activities the subject regards the platform as being not only stable but also level and the walls upright. Under special circumstances the room may appear tilted (the oculogyral illusion, ref. 25), and, seated in the dark, the subject may regard the chair

on which he is seated as being tilted but always with respect to a level stable platform. Under most conditions, however, the visual environment appears the same as when the room is not rotating.

The effects of rotation already have been discussed under three headings: the clinical symptoms, clinical laboratory findings, and psychophysiological performance. They will now be discussed briefly in terms of the significant changes along the time axis of the experiment, and some of the practical and theoretical implications will be summarized.

The unexpected finding of two quite distinct perrotation periods is explained thusly: In the early perrotation period the subjects restricted their head movements and consequently their physical activity to prevent the nausea syndrome. This subject-paced stimulus level was well below the unpaced level and was individually adjusted to the time-course of adaptation to this unpleasant syndrome. With the disappearance of nausea this restriction on head movements was lifted, thus increasing the bizarre stimulus to the semicircular canals. This paved the way for the appearance of effects which either (1) necessitated a longer time-course of adaptation than did nausea; (2) required a stronger stimulus for their exhibition; or (3) had different transfer patterns in terms of levels of stimulation. The restriction on physical activity, however, was partially maintained inasmuch as it was now paced by such symptoms as drowsiness and fatigue. Thus the late perrotation period while initiated by the lifting of restrictions on head movements was characterized by a rising level of physical activity which, although never reaching the pre-rotation level, may nevertheless have exceeded it in terms of muscular work. The symptomatology of the two distinct perrotation periods revealed many interdigitations both for the individual and for the group and in all likelihood was complicated by symptoms referable to confinement and level of physical work.

The first day of rotation made the great-

est overall impact on the subjects. The sudden onset, marking the rather dramatic beginning of a long exposure to unfamiliar stresses, was, after the days spent in carrying out preliminary arrangements and baseline studies, keenly anticipated. Such immediate effects as difficulty in walking and the strange feeling associated with head movements were not unpleasant, and all except LI were lulled into making a false estimate of their susceptibility to the nausea syndrome. Within an hour MO experienced the first of eight vomiting episodes and SH complained of nausea. Before long quiet fell over a previously noisy group, and all except WI attached the head brace to minimize movement between head and thorax. Thus the findings on the first day reflected not only differences in susceptibility to a given level of stress but also differences in self-appraisal of the susceptibility, which influenced the subjects' self-paced exposure to stress. This accounted in part for the severity of symptoms the first day and for certain differences in pattern of response between Day 1 and Day 2.

Adaptation to the nausea syndrome was the main guide in defining the early perrotation period, a period characterized not only by typical symptoms of canal sickness but also, except on Day 1, by a low level of physical activity. Individual differences in severity of symptoms and rate of adaptation were revealed. Moreover, there were significant alterations in the clinical laboratory findings reflecting in part the low level of physical activity and in part the stress of the force environment. All of the subjects carried out the performance tests on the first day although MO and SH did not participate in all of the clinical tests. Slight decreases in performance were recorded in several tests especially on Day 1 and notably in hand dynamometry.

The late perrotation period was characterized by the fatigue syndrome, a changing pattern in the laboratory findings, and slight decrements in certain performance tests. It was necessary to take into account three

etiological factors, namely, vestibular stimulation, increasing physical work, and confinement.

Such symptoms as drowsiness and fatigue were complaints even in the early perrotation period but were overshadowed by nausea. With the disappearance of nausea, however, they became the presenting symptoms. The subjects reported a gradual decrease in drowsiness but continued to complain of fatigue. It is difficult to distinguish between boredom and fatigue, and the increase in physical work may have been a factor.

The unexpected biochemical findings were the appearance for the first time of stress hormones in the urine, which were almost surely attributable to stimulation of the canals, and striking increases in glucose utilization and in the serum enzyme LDH.

All the tasks were carried out in the late perrotation period. With regard to the performance tests, there was a drop in score in hand dynamometry on Days 11 and 12 which was probably significant and a slight drop in scores in digit span on Days 7 and 8, in the Complex Counting Test on Day 8, and in the Math Test on Day 11.

Cessation of rotation had less effect than anticipated on the symptomatology. Only SH experienced a mild return of nausea (stomach awareness) lasting a short time, and while the subjects were surprised over the difficulty in walking, especially on rotating the head, this soon disappeared. None of the changes in the clinical laboratory findings were unexpected. There were slight decrements in some of the performance scores but an increase in score in hand dynamometry.

The onboard observer KE presented no history of motion sickness and had amassed > 500 hours at different velocities in the Slow Rotation Room. In his first few exposures dizziness and slight discomfort were his only complaints. In subsequent experiments he remained symptom free. Audiometric exam, caloric response, and postural equilibrium were within normal

limits and there was no history of disease of the sensory organs of the inner ear.

During the present experiment KE reported slight dizziness only during the first hour. This rapidly subsided and with the exception of drowsiness was the only characteristic symptom reported throughout the rotation period. The first day KE had only slight difficulty walking and (unlike all the subjects) did not restrict his movements at all. From all indications adaptation was accomplished quickly. Although KE's log was sprinkled with comments on "sleepiness," his fatigue was probably not so great as that of the other four men. This freedom from difficulties may be the result of KE's initial insusceptibility but is more probably the result of his conditioning from previous experiments.

KE remained on board for the duration of the experiment and his exposure was equivalent at least to that of the subjects, with the exception that during the short daily stops KE left the SRR (and consequently moved his head). This was done, as a probe, to discover the feasibility of maintaining adaptation to 10 rpm and to zero simultaneously. This proved quite successful (at least with respect to postural equilibrium) since KE did not experience ataxia problems when rotation finally ceased. On the other hand, the additional stress of adaptation to both environments may have contributed to the fatigue KE reported. Verification of these findings awaits future experimentation.

All the aviators were asked whether, in their opinion, they were unfit at times during the run to carry out tasks which might be expected of astronauts in orbital flights. All agreed that this was the case; even WI who was quite fit in the early period considered he was less fit later due to prolonged fatigue. This opinion is fairly well supported by the objective findings. The most severe symptoms were experienced by the two subjects who were nauseated, and their performance was adversely affected. Even this did not seriously affect vital homeostatic

mechanisms, and at no time were the subjects in need of medical care. Such countermeasures as selection, adaptation, and drugs would have greatly minimized the nausea syndrome, but fatigue might present a more difficult problem.

In attempting to apply the findings obtained in the SRR to a rotating orbiting spacecraft it is necessary first to consider important differences between the two with regard to the force environment (refs. 26 and 27). A spacecraft with a radius of 30 feet and rotating at a velocity of 10 rpm would generate a centripetal force of about 1.0 G unit at the periphery. The disturbances in neuromuscular coordination resulting from changes in the linear gravito-inertial vector associated with "body movements" would be comparable to those in the SRR. The disturbances resulting from "head movements" might be worse due to the fact that the "upright" position of the astronaut would be at right angles to the axis of rotation rather than parallel to it as when in the SRR. At least this would be true to the extent that swiveling rotations of the head, which can be made with impunity in the SRR, would prove to be more stressful than nodding motions.

If the radius of the spacecraft were 15 instead of 30 feet, thus generating only 0.56 G unit at the periphery, the smaller magnitude of the linear force vector and the greater changes in direction associated with body movements would adversely affect equilibrium. Although the gyroscopic accelerations stimulating the semicircular canals would be the same, the possible decrease in the modulating influence of the otolith organs might be significant.

It should be pointed out that, in the SRR, the subject's head, when he is bending over, reclining, or recumbent, is nearly at right angles to the axis of rotation and that he adapts to whatever rotations of the head are made in these positions. Although plans have been made for a systematic study comparing the effects with subjects parallel and at right angles to the axis of rotation,

it is safe to predict that the differences will not be great and that adaptation in one circumstance will provide a large measure of "protection" in the other.

The fact that the onboard observer did not experience nausea and was never more than moderately fatigued suggests that much can be accomplished in the areas of selection, training, and habituation with regard to the prevention of canal sickness. It would be worthwhile to explore the effect of slowly increasing the angular velocity of the SRR in order to minimize or even prevent unwanted effects. Moreover, adaptation at a higher angular velocity than that planned for a space flight might be advantageous.

Additional countermeasures might include such practical measures as the location of hand rails for support, the arrangement of instrument panels to take advantage of the fact that head movements in the plane of rotation are not stressful, and the identification of the most effective drugs to prevent nausea and possibly other symptoms.

It is also of more than passing interest that the onboard observer who left the SRR for short periods was not seriously disturbed by such transitions, suggesting that simultaneous adaptation to rotating and stationary environments is feasible.

In the present experiment, especially in the late perrotation period, it was difficult or impossible properly to evaluate the etiological roles of confinement, muscular work, and vestibular stimulation responsible for the symptomatology. The significance of factors other than the force environment could be determined, in part at least, by comparing the responses once with the SRR stationary and again with the room rotating. The role of the vestibular organs could be determined by a comparison of the effects between normal subjects and persons with loss of vestibular function.

From a more theoretical standpoint several findings deserve mention. The changes observed in fusion frequency of flicker are of interest whether they represent specific or nonspecific effects of stimulation of the

semicircular canals. In either case the value of such a delicate indicator is emphasized and it points up the need to investigate not only its advantages and limitations but also its implications in terms of central nervous system mechanisms. If the changes in CFF were specific, the findings indicate that decrements in performance were associated with manifestations of the nausea syndrome but not with drowsiness and fatigue in the late perrotation period when, in fact, mean performance was above the prerotation level. It is possible that changes in CFF and the time-course of adaptation to these changes may have practical application. It would be interesting to compare these with nystagmographic findings.

The present experiment did not permit a distinction between the etiological roles of the semicircular canals, otolith organs, and nonotolith gravireceptor mechanisms in the causation of and adaptation to ataxia. It did provide, however, a forceful reminder of the dominance of the visual upright over the gravito-inertial force upright under the experimental conditions, i.e., relatively weak inertial force and strongly structured visual field.

An attempt might have been made to explain the changes in serum level of the enzyme LDH by the changing level of muscular work were it not for the finding that on Day 1 two subjects had low and two had high values. The fact that on Days 2 and 3 all subjects had low values suggested that restraint in making either head or body movements or both was responsible. The relatively high values manifested by two subjects (MO and WI) on Day 1 are best explained if it is assumed that two factors were operating, namely, restriction in

physical activity tending toward a decrease and vestibular stimulation tending toward an increase in serum LDH. MO who restricted his activities considerably was most susceptible to typical symptoms of canal sickness. WI, who restricted his activities least, necessarily stimulated the labyrinth most among the four subjects. Whatever the etiological factors involved, it is worthwhile to investigate the underlying mechanisms. Although it has been shown that serum level of LDH is related to muscular work, clear-cut changes are manifested only with large changes in work load (ref. 28). With regard to stimulation of the labyrinth, it has been shown that it is an important factor modulating muscle tonus (ref. 20).

One of the most striking and unexpected findings observed was the increase in rate of glucose utilization which, directly or indirectly, must have had its genesis in exposure to the unusual force environment. The most likely etiologic factors and underlying mechanisms involved have been discussed above in some detail. Here it is important only to emphasize that the elucidation of this phenomenon might represent a significant scientific contribution.

It may be concluded that the present experiment, although it constituted only an initial probe into the problems incidental to adaptation at a velocity of 10 rpm, has advanced our knowledge in two main respects. First, it has shown that countermeasures in addition to adaptation are needed if rotational velocities of 10 rpm are required. Secondly, it has demonstrated the usefulness of the rotating room for the further exploration of vestibular and central nervous system mechanisms.

#### REFERENCES

1. GRAYBIEL, A.; CLARK, B.; AND ZARRIELLO, J. J.: Observations on Human Subjects Living in a "Slow Rotation Room" for Periods of Two Days. *Arch. Neurol.*, 3, 1960, pp. 55-73.
2. CLARK, B.; AND GRAYBIEL, A.: Human Performance During Adaptation to Stress in the Pensacola Slow Rotation Room. *Aerospace Med.*, 32, 1961, pp. 93-106.
3. GUEDRY, F. E., JR.; AND GRAYBIEL, A.: Compensatory Nystagmus Conditioned During Adaptation to Living in a Rotating Room. *J. Appl. Physiol.*, 17, 1962, pp. 398-404.
4. GRAYBIEL, A.; GUEDRY, F. E., JR.; JOHNSON, W. H.; AND KENNEDY, R. S.: Adaptation to Bizarre Stimulation of the Semicircular Canals

- as Indicated by the Oculogyral Illusion. *Aerospace Med.*, 32, 1961, pp. 321-327.
5. KENNEDY, R. S.; AND GRAYBIEL, A.: Validity of Tests of Canal Sickness in Predicting Susceptibility to Airsickness and Seasickness. *Aerospace Med.*, 33, 1962, pp. 935-938.
  6. STONE, R. W., JR.; AND LETKO, W.: Effects of Rotation on the Ability of Subjects to Perform Simple Tasks. In: *A Report on the Research and Technological Problems of Manned Rotating Spacecraft*. NASA TN D-1504, 1962, pp. 85-90.
  7. MCLEOD, M. E.; AND MEEK, J. C.: A Threshold Caloric Test for the Horizontal Semicircular Canal. NSAM-834 and NASA Order No. R-47, Naval School of Aviation Medicine, 1962.
  8. BECK, S. J.: Rorschach's Test. Volume II. Grune & Stratton (New York), 1945.
  9. McMICHAEL, A. L.; AND GRAYBIEL, A.: Rorschach Indications of Emotional Instability and Susceptibility to Motion Sickness. *Aerospace Med.*, 34, 1963, pp. 997-1000.
  10. HARRIS, C. S.; AMBLER, R. K.; AND GUEDRY, F. E., JR.: A Brief Vestibular Disorientation Test. NSAM-856 and NASA Order No. R-47, Naval School of Aviation Medicine, 1963.
  11. GUEDRY, F. E., JR.; KENNEDY, R. S.; HARRIS, C. S.; AND GRAYBIEL, A.: Human Performance During Two Weeks in a Room Rotating at Three RPM. *Aerospace Med.*, 35, 1964, pp. 1071-1082.
  12. SIMONSON, E.; FOX, M. S.; AND ENZER, N.: Influence of Vestibular Stimulation on the Fusion Frequency of Flicker in Normal Subjects and in Patients with Postconcussion Syndrome. *Arch. Otolaryng.*, 38, 1943, pp. 245-251.
  13. SCHALES, O., AND SCHALES, S. S.: Simple and Accurate Methods for Determination of Chloride in Biological Fluids. *J. Biol. Chem.*, 140, 1941, pp. 879-884.
  14. DIEHL, H.; AND ELLINGBOE, J.: Indicator for Titration of Calcium in Presence of Magnesium Using Disodium Dihydrogen Ethylenediamine Tetraacetate. *Anal. Chem.*, 28, 1956, pp. 882-884.
  15. FISK, C. H.; AND SUBBAROW, Y.: Colorimetric Determination of Phosphorous. *J. Biol. Chem.*, 66, 1925, pp. 375-400.
  16. WEIL-MALHERBE, H.; AND BONE, A. D.: The Chemical Estimation of Adrenaline-Like Substances in Blood. *Biochem. J.*, 51, 1952, pp. 311-318.
  17. CROUT, J. R.: Catechol Amines in Urine. In: *Standard Methods of Clinical Chemistry* (Seligson, D., ed.), Volume 3. Academic Press, 1961, pp. 62-80.
  18. KORNEL, L.: An Improved Rapid Method for Free and Conjugated 17-Hydrocorticosteroids in Urine. *Metabolism*, 8, 1959, pp. 432-440.
  19. TAYLOR, N. B. G.; HUNTER, J.; AND JOHNSON, W. H.: Antidiuresis as a Measurement of Laboratory Induced Motion Sickness. *Can. J. Biochem. Physiol.*, 35, 1957, pp. 1017-1027.
  20. GERNANDT, B. E.: Somatic and Autonomic Motor Outflow to Vestibular Stimulation. In: *Neurological Aspects of Auditory and Vestibular Disorders* (Fields, W. S., and Alford, B. R., eds.), Charles C Thomas (Springfield, Ill.), 1964, pp. 194-215.
  21. GAIN, P.; AND FITTS, P. M.: Simplified Electronic Tracking Apparatus. WADC 59-54, Wright Air Development Center, 1959.
  22. WECHSLER, D.: The Manual for Adult Intelligence Scale. New York Psychological Corporation, 1955.
  23. JERISON, H. J.: Effect of a Combination of Noise and Fatigue as a Complex Counting Task. WADC 55-360, Wright Air Development Center, 1955.
  24. BROWN, J. I.: *Efficient Reading*. D. C. Heath & Co. (Boston, Mass.), 1952.
  25. GRAYBIEL, A.; AND HUPP, D. I.: The Oculo-Gyral Illusion. A Form of Apparent Motion Which May be Observed Following Stimulation of the Semicircular Canals. *J. Aviat. Med.*, 17, 1946, pp. 3-27.
  26. LORET, B. J.: Optimization of Space Vehicle Design With Respect to Artificial Gravity. *Aerospace Med.*, 34, 1963, pp. 430-441.
  27. THOMPSON, A. B.; AND GRAYBIEL, A.: Effects of Long-Term Weightlessness and Requirements for Artificial Gravity in Manned Space Systems. Presented at 20th Aero Space Medical Panel Meeting, AGARD, NATO (Athens, Greece), July 5-12, 1963.
  28. HALONEN, P. I.; AND KONLTINEN, A.: Effect of Physical Exercise on Some Enzymes in the Serum. *Nature*, 193, 1962, pp. 942-944.

#### DISCUSSION

WENDT: At various times during the meeting I have heard people refer to the emotional state of subjects in a rotating room. I would like to make a suggestion of a so-called "instrument" that we use in our drug work to assess emotional and motivational state. It is an adjective check list that consists of 133 adjectives descriptive of emotional states. It is

highly sensitive to drug effects and is apparently quite valid. It takes 5 to 10 minutes to administer. It could be administered as many times during the day as you liked. There are many normative data available in our files on hundreds of subjects used many times. I think instead of talking only of apathy or depression, the adjective check list which

consists essentially of about 12 different classes of adjectives like cheerful, genial, confident, easygoing, important, sluggish, apprehensive, uneasy, depressed, apathetic, and so on, would yield a more discriminative assessment of the emotional and motivational changes that were taking place, at remarkably little cost in time.

**YORK:** Dr. Graybiel, do you have any suggestion about the screening tests and how they correlate with the subsequent behavior you mentioned?

**GRAYBIEL:** Prior to the onset of rotation the subjects were evaluated with regard to susceptibility to motion sickness on the basis of response to the threshold caloric test, interview, standardized acrobatics, brief vestibular test, et cetera. The vestibular test consisted essentially of having the subject rotate his head while turning in a Bárány type chair and scoring the responses. The results ranked the subjects with respect to susceptibility to the so-called nausea syndrome. None of the subjects were susceptible to air sickness and only two had a history, one incident each, of motion sickness. I rated one subject as less susceptible than the others on the basis of a slightly

depressed function of the semicircular canals on one side inasmuch as our experience demonstrated such a relationship. Actually, however, this subject, MO, proved to be the most susceptible. Rankings based on susceptibility to the nausea syndrome did not hold up during the latter part of the perrotation period when the subjects complained of what we termed the fatigue syndrome; here, there was little difference between the subjects.

**M. JONES:** In a situation like this, one might expect incorrect automatic vestibulo-ocular "stabilizing" signals to actively *destabilize* the retinal image; in which case one would expect reduced visual acuity during head movements having frequencies above the cutoff value for visual tracking, i.e., above about 1 cps. If this were the case one might imagine that the labyrinthine defective subjects would actually see better than normals during head movement in the rotating room. Have you any evidence to suggest whether this is the case?

**GRAYBIEL:** No, we haven't carried out tests along this line but we are planning to do so.



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## Stepwise Adaptation to a Velocity of 10 rpm in the Pensacola Slow Rotation Room

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*Umea University Medical School, Sweden*

Previous experimentation in Pensacola by Graybiel and coworkers (ref. 1) demonstrated that sudden exposure to 10 revolutions per minute (rpm) in the Slow Rotation Room (SRR) (fig. 1) induces considerable disturbances even in relatively unsusceptible individuals. Earlier experiments (ref. 2) have, however, shown that the lower values of rpm are more easily tolerated; therefore, it seemed reasonable to attempt a stepwise procedure in order to allow the test subjects to adapt to one rpm level before going to the next.

Earlier experiments had also demonstrated that after remaining in a rotating environment, the readaptation to zero rpm also presents problems. This readaptation period was short, but seemed to be more pronounced after longer periods in the rotating environment. An incremental procedure should also be attempted for the decrease from 10 to 0 rpm.

Underlying the three experiments presented here was also the question of eventual preadaptation for a rotating environment while maintaining the adaptation to 0 rpm. A rotating chair was mounted inside the SRR and used to assess adaptation in these three experiments (fig. 2). The chair could be set to any desired resultant speed—clockwise or counterclockwise. With the subject in this chair the Coriolis oculogyral test (ref. 3) was performed and "Coriolis nystagmus" (ref. 4) was recorded. Postural equilibrium tests (walking and standing on

rails) according to Graybiel and Fregley (a preceding paper in this symposium) were performed. Several other tests were also performed throughout the experiments. The mental and physiological fitness of the crew was assessed by two types of psychological tests—the Rater and Logit tests according to French (ref. 5). Cardiovascular tests and other tasks were performed pre-, during, and post-run. Every day was divided into a 4 hour period of morning tests and—after a lunch break—4 hour afternoon tests. A typical work day lasted about 9 hours, and the subjects remained busy with testing and being tested. Testing was usually completed by 1630, at which time the subjects were free to follow their own interests, make log entries, prepare supper, watch TV, listen to music, etc., after which they retired and usually slept about 8 hours. The idea was to keep them busy during working hours by different tests, etc., and also during their free time but to a lesser degree.

In this presentation it will naturally be impossible to describe all details. Instead, I will briefly describe the experiments and give the main result of each.

In the first experiment it was our intention to make the first step larger than the following ones. This was a result of earlier experience where higher levels of the rpm are more difficult to adjust to. The intention was to begin at 5 rpm but by mistake 6.4 rpm became the starting level. The crew of four less-than-normal susceptible indi-

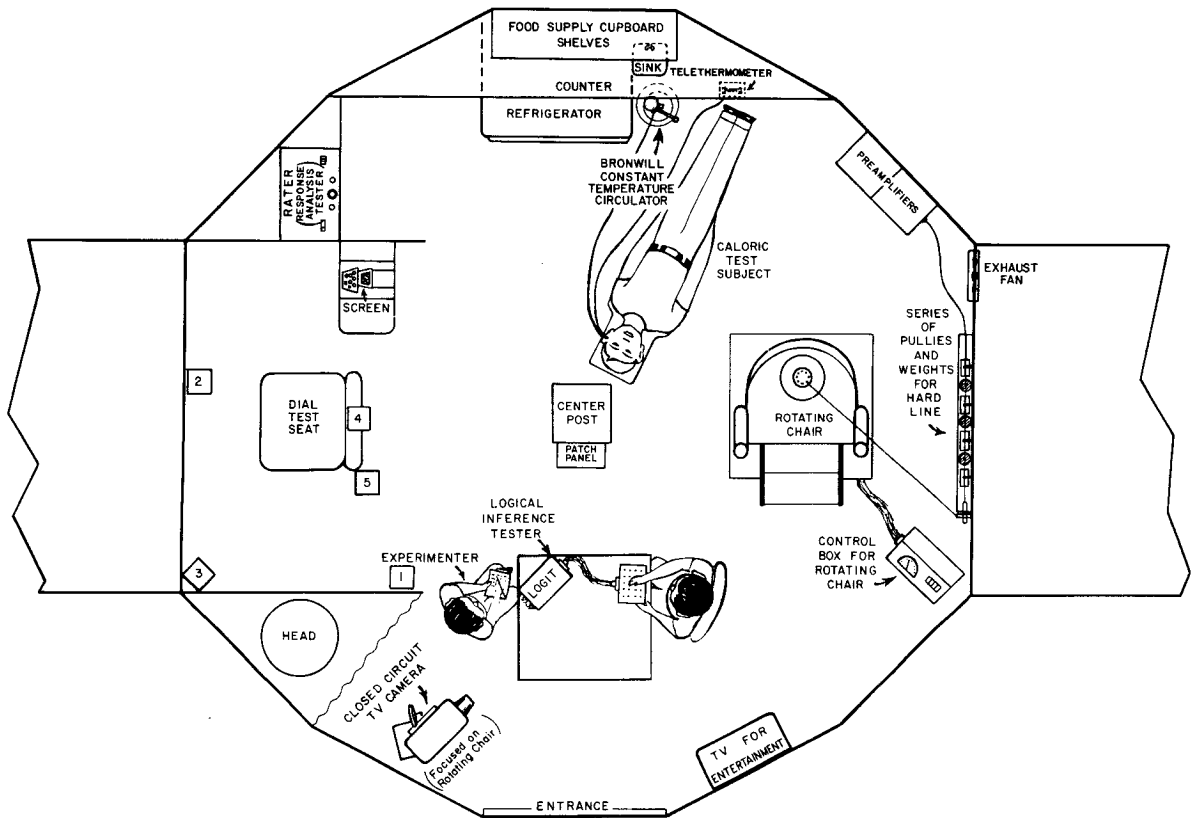


Figure 1.—Interior of SRR. Schematic drawing from above showing location of different facilities.

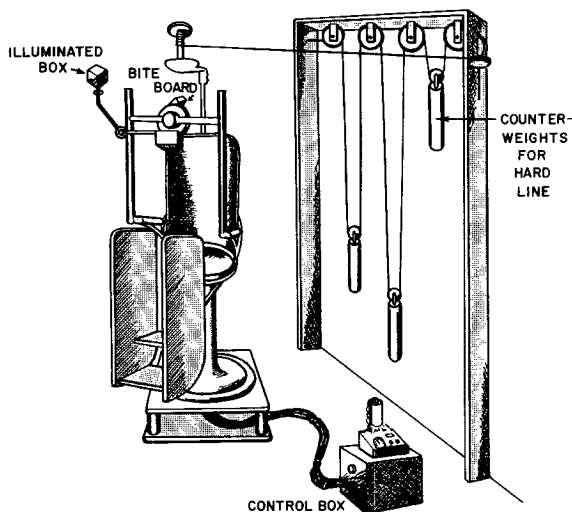


Figure 2.—Rotating chair in SRR with bite board arrangement and hardline for recording of Coriolis nystagmus.

viduals experienced the nausea syndrome. They adapted, however, and 36 hours after rotation began they could then proceed to the next higher level—8.5 rpm. This step from 6.4 to 8.5 did not increase significantly unpleasant symptoms although walking became more difficult. After 1 day they were ready for the 10 rpm level. This step was tolerated quite well. As soon as we observed *the readiness with which they appeared to adapt for nausea*, there was no reason for this experiment to continue, and after 1 day the decrease in rotation started. There was little, if any, difficulty in the descent to 6.4 again and it was almost relaxing after the much more stressful 10 rpm level. The next lower level, 3.6, was no problem either. We continued on until 0900 of the last day. For the after-run tests it was to our advantage to perform the post-run tests during the time of the day when the morning tests gen-

erally started. The step from 3.6 rpm to 0 showed a certain change concerning the equilibrium tests and slight changes in the cardiovascular tests. The whole crew was in good order but looked somewhat "exhausted" after the experiment.

The main result of the first experiment was that the stepwise procedure is to be preferred instead of going directly from 0 to 10 rpm. The differences in rpm between each step were (fig. 3): Up—6.4, 2.1, and 1.5; Down—3.6, 2.8, and 3.6. Scrutinizing the main result for fitness of the men, it was also clear that the 6.4 rpm step was too high, the 3.6 was an acceptable maximal value, and that 2.8, 2.1, and especially 1.5 rpm, were all easy to become adapted to. The experimental data suggest that, when adapted to a certain level, it is *the difference to next level* of rpm which is more significant than the absolute value of rpm.

For this second experiment we wanted to study equal steps of 3.3 rpm with another group of test subjects. In a pre-run of short duration in the SRR, they showed greater susceptibility than the subjects in the preceding experiment. When a group of volun-

teers accepted, unfortunately no one could be excluded. There was a need to confirm the result from the preceding experiment that it is the value of change of rpm which is essential for the amount of disturbances and not the level per se. By a stepwise procedure with equal steps (fig. 4) this could be more clearly studied. Each level of rpm was maintained for 24 hours or a longer time if necessary to determine adequately the test subject's ability to adapt for that level.

Earlier studies had shown that, in general, 24 hours or a little shorter time seems to be sufficient for adaptation for a 0 to 5.4 rpm step. This time for adaptation is in good agreement with other tests and subjective descriptions.

In order to compare whether the return is "easier," we wanted to make the experiment "symmetrical" for the going up and down procedure. As seen from the preceding experiment, we expected the return to be less stressful. The plans were to keep the subjects rotating at 10 rpm for periods longer than 1 day. One of the subjects, however, had great difficulty in adapting, and

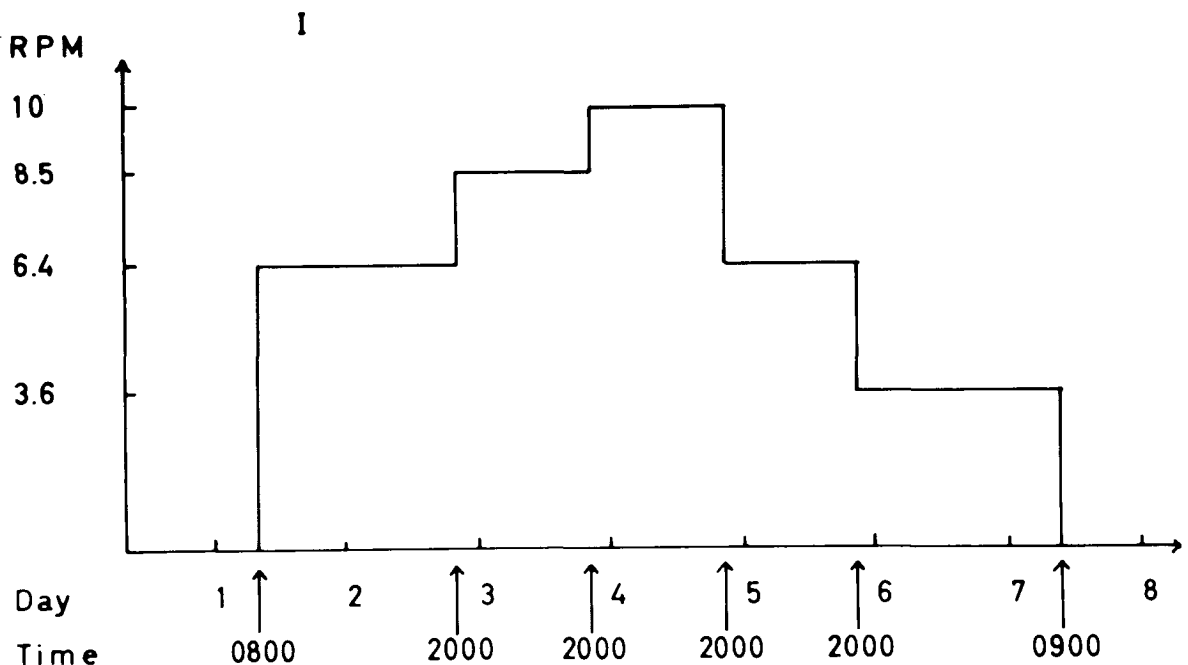


Figure 3.—Profile of first experiment. Revolutions per minute (rpm) of SRR versus time.

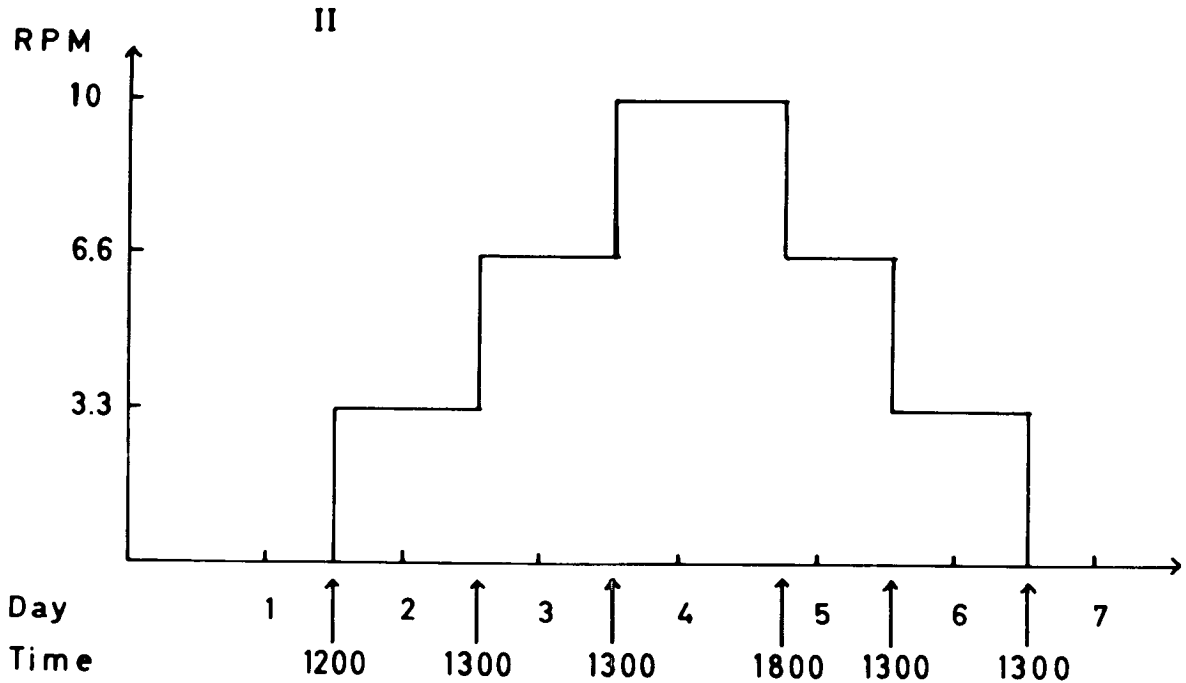


Figure 4.—Profile of second experiment.

nausea and stress became so intense and tiring for him on the third and fourth day that the going down procedure had to start. The going down procedure was generally easy. The step from 3.3 rpm to 0 differed a little from the others and was the more disturbing, but the readaptation period was short lasting.

The principal finding of the second experiment was that it was possible to reach the 10 rpm level with almost but not full efficiency of two of the men. On the way up they became too nauseated the first evening (about 3 hours after start) to be acceptable. Later they adapted more easily to change of rpm, and, in summary, those two who were more susceptible than average had great difficulty in performing the tests. The chair test with oculogyral tests and "Coriolis nystagmus test" showed little or no adaptation through the whole run.

This second experiment showed that, for a stepwise progression, a change of 3.3 rpm is an upper limit for above average susceptible individuals. The results lend support to

the idea that the difference in rpm is essential and not the absolute value of rpm.

These two experiments in the SRR pointed out that for progression to 10 and back to 0 rpm, a stepwise procedure could be used with equal steps of about 2 to 3 rpm. For the going down procedure the duration of the stay at each level of rpm could presumably be shorter than during the going up procedure. Another result was that if the step was small, the subject seemed to adapt in shorter time. This result could speak in favor of a *procedure of many small steps*, with the possibility of adapting after a relatively short time. Discussing the earlier results, the idea of a continuous increase and decrease of rpm occurred. Judging from earlier experiments, adaptation for a certain level with a limited increase of rpm of 2-3 rpm takes about 5-24 hours. A short preliminary test lasting a couple of hours was performed from 0 to about 1 rpm, after which 40 hours were chosen for the increase from 0 to 10 rpm, that is, 0.25 rpm per hour (fig. 5).

This made it necessary to increase rotat-

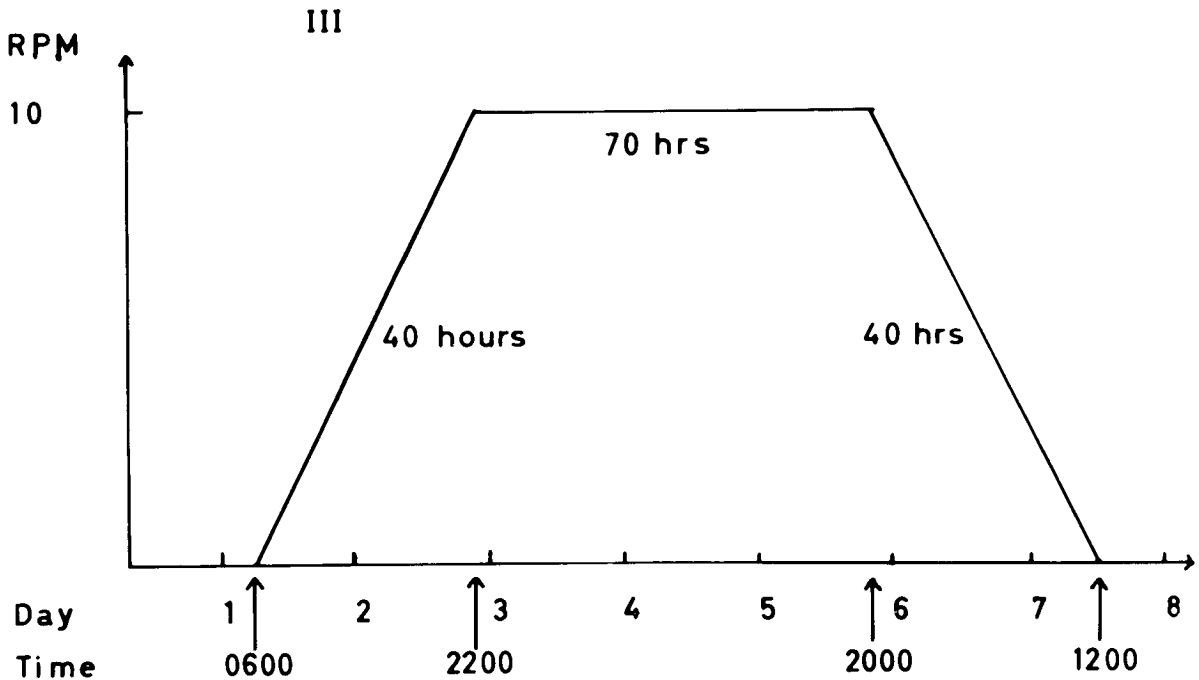


Figure 5.—Profile of third experiment.

ing during 1 night's sleep. The subjects would presumably not adapt during sleep, and we were highly interested in studying the effect during a period of sleep when they would wake up the following morning with rotation increased about 2 rpm. This had not been tried before. For a planned space station with several men on board and presumably a watch system with some men asleep and some awake, it seems necessary to know more about vestibular adaptation and sleep.

To make it possible to decide if the going down procedure is easier to "take" than the going up, we wanted to perform the continuous decrease also during 40 hours. We expected, however, that the going down would be easier to make.

The principal finding of this third experiment was that the continuous change of rpm of 0.25 each hour was well tolerated by the subjects from the start up to about 3.0 rpm and so long as they moved around performing their tasks. As soon as they rested during the free time in the evening,

problems occurred, and there were still more pronounced effects after 1 night's sleep period; all had difficulties adapting to this steady increase of rpm. Going down was better tolerated overall, and now 1 night's sleep bothered them less than during the going up period.

All subjects became incapacitated when they woke up and started moving around. Spinning at 10 rpm, all subjects started to adapt for this level and reached reasonable efficiency in 3 days. There was no adaptation for the increase by itself. Compared with the last foregoing experiments, the continuous increase of rpm in 40 hours does not seem to shorten the time of adaptation for reaching the 10 rpm level. A similar going down procedure is better tolerated than going up after a stay of 3 days at 10 rpm. The pronounced efficiency of the subjects during the continuous increase from 0 to about 3.0 rpm in 12 hours was very interesting and could point to a method of reaching a 3 rpm level in a short time and perhaps of increasing the rotation speed

when the crew is busy and of keeping the speed constant during resting time and sleep.

The results of these three experiments show:

- (1) A stepwise procedure to reach 10 rpm seems favorable.
- (2) The steps can mostly be 2-3 rpm.
- (3) Despite a stepwise procedure it seems

difficult to reach or adapt to 10 rpm in shorter time than 5 days.

- (4) The 10 rpm level is really "difficult" and stressing.
- (5) A rotating chair for oculogyral illusion tests and recording of Coriolis nystagmus can be used for estimation of adaptation.

#### REFERENCES

1. GRAYBIEL, A.; CLARK, B.; AND ZARRIELLO, J. J.: Observations on Human Subjects Living in a "Slow Rotation Room" for Periods of Two Days. *A.M.A. Archives of Neurology*, 3, 1960, pp. 55-73.
2. KENNEDY, R. S.; AND GRAYBIEL, A.: Symptomatology During Prolonged Exposure in a Constantly Rotating Environment at a Velocity of One Revolution Per Minute. *Aerospace Med.*, 33, 1962, pp. 817-825.
3. GRAYBIEL, A.; GUEDRY, F. E.; JOHNSON, W. H.; AND KENNEDY, R. S.: Adaptation to Bizarre Stimulation of the Semicircular Canals as Indicated by the Oculogyral Illusion. *Aerospace Med.* 32, 1961, pp. 321-327.
4. GUEDRY, F. E.; AND MONTAGUE, E. K.: Quantitative Evaluation of the Vestibular Coriolis Reaction, *Aerospace Med.*, 32, 1961, pp. 487-500.
5. FRENCH, R. S.: Rater and Logit: Devices for Assessing Crew Efficiency Under Stress. *General Dynamics/Astronautics*, San Diego, Calif.

#### DISCUSSION

HUERTAS: It is a fact that going by steps, adaptation might occur at a faster rate than going at large jumps. In view of your findings, where do you think that adaptation occurred compared with the known physiology of the vestibular system—at the end organ or at the central nervous system?

BERGSTEDT: I think all the feedback cycles are involved in this adaptation process.

MAYNE: I wonder whether it might be possible to consider two types of adaptation. An adaptation first as to the proper interpretation or spatial orientation, and second, an adaptation to nausea. There doesn't appear to be complete correlation between disorientation and nausea. Dr. Graybiel mentioned previously that some of the subjects have been quite successful in overcoming nausea and still show significant disorientation in the performance of various tasks. I am wondering if a study is being made of this problem.

BERGSTEDT: I think the question was best answered already by Dr. Graybiel's paper. In these studies we did not continue long enough to really answer this question. But I agree that there are different components in the adaptation process and they presumably have different, what we can call, delay times.

VON GIERKE: Did the subjects know what the program would be for their exposure and what the steps would be that they would be exposed to?

BERGSTEDT: Yes, they knew.

THOMPSON: If you wanted to operate at 6.4 rpm, to adapt the subjects up to 10, and then step back down to 6.4—is there any better adaptation in that method than by stepwise going up to 6.4 and just holding it there. In other words, go up to 6.4 and

hold it versus over adapting and then going back down to 6.4?

BERGSTEDT: Yes. They find the level of 6.4 rpm easy when they have stayed at 10 for 1 or 2 days. Whether this shortens the total time for adaptation to 6.4 rpm is not sure, however.

THOMPSON: Is there any difference in performance at 6.4 going up over and coming down or just holding it?

BERGSTEDT: We didn't make this comparison. I can't fully answer your question. Going down they performed well and especially subjectively they found it almost pleasant.

JONES: I was particularly interested in the effects of sleep upon the adaptive processes you have demonstrated. The results of a series of recent, as yet unpublished, experiments conducted in my laboratory to examine the effect of sleep upon the vestibulo-ocular reflex in man may have some bearing on your observations. Initially, on going to sleep a good nystagmoid pattern of vestibulo-ocular response to sinusoidal rotational stimulus was obtained. Subsequently, as the depth of sleep increased, there was (a) loss of the saccadic component of eye movement, accompanied by out-of-phase oculomotor response, (b) the development of slow wandering eye movements having no relation to the stimulus, and (c) the return of compensatory mode of response but one which was imprecise and without saccades. It occurred to me that the obvious modification of vestibulo-ocular response seen in these experiments might be related to the altered adaptive behavior due to sleep in your experiments.

HUERTAS: Paradoxical sleep is characterized among

other things by random motions of the eyes. How did you keep your records free of this random interference during paradoxical sleep and those eye roving motions that are present during this type of sleep?

**JONES:** The eye movement and the EEG records were completely separate from one another; thus, the "cleanliness" of the former record did not depend upon the state of sleep.

**C. SMITH:** I would like to point out something that I don't think has been emphasized before. It seems probable that there is some central control of the peripheral vestibular organs. Rasmussen and Gacek pointed out that there are efferents going from the lateral vestibular nucleus out to the vestibule, and Rossi recently has confirmed this, i.e., that there are efferents going from the lateral vestibular nucleus as well as from an interposed nucleus. We don't know where they end but it is probable that they are related to the small granulated nerve fibers or nerve endings Dr. Engström discussed in his paper. We have found that both in rodents and in the squirrel monkey these nerves have synaptic terminals or synaptic contact with the vestibular hair cells, with the vestibular nerve fibers, and also with

the vestibular nerve endings. Even though the endings of these efferent nerves have not been well worked out yet, I think we might speculate that probably there is control of the peripheral organ from a central source.

**BERGSTEDT:** Another detail in this slow rotation experiment in relation to what Dr. Jones said is that if we stop the SRR, as we usually have done, two times a day and the subjects keep the head immovable, they have no unpleasant feeling of vertigo as long as they keep the head immovable. But as soon as they move the head even the slightest, they feel nauseated. So in these experiments we used a new procedure and allowed them to stay in the room when it stopped and keep the head immovable. Then we took the first man and allowed him to perform the aftertests. After this the No. 2 man started and so on. The general impression from early experiences seems to be that by keeping the head immovable you also preserve the adaptation for the Slow Rotation Room. How long a time it takes to subside or go down, we do not know. Perhaps it would last for 1, 2, or 3 days if the subjects kept their heads immovable. But at least for several hours the adaptation seems to be preserved.

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# Biochemical Responses to Vestibular Stimulation

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## SUMMARY

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This paper reports a series of biochemical parameters which was measured in an attempt to focus critical areas of biochemical responses to motion sickness attending prolonged exposure to a moving room environment. The most striking finding in the study is the demonstration of an increased glucose utilization by normal subjects in comparison with labyrinth-defective volunteers. These findings are consistent with a hypothesis drawn from a previous study (pilots, 1962) that changes in glucose tolerance may be caused by increased muscle tonus in normal subjects. The increase in tonus may be a direct result of labyrinth stimulation. This hypothesis is supported by determination of pH which showed a decrease, by an increased  $pCO_2$ , and by a steady or slightly decreased  $pO_2$  value for normal subjects. The LDH value was also increased in the pilot group only. These are the types of responses that would be expected. The L-D group should not show such a response. With a possible exception to an unexplained response in  $pO_2$  in the pilot 1964 group, this hypothesis is borne out. An additional definitive experiment with especially careful control of diet, work regimen, and subjective observations is indicated to assess further the differences between the responses of normal and labyrinth-defective men when subjected to vestibular stimulation.

Author

## INTRODUCTION

A search of the literature has not revealed any extensive biochemical studies on man wherein vestibular stimulation of an assessable magnitude with a prolonged exposure was performed. The reports of Guedry et al. (ref. 1), Collins (ref. 2), and Collins and Guedry (ref. 3) describe the psychological and general performance changes in normal men when the conditions of the experiment ranged between 3 and 10 rpm.

Tyler and Bard (ref. 4) in their review article detail much of the physiology of the etiology of motion sickness and point out the resistance of men, whose labyrinths had been destroyed by disease, to motion sickness. Colehour and Graybiel (ref. 5) report the excretion of steroids and catechol amines on both normal persons and deaf subjects following parabolic flight stress. These

findings likewise show a difference between the two groups with normal subjects showing a greater response than the labyrinth deficient men.

The general lack of biochemical data indicates a definite requirement for more information regarding the biochemical responses which accompany the changes in ability to perform tasks satisfactorily in the early phases of the prolonged experiment and to help account for the ability of the individual to adapt to the abnormal environment after 2 or 3 days exposure. In 1962, four young healthy student pilots volunteered for a two week "tour" in the Coriolis Chamber earlier described in a preceding paper by Graybiel et al. This run was for 12 days of rotation at 10 rpm with samples of blood and urine collected at intervals. It was expected that 10 rpm would



produce a certain degree of motion sickness in the pilots, and the biochemical parameters were designed to measure such conditions as intestinal absorption, excretion of calcium and electrolytes, blood catechol amine assays, serum enzyme activities, rates of glucose metabolism, and other parameters which might assist in evaluating the observed responses and work performance capability during the tests. The results of these preliminary evaluations were sufficiently encouraging that a more elaborate experiment was planned to include a comparison of the apparent normal individual with labyrinth defective (L-D) subjects.

The labyrinth defective group was composed of four totally deaf male students at Gallaudet College. Also with this group was included one man who had demonstrated a high level of resistance to motion sickness during previous short runs in other experimental programs.

#### DESIGN OF EXPERIMENT

The subjects were required to be available for both pre-test and post-test evaluations with a 3-day period in each case stipulated. Prerotational data on each man served as a base for comparison of the responses of the rotational period during which the vestibular apparatus was being stimulated. The period after the termination of the rotational experience served as a further comparison for evaluating rates of return to normal and also to determine the degree of response during that period when the subject was readjusting to his normal environment.

Two blood and three urine collections were made during the "control" periods with blood samples being taken on the first (complete 24 hour) day and again on the fourth, seventh, tenth, and twelfth days of rotation. Glucose tolerance tests were also done at these same time intervals.

The diet fed the subjects during the experiment was as closely regulated as possible with major variations in the quality of the diet eliminated. It was not possible, however, to weigh the individual portions of

the food but these were estimated as closely as possible and recorded. Fluid intake records were kept for each 24 hour period.

Urine collections were made to allow total collection of voidings, extending between 0800 one day through 0800 the following day. Fasting blood specimens were collected at 0830-0900 on each day of sampling to minimize diurnal variations in blood chemistry responses. During the time when it was necessary to stop rotation for servicing or other requirements, the subjects were fixed in a neck harness arrangement which did not allow movement of their heads. In the room the men performed normal work cycles.

#### EXPERIMENTAL OBSERVATIONS

In general it was noted that both the pilot groups of 1962 and 1964 showed similar responses to the rotational environment. The rates of rotation were the same (10 rpm); however, the 1962 study was made in a room which has approximately one-half the diameter of the newer Coriolis Chamber Facility. For this reason the gravitational effects are somewhat greater in the second experiment. In both experiments all subjects showed a varying degree of motion sickness early in the first day with nausea and vomiting during the first 24 hours in most cases. The feeling of discomfort persisted for 2 to 3 days at which time an adjustment to rotation was noticed. No motion-sickness drugs were used. In general, the normal subjects drastically cut their fluid intake during the first 3 days of rotation and thereafter resumed liquid intake at approximately the same level as during the pre-test control period. Food intake followed a similar pattern; however, during the rotational phase most of the men reported an increased desire for starchy foods, bread, and fats. The normal subjects also complained of a great feeling of fatigue while on board and it was readily apparent to all observers that the degree of alertness of the men was reduced during the rotational period. These changes have also been reported separately as re-

gards the ability of the men to perform their assigned tasks and to respond to psychological testing programs with the facility, observed during the pre-test control period. The biochemical studies in part offer an explanation for these changes.

#### URINE VOLUMES AND EXAMINATION

Both groups of pilots showed a similar response throughout the program. During the first 24 hours of rotation all decreased their urinary output with the decrease maintaining for 3-5 days. In those pilots who experienced vomiting, the urine output decreased to the greatest degree. This probably reflects the reluctance on the part of the individual to increase his fluid intake since it was during this period that the smallest fluid intake volumes were recorded.

In comparison, the L-D subject group showed a urine volume which appeared consistent with their fluid intake which remained near the control level during the entire experimental period. Midway through the experiment, an increased fluid intake was recorded with urine levels remaining near control levels. This was accounted for by partial failure of the air conditioning system in the Coriolis Chamber with the increased temperature resulting in profuse sweating by the subjects.

The examination of the urine for specific gravity, total solids, pH, protein, sugar, ketones, and microscopic examination did not reveal anything remarkable in any test group although one pilot did show a tendency for albuminuria which showed approximately 30-40 mg protein per 100 ml urine during the first 2-5 days of rotation.

Excretion of sodium, potassium, and calcium in the urine did not show any changes which might be described as significantly altered. In the absence of a fully controlled balance study, observations at this point would indicate that electrolyte and calcium excretion are the result of a normal excretion pattern attending the usual conditions of nausea and anorexia. No tendency for increased calcium excretion was noted. The

greatest change in calcium excretion (a decrease) was shown by the pilot who had the strongest response to nausea and at the same time the largest reduction in 24 hour urine volume.

#### BLOOD CHEMISTRY EXAMINATIONS

##### Catechol Amines

During the 1962 test the blood catechol amines (epinephrine and norepinephrine) were included as a potential measure for prolonged stress. The determinations did not show any significant changes at any point of the test program. In many collections, the level of epinephrine was below detectable limits. This examination—costly in blood requirements and difficult to perform—was dropped from the 1964 program in favor of the urinary determinations for stressor substances.

##### Xylose Tolerance

This test, again performed on the 1962 subjects, utilized the 5 gram oral dose of xylose and was included as a possible measure of the ability of the intestine to absorb food. Five hour excretions of xylose for the various test days for the subject did not show any significant variation and were considered to be within normal experimental limits of variation.

#### BLOOD ENZYME EXAMINATIONS

Due to the early difficulty in accomplishing adequate muscular coordination in the moving room it was expected that some response would appear with measurement of lactic dehydrogenase (LDH), glutamic-oxaloacetic acid transaminase (SGOT) and alkaline phosphatase. In the 1962 study the maximum changes were shown in the LDH component with increases demonstrated within the first 24 hours of rotation and again at the end of the experimental period. The order of magnitude was from a base average of 175 units LDH to 425 following 24 hours of rotation. Levels had dropped to control values by the second day but began to rise again on the seventh day of rotation. By the eleventh day of the experi-

ment the LDH level attained a peak of 745 units. During the 7–11 days of rotation the men were able to move freely in their environment, and the testing program for job performance was likewise increased. An abrupt increase in LDH levels was again shown when rotation was stopped and the pilots went through a period of adjustment to the nonrotational environment. During this period it was observed that all normal men experienced considerable difficulty in locomotion, with a matter of hours being required before any feeling of security was felt for walking.

The experimental subjects of 1964 showed essentially the same pattern except that the first peak in LDH activity was not shown. Since there was a 24 hour difference in sample collection, it is possible that the very early response may have been missed. For comparison with the L-D subjects, figure 1 shows the responses between test groups in the 1964 study. It is noted that the L-D group did not show the abrupt response which was shown by both the 1962 group and the 1964 normal subjects. Method used for LDH determination was that of Wroblewski and LaDue (ref. 6), with normal ranges being recorded between 200–400 delta optical density units (1 ml serum) per minute  $\times$  1000.

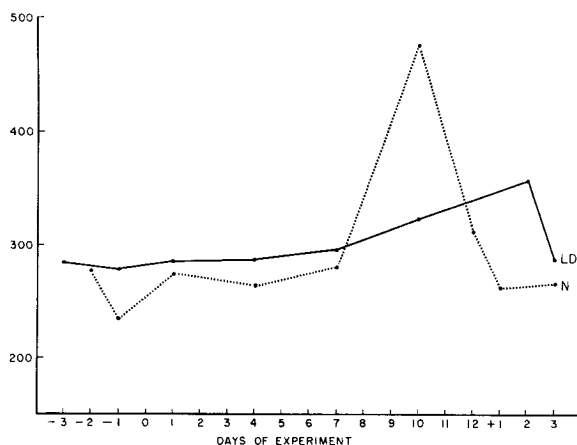


Figure 1.—Comparison of lactic dehydrogenase ( $\Delta_{OD}$ , 1 ml serum/min  $\times$  1000). Responses from normal and labyrinth deficient subjects during experimental period (1964).

Further evaluation of this response was made by Papadopoulos (unpublished data obtained at Walter Reed Army Institute of Research) who performed isoenzyme separations of LDH. It was apparent from his preliminary examination that the labile components of the plasma, generally attributed to muscle in origin, were considerably reduced in the stored specimens.

The alkaline phosphatase, (ref. 7), shows normals ranging between 17 and 40 micromoles substrate consumed per liter per minute. In figure 2 the responses of the 1964 normals versus L-D subjects are shown. The L-D group was slightly higher and more steady throughout the test period. In light of what is presently known concerning these responses it is questionable if significance should be attached to the minor fluctuations of the alkaline phosphatase observed in the normal subjects. A very recent release by Gardner et al. (ref. 8) reports that, in athletes on treadmill exercise, the serum enzyme levels increase with increases dependent to considerable degree on the physical fitness of the subject. They report, using a treadmill at 11° angle at 5 mph, that SGOT, malic dehydrogenase (MDH), aldolase, and LDH increase with the greatest changes being shown in the untrained subjects. Perhaps there is a corollary between these experiments and the different responses observed in the moving room by the two groups reported herein.

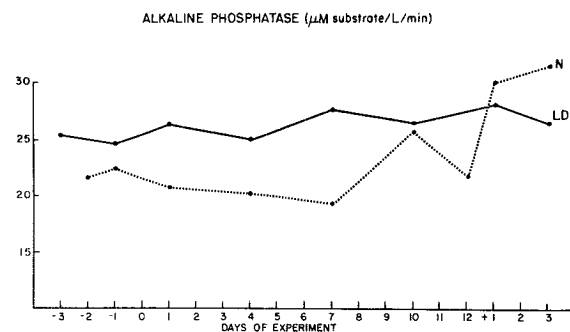


Figure 2.—Alkaline phosphatase ( $\mu\text{M}$  substrate/L/min) responses in normal and labyrinth deficient subjects during experimental period (1964).

GLUCOSE METABOLISM

It was felt that a measurement of glucose utilization by the various groups would serve as a measure of changing metabolic requirements. Evaluations were made by Mertz (ref. 9). Fifty milliliters of 50 percent dextrose were injected intravenously after an overnight's fast, and the subsequent decline of excess blood sugar levels was determined. The gross composition of diet was unchanged during the experiments and with the exception of rotation all tests were conducted under as near identical conditions as possible. With these precautions, rates of glucose removal remain unchanged in individual subjects; thus, any significant changes must be attributed to the effects of rotational environment.

The qualitative responses of the groups were quite uniform. These are shown in figure 3. The fasting blood glucose levels in neither the L-D nor normal groups showed anything of a spectacular nature although the L-D group showed less fluctuation. However, there were definite changes shown in the rates by which the two groups of pilots removed excess glucose from the blood. In each case there was a decrease in utilization on the first day of the experiment, with a considerable increase in utilization shown by the fourth day of rotation. These rates

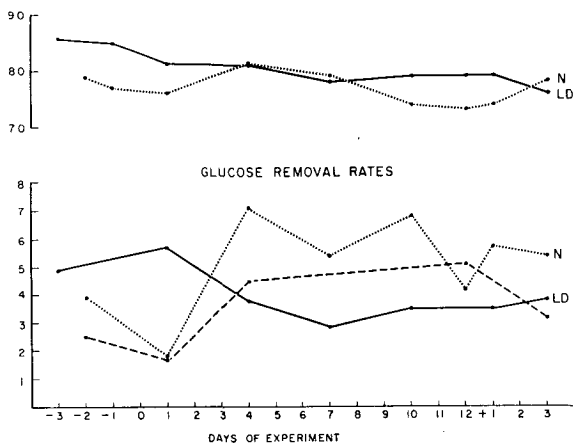


Figure 3.—Glucose utilization in normal and labyrinth deficient subjects during experimental period (1964).

were maintained at elevated levels throughout the stress period. The L-D group did not elicit this response but instead showed an increase during the first day of rotation and a decreased utilization during the balance of the testing period. These changes are further shown in figure 4 where the percentage change from baseline is recorded. It is amply demonstrated that there are gross differences between the L-D group and the two pilot groups which show essentially the same response. Although results reported are an average of the four for each test group, the pattern was found for every subject, with the only difference being the time of the change occurring during the test. It was also interesting to note that the "functional L-D"—the man who was highly resistant to motion sickness—responded in a manner parallel with the pilot group, however to a somewhat lesser degree.

BLOOD pH, pCO<sub>2</sub>, and pO<sub>2</sub>, OBSERVATIONS

These parameters were determined by the use of the Spinco Multiple Cuvette (ref. 10) with the usual precautions of calibration of equipment and electrode responses before each run. In figure 5 the observations are shown of the pCO<sub>2</sub> changes of the L-D and pilot (1964) group. During the early stages of the stress period the normal subjects showed an abrupt increase in pCO<sub>2</sub> which by the fourth day of rotation had reached a value of 70 mm Hg pCO<sub>2</sub>. Thereafter the level stabilized at a level in excess of the control value (55 mm Hg) and showed

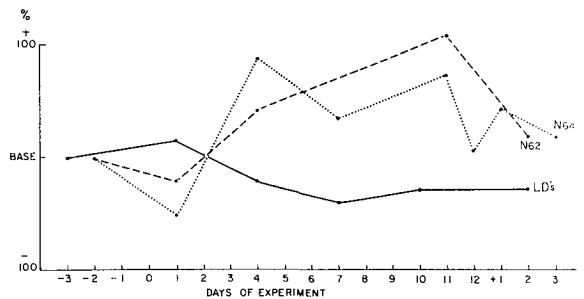


Figure 4.—Percentage change in glucose utilization in two groups of normal subjects (1962, 1964) and labyrinth deficient subjects.

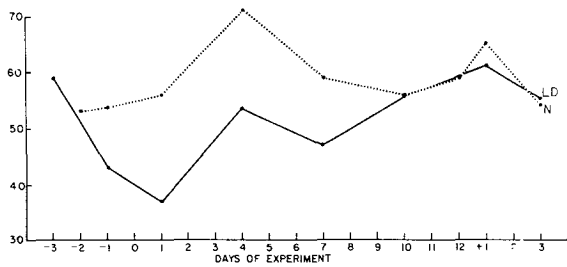


Figure 5.—Blood carbon dioxide (pCO<sub>2</sub>-mm Hg) changes in normal and labyrinth deficient subjects during experimental period (1964).

a second increased response when the men were released from the room. In contrast, the L-D subjects showed a drop of pCO<sub>2</sub> on the first day of rotation and thereafter remained within the normal limits of the determination, until toward the end of the rotating period when they too showed an increase to the level of 60 mm Hg pCO<sub>2</sub>.

Figure 6 lists the values of pH determined. It is noted that the pH increase in the pilot group is slight when compared with the pCO<sub>2</sub> of the fourth day; therefore, it is apparent that the alkalosis is compensated. Such appears to be the case for other fluctuations of pCO<sub>2</sub> throughout the rest of the test period. Again the L-D's do not show the same pattern of change shown by the pilots, but actually show an opposing response.

It was hoped that a determination of pO<sub>2</sub> (venous) would help explain the variation shown in the glucose utilization. The re-

sponses of the two pilot groups in this case were not the same (fig. 7) since the 1964 pilots showed an abrupt increase in pO<sub>2</sub> on the seventh and tenth days of the experiment. The 1962 pilots did not elicit any such response and after an initial increase at the first day of the run dropped to a low normal level which they maintained throughout the balance of the test period. The L-D group did show a marked difference from either pilot group. The venous oxygen levels for the L-D's showed a continuous increase through the test period, with a drop upon coming from the room but a rebound upward at the end of the evaluation. At the present there is not adequate information regarding activity patterns and so forth to assess the differences in pO<sub>2</sub> between the various groups; however, it is apparent that some difference in metabolic pattern or work regimen has an important role in the responses shown.

Total protein measurements were done by the Biuret method (ref. 11). The total protein value in the L-D group showed a gradual decrease (fig. 8) through the fourth day of rotation, with a return to a steady normal level by the seventh day of rotation. The pilot (1964) group showed the increase which would be expected with a moderate hemoconcentration at the end of the first day of rotation; however, they continued to drop throughout the balance of the test period, with a net decrease of approximately one gram of protein per 100 ml plasma dur-

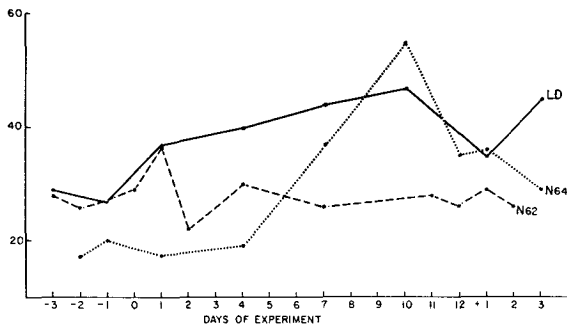


Figure 6.—Blood pH values in normal and labyrinth deficient subjects during experimental period (1964).

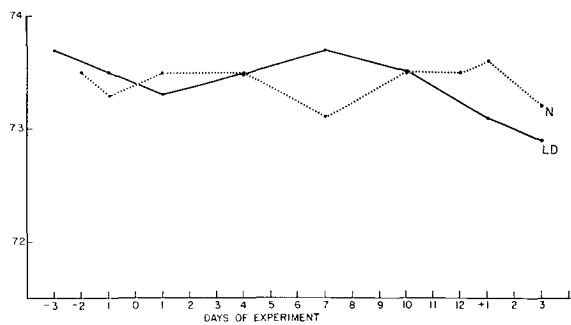


Figure 7.—Blood oxygen (venous, pO<sub>2</sub>-mm Hg) in two groups of normal subjects (1962, 1964) and labyrinth deficient subjects.

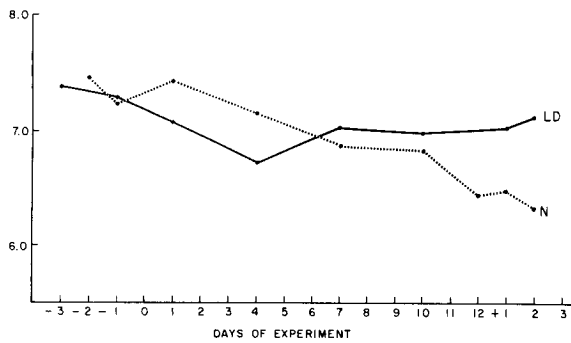


Figure 8.—Total protein measurements (grams/100 ml plasma) in normal and labyrinth deficient subjects during experimental period (1964).

ing the test period. In this examination, no electrophoresis was accomplished; therefore, nothing can be said regarding the variation of protein components.

ADDITIONAL EXAMINATIONS

The hemoglobin (fig. 9) was determined during the program on all samples drawn. There was an increase in hemoglobin concentration at the beginning of rotation and again when the subjects entered their normal environment. Both the L-D's and the pilots (1964) showed this response, with the magnitude being essentially the same in each case.

The absolute eosinophil count was done as a means of assessing the degree of stress (fig. 10). Again both the L-D's and pilots showed a response; however, the pilots' response was delayed in comparison with the L-D's and remained higher throughout the

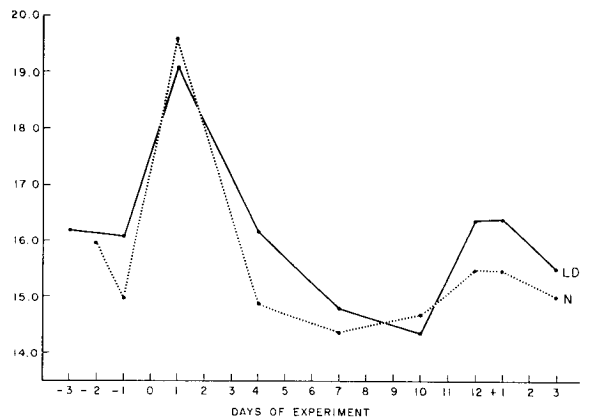


Figure 9.—Hemoglobin determinations (grams/100 ml blood) in normal and labyrinth deficient subjects during experimental period (1964).

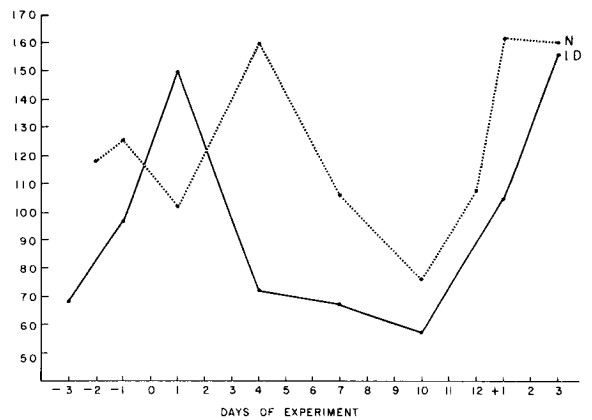


Figure 10.—Absolute eosinophile count in normal and labyrinth deficient subjects during experimental period (1964).

experiment. Both groups show a response to stress since at each of the stress points a positive response was shown.

REFERENCES

1. GUEDRY, F. E., JR.; KENNEDY, R. S.; HARRIS C. S.; AND GRAYBIEL, A.: Human Performance During Two Weeks in Room Rotating at 3 RPM. *Aerospace Med.*, 35, 1964, pp. 1071-1082.
2. COLLINS, W. E.: Task Control of Arousal and the Effects of Repeated Unidirectional Angular Acceleration on Human Vestibular Responses. *Acta Otolaryngol*, 190, Stockholm, 1964, pp. 1-34.
3. COLLINS, W. E.; AND GUEDRY, F. E., JR.: Arousal Effects and Nystagmus During Prolonged Angular Acceleration. *Acta Otolaryngol* 54, Stockholm, 1962, p. 349.
4. TYLER, D. B.; AND BARD, P.: Motion Sickness. *Physiol. Rev.*, 29, 1949, p. 311.
5. COLEHOUR, J. K., AND GRAYBIEL, A.; Urinary Excretion of Corticosteroids and Catecholamines in Normal Persons and Deaf Subjects With Bilateral Vestibular Defects Following Acrobatic Flight Stress. *Aerospace Med.*, 35, 1964, pp. 370-373.
6. WROBLEWSKI, F.; AND LADUE, J. S.: Lactic Dehydrogenase Activity in Blood. *Proc. Soc. Exp. Biol.*, 90, 1955, p. 210.
7. Alkaline Phosphatase. Sigma Chemical Company Tech. Bull. 104, rev. 1960.
8. GARDNER, G. W., BRATTON, R.; CHOWDBURY, S.

- R., FOWLER, W. M.; AND PEARSON, C. M.: Serum Enzyme Levels in Athletes After Exercise. *J. Sports Med. and Phys. Fitness* (reported in *Med. Tribune*, Dec. 28, 1964).
9. MERTZ, W.; AND SCHWARTZ, K.: Impaired Intravenous Glucose Tolerance as an Early Sign of Dietary Necrotic Liver Degeneration. *Arch. Biochem. Biophys.*, 58, 1955, p. 504.
10. Beckman Instruments. Spinco Division, Palo Alto, Calif.
11. HENRY, R. J.; SOBEL, C.; AND BERKMAN, S.: Interferences with Biuret Methods for Serum Proteins. Use of Benedict's Qualitative Glucose Reagent as a Biuret Reagent. *Anal. Chem.*, 29, 1957, p. 1491.

### DISCUSSION

HUERTAS: I think this study is an eye opener because so far we have heard in most papers that the dramatic symptoms of vestibular stimulation and motion sickness are taken as the end of the stress. If a person is not vomiting or with a stomach ache or a loss of equilibrium, then he is adapted to rotation and any work ends. You have shown us that the different biological parameters in the organism change and that some of them change even after 7 days. This intelligence, I think, is of paramount importance and constitutes a word of caution as to when we are going to call an organism adapted.

GUALTIEROTTI: You have a large change in  $pCO_2$  in the blood and apparently no change of pH which is rather strange, isn't it? Was it because in the meantime you have a large increase in muscular work? That means you have a very large ventilation increase. Is that the case?

NOBLOCK: I have consulted with our respiratory physiologist regarding this. This looks like our compensating mechanisms have been able to handle it. It's probably a relatively acute change and one that the buffering action has been able to handle.

GUALTIEROTTI: Did you make any hemoglobin separation tests?

NOBLOCK: We did not do hemoglobin electrophoresis. There are additional tests to be made and this is one of them.

GUALTIEROTTI: What about metabolic rate? Did you do a BMR?

NOBLOCK: No. Dr. Graybiel and I have discussed this whole problem. We would really like to know the entire energy utilization pattern during this period of time. Peter Beck (School of Aviation Medicine) will be a great asset to us in this area. Probably he will do the work, and we'll look over his shoulder.

GUALTIEROTTI: The last point. You probably had a considerable sweating. That means a very large loss of sodium. Of course, if you drink water, you lose sodium; and if you don't have additional sodium to the diet, you will get the very important syndrome of sodium depletion. Is that the case?

NOBLOCK: Unfortunately I can't tell you, and this is one of the problems that we are all aware of. If we don't really know the dietary intake on these patients, I can't answer that.

GUALTIEROTTI: I would suggest that you give sodium tablets just to avoid the low sodium syndrome which

is extremely important to the metabolism and in general.

NOBLOCK: Without prior data it was difficult to anticipate all the areas needing study. In the future we will have these data to fall back on and they will help suggest what other steps we should take.

C. CLARK: Do you feel that any of these curves with changes toward the end of the experiment indicate beginning signs of decompensation?

NOBLOCK: Let's put it this way. I couldn't say at this point if it is a sign of decompensation because the actual activity patterns of the people changed considerably during this period of time.

WHITE: Is there any relation worked out between this glucose tolerance test and the PBI (the protein bound iodine)?

NOBLOCK: Most of the work that has been done by this test has been for evaluation of diabetes and for the diagnosis of acute liver disease. I don't think Walter Mertz has done any work in terms of assaying this against the PBI or the thyroid TSH, or various other tests.

LEVINE: Some years ago I learned a very lovely word iatrogenic, as in iatrogenic disorder, which refers to diseases induced by the physician. This was in connection with a sensory deprivation study, but I would like to know what sort of pre-indoctrination all the subjects received. Were they given to understand that they might get sick or in the case of the L-D's were they led to understand that they would have no trouble at all?

KENNEDY: The subjects were all Naval Academy graduates. They were here awaiting flight training because there were some hurricanes during the summer, and it set back the flight program. They were assigned to School of Aviation Medicine to serve as tabulators, subjects, whatever there was reason to use them for. From this population we requested as many volunteers as possible for this particular run. They were given a complete run-down of what was going to happen to them. We told them that people in the past had gotten sick. This had happened at this rpm. These particular things can happen to you. We made every effort to maintain motivation at a very high rate by publicity and good food; we told them that we would give them time off afterwards. We told them that the fact they volunteered would go into their service

records and the fact that they got sick would not. In effect, we attempted to maintain as high a motivation as possible. They were representative of the population from which they were drawn, I think, but if anything, they were a little bit brighter and a little bit more highly motivated. They were in absolutely tip-top physical condition because they had all graduated from Naval Academy about a month previous.

I wanted to say one more thing about perspiration. At least on the first day there probably was an increase as one of the motion sickness symptoms. The room temperature throughout the entire run was maintained at very comfortable levels. As to terms of the amount of salt they took in, the food intake might serve as an index. Their normal breakfast was increased as the run progressively increased. They ended up with, for instance, half a pound of bacon or pork sausage for breakfast, three or four eggs, a quarter pound of butter split five ways, and a couple glasses of milk.

LEVINE: How long did these symptoms last from the first day?

KENNEDY: They all ate the first day but differing amounts depending on how uncomfortable they were and how sick they were. In a day or two they were all eating very well; certainly by the third day.

LEVINE: How about vomiting? How much did they vomit?

KENNEDY: SH vomited once, WO eight times, LI and WI did not vomit.

POLLACK: The averaging out of these values in vomiters and nonvomiters, I think, puts you at a distinct disadvantage in attempting to interpret some of the results, particularly when it comes to sodium excretion, etc.; so, I am not at all sure that you are being fair with yourself in presenting the data in this type of expression. Were these men weighed daily? Is there any indication of change in body weight?

KNOBLOCK: Yes, they were weighed. Unfortunately when I wrote this, I did not have those data available. Certainly you are quite right. I was interested more here in presenting trends, and if you took extremes in this case or in the second case (the one man that got quite ill), his patterns represent the extreme. The data do represent a mean value for each group; however, there was no overlap on an individual basis between members of the normal and L-D groups. The other thing I forgot to mention. A subject in one experiment had slightly suppressed function of the semicircular canals, and he was highly refractory to motion sickness. This man's response is somewhere intermediate between the L-D's and the normal group when it is graphed out.



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## Stress Measurements in Normal and Labyrinthine Defective Subjects in Unusual Force Environments

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Some of the most stressful effects of space-flight are produced by the changing force environment in the transition from one to zero gravity after lift-off and the reverse cycle in reentry. Although these exact conditions are impossible to attain on Earth, many reports have been made of changes in blood and urine composition on similarly stressed occupants of fixed wing aircraft (refs. 1, 2, 4, 8, 10, 16, 17, 22-24, 26, and 27). The effects of the systemic and neurogenic stresses in these airmen have been measured as increased levels of 17-hydroxycorticosteroids and catechol amine compounds. In this report three experiments will be described in which subjects were exposed to different force backgrounds, and the excretion patterns of the above compounds measured.

Although it is difficult to define specific causative factors for the increases in corticoids, Mason has concluded (ref. 25) that it is due to influences of a general psychological nature in which undifferentiated stress is sufficient to cause ACTH release. More recently, in conditions of chronic stress, it has been described (ref. 9) in terms of the individual's "coping" ability.

The conditions for the release of the catechol amines, epinephrine (E) and norepinephrine (NE), have been more specifically defined. Several years ago Elmadjian noted (ref. 6) that the increase in catechol excretions was related to specific emotional and physiological states. Goodall has shown

(refs. 11 to 13) in centrifugation and in simulated zero gravity, water immersion experiments that epinephrine elevations are related to anxiety, as was described by Von Euler (ref. 8), while norepinephrine release can be associated more closely to peripheral effects. That NE can be stored and released at the sympathetic nerve endings, as originally discovered also by Von Euler (ref. 7), appears to be further verified in the pathological condition of neuroblastoma (refs. 15 and 29). Even with a subtle change in force environment which produces only mild circulatory effects, changes in norepinephrine excretion have been noted in orthostatic hypotension (refs. 18 and 21), tilt table tests (refs. 18 and 28), and in simulated zero gravity by water immersion (ref. 13) as well as in the more stressful condition of centrifugation (ref. 11).

In one of the experiments reported here determinations of leukocytic elements in the blood were made as secondary indicators of stress presumably as a result of ACTH and corticoid release. These effects, described some years ago in the "alarm reaction" of Selye (ref. 5), are well known and follow the stressful incident after a few hours' interval.

Although the experiments to be described were somewhat different in nature, all were designed to produce bizarre stimulation to vestibular mechanisms. At least one and possibly more stimuli were present in each

experiment, but no attempt was made to establish a cause-effect relationship between a particular stimulus and its specific response in the organism. Rather, the purpose was to expose the distinct subject groups to various accelerative forces and measure the overall response in the parameters described above.

The test subjects for the experiments fell into two distinct classifications: (1) a normal group from the healthy, young-adult, male population and (2) a group of men of similar age range, most of whom had had meningitis at an early age and one individual who had had mastoiditis. The latter group required very high sound levels for perception, were negative to caloric testing, and, therefore, are presumed to have nonfunctioning vestibular organs. This group is referred to as the labyrinthine defective or L-D group.

The methods used in the experiments for measuring urinary excretion rates were that of Crout for the catechol amines (ref. 3) and that of Kornel for the corticoids (ref. 19). In one experiment uropepsin assays were made, and these were done by the method of Gray (ref. 14). Leukocyte counts were done by conventional methods.

#### EXPERIMENT 1

In this experiment both subject groups were exposed to an intensive set of acrobatic flight maneuvers lasting approximately 30 minutes. All the subjects were naive as far as this type of flying was concerned. Not only was disorientation complete during this first time experience but the four G pullout in one of the maneuvers was a very stressful incident for the uninitiated subjects. The flight was terminated in the case of three normal subjects because of nausea and four others of the same group complained of malaise at the termination of the flight. That the flight was distressing to the normals was evidenced by statements to the effect that "flying was not for me" although all had intended to make flying a career at an earlier time. The L-D subjects, on the other

hand, were asymptomatic during and after the flight and some even expressed desire to take another ride at the conclusion of their first experience.

In order to make a biochemical evaluation of the effects of the flight, the stress hormone determinations were made on urine samples taken on the night before the flight and compared to 6-hour excretions immediately postflight and also to similar measurements on a nonflight day. Samples were collected in this way because it had been shown that (1) the night resting samples made consistent reference points for each individual, and (2) practically all the increase in stress hormones was excreted within a 6-hour period after a single stressful incident. Figure 1 shows the catechol and steroid excretions plotted as micrograms per hour. The normals and L-D's both had slight increases in catechol excretion rates in the afternoon compared to night resting samples on the control days. On flight day the normals had elevated levels postflight, but the L-D rates were about the same. The corticoid excretions told about the same story except that the L-D's had relatively great increases between night resting and afternoon samples on the control day but increases of only the same order on flight day. In this experiment, uropepsin measurements were made, and although the afternoon rates were generally higher than the night resting values, the changes could not be related to the flight nor to the difference in subject groups.

Table I shows the statistical evaluation of the results. Although slight mean differences for the catechol excretions were seen in figure 1 between resting and afternoon samples on the control day, statistical analyses did not permit significance with  $p$  values greater than 0.1. On flight day the normals had highly significant increases in catechols while the L-D's did not show change. Resting samples from both days were compared also in order to see if there was a measurable anxiety on the night before the flight; such was not the case. Probably the most meaningful comparison is shown in

Table I.—Statistical Evaluation of Urinary Stress Hormone Levels in Normal and Labyrinthine Defective Subjects After Acrobatic Flight

[E = epinephrine; NE = norepinephrine; T = total catechol amines; F = free 17-OH corticosteroids; C = conjugated 17-OH corticosteroids; TC = total 17-OH corticosteroids; TU = total uropepsin; FD = flight day; NF = nonflight day]

Subjects	Mann-Whitney "p" Values											
	Resting (NF) vs. afternoon (NF)			Resting (FD) vs. afternoon (FD)			Resting (NF) vs. resting (FD)			Afternoon (NF) vs. afternoon (FD)		
Catecholamines												
Normal Lab. Def.	E	NE	T	E	NE	T	E	NE	T	E	NE	T
		>.1	>.1	>.1	.005	.005	.005	>.1	>.1	>.1	.05	.05
	>.1	>.1	>.1	>.1	>.1	>.1	>.1	>.1	>.1	>.1	>.1	>.1
17-OH corticosteroids												
Normal Lab. Def.	F	C	TC	F	C	TC	F	C	TC	F	C	TC
	>.1	>.1	>.1	.01	.005	.005	>.1	>.1	>.1	.10	.02	.02
	>.1	.05	.04	>.1	.005	.005	>.1	>.1	>.1	>.1	>.1	>.1
Uropepsin												
Normal Lab. Def.	TU			TU			TU			TU		
	>.1			>.1			>.1			>.1		
	>.1			>.1			>.1			>.1		

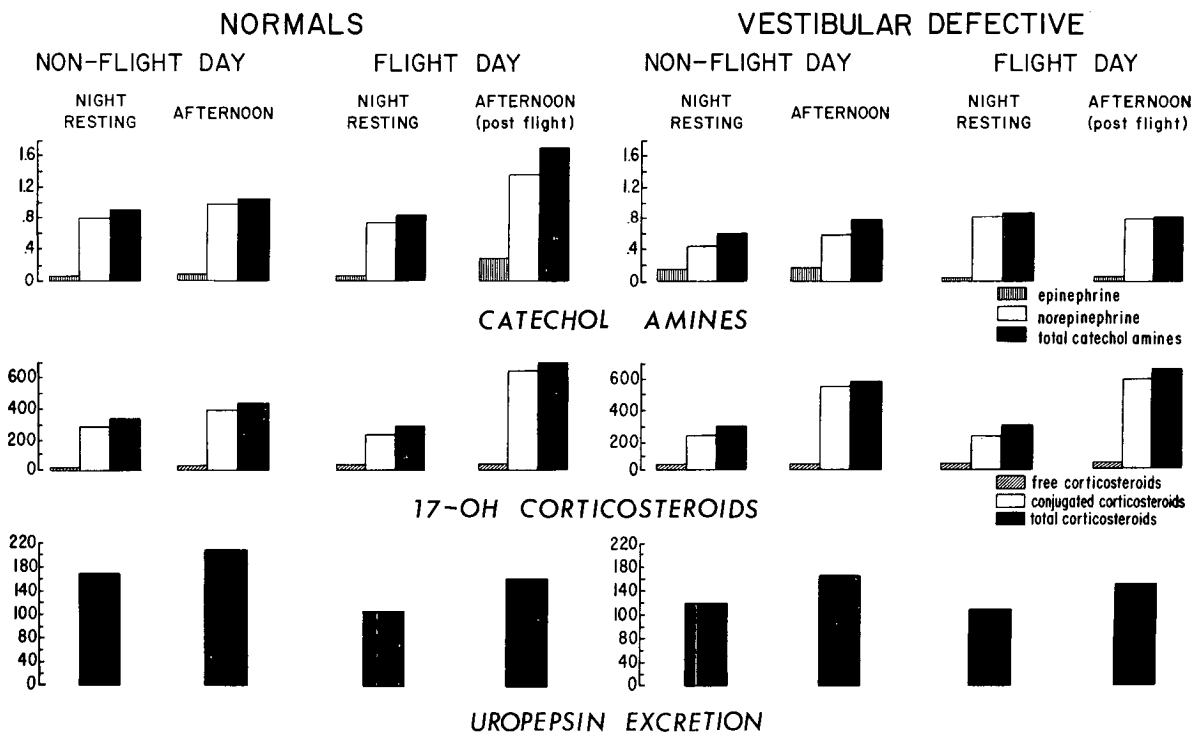


Figure 1.—Stress hormone excretion after acrobatic flight.

the last column of table I in which afternoon excretion rates for both days were compared; the normals had significant increase while the L-D's did not. In such a comparison the circadian rhythm variation is canceled out and therefore this increase should be the true experimental change.

The statistical analyses of the corticoid results in the central part of the table show similar findings with one exception. The L-D's had significant increases on both the control and flight days when resting and afternoon samples were compared. However, when afternoon samples on the nonflight day were compared to those of the flight day, only the normals had significant change.

The statistical information on the uropepsin values is shown in the lower part of the table and is unrevealing. If any change in uropepsin excretion occurred as the result of the flight, it was lost in normal diurnal variation.

#### EXPERIMENT 2

The greatest stress imposed in this experiment was Coriolis acceleration during zero gravity flight. Through the cooperation of the Flight Group at Wright-Patterson Air Force Base, Ohio, the zero gravity condition was produced by means of parabolic flight. The unusual Coriolis force was achieved by rotating the subject in a modified Bárány chair with the subject tilting his head from side to side, all during the zero G phase of the parabola. As before, urinary excretion rates of corticoids and catechol amines were determined and, in addition, the following tests were made on blood samples: total leukocyte, neutrophil, lymphocyte, and eosinophil counts. All determinations were made on both the normal and L-D subject groups each time they were exposed to the following two experimental procedures: (1) during zero gravity alone and (2) while Coriolis acceleration was imposed during zero gravity. Control blood determinations were made just before each flight and the experimental values established immediately at the end of the flight. Urines were collected for 6 hours postflight and compared to

samples collected at the same time on a non-flight day.

The results of the determinations on the formed elements in the blood are shown in figure 2. The normals had small but significant increases in total leukocytes due to increases of neutrophils, decreases in eosinophils after exposure to zero gravity alone, and highly significant similar changes following zero gravity plus Coriolis acceleration. Only lymphocyte changes were not significant. In addition, most of the normals suffered malaise from the acceleration experience. The L-D's had no significant changes in the same parameters and did not evidence signs of motion sickness.

Figure 3 shows the excretion rates of corticoids and catechol amines for 6 hours postflight compared with the control period. The results appear to be contradictory to former findings in which increases in corticoids appeared in the normals after the stressful incident and, as a matter of fact, contradictory to the leukocytic measures of stress indicated in this experiment in figure 2. However, the nature of this experiment required that the total flight time be from 3 to 4 hours so that some of the postflight urine collection period was extended as much as 10 hours poststress. It is possible that this extended collection period could represent a period of adrenal depletion and perhaps coincide with what Mason has described (ref. 25) as a very sharp decline in late morning, poststress plasma values. It also may be related to the feedback mechanism described by Liddle (ref. 20) in which high corticoid levels within a matter of minutes restrict ACTH production so that further 17-OH secretion is also greatly restricted. Whatever the explanation, it is clear that the 17-OH response is quite different in the two groups.

Excretion patterns of the catechols were not significantly different between the two groups except in one instance in which epinephrine was increased in the L-D's. Why this occurred we are not prepared to say although it is possible that anxiety may have been a

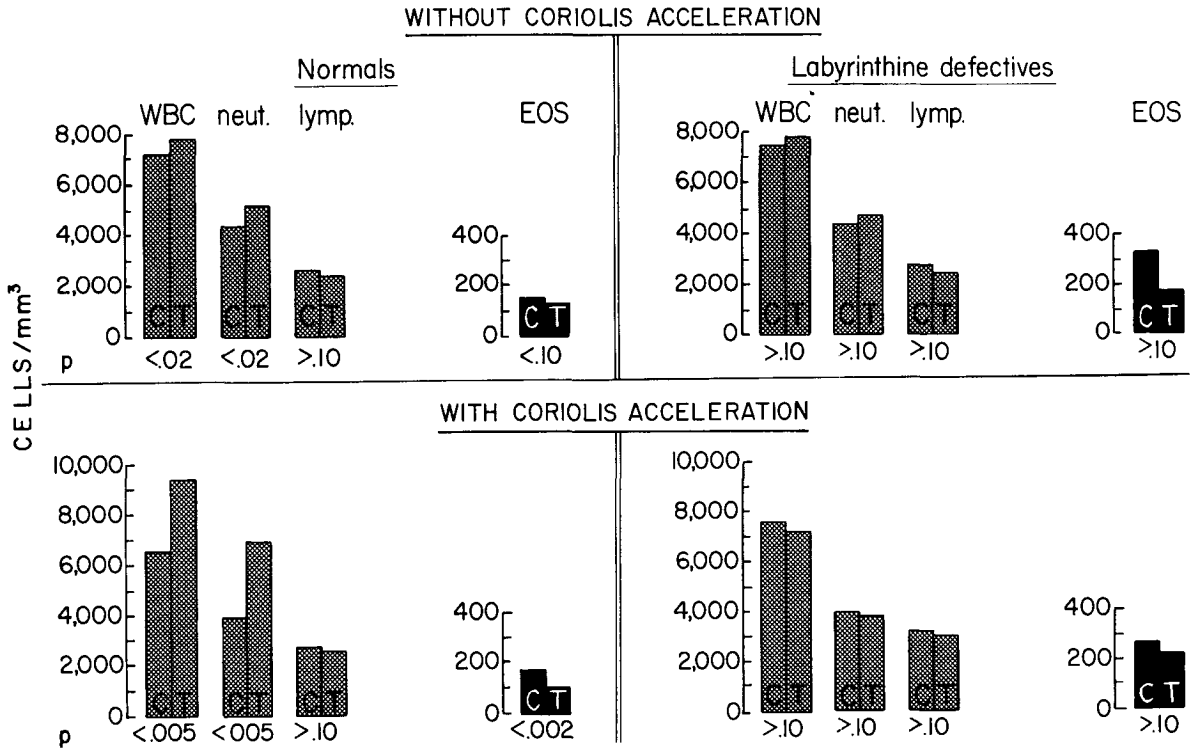


Figure 2.—Variations in leukocytic elements in the blood after zero-G parabolic flight.

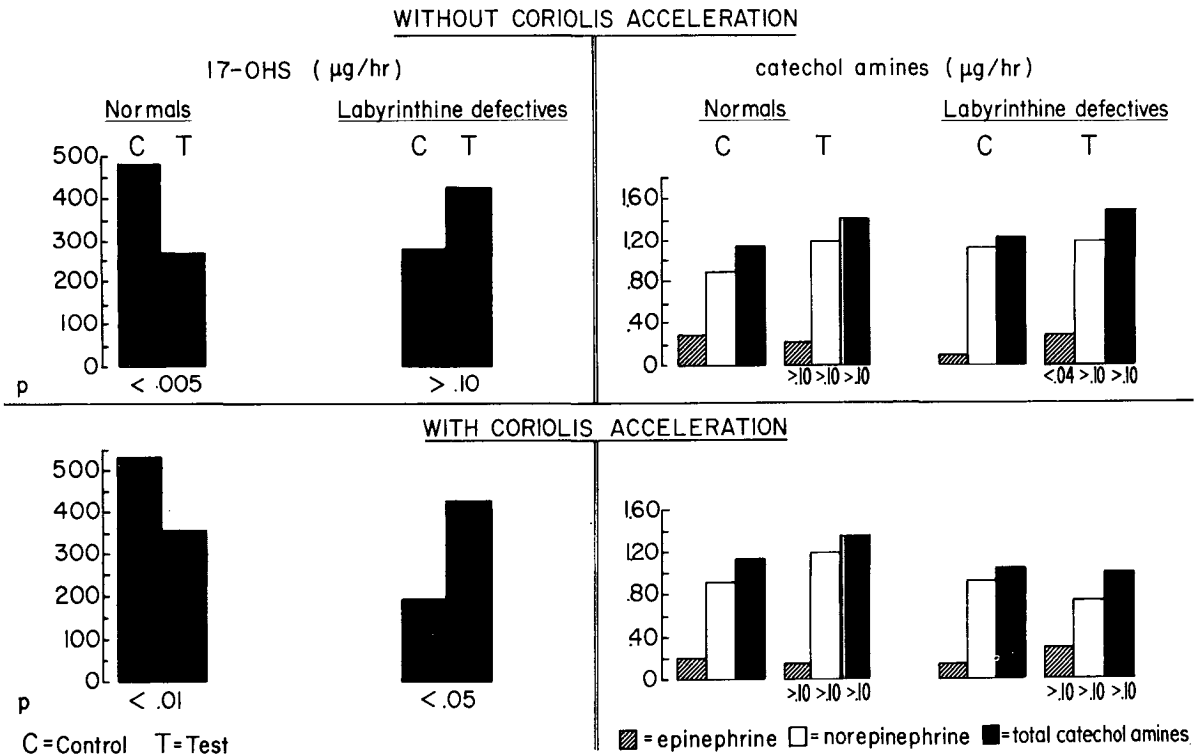


Figure 3.—Stress hormone excretion rates after zero-G parabolic flight.

factor. However, postflight conversations with both subject groups revealed that fear was not a factor in these flights as it had been in the former acrobatic flight experiment and the unremarkable catechol results would bear this out.

Table II contains the statistical summary of the measured parameters. With two minor exceptions no significant changes were found in the L-D measurements while stress was indicated by several significant changes in the formed elements of the blood of the normals especially when they experienced Coriolis acceleration during zero gravity.

### EXPERIMENT 3

This experiment was a sea-going mission in which the pitch and roll of a relatively small ship was the motion variant. Eight subjects from each group were tested. The site of the experiment was an approximately 100-mile stretch of open sea at the point where the Gulf of St. Lawrence opens into the Atlantic Ocean. The craft used was a 145-foot wooden-hulled vessel that made weekly round trips between North Sydney, Nova Scotia, and St. Pierre of the Miquelon Islands with general cargo.

Control samples were taken during the 24 hours before going aboard the ship. The conditions suitable for the experimental phase of the mission occurred on the return trip from St. Pierre when the ship entered

a storm characterized by winds which reached peaks of 70-75 miles per hour. The skipper, who had spent much of his life on the sea, described the storm as not the worst, but one of the worst he had ever experienced. No measurements were made of the motion of the ship but unsecured objects on the cabin decks would easily crash from one bulkhead to the other with each roll of the vessel. The intensity of the storm was such that all of the normal subjects were seasick within an hour or two of entering the open sea. Typical symptomatology of seasickness, consisting of repeated episodes of vomiting relieved only by complete relaxation, was observed in the normals for the next several hours. Among the crew, the second engineer and cook, both of whom were accustomed to the crossing, were also nauseated.

Twenty-four-hour urine samples were collected from the 16 subjects during a period which included the storm at its maximum intensity. Figure 4 is a record of the volumes. The normals had a highly significant decrease in output due not only to loss of fluid but also due to restricted fluid intake. The L-D's had no significant change from control values. During the 8-hour poststorm period some of the normals voided only 30-50 ml of urine, which, with the other information, indicated a condition of dehydration. In two former Slow Rotation Room (SRR) experi-

Table II.—*Summary of Confidence Levels of Blood and Urine Changes after Repetitive Zero G Flight*

[Confidence levels of greater than 0.10 considered not significant]

Subjects		Mann-Whitney "p" values <sup>a</sup>							
		Blood values				Urine values			
		WBC	Neut.	Lymp.	Eos.	17 OHS	E	NE	E + NE
Normals	Zero G	<.02	<.02	NS	<.10	<.005	NS	NS	NS
	Zero G + Cor. accel.	<.005	<.005	NS	<.002	<.10	NS	NS	NS
L-D	Zero G	NS	NS	NS	NS	NS	<.04	NS	NS
	Zero G + Cor. accel.	NS	NS	NS	NS	<.05	NS	NS	NS

<sup>a</sup> Auble, D.: Bull. Inst. Ed. Research at Indiana U., vol. 1, no. 2, 1953.

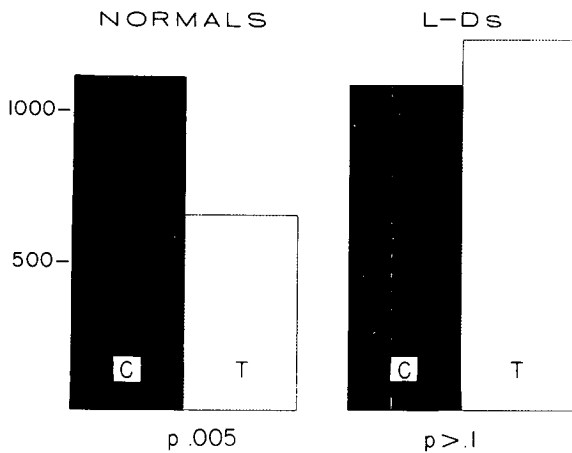


Figure 4.—24 hr urine volumes. C = control; T = test.

ments we had found that, when continued vomiting did take place with resulting fluid imbalance, adrenal function, in terms of hormone excretion rate, could not be related to the stress from the motion itself. The side effects from the vomiting in the SRR experiments included electrolyte imbalance, lowered  $\text{CO}_2$  tension in both blood and alveolar air, and alkalosis due to loss of gastric HCl. Although corticoid excretions are not dependent, as a rule, on normal day-to-day variations in fluid balance, the physiological condition of the normals in this experiment was so far removed from normal that it would be misleading to interpret their inconsistent changes in excretion rates to the motion of the ship. For that reason the biochemical measurements on the normals are not given. An evaluation of the results of samples collected from the L-D's presents a completely different picture. As noted above, their 24-hour volumes were not different from their controls because none vomited and they maintained normal fluid intake. Figure 5 shows the L-D corticoid and catechol amine excretions; as with the volumes, the stress hormone outputs were not significantly different from those determined during the control period.

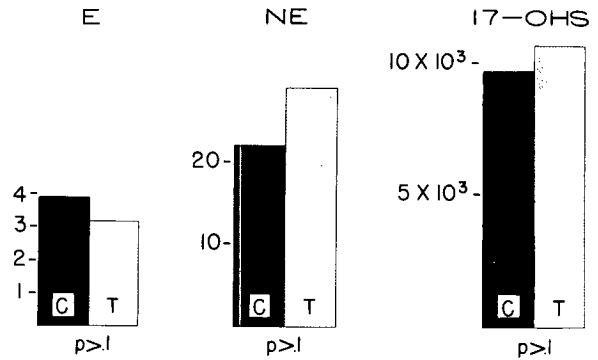


Figure 5.—Excretion of stress hormones from L-D subjects following rough-sea experience.  $\mu\text{g}/24$  hr; C = control; T = test.

In summarizing the results from the three experiments the following observations can be made:

(a) When normal subjects and those with nonfunctioning vestibular organs were exposed to force environments which produce unusual stimuli to the mechanisms of the inner ear, differences in endocrine response were observed when excretion rates of 17-hydroxycorticosteroids and catechol amines, and leukocytic changes were used as the indicators.

(b) The differences were noted in the normals following exposure to one or more of the following conditions: that producing disorientation and changing force environment, zero G parabolic flight sequence especially when Coriolis acceleration was present, and the violent pitch and roll of a 145-foot boat.

(c) Psychological stress was probably involved also since, in one of the experiments, the normals expressed fear, and in these experiments not only corticoid but also catechol excretions were significantly higher.

(d) Varying degrees of nausea were common among the normals in all of the experiments.

(e) The L-D subjects were asymptomatic in the experiments as far as nausea was concerned, and, with two minor exceptions gave no biochemical evidence of stress.

#### REFERENCES

1. BERMAN, M. L.: Quantitative Estimation of Urinary Metabolite of Adrenaline and Noradrenaline as Possible Indication of Tolerance to Gravitational Stress. *Aerospace Med.*, 30 1959, pp. 580-584.
2. CRAVEN, C. W.; AND SMITH, C. S.: Steroid Ex-

- cretion in Airmen Under Stress. *J. Aviat. Med.*, 25, 1955, pp. 200-205.
3. CROUT, J. R.: Catechol Amines in Urine. In: *Standard Methods of Clinical Chemistry* (Seligson, D., ed.). Vol. 3. Academic Press, 1961, pp. 62-80.
  4. DAHL, E. V.; FRANKS, J. J.; PRIGMORE, J. R.; AND CRAMER, R. L.: Adrenal Cortical Response in Motion Sickness. *Arch. Environ. Health*, 7, 1963, pp. 92-97.
  5. DALTON, A. J.; AND SELYE, H.: The Blood Picture During the Alarm Reaction. *Folia Haematol.*, 62, 1939, pp. 397-407.
  6. ELMADJIAN, F.; HOPE, J. M.; AND LAMSON, E. T.: Excretion of Epinephrine and Norepinephrine Under Stress. In: *Recent Progress in Hormone Research* (Pincus, G., ed.). Vol. 14. Academic Press, 1958, pp. 513-553.
  7. VON EULER, U. S.: Identification of the Sympathomimetic Ergone in Adrenergic Nerves of Cattle (Sympathin N) with Laevo-Noradrenaline. *Acta Physiol. Scand.*, 16, 1948, pp. 63-74.
  8. VON EULER, U. S.; AND LUNDBERG, U.: Effect of Flying on the Epinephrine Excretion in Air Force Personnel. *J. Appl. Physiol.*, 6, 1954, pp. 551-555.
  9. FRIEDMAN, S. B.; MASON, J. W.; AND HAMBURG, D. A.: Urinary 17-Hydroxycorticosteroid Levels in Parents of Children With Neoplastic Disease. A Study of Chronic Psychological Stress. *Psychosomatic Med.*, 25, 1963, pp. 364-376.
  10. GOFTON, J. P.; AND McGRATH, S. D.: Studies of Adrenocortical Function in Long-Distance Fliers. A Preliminary Report. *J. Aviat. Med.*, 24, 1953, pp. 107-112.
  11. GOODALL, MCC.: Sympathoadrenal Response to Gravitational Stress. *J. Clin. Invest.*, 41, 1962, pp. 197-202.
  12. GOODALL, MCC.; AND BERMAN, M. L.: Urinary Output of Adrenaline, Noradrenaline, and 3-Methoxy-4-Hydroxy-Mandelic Acid Following Centrifugation and Anticipation of Centrifugation. *J. Clin. Invest.*, 39, 1960, pp. 1533-1538.
  13. GOODALL, MCC.; MCCALLY, M.; AND GRAVELINE, D. E.: Urinary Adrenaline and Noradrenaline Response to Simulated Weightless State. *Amer. J. Physiol.*, 206, 1964, pp. 431-436.
  14. GRAY, S. J.; RAMSEY, C. G.; AND REINFENSTEIN, R. W.: Clinical Use of the Urinary Uropepsin Determination in Medicine and Surgery. *New Eng. J. Med.*, 251, 1954, pp. 835-845.
  15. GREENBERG, R. E.; AND GARDNER, L. I.: Catecholamine Metabolism in a Functional Neural Tumor. *J. Clin. Investig.*, 39, 1960, pp. 1729-1736.
  16. HALE, H. B.; ELLIS, J. B., JR.; AND KRATOCHVIL, C. H.: Effects of Piloting Supersonic Aircraft on Plasma Corticosteroids and Bicarbonate. *J. Appl. Physiol.*, 14, 1959, pp. 629-631.
  17. HALE, H. B.; KRATOCHVIL, C. H.; AND ELLIS, J. B., JR.: Plasma Corticosteroid Levels in Aircrewmen After Long Flights. *J. Clin. Endocrin.*, 18, 1958, pp. 1440-1443.
  18. HICKER, R. B.; WELLS, R. E.; TYLER, R.; AND HAMLIN, J. T.: Plasma Catechol Amine and Electroencephalographic Responses to Acute Postural Changes. *Am. Jour. Med.*, 26, 1959, pp. 410-423.
  19. KORNEL, L.: An Improved, Rapid Method for Free and Conjugated 17-Hydroxycorticosteroids in Urine. *Metabolism*, 8, 1959, pp. 432-440.
  20. LIDDLE, G. W.; ISLAND, D.; AND MEADOR, C. K.: Normal and Abnormal Regulation of Corticotropin Secretion in Man. In: *Recent Progress in Hormone Research* (Pincus, G., ed.), Vol. 18. Academic Press, 1962, pp. 125-166.
  21. LUFT, R.; AND VON EULER, U. S.: Two cases of Postural Hypotension Showing a Deficiency in Release of Norepinephrine and Epinephrine. *J. Clin. Invest.*, 32, 1953, pp. 1065-1069.
  22. MARCHBANKS, V. H., JR.: Effect of Flying Stress on Urinary 17-Hydroxycorticosteroid Levels. Observations During a 22½-Hour Mission. *J. Aviat. Med.*, 29, 1958, pp. 676-682.
  23. MARCHBANKS, V. H., JR.: Flying Stress and Urinary 17-Hydroxycorticosteroid Levels During Twenty-Hour Missions. *Aerospace Med.*, 31, 1960, pp. 639-643.
  24. MARCHBANKS, V. H.; HALE, H. B.; AND ELLIS, J. P.: Stress Responses of Pilots Flying 6-Hour Overwater Missions in F-100 and F-104 Aircraft. SAM-TDR-62-112. Brooks Air Force Base, Texas: School of Aerospace Medicine, 1962.
  25. MASON, J. W.: Psychological Influences on the Pituitary-Adrenal Cortical System. In: *Recent Progress in Hormone Research* (Pincus, G., ed.), Vol. 15. Academic Press, 1959, pp. 345-389.
  26. MEEHAN, J. P.; JACOBS, E.: Physiologic Production of Catechol Amines in Response to Several Physical Stresses. WADC 59-534, Wright Air Development Center, 1959.
  27. PINCUS, G.; AND HOAGLAND, H.: Steroid Excretion and the Stress of Flying. *J. Aviat. Med.*, 14, 1943, pp. 173-193.
  28. SUNDIN, T.: The Effect of Body Posture on the Urinary Excretion of Adrenaline and Noradrenaline. *Acta. Med. Scand. Supp.* 161, 1958, p. 336.
  29. VOORHESS, M. L.; AND GARDNER, L. I.: Studies of Catechol Amine Excretion by Children With Neural Tumors. *J. Clin. Endocrin.*, 22, 1962, pp. 126-133.



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# Antimotion Sickness Drugs for Aerospace

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The space flights of long duration which are planned for the future will require carefully selected medications to increase the comfort, efficiency and even survival of the crews. These medications must be scientifically selected by tests in simulated space conditions. The antimotion sickness drugs which we have been testing at Pensacola illustrate this point. A review of the literature on a clinical preference is not sufficient to predict the relative effectiveness of these drugs.

The facilities at the Naval School of Aviation Medicine are well suited for research on the antimotion sickness drugs. The Slow Rotation Room provides a simulation of the effects of artificial gravity for prolonged periods. When the dial test which requires a prescribed series of head movements to set a series of dials is added, this facility provides a well standardized method of producing motion sickness. A constant G force can be established and then the head movements produce a stimulus to the vestibular receptors such as that from the roll, pitch, yaw, and heave of a surface vessel or aircraft. A group of highly selected subjects are also available here. The same approximate age, size, health, intelligence and living conditions are represented by the Navy personnel available to act as subjects.

### METHODS

On test days the subjects were given a light breakfast of milk, cereal, and juice. The drugs were administered in matched oral capsules 1½ to 2 hours before the ex-

periment. Double blind and placebo procedures were used throughout the study. A rest period of from 48 to 72 hours was allowed between administration of each drug. All subjects were tested with each of the drugs used. A total of 112 separate experiments were performed using eight different drugs. The results were compared with the previous study which involved 15 subjects and utilized the recommended dose of these same drugs.

The drugs and the doses used were as follows:

	1st study	2nd study
Meclizine (Bonamine), mg-----	50	150
Hyoscine (Scopolamine), mg-----	0.6	.06
Hyoscine (Scopolamine), mg-----	---	1.2
d-Amphetamine (Dexadrine), mg---	10	20
Thiethylperazine (Torecan), mg---	10	30
Trimethobenzamide (Tigan), mg---	250	750
Prochlorperazine (Compazine), mg--	5	15
Placebo (Lactose), mg -----	750	750

A combination of 1.2 mg hyoscine and 20 mg of d-amphetamine was also used.

The subjects were given questionnaires to record the side effects produced by the drugs. Medical surveillance was maintained for 8 hours following administration of the drugs. Blood pressures and pulse rates were recorded periodically.

To avoid conditioning the subjects against the experiments a scale of signs and symptoms of motion sickness was adopted from previous research (ref. 9). This enabled a

definite diagnosis of motion sickness to be made short of emesis.

The subjects were required to continually set a sequence of five dials to numbers given at 4-second intervals by a tape recorder. The dials were arranged in such a manner as to require head movements which approximated the stimulation received by the vestibular receptors from the roll, pitch, yaw, and heave of a surface vessel or aircraft. The subjects were spun at increasing rpm in the SRR until they developed the malaise III condition (ref. 6) of motion sickness in 50 head movements or less. This was considered to be the subjects' basal susceptibility to motion sickness. Two control runs and a placebo run were then taken for each subject to confirm this baseline before administration of the drugs. Placebo runs were given

periodically throughout and at the end of the experiment to determine any adaptation of the subjects to the stimulus. If a subject completed 300 head movements without developing malaise III the test was halted.

The subjects were isolated in the SRR during the test periods except for a television monitor and a trained observer who recorded signs and symptoms of motion sickness.

#### RESULTS

The results of the first study are shown in figure 1.

*Results of second study (fig. 2).*—The therapeutic effect was enhanced by the increased dose with only two of the antimotion sickness preparations used in this project. An increased effectiveness was seen with d-amphetamine (20 mg) and with the com-

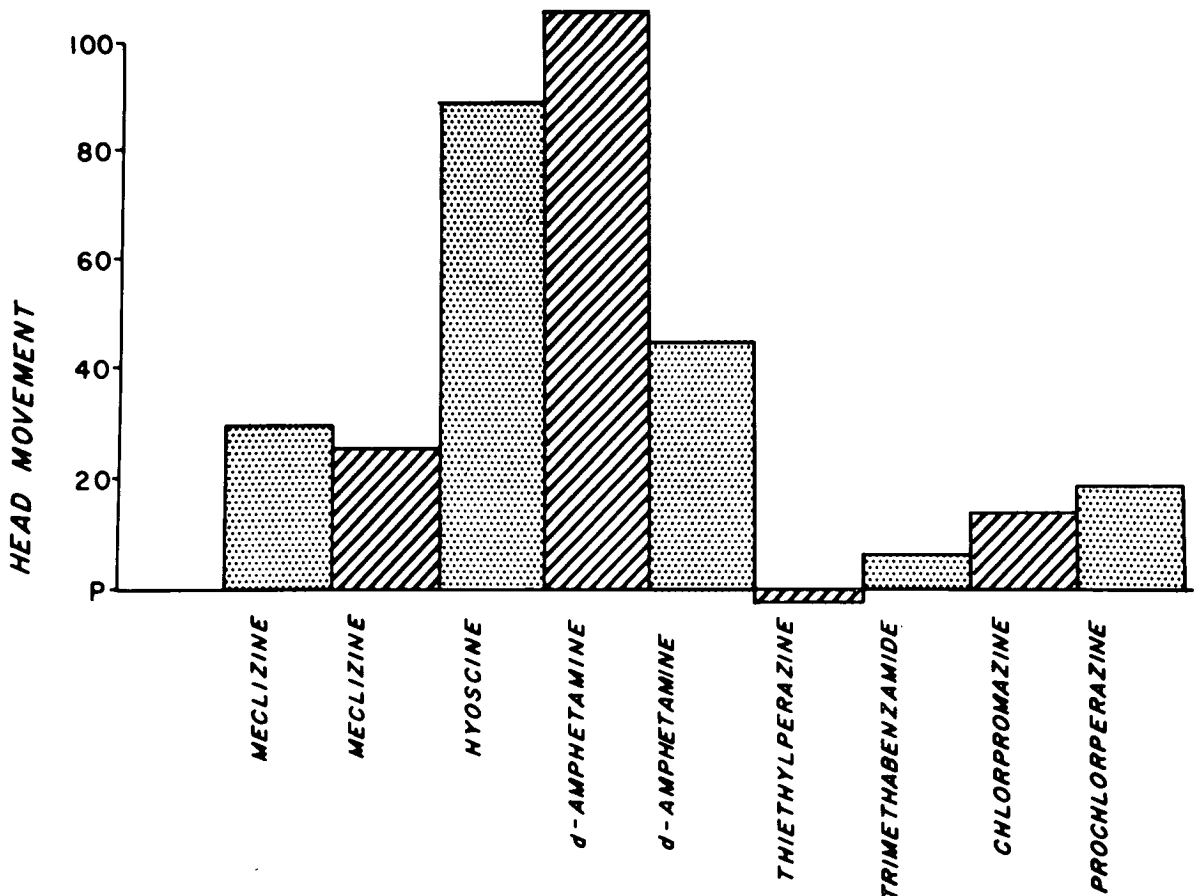


Figure 1.—Increases in tolerated head movements compared for experimental and control group to determine relative effectiveness of antimotion sickness drugs. Baseline is level tolerated on placebos.

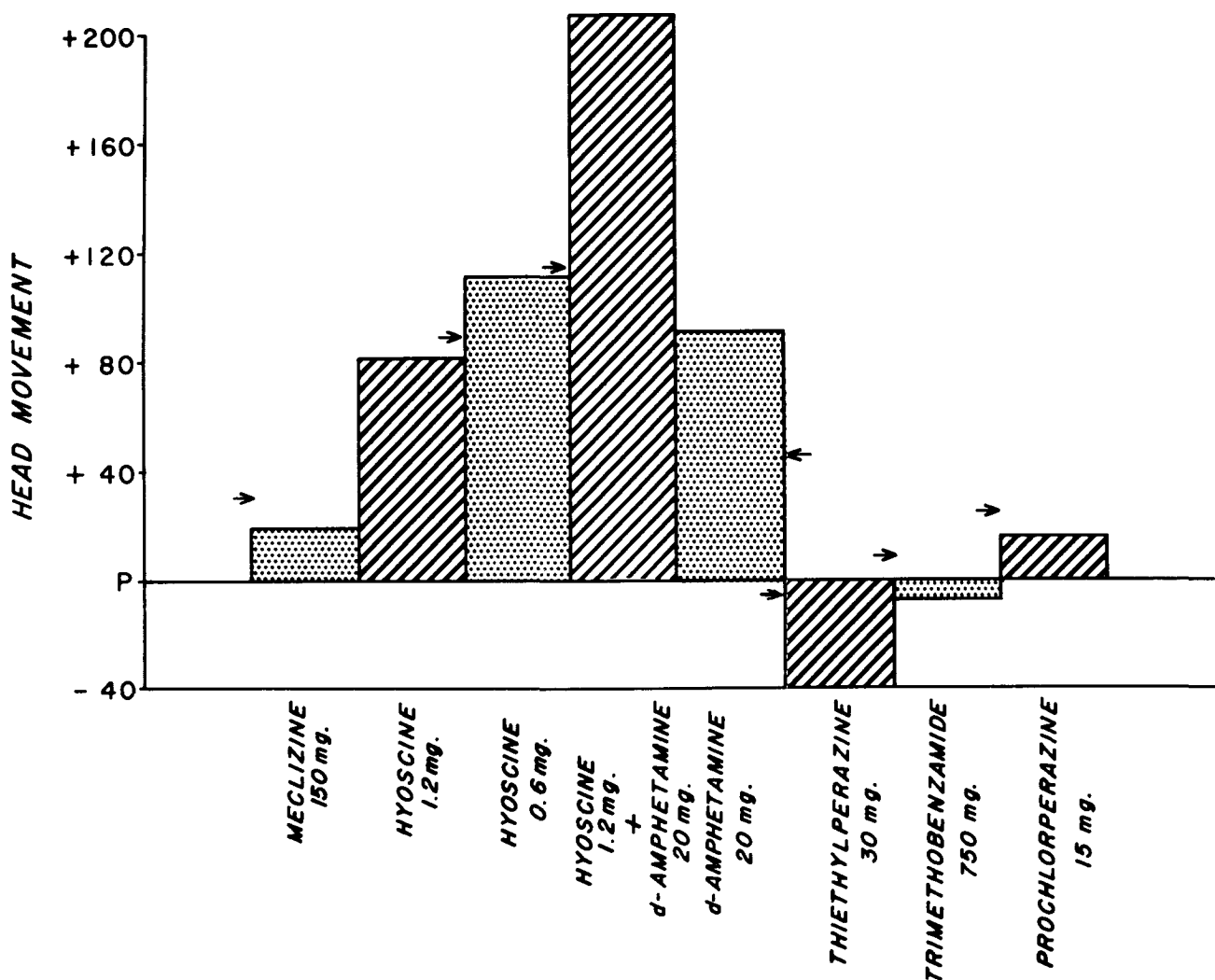


Figure 2.—Average number of tolerated head movements is shown for each of drugs tested. Drugs were meclizine, hyoscine, d-amphetamine, thiethylperazine, trimethobenzamide, and prochlorperazine. Arrows indicate effectiveness of recommended dose of these drugs in a previous study.

bination of d-amphetamine (20 mg) and hyoscine (1.2 mg). In a previous study 10 mg of d-amphetamine increased the tolerance to motion by 80% or an average increase of 44 head movements over the placebo level. In this study the dose of 20 mg of d-amphetamine increased this protection to a level of 170% or an average of 92 head movements over the placebo level.

The combination of hyoscine and d-amphetamine was the most effective preparation in both studies. Earlier, 0.6 mg of hyoscine with 10 mg d-amphetamine increased the

tolerated head movements by an average of 133 movements or by 240%. A dose of 20 mg d-amphetamine with 1.2 mg hyoscine increased this to an average of 206 movements or by 375% over the corresponding placebo level.

Hyoscine (0.6 mg) increased the tolerated head movements by an average of 88 or by 160% in the first study. Doubling the dose of hyoscine (1.2 mg) in the present research failed to produce an increase in resistance to motion sickness. This failure to increase therapeutic effectiveness with increased dos-

age was also seen with all other drugs used in this study, with the two exceptions which were mentioned above. A decrease in effectiveness was seen with increased doses of prochlorperazine (Compazine) 15 mg, trimethobenzamide (Tigan) 750 mg, and meclizine (Bonamine) 150 mg. Thiethylperazine (Torecan) 30 mg showed the most marked drop in effectiveness as the number of tolerated head movements fell from an average of four less than the placebo to an average of 40 less with the increased dose. The effectiveness of the antimotion sickness drugs as reported in the literature are shown for comparative purposes in the following table:

	Dose, mg	Reported peak effective- ness, %
Hyoscine (Scopolamine)-----	0.6	90
Meclizine (Bonamine)-----	50	85
Thiethylperazine (Torecan)-----	10	78
Prochlorperazine (Compazine)-----	5	75
Chlorpromazine (Thorazine)-----	25	--
Trimethobenzamide (Tigan)-----	250	?
d-Amphetamine (Dexadrine)-----	10	39

The results of the questionnaire on side effects are in table I and are reported in percentages to correspond with other side effect studies. The dose of 1.2 mg of hyoscine produced marked drowsiness, blurring of vision and nervousness. Hyoscine in the 0.6 mg dose produced a less severe state of drowsiness, but a greater incidence of vertigo was reported. The combination of hyoscine (1.2 mg) and d-amphetamine (20 mg) caused the largest number of subjects to report vertigo and stomach awareness. The drowsiness reported with this combination was milder than with hyoscine alone. When d-amphetamine (20 mg) was administered, some blurring of vision, vertigo and nervousness were reported. One subject reported a headache which appeared to be due to an increase in blood pressure from 125/80 to 140/90. This was the greatest alteration of blood pressure noted in the study and the only re-

port of headache from this dose of d-amphetamine. The mildest drug as indicated by the questionnaire was meclizine in spite of the dose of 150 mg. One half of the subjects reported no side effects with this drug. Trimethobenzamide (Tigan) had only slightly more side effects reported than did meclizine. Thiethylperazine (Torecan) in the 30 mg dose produced considerable drowsiness and headache but no vertigo was reported. When the lactose placebo was given, one-third of the subjects reported drowsiness and one-sixth reported a headache.

#### DISCUSSION

The Slow Rotation Room appears to offer an excellent test situation for the antimotion sickness drugs. The standardized head movements and rate of spin permitted the same vestibular stimulus to be repeated for each subject through a series of experiments. This controlled test situation permits a more exact comparison of the effectiveness of each of a group of antimotion sickness drugs. Such an exact comparison is not possible under the uncontrolled conditions of field tests (ref. 8). The results of this study are in general agreement with the literature in that the well established drugs such as hyoscine and meclizine were most effective (ref. 13). Preparations which were ineffective in this research were those which have not had extensive testing as antimotion sickness drugs such as thiethylperazine (Torecan) (ref. 11), trimethobenzamide (Tigan) (ref. 12) and prochlorperazine (Compazine) (ref. 1). The exception to this was d-amphetamine which has had only a few reports of effectiveness as an antimotion sickness remedy. Prior to World War II it was reported to have antimotion sickness activity by Hill (ref. 7). Blackham shortly thereafter reported it to be one of the best drugs in his study (ref. 2). Since that time it has apparently not been used against motion sickness. In the present research it was the second most effective drug. In our previous study it also proved to be effective in the 10 mg dose. The fact that it has proven

effective suggests that the sympathomimetics may be a promising area for future development of antimotion sickness drugs.

The most effective single drug in this study was hyoscine (0.6 mg). Doubling this dose failed to increase the therapeutic potency; however, in combination with d-amphetamine the increase in tolerance to motion exceeded the sum of the effect of these drugs when used separately. This preparation would have shown an even greater effect except for the fact that five of the eight subjects on this drug completed the full 300 head movements and were halted there. In two of the remaining subjects this drug combination produced the best therapeutic effect of the preparations tested.

The signs and symptoms of motion sickness closely resemble the effect of overactivity of the parasympathetic nervous system. An overdose of neostigmine, which protects acetylcholine the mediator of the parasympathetics, produces similar reactions. The effective drugs discussed above included a parasympathetic blocking agent and a sympathomimetic; this with the aforementioned facts would suggest that part of the mechanism of action of these drugs could be the result of a shift of autonomic activity toward the sympathetics. Further support is lent to this contention by the report that dibenzoline, which blocks the sympathetics, increases susceptibility to motion sickness (ref. 4).

The increased dose of meclizine, thiethylperazine, trimethobenzamide, and prochlorperazine produced less of a therapeutic effect than did the recommended dose. This has also been observed in other studies on the antimotion sickness drugs (ref. 10). It is well established that any factor that irritates the stomach lowers the tolerance for motion and this could be a factor with the large doses used here (ref. 3). Nausea is a common side effect with overdose of various drugs. It was reported for several of the preparations on the side effects questionnaire which was completed by the subjects before entering the SRR. The decrease in potency

with increased dose may have been related to these facts.

A review of the literature on antimotion sickness drugs indicated that hyoscine and meclizine should be of about equal potency. A very significant difference in favor of hyoscine was found in this and in our earlier study. The British investigations have long held that hyoscine is the superior drug (ref. 5) while several U. S. reports favor the antihistamines as being the most effective (ref. 10). A difference in strength of stimulus in these studies may be responsible for these divergent findings (ref. 8). Our results strongly support the view that hyoscine is the drug of choice for prevention of motion sickness.

Subjects with defective labyrinths have been studied and have been found to be resistant to motion sickness even under the most extreme conditions (ref. 6). The side effect of vertigo in this study was roughly correlated to potency of the drugs against motion sickness. These observations suggest that these drugs may act at the vestibular receptor sites by lowering their sensitivity. It is difficult, however, to visualize d-amphetamine as having this mechanism of action.

The scale of signs and symptoms used to determine motion sickness gave very good results with trained subjects and observers. It would most likely be difficult to apply it to untrained personnel under field conditions. The slight but steady rise in the basal (placebo) tolerance for motion indicated that no conditioning against the test occurred. This enabled each subject to be tested on all the drugs used and also to serve as his own control.

The human centrifuge served as an excellent laboratory device for testing the antimotion sickness drugs. When the semicircular canals had stabilized to the constant rate of spin, no motion was perceived as long as the head remained still. With head movements the stimulation to the otoliths approximated that received from rough conditions in a plane or surface vessel.

Table I.—Results of Questionnaires on Side Effects

Side effects	Hyoscine & d-Amphetamine	Hyoscine, 1.2 mg	Hyoscine, 0.6 mg	d-Amphetamine	Meclizine	Thiethylperazine	Prochlorperazine	Trimethobenzamide	Placebo
Fatigue, %	22	56	40	10	13	42	10	13	4
Drowsiness, %	60	86	80	10	13	56	22	13	32
Headache, %	0	28	30	10	13	28	33	13	16
Blurred vision, %	22	56	40	33	0	0	0	13	0
Vertigo, %	55	42	50	22	13	0	0	0	4
Nervousness, %	10	42	0	33	0	28	10	13	8
Dry mouth, %	10	28	30	10	0	0	10	0	0
Nausea, %	33	28	30	10	13	0	22	0	4
None, %	10	0	0	22	50	0	33	36	36

## REFERENCES

- BLACKENSTOE, G. S.: Prochlorperazine in Air-sickness 70% Control. *Pennsyl. Med. J.*, 62, 1959, pp. 1341-1343.
- BLACKHAM, R. J.: Seasickness. *Brit. Med. J.*, 2, 1939, pp. 163-167.
- CHINN, H. I.; AND SMITH, P. K.: Motion Sickness. *Pharmacol. Rev.*, 7, 1955, pp. 33-82.
- CHINN, H. I.; ET AL.: Evaluation of Some Drugs in Seasickness. *J. Pharmacol. and Exper. Therap.*, 108, 1953, pp. 69-79.
- GLASER, M. C.: Prevention and Treatment of Motion Sickness. *Proc. Roy. Soc. Med.*, 52, 1959, p. 965.
- GRAYBIEL, A.: Vestibular Sickness and Some of its Implications for Space Flight. 11th Annual Scientific Meeting of the Houston Neurological Society, Baylor Univ. Col. of Med. (Houston, Tex.), 1963.
- HILL, J.: Benzedrine in Seasickness. *Brit. M. J.*, 2, Dec. 4, 1937, pp. 1109-1112.
- JOHNSON, C.; AND WENDT, G. R.: Studies of Motion Sickness—The Efficiency of Laboratory Tests of the Preventive Action Drugs. *J. of Psychol.*, 57, 1964, p. 71.
- KENNEDY, R. S.; AND GRAYBIEL, A.: Symptomatology During Prolonged Exposure in a Constantly Rotating Environment at a Velocity of One Revolution Per Minute. *Aerospace Med.*, 33, 1962, p. 817.
- TRUMBULL, R., ET AL.: Effect of Certain Drugs on the Incidence of Seasickness. (Army, Navy, and Air Force Motion Sickness Team.) *Clin. Pharmacol. and Therap.*, 1, 1960, pp. 280-283.
- VINAGUERRA, G.: Esperienze sull'azine anticinetosice del Torecan. *Inform. Med. (Genova)*, 16, 1961, pp. 94-95.
- WOOD, C. D.; GRAYBIEL, A.; KENNEDY, R. S.; AND McDONOUGH, R.: Evaluation of Some Antimotion Sickness Drugs on the Slow Rotation Room No. I. NSAM-923. NASA R-93. U.S. Naval School Aviation Med., 1965.
- WOOD, C. D.: Review of Antimotion Sickness Drugs from 1954-1964. Special Report 64-2, U.S. Naval School of Aviation Med., 1964.

## DISCUSSION

FIELDS: Dr. Wendt and I did some drug testing nearly 25 years ago with somewhat more primitive facilities. However, since then he has done a great deal to perfect his apparatus. After several years of testing of various drugs in a crash program to find one which could be used in amphibious operations during World War II, we also came to the conclusion that hyoscine was the best available agent. Did you try experimenting with double the amount of amphetamine but with the same dose of hyoscine?

WOOD: No, we didn't. We doubled the dose of both in combination, and we doubled them each separately, but we didn't use that dosage.

WENDT: I don't think I agree that these drugs have a common mechanism. In our eye movement experiments we found that hyoscine decreases the velocity of the slow phase of the nystagmus. Dramamine, which is somewhat similar to your Bonamine, does not do so but instead brings in the wandering eye movement, which I think is associated with sleep and which competes with nystagmus. I suspect that

the one acts on the vestibular nuclei and the other acts on the reticular activating system.

I believe that amphetamines operate by making your subjects more optimistic, cheerful, self-confident, and egocentric. When I went to Wright-Patterson Field to fly their weightless patterns and didn't want to get motion sick, I took hyoscine to depress my vestibular nuclei and Marezine to depress my reticular activating system. I mistakenly took tigan hydrochloride to depress my vomiting center, and I took paregoric to keep stomach sensations from bothering me. I took amphetamine to make me cheerful and a very small amount of Seconal to make me self-confident. In the aggregate, there were no significant side effects of any kind, but as a motion sickness preventative, this mess of stuff actually turned out to be rather effective.

We have recently published a paper on the use of the vertical accelerator for evaluation of drugs. We find that it's much more efficient than sea trials; a group of 24 subjects for each treatment seems to be enough to replicate the efficacy order of drugs where we are using hyoscine, dramamine, placebo, and one other weak motion sickness preventative. The use of machines for screening motion sickness preventatives for a purpose of this kind is quite valid because it gives similar results to those obtained from sea trials.

WOOD: I agree with Dr. Wendt on the mechanisms. This was, as I said, an attempt to find a common ground for the drugs. It certainly is not necessary to find it, and there are many factors working in motion sickness. As far as the sea trial is concerned, I can imagine no more uncontrolled situation than to have the observer sick also in sea trials. I am highly in favor of the laboratory methods, if we can get the purist to accept them.

POLLACK: What you present is perfectly applicable at 1 G, but you are not extrapolating this at zero G, are you?

WOOD: No, we're not.

RUBIN: It seems that Dr. Wood made use of some motion sickness drug evaluation techniques done in the Slow Rotation Room that are not in common use. It would be well if the complete method of evaluation were published for all to understand. Different drug results are obtainable when, say, vomiting is used as the end point in a vertical swing apparatus, or when ENG recorded nystagmus is used as the end point following caloric stimulation. In well controlled studies, when both double blind and triple blind drug administration techniques are used, drugs in the phenothiazine group and in the antihistamine group show repeatedly better response than do the drugs in automatic drug group. Further, I am wondering whether Dr. Wood has made any attempt to correlate drug localization in normal or other tissue to corroborate his motion sickness control studies. We have done drug localization studies in brain tissue of both rabbits and dogs and we are in the process of further following this drug accumulation into subcellular portions of the areas of the brain with the greatest concentration of drug. We have found most interesting accumulation of one of the phenothiazines in the vestibular nuclei and cerebellum. There are other studies now in progress for following drug accumulation in the brain tissue of other drug types with vomiting suppression response in the vertical swing technique. It is evident that only part of the motion sickness drug evaluation story can be told if only Dr. Wood's method is followed. I believe that other techniques, such as vomiting suppression or nystagmus decline, should be considered in order to elicit the complete picture.

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## Temporary Suppression of Semicircular Canal Function in Squirrel Monkeys After Streptomycin Sulfate

MAKOTO IGARASHI

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The evidence of the role of the vestibular organs in causing motion sickness in unusual force environments is seen in comparing the distress of normal subjects with the carefree attitude of those who have lost the function of these organs. Even partial loss of vestibular function affords protection. Among those so protected are patients who have received streptomycin for the treatment of their Ménière's disease. The vestibular ablation technique for Ménière's disease has been established by Fowler (ref. 1), Hamburger et al. (ref. 2), Ruedi (ref. 3), Hanson (ref. 4), and Schuknecht (ref. 5). When administered either intratympanically or parenterally, vestibular ablation was successful in most of the cases. Schuknecht administered streptomycin to patients with Ménière's disease in amounts just sufficient to result in ataxia. According to him, no one suffered a loss in hearing, suggesting a selective affinity for the vestibular labyrinth. Four of his patients were studied 10 years later; hearing had improved in three and was unchanged in one (Graybiel, personal communication).

Although studies of ototoxic effects of streptomycin sulfate in lower animals by Schuknecht (ref. 5) Hawkins (refs. 6 and 7), Winston and colleagues (refs. 8 to 10), Ruedi and colleagues (refs. 11 and 12), Christensen et al. (ref. 13), Berg (ref. 14), and McGee et al. (ref. 15) had revealed the hair cell loss in the cochlea as well as semicircular canals, the possibility still existed

that, in man, small doses might suppress the vestibular function without damaging hearing end organs.

As a first step in exploring this possibility, squirrel monkeys were chosen among the subhuman primates inasmuch as some vomit when exposed to the inertial forces in a rotating environment. In addition to the primary purpose, the opportunity also existed to study the relative roles of the semicircular canals and otolith end organs in causing vestibular sickness, because streptomycin sulfate has generally been known to have less effect on the maculae of the otolith organs.

Healthy squirrel monkeys were screened on the basis of normal semicircular canal function as repeatedly measured by the threshold caloric test and susceptibility to vomiting in the Pensacola Slow Rotation Room. Hearing testing was not attempted. Eight animals selected were given one or more courses of streptomycin sulfate for a period of three weeks. During this period, both tests were performed continuously. Treatment was continued until there was a sign of suppression of canal function.

Six months after the suppression, pathological studies were carried out. All animals were sacrificed by means of intravital cardiac perfusion, and the temporal bones were prepared following the standard preparation procedure. The horizontal serial sections were examined light-microscopically. The pathological findings of both vestibule



and cochlea were graphically reconstructed thereafter.

All functional test results, and the pathological findings of both vestibule and cochlea, are summarized in table I.

The complete or almost complete suppression of caloric threshold was observed in five animals, while the rest showed partial suppression. Six months after the suppression, the caloric test indicated normal or nearly normal function in all animals.

Six monkeys tested in the Slow Rotation Room (10 rpm) did not show any canal sickness symptoms while their caloric thresholds were depressed, but all except one demonstrated canal sickness symptoms with vomiting when the caloric thresholds were recovered. Figures 1, 2, and 3 demonstrate representative data of threshold caloric tests and canal sickness tests in the Slow Rotation Room. The suppression of caloric threshold occurred rapidly, but the recovery was rather gradual.

The hair cell pathology in semicircular canal cristae was severe in two animals which received rather small total doses of streptomycin sulfate, moderate in two animals, and slight or very slight in others.

Two of three monkeys which showed partially suppressed caloric threshold had slight

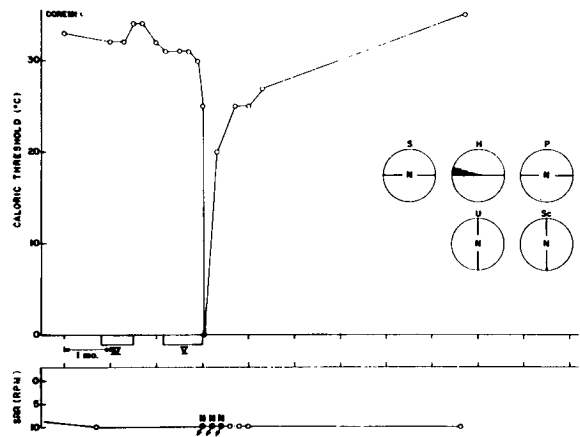


Figure 1.—The course of caloric threshold and canal sickness threshold change in Pensacola Slow Rotation Room after administration of 4600 mg of streptomycin sulfate. Open circles in the caloric column indicate the testing points. Open circles in Slow Rotation Room test column indicate the positive canal sickness at 10 rpm. The roman numerals and the rectangles below horizontal lines, with one month step abscissa, indicate the numbers of streptomycin courses. The five large circles are graphic representations of vestibular hair cell pathology. S: Superior semicircular canal crista; H: Horizontal semicircular canal crista; P: Posterior semicircular canal crista; U: Utricular macula; Sc: Saccular macula; N: No pathological findings.

and very slight hair cell pathology in cristae, but the other monkey had severe pathology.

Figure 4 is a view of normal horizontal

Table I.—Functional Tests Data and Pathological Findings in Eight Squirrel Monkeys after Streptomycin Sulfate Injection

[Pathological findings: +++, severe; ++, moderate; +, slight; ±, very slight; —, none. Signs indicate the estimated average of findings from each animal]

	Total streptomycin dose, mgr	Functional testings				Pathological findings				
		Caloric		SRR		Paral-lelism caloric I SRR	Cristae	Maculae	Organ of corti	Spiral ganglion
		Lowest threshold, °C	Complete recovery	No symptom at 10 rpm	Recovery					
ET	1100	<10	+				+++	—	+	—
DR	1150	< 7	+				++	—	++	±
EP	2800	13	+	+	—	±	+++	+	+++	++
FC	3000	20	+	+	+	+	+	—	++	—
DH	3000	< 2	+	+	+	+	±	—	++	—
FN	3350	2	+	+	+	+	++	±	++	—
DM	4050	24	+	+	+	+	±	±	++	++
ES	4600	< 2	+	+	+	+	±	—	++	±

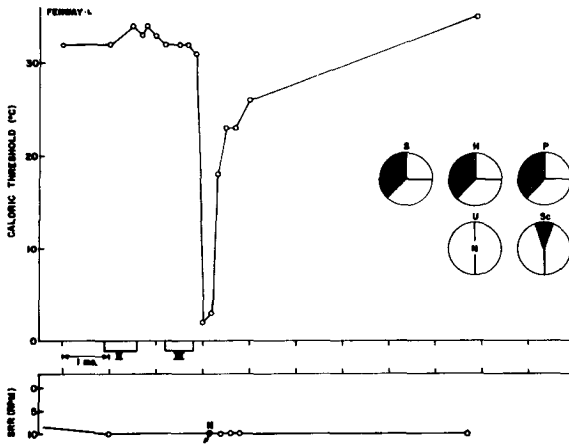


Figure 2.—Course of caloric threshold and canal sickness threshold change after administration of 3350 mg of streptomycin sulfate. Lowest caloric threshold was 2°C. (See fig. 1.)

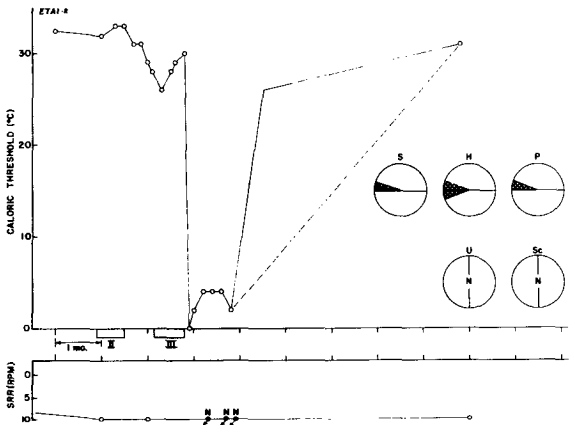


Figure 3.—Course of caloric threshold and canal sickness threshold change after administration of 3000 mg of streptomycin sulfate. (See fig. 1.)

semicircular canal crista, and figure 5 is representative of the end organ pathology in the crista. The hair cell pathology was usually more significant on the summit of the crista than on the sides.

Even with severe hair cell pathology in the crista, the recovery of the caloric threshold was almost complete in all monkeys investigated. Temporary suppression and recovery of canal function after the parenteral administration of streptomycin sulfate, by measuring caloric nystagmus duration, was reported by Schuknecht (ref. 5) in some of

his Ménière's patients. Investigating postrotatory nystagmus duration, McGee and Olszewski (ref. 15) reported partial recovery of canal function with severe hair cell pathology in crista, in a few of their streptomycin

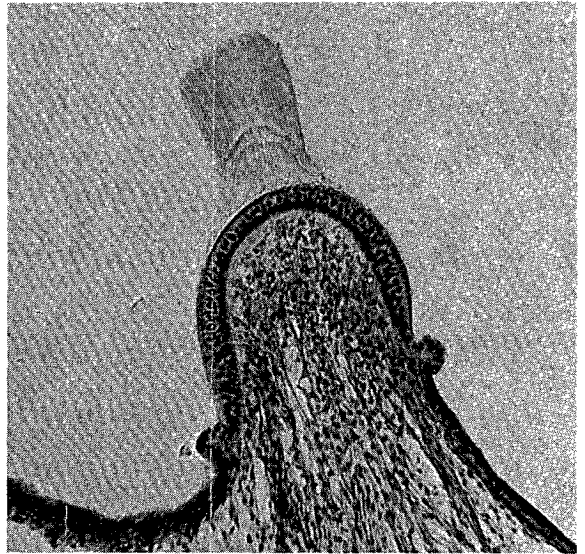


Figure 4.—Photomicrograph shows a normal horizontal semicircular canal crista of squirrel monkey. × 260.

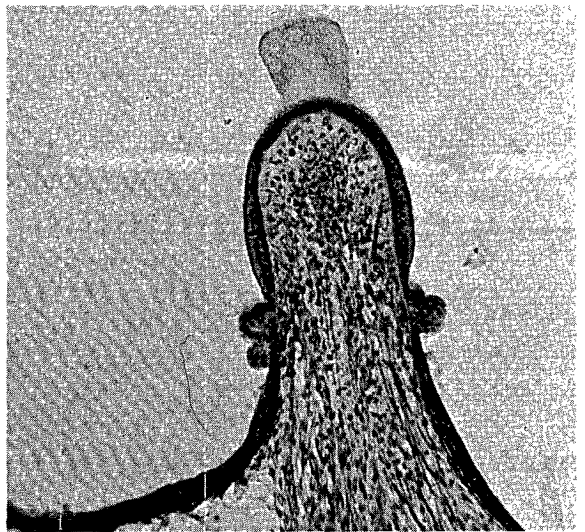


Figure 5.—Photomicrograph demonstrating a view of severe end organ pathology in horizontal semicircular canal crista from squirrel monkey after administration of 1100 mg of streptomycin sulfate. Hair cell damage is more prominent on summit of crista. × 235.

sulfate cat series. It is therefore feasible that functional suppression with recovery can be expected in the vestibular end organs after the streptomycin sulfate administration, which may possibly involve the biochemistry of endolymph and hair cells first, followed by morphological destruction. The neurotoxicity of streptomycin sulfate is still unknown.

Maculae in all of our monkeys showed minimal end organ pathology. Figure 6 is a view of the almost intact saccular macula and destroyed upper basal turn after streptomycin sulfate injection. Representative graphic reconstructions of cochlear and vestibular end organs are demonstrated in the figures 7-11. Minimal difference was observed between two ears of each individual monkey.

All cochleas investigated demonstrated

hair cell lesions without any exception. The moderate spiral ganglion cell lesions were observed in two monkeys, and very slight ganglion cell loss was observed in another two monkeys. The hair cell losses in the cochleas were extended to the average of 12 mm from the basal ends, regardless of the total doses of streptomycin sulfate. Figure 12 demonstrates a normal and figure 13 a streptomycin destroyed organ of Corti from upper middle and upper basal turns of squirrel monkey cochleas. It is possible that these animals had high frequency hearing losses. Very minimal pathology was observed in all other structures in the cochlea.

Since the hair cell involvement always started from the basal end towards the apex and decreased gradually in the present investigation, the probability of hearing pres-

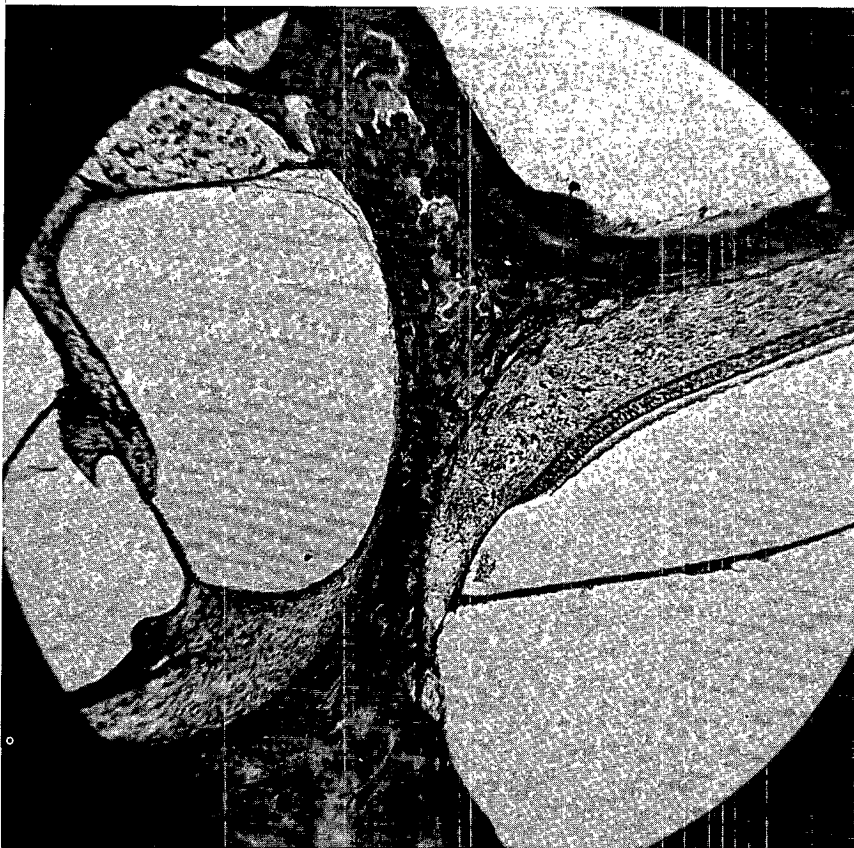


Figure 6.—Photomicrograph showing intact saccular macula and destroyed upper basal turn of cochlea after administration of 4050 mg of streptomycin sulfate.  $\times 160$ .

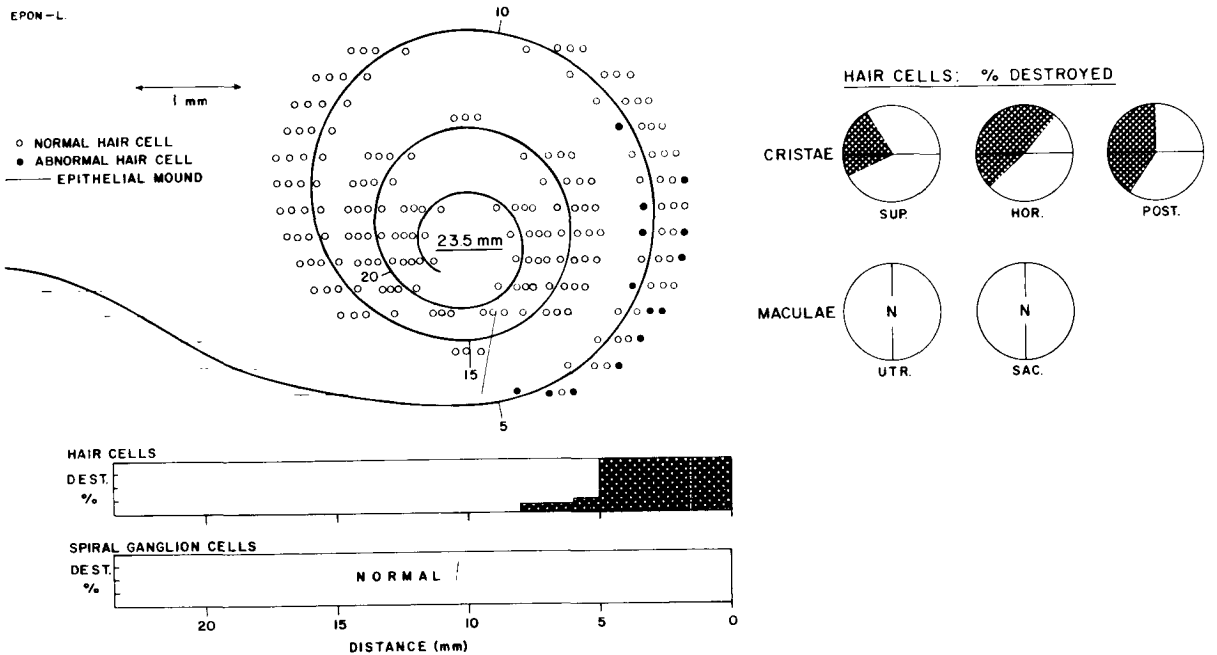


Figure 7.—Graphic display showing moderate to severe hair cell pathology in cristae, and slight cochlear lesion after administration of 1100 mg of streptomycin sulfate.

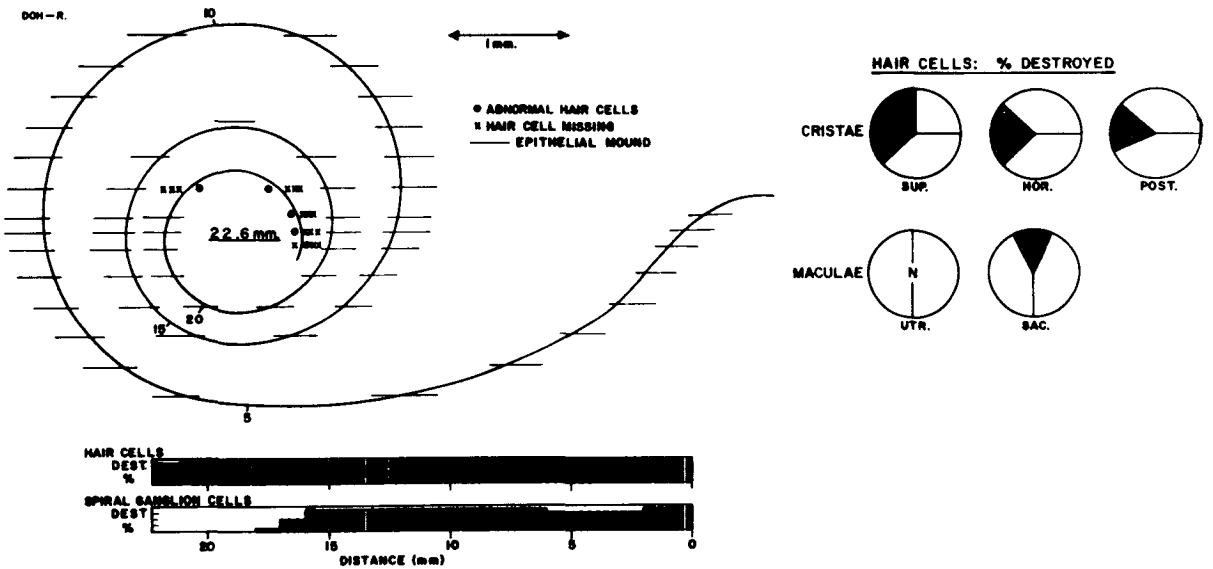


Figure 8.—Spiral display and barograms demonstrating severest cochlear destruction after administration of 2800 mg of streptomycin sulfate. Pathology in cristae was also moderate to severe.

ervation, except at very high frequencies, still remains. Figure 14 demonstrates a severely damaged organ of Corti in basal turn and a morphologically intact organ of

Corti in middle and apical turns, after 4050 mg of streptomycin sulfate injection. The hearing improvement reported by Schuknecht (ref. 5) in some Ménière's cases after

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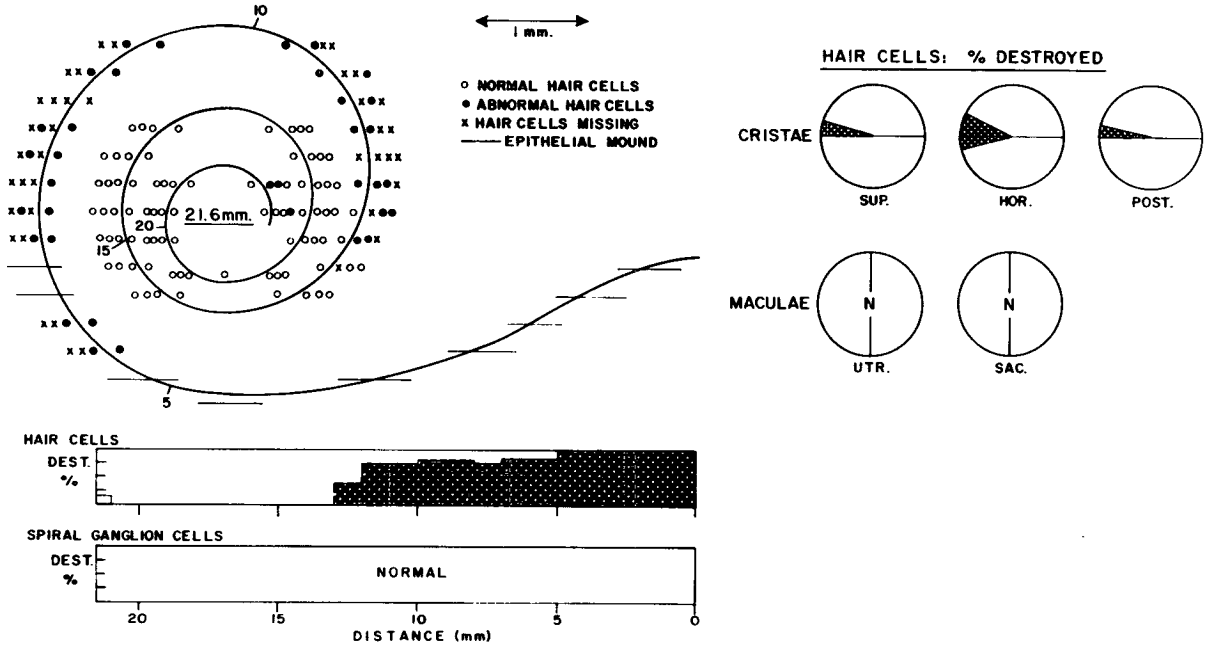


Figure 9.—Graphic reconstruction showing slight vestibular pathology and moderate cochlear damage after administration of 3000 mg of streptomycin sulfate.

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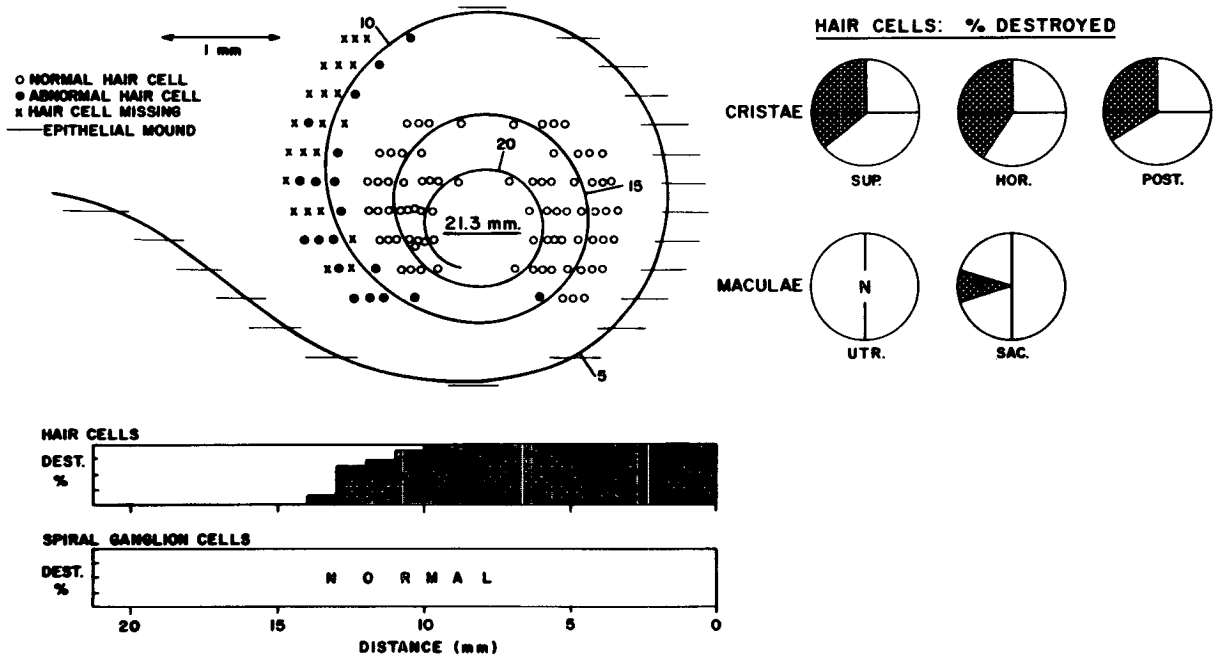


Figure 10.—Graphic display showing moderate to severe pathology both in cristae and in organ of Corti after administration of 3350 mg of streptomycin sulfate.

the parenteral administration of streptomycin sulfate, was quite remarkable in middle and low frequencies; however, it was not quite clear in high frequencies. McGee et al.

(ref. 15) reported cochlear pathology with hearing losses in four of their seven cats after the parenteral use of streptomycin sulfate (75–200 mg/kg daily, total 1800–

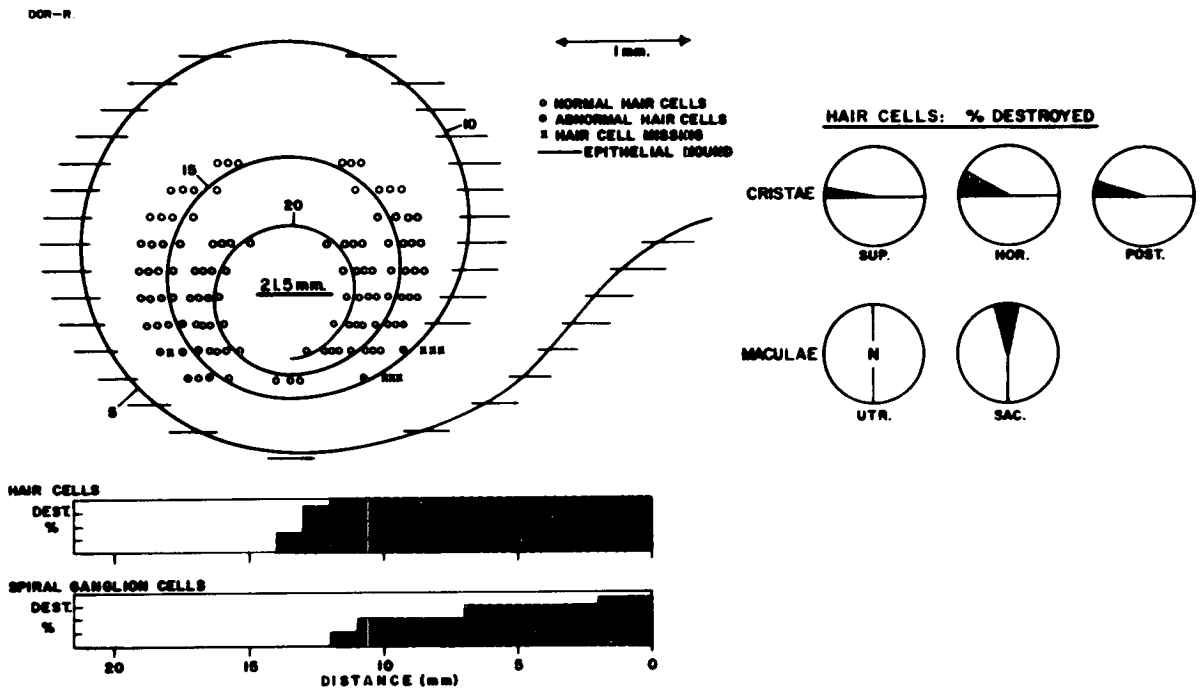


Figure 11.—Graphic reconstruction demonstrating very slight vestibular end organ pathology after administration of 4050 mg of streptomycin sulfate injection.



Figure 12.—Photomicrograph showing normal organ of Corti from upper middle and upper basal turns in a normal squirrel monkey.  $\times 260$ .

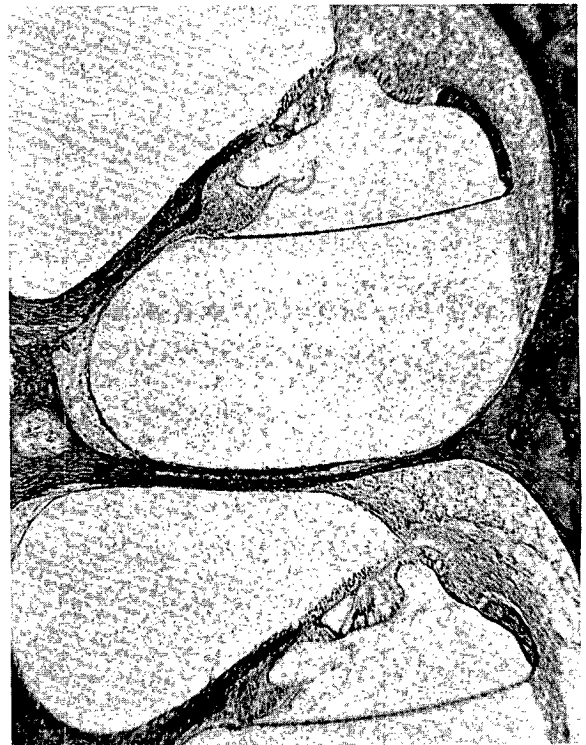


Figure 13.—Photomicrograph demonstrating partial outer hair cell loss in upper middle turn, and total loss of outer hair cells in upper basal turn, after administration of 3000 mg of streptomycin sulfate injection.  $\times 240$ .



Figure 14.—Microphotograph demonstrating entire cochlea, with moderate pathology in organ of Corti and spiral ganglion, after administration of 4050 mg of streptomycin sulfate. Notice loss of primary neurons in osseous spiral lamina of basal turn. Organ of Corti and spiral ganglion are morphologically intact in middle and apical turns.  $\times 30$ .

5600 mg); and two of these were accompanied with spiral ganglion cell lesions. Our present data demonstrate cochlear damage similar to their findings. The severity of the cochlear lesions was not always parallel to that of the vestibular end organs.

A difference may exist among different antibiotic concentration levels in the serum, among different species, or different individuals, etc. And the reactions which were exhibited by Ménière's diseased ears and normal ears may not be the same. However, extreme caution should always be used in the parenteral administration of streptomycin sulfate to primates, for the purpose of vestibular ablation, since some monkeys in our present study exhibited evident cochlear damages.

The significance of these findings may be

summarized as follows: Damage to the hearing end organ occurred following the administration of the smallest dose of streptomycin sulfate sufficient to depress the semi-circular canal function. However, the amount of streptomycin required to produce ataxia in monkeys was much greater than in the case of man.

In the squirrel monkey, neither a normal caloric threshold nor susceptibility to vomiting in the Slow Rotation Room was a reliable indicator of morphological normality of the canal crista. From the clinical standpoint, the fact that the effects of treatment were almost reversible suggests that either initially some of the morphological or biochemical changes were of a reversible nature, or that a compensatory function from

higher vestibular system occurred, or both.

Streptomycin sulfate did not result in any severe morphological destruction in the maculae in most monkey ears. Therefore, the loss of susceptibility to vomiting in the Slow Rotation Room was possibly due to suppression of the canal function, and the term "canal sickness" is appropriate, because the essentiality of the canal function in the

etiology of vomiting was demonstrated. The appearance of ataxia in some monkeys with no pathology in the maculae, and its subsequent disappearance, indicates that this disturbance also had its possible genesis in the canal crista. The administration of streptomycin sulfate is a useful method of selectively suppressing the semicircular canal function.

#### REFERENCES

1. FOWLER, E. P.: Streptomycin Treatment of Vertigo. *Trans. Amer. Acad. Ophth. and Otolaryng.* 52, 1948, pp. 239-301.
2. HAMBERGER, C. A.; HYDEN, H.; AND KOCH, H.: Streptomycin bei der Meniereschen Krankheit. *Arch. f. Ohren-, Nasen- u. Kehlkopfh. 155*, 1949, pp. 667-682.
3. RUEDI, L.: Therapeutic and Toxic Effects of Streptomycin in Otology. *Laryngoscope* 61, 1951, pp. 613-636.
4. HANSON, H. V.: The Treatment of Endolymphatic Hydrops with Streptomycin. *Ann. Otol.* 60, 1951, pp. 676-691.
5. SCHUKNECHT, H. F.: Ablation Therapy in the Management of Menière's Disease. *Acta Otolaryng. (Stockh.) Suppl.* 132, 1957, pp. 1-42.
6. HAWKINS, J. E., JR.: Cochlear Signs of Streptomycin Intoxication. *J. Pharm. Exper. Therap.* 100, 1950, pp. 38-44.
7. HAWKINS, J. E., JR.; AND LURIE, M. H.: The Ototoxicity of Streptomycin. *Ann. Otol.* 61, 1952, pp. 789-809.
8. WINSTON, J.; ET AL.: An Experimental Study of the Toxic Effects of Streptomycin on the Vestibular Apparatus of the Cat; Part 1. The Central Nervous System. *Ann. Otol.* 87, 1948, pp. 738-753.
9. WINSTON, J.; ET AL.: Further Experimental Studies of the Toxic Effects of Streptomycin on the Central Vestibular Apparatus of the Cat. *Ann. Otol.* 58, 1949, pp. 988-996.
10. WINSTON, J.: Clinical Problems Pertaining to Neurotoxicity of Streptomycin Group of Drugs. *Arch. Otolaryng.* 58, 1953, pp. 55-61.
11. RUEDI, L.; FURRER, W.; ESCHER, F.; AND LUTHY, F.: Toxische Wirkungen des Streptomycins. *Acta Otolaryng. (Stockh.) Suppl.* 78, 1949, pp. 66-77.
12. RUEDI, L.; ET AL.: Further Observations Concerning the Toxic Effects of Streptomycin and Quinine on the Auditory Organ of Guinea Pigs. *Laryngoscope* 62, 1952, pp. 333-351.
13. CHRISTENSEN, E.; HERTZ, H.; RISKÆR, N.; AND VRA-JENSEN, G.: Histological Investigations in Chronic Streptomycin Poisoning in Guinea Pigs. *Ann. Otol.* 60, 1951, pp. 343-349.
14. BERG, K.: The Toxic Effect of Streptomycin on the Vestibular and Cochlear Apparatus. *Acta Otolaryng. (Stockh.) Suppl.* 97, 1951, pp. 1-77.
15. MCGEE, T. M.; AND OLSZEWSKI, J.: Streptomycin Sulfate and Dihydrostreptomycin Toxicity; Behavioral and Histopathologic Studies. *Arch. Otolaryng.* 75, 1962, pp. 295-311.

#### DISCUSSION

C. SMITH: There are two points that I would like to make in discussing Dr. Igarashi's paper. One is in confirmation of the data that he has presented and the second is to add a little bit more about the otolithic organs. I also have been studying sections of the ears from a series of squirrel monkeys that were treated with streptomycin in Pensacola by Dr. Graybiel, Lt. Meek, and Lt. McLeod. They were both treated and tested here, and the pathological results are mostly in agreement with Dr. Igarashi's results.

The damage in the cristae varied from nuclear pyknosis in the hair cells and supporting cells to loss of about three-fourths of the hair cells in some of the cristae. There was hair cell loss in the organ of Corti in each of these animals. In my series this

was restricted to the basal turn and often restricted to an area close to the round window. Many of the maculae of the saccules and utricles were normal in appearance, as in Dr. Igarashi's animals. In some there seemed to be hair cell damage as evidenced by nuclear extrusion from some of the hair cells and perhaps some of the supporting cells. In one animal the cell count of hair cells in the utricle was about 20% lower than that counted in comparable sections from a normal utricle. However, the value of such a cell count would be questionable because it would have to be based on a great deal of normal data, and I don't have that data at the present time.

It is the changes in the otolithic organs that I would like to discuss in a little more detail. First,



it is not too difficult to detect hair cell loss in the cristae of the semicircular canals or in the organ of Corti because the hair cells in the organ of Corti are arranged in four rows, and one can easily determine if the hair cells are present or absent. In the cristae they are arranged in a single layer and here again one can determine, especially across the top of the crest, when hair cells are present or absent and when the supporting cells only are left.

The hair cells in the maculae, on the other hand, are sort of "pseudo stratified," and it is more difficult to determine when a few hair cells would be lost in these organs. In an osmic acid fixed ear, embedded in epon and stained with a basic fuchsin, the basal layer of supporting cell nuclei and the nuclei above, which belong to the hair cells, can be seen. All the hair cells reach the surface of the macula, and for this reason I said they were "pseudo-stratified." They only appear to be stratified because some are shorter and others are longer. There are supporting cells in between. If such a hair cell, which is very long and constricted in its supranuclear portion, were lost, the supporting cells could very easily fill in the space and the loss of a few of these cells might not be readily evident. I find it difficult to believe that there should be the hair cell loss we have found in the cristae of semicircular canals and in the organ of Corti without any pathology at all to the maculae and, in fact, recently Wersäll and Duval, who used a somewhat stronger dose of streptomycin sulfate in the guinea pig, showed that there were similar changes in the hair cells of both cristae and maculae as well as the organ of Corti. We thought that if we should examine the maculae at high resolution perhaps something more would be evident. Thus we examined material from two streptomycin treated animals and from two normal animals. The normal animals were used as controls. The basic fuchsin stained the hair cells and the supporting cells rather evenly and the only more densely stained materials were the nuclei and the mitochondria.

Figure D1 shows material taken from the utricle from the second control animal. This is a rather tangential section as evidenced by the double rows of supporting cell nuclei. On the right we see that the supporting cells are evenly stained but on the left there is some uneven staining and the supporting cells seem not to be stained so densely.

We also obtained a basic fuchsin stained, osmic acid fixed, preparation from the utricle of a squirrel monkey that received 60 milligrams of streptomycin sulfate, per kilo, per day for 26 days. The hair cells appeared to be slightly shrunken. The animal was fixed 8 months after cessation of treatment. The supporting cells were much less densely stained.

Figure D2 shows material from the saccule of this animal, and here again we see that the supporting cells seem to be less densely stained than those

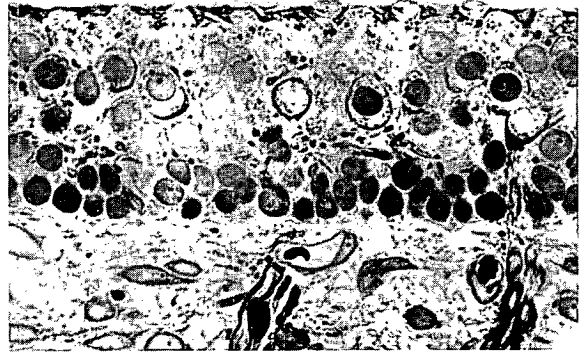


Figure D1.—Photomicrograph of section from utricle of normal squirrel monkey ear.  $\text{OsO}_4$  fixed; epon embedded; basic fuchsin stain.  $\times 600$ .

in the normal. This is not a consistent finding; it is only intermittently found in these maculae. The electronmicrographs which are made on much thinner sections do not show this nearly so well.

I wish to note some structures in order to give a base value for comparison with the pathological material. In the entire thickness of the saccule from a normal control animal, the outline of the hair cells is fairly regular. The supporting cells are filled with normal cell organelles. This supporting cell seems to have fewer organelles. With a somewhat higher magnification of hair cells its regular outline and two supporting cells can be

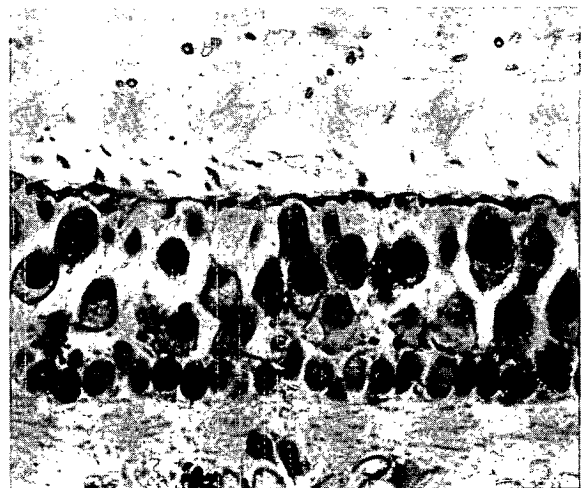


Figure D2.—Photomicrograph of section from saccule of streptomycin-treated squirrel monkey ear.  $\text{OsO}_4$  fixed; epon embedded; basic fuchsin stain.  $\times 500$ .

detected. The base of supporting hair cells does not seem to contain as many cell organelles as one finds up at the apex.

The hair cells from the ear of a streptomycin treated squirrel monkey appear to be much more irregular in outline, as if they are shrunken. The supporting cells seem no different from the normal supporting cells. In another section from this same animal the outline of the hair cells appears to be more irregular than in the normal, as if they were shrunken. There is no difference in the cytoplasmic contents of these supporting cells as shown by electronmicroscopy. We find the same type of cell organelles as are found in the normal maculae. There does not seem to be a deficiency in any particular type of cell organelles. And I would expect that the lighter staining in the basic fuchsin stains is probably due to a greater water content and a greater dispersion of the usual cytoplasmic contents.

Certainly this is not any definite proof for pathology in the otolith organs. However, as I said, one would hardly expect to find active cell degeneration at this period. I feel, however, that we should look for some active pathological processes in earlier animals before concluding that the hair cells of the cristae are selectively injured by streptomycin or by other drugs.

IGARASHI: It is quite true that to evaluate the hair cell pathology accurately is very difficult. I certainly agree with Dr. Smith that if we use the electronmicroscope we might detect more pathological findings in the maculae. Also if we increase the doses of streptomycin, we would expect higher serum concentrations of the drug, and hence in the vestibular organs, and then we might expect more severe injury to the maculae and to the cristae. However, our initial purpose was how to abolish canal sickness, at the same time avoiding injury to the organ of Corti. This was our initial purpose in the streptomycin experiment.

Talking about the vestibular ablation, if we try to destroy all vestibular end organs, that can be done surgically. Or even some part of the otolith organs can be destroyed surgically. However, there is no other way to take out canal function completely and preserve otolith function, without using this streptomycin sulfate technique. Streptomycin sulfate may be a means of completely suppressing canal functions with little or no demonstrable loss of function of the otolith organs.

SPOENDLIN: There are important differences in the action of different types of streptomycin in humans and different animals. In humans, streptomycin has a much more selective action on the vestibular apparatus than on the cochlea. The opposite is true for d-hydrostreptomycin which has in humans a much stronger action on the cochlea than on the vestibular apparatus. As far as I understood, you

did your pathological studies only after the recovery time. Is that correct?

IGARASHI: Right.

SPOENDLIN: Of course, the thing to do now would be to carry out pathological studies at successively increasing intervals following streptomycin sulfate administration.

IGARASHI: This certainly should be done and we are planning to do it. Talking about the difference between species, I agree with you. In some countries with a high incidence of tuberculosis, streptomycin is used quite extensively. Even though we don't have so many deaf persons, maybe less than 1% of streptomycin treated people, I know that this drug has very different effect in different species.

GRAYBIEL: I should like to ask a question which is based on the fact that we can measure counterrolling in persons fairly accurately but we don't have any good notion as to what a certain loss of counterrolling function means in terms of the ability of the person to perform any of his purposeful activities. To a lesser extent this is also the case for the functions of the semicircular canals. The question is whether such clinical studies in the squirrel monkey supported by pathological findings would have worthwhile application to man? The clinical-pathological correlations in squirrel monkeys given streptomycin sulfate disclosed the unexpected finding that very considerable loss of sensory epithelium of the cristae was associated with little or no loss of function as determined by clinical tests. It would be important to know how far this is true for man.

WOLFSON: We were very surprised in attempting selective destruction of the labyrinth by using ultrasonic techniques in humans for Menière's disease and in this case, we also find, very amazingly, that in several cases immediately after ultrasonics you have a marked suppression of vestibular function to caloric testing. And after a period of months you find the vestibular function returning right back to normal levels in several cases. And again, this is the most amazing thing because in animals after they have been sacrificed 4 to 6 weeks after ultrasonics has been applied to a labyrinth you find a complete destruction of the neuroepithelium, and it is amazing how in humans this recovery may take place. Apparently the vestibular apparatus has a great recovery power. We have been working for the last year and a half along these lines using cryogenic surgery applied to the labyrinth of animals, primarily monkeys. In using this technique we were able to apply temperatures down to about minus 180°C and for varying durations. In monkeys we can destroy their labyrinths or obtain a marked suppression of vestibular function. We have not up to this time finished the histological studies.

SPOENDLIN: In studying normal maculae I have

been surprised to find how many sensory cells have been lacking. In my paper, I presented a figure that features a "hole" where the usual normal sensory cells should be. This indicates that even under normal conditions some sensory cells wear out and

break down. And I keep asking myself if eventually a regeneration of sensory cells could take place, a phenomenon which was demonstrated for instance in the taste buds but which was never thought to occur in the vestibular sensory epithelia.

**APPENDIX**

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