NASA SP-314

FIFTH SYMPOSIUM ON THE ROLE OF THE VESTIBULAR ORGANS IN SPACE EXPLORATION

NAVAL AEROSPACE MEDICAL INSTITUTE
NAVAL AEROSPACE MEDICAL CENTER
Pensacola, Florida

August 19–21, 1970

NATIONAL AERONAUTICS AND SPACE ADMINISTRATION

NASA SP-314

FIFTH SYMPOSIUM ON

THE ROLE OF THE VESTIBULAR ORGANS IN SPACE EXPLORATION

Held under the auspices of the Committee on Hearing, Bioacoustics, and Biomechanics, National Academy of Sciences–National Research Council, and assisted by the Office of Advanced Research and Technology, National Aeronautics and Space Administration.

Naval Aerospace Medical Research Laboratory
Naval Aerospace Medical Institute
Naval Aerospace Medical Center

Pensacola, Florida

August 19–21, 1970

General Chairman: ASHTON GRAYBIEL

Scientific and Technical Information Office 1973
NATIONAL AERONAUTICS AND SPACE ADMINISTRATION
Washington, D.C.

For sale by the Superintendent of Documents,
U.S. Government Printing Office, Washington, D.C. 20402
Price $4.05 Stock Number 3300-00498
Library of Congress Catalog Card Number 72-600319

Foreword

The fifth symposium emphasizing vestibular problems in space exploration divides into two parts. The first part is operationally oriented, and the second is devoted to recent contributions to our knowledge of the vestibular system.

More than 100 attendees representing six countries were welcomed by Dr. Walton L. Jones on behalf of CHABA, National Academy of Sciences–National Research Council, and the National Aeronautics and Space Administration. Dr. Jones emphasized the unique opportunity in space flight to investigate vestibular function when the stimulus due to gravity is lifted. He noted that this was one of many related problems of a complex nature, requiring a multidisciplinary approach if artificial gravity is deemed necessary on extended space missions. Dr. Jones pointed to the proceedings of the vestibular symposiums as an example of the fading lines of demarkation among life scientists, physicians, and engineers.

The keynote address by Mr. Douglas R. Lord on "The Space Station" was an authoritative account, beautifully illustrated, of an artificial satellite of modular construction that would serve admirably as a place to live and work. Planning the space station represented an enormous undertaking, and never before was so much deliberation and research given to "human habitability" in an exotic environment. Mr. Lord's presentation was backed up by Mr. George A. Keller's description of a proposed rotating space station for the study of effects of fractional *g*-loads. Other speakers dealt with problems likely to be encountered in a rotating space station, including locomotion, performance of a variety of tasks, physical fitness, and vestibular side effects.

Dr. Charles A. Berry, in opening the first session, presented a detailed account of the motion sickness experienced by nine astronauts in orbital flight. The conclusion was reached that motion sickness, although a problem, was not a serious problem in any of the nine astronauts. The need to take into account benefits as well as undesirable side effects resulting from exposure to weightlessness was emphasized.

If a decision is made to rotate a portion of a space station, a second cause of vestibular side effects must be taken into account. Although motion sickness is, in all likelihood, the chief side effect in the weightless and rotating environments, the problems posed are different. On transition into weightlessness it appears that many persons are less rather than more susceptible to motion sickness, and preflight identification and adaptation are not readily achieved. Moreover, the "worst case" situation is experienced immediately if head movements are made and the problem may involve treatment rather than prevention of motion sickness. The rotation of a spacecraft is easily simulated under ground-based conditions; thus preflight determination of susceptibility to rotation and, if need be, adaptation to rotation are readily achieved. Moreover, by the use of a rotating chair aloft, counterrotation makes it possible to use an incremental adaptation schedule that would avoid eliciting symptoms. Thus, in terms of coping with motion sickness aloft (as distinct from the

overall magnitude of the problem), the greater difficulty is expected in the weightless rather than in the rotating environment.

Presentations in the second part of the symposium dealing with the vestibular system included reports on the morphology of the end organs, central nervous system connections, habituation of vestibular side effects, and models based on side effects of canalicular and otolithic origin.

Recent additions to our knowledge of central nervous system connections deserve special attention, for the reason that the reflex vestibular system revealed by classical morphological techniques is not extensive, and it is clearly evident that vestibular activity (or influences) reaches far beyond these anatomically defined boundaries. This has encouraged the use of other than classical morphological techniques to reveal additional vestibular pathways. When, for instance, the electrophysiologist stimulates (often in a highly abnormal manner) the vestibular nerve or a smaller component of the vestibular nervous system, the question arises whether the stimulus condition is analogous to that used in eliciting vestibular side effects in normal persons. If we have not used a wrong analogy, care must be used in categorizing (from normal to abnormal) vestibular pathways defined by other than classical morphological techniques.

The question has been raised by a number of investigators whether a sixth symposium will be held in Pensacola. During the 6 years that have elapsed between the first and fifth symposiums in this series, the opportunities to participate in meetings devoted wholly or in part to the vestibular system have greatly increased. In consequence, the reasons for holding a sixth symposium in Pensacola would depend mainly on the desirability to see and learn about equipment, either not available or not so readily available elsewhere, or to focus attention on specific operational problems. The fact that the vestibular problems have been identified (that is, given names) does not increase our understanding of the problems.

Rather than excuses for the delay in publication of the proceedings and for the failure to include the discussions, we offer our apology.

ASHTON GRAYBIEL
Naval Aerospace Medical Research Laboratory
Naval Aerospace Medical Institute
Naval Aerospace Medical Center
Pensacola, Florida

Contents

SESSION III

Chairman: Ralph W. Stone, Jr.

SESSION IV

Chairman: Brant Clark

SESSION V

Chairman: John Billingham

SESSION VI

Chairman: César Fernández

SESSION VII

Chairman: Laurence R. Young

Welcoming Address

WALTON L. JONES
Office of Advanced Research and Technology, NASA

On four previous occasions I have had the privilege of welcoming you, on behalf of CHABA, National Academy of Sciences–National Research Council, and the National Aeronautics and Space Administration, to a symposium on The Role of the Vestibular Organs in Space Exploration. This is a pleasure that I find is not dimmed through repetition. Today, as we gather for the fifth of these symposia, we will be presented with some of the most advanced information extant in the general field of vestibular physiology. We also will deal with some very practical issues of importance for the design of future spacecraft. In all, I anticipate that the present meeting will be every bit as productive and rewarding as the previous symposia.

The scientist of tomorrow—and I hesitate to use such terms as physiologist, physician, and engineer, in view of the rapid trend toward multidisciplined research, which is fading the lines of demarcation among these professions—will view the combined reports of these meetings as the single most comprehensive treatise dealing with the vestibular system and its importance in space-age living. The file of these reports will for the foreseeable future occupy a place of prominence on the desks of individuals conducting research in this field. The use of these documents also will tend to accelerate the trend toward multidisciplined research I noted a moment ago. Here the final product has not resulted from the labors of a single individual, as was the case with Helmholtz's magnificent opus on physiological optics, but is rather the result of research efforts in all of the biological and physical sciences, including even the new science of computer modeling.

Now let us turn for a moment to the manner in which I feel these symposia are contributing to the field of medicine. In 1822 a Canadian named Alexis St. Martin suffered an accidental gunshot wound that caused a great opening in his side. Miraculously, St. Martin survived the accident, although he was left with a 6.3-cm-diameter fistula that led directly through skin and muscle into the stomach. The doctor attending St. Martin recognized that this peephole into his stomach presented a unique and priceless opportunity for a systematic study of the digestive system in operation. Invaluable medical knowledge was gained through the exploitation of this opportunity.

In a similar matter the medical profession again is presented with a unique opportunity for an increased understanding of physiological functioning. Orbital flight represents the first instance in which it is possible to study in any detail the functioning of the vestibular system in the absence of normal gravity. Plans have been under way for several years to take advantage of these circumstances through an orbital experiment in which the direct output of the otolith will be measured during weightlessness. Dr. Torquato Gualtierotti had planned to speak to you on this experiment. However, he is in the final preparation for the launch. There are booklets available describing the experiment. The impetus for this flight experiment comes, at least in part, from discussions held during the course of previous vestibular meetings.

Now let us turn to a consideration of the value of these meetings in direct support of NASA objectives. A major issue throughout the biomedical community, and also throughout NASA, can be expressed quite simply: What is the definitive role of man in space? We need to know what man can do best in the space environment. What are his performance capabilities in roles for which

automated hardware systems are not feasible? Finally, and of particular importance, what support must be provided for the man in order to ensure that he will perform optimally in space? It is on this latter point that the discussions in these meetings will be of value to NASA. At the moment, one of the major unresolved issues in the area of human support deals with the possible need for an artificial-gravity system in spacecraft. Dr. Charles Berry of the Manned Spacecraft Center will present some excellent information from recent Apollo missions, which should serve to trigger extensive discussion on this point. The NASA Office of Manned Space Flight and the Manned Spacecraft Center require just such a careful consideration of these data as they deal with design decisions concerning the next generation of spacecraft. The Office of Advanced Research and Technology also benefits as these meetings pinpoint gaps in our knowledge and then point the way for OART research programs that, in turn, will be able to supply the information required by OMSF and MSC 5 to 10 years from now. Thus the information presented at these meetings, and the subsequent critiques, are valuable in supporting the various internal functions of NASA.

As one remaining point, I should like to note that these meetings give us in NASA, in DoD, and in industry a chance to interact at some length with members of our leading universities. This close working relationship, leaning as it does on the tremendous capability residing in the academic world, is very important for NASA and must be maintained through meetings such as these.

Finally, I should like to take just a moment for a different thought. Several times I have mentioned the contribution of persons from various disciplines and from various working environments and have stated that the results of these meetings come through the efforts of many persons. However, there is one individual without whose drive, powers of organization, and scientific prestige the meetings never would have occurred. I refer, of course, to Dr. Ashton Graybiel. We all owe him a note of genuine appreciation.

KEYNOTE ADDRESS

The Space Station: Key to Living in Space

DOUGLAS R. LORD
Office of Manned Space Flight, NASA

It is an honor to be given this opportunity to make the keynote talk. As I mentioned to Admiral Gray, the last time I was in Pensacola was a few years ago when I was a 20-year-old ensign being discarded by the Navy presumably because I was too old to cut the mustard. It is a real pleasure to be back here in Pensacola again and to see so many Navy people. I chose the subject "The Space Station: Key to Living in Space" because, as I looked at your agenda and saw so many medical subjects, being an engineer, I felt the one thing I definitely could not say anything about was anything medical. I could get nothing but trouble from you. As you know, I have been devoting much of my last several years to the subject of the space station, and I would like to give you a status report of where we stand today, how we got to where we are, and what we are looking forward to in the future.

Last year, you may remember, as a result of the successful lunar landing mission of Apollo 11, the President appointed a Space Task Group and asked them to take another look at the space program and to give him advice as to where we should be going in the future. The task group recommended to the President that our main purposes in space should be three: exploration, the accumulation of scientific knowledge, and deriving practical applications to benefit man on Earth. The specific objectives related to those major purposes were to continue the exploration of the Moon, to go on and explore the planets and the rest of the universe, to reduce substantially the cost of space operations from what it has been in the past, to extend man's capabilities to live and work in space, to convert these benefits that we have talked about to the usefulness of man, and to encourage greater international cooperation.

I should like to point out that the space station can be related to each one of these efforts. In the area of exploration of the Moon, it may be that later, as the approach to exploration of the Moon evolves, we can consider putting a station in orbit around the Moon and using that as our base of operations rather than a site on the surface of the Moon itself. Thus, while we are conducting surface explorations, we can be readying the follow-on missions and sending exploratory probes down to places of lesser interest on the Moon, but of significance to the overall exploration. I will give you an idea later how our present designs can be adapted to manned planetary missions, how they can serve as way stations for unmanned explorations of the planets, and how they can be used by astronomers in looking at the universe. To reduce the cost of space operations, we have to incorporate features of reuse and long life. Both of these are essential qualities of the station. To extend man's capabilities to live and work in space, we need to acquire experience. This is a key to expanding the practical applications of space. By taking very advanced sensors to a station and using them with men aboard, able to make changes on a real-time basis, we believe we can hasten the development of these types of equipment. Finally, a station that can accommodate 10, 12, or perhaps 50 men in space will give us the opportunity for the first time to have international crews.

The first purpose of the station is to conduct scientific investigations, technological experiments, and research in beneficial applications; so it is a multidisciplinary facility in space, not unlike a

laboratory here in Pensacola or any other place in this country. Second, it would be used to develop space subsystems which we would need for very much longer missions, such as to the planets. Third, the station will enable us to develop new operational techniques and equipment so that we can reduce operating costs. Fourth, it would be useful in developing systems that are applicable to lunar and planetary exploration. Finally, although I should not have put this one last for today's meeting, because I am sure this is the key to the purpose of your sessions this week, it will enable us to better understand man in space.

Over the past decade we have studied many kinds of stations. Figure 1 represents a panorama of the types of space stations that have been considered. We started early thinking very ambitiously: 24 to 36 men, even as many as 100 men, in rotational concepts. You have seen these pictures in *Colliers* and in old books by Wernher Von Braun and others for many years. But we first took serious engineering looks at these in the early Sixties. We then looked at ways of modularizing stations. We looked at ways of extending the Apollo by attaching either the LM or some kind of a small canned laboratory to the command and service module. We looked at the Manned Orbiting Research Laboratory, which was a six- to nine-man station. There was a very substantial effort over several years by Langley Research Center in this area. We have gone on up to single launches of the Saturn V, and we looked at various other configurations.

Two systems got as far as an actual approved program stage. One was the Manned Orbiting Laboratory, a two-man, 30-day mission which the Air Force undertook, until it was canceled last year. The second, of course, is Skylab, our first actual space station, which still is approved and planned for flight in the latter part of 1972. It will be a three-man system. Originally it was planned to use the hydrogen tank of the S–IVB

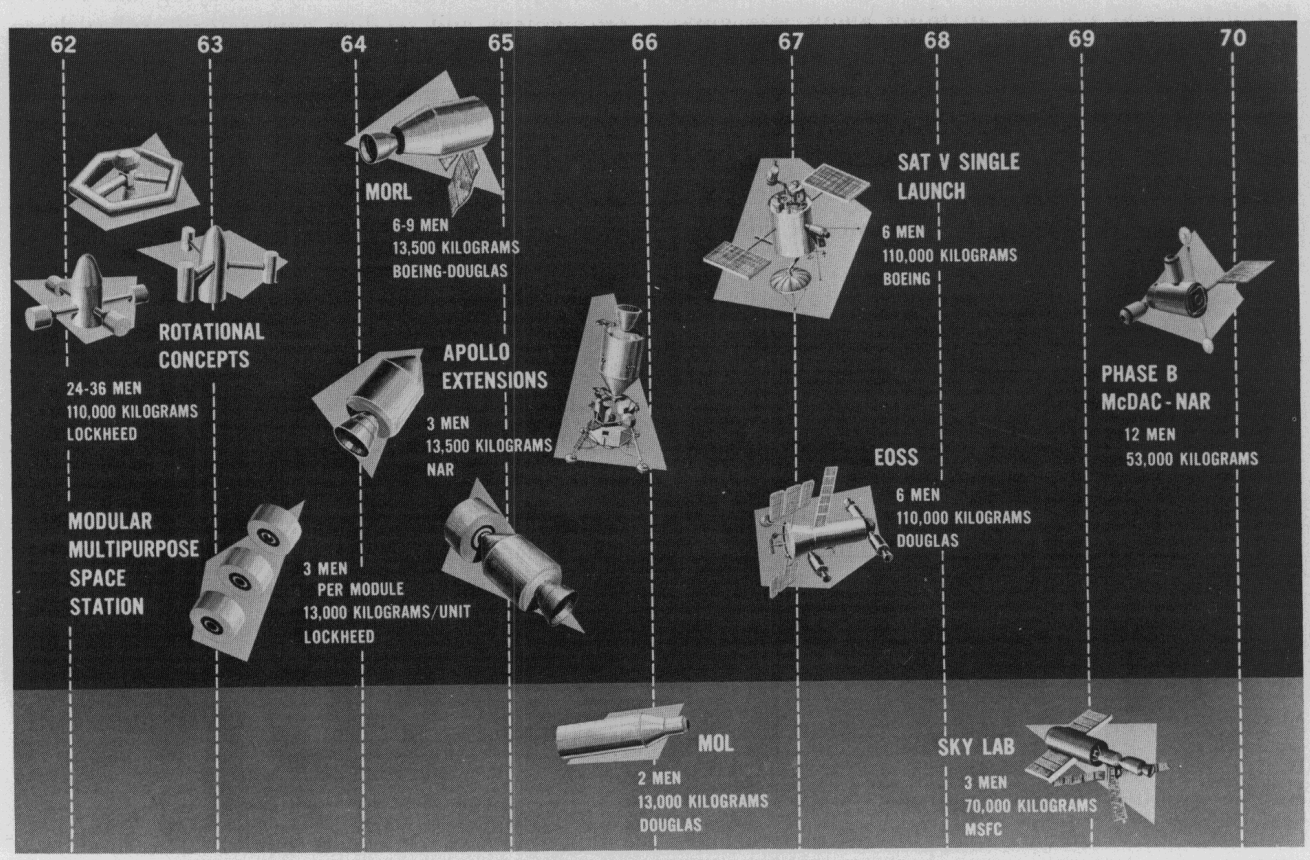


FIGURE 1.—*Evolution of space station concepts.*

stage after it had been used as a propulsion stage. Now it is a completely ground-outfitted stage and is in a sense an embryonic space station, with mission durations of first 1 month and then 2 months. The major emphasis of these missions, from a scientific viewpoint, is on, first, medical experiments for these longer durations, and, second, the Apollo telescope mount, which will be used to conduct a series of solar observations.

Based on all of the past work, we decided this last year to go into a phase-B-level study in depth of the space station. We pooled all the resources we had at that time and said, "Okay, knowing all the facts we have, what kind of guidelines do we want to give these design studies?" These, then, are the guidelines for the phase-B program definition studies that were to end up in preliminary designs. We said we thought the station should have a 12-man crew. The 12-man level gives you enough variation in trained personnel, enough of a crew so that you can have shifts, and a reasonable statistical sample on the men. You can accomplish a reasonable amount of work because all of your crew are not busy just trying to keep the systems going and staying alive. We said we wanted it to be a permanent facility, so it would have to operate for at least a 10-year period. We said we would like it to be able to fit on a Saturn V, whose payload in orbits we were talking about (around 320-km altitude and 55° inclination) is about 82 000 kg. We said, "All right. We will give you a target of 50 000 kg. We will allow 50-percent weight growth, so we do not have to worry about running out of weight." It should not be any bigger than the S–IC and S–II stages, so we gave 10 m as a maximum diameter. The studies quickly went to that maximum diameter, for a number of reasons. You will see later one of the problems of trying to put square rooms in round cans; it is much easier with a larger diameter. But frankly, the problem of people moving around on the station is one of the major reasons for specifying this dimension. We have some second thoughts now that perhaps we should go back and reexamine the 6.7-m diameter, but for the time being I would like to concentrate on the larger one.

We felt that each crewman should have his own private compartment. If we are expecting 12 men to live there for extended periods, 3 to 6 months, we have to make the station a desirable place to go, not just a convenient place or a required place, but really desirable. We want essentially a normal sea-level atmosphere. We wanted to be able to operate without resupply in emergency modes, so that we would not have to abandon in case one of our logistic vehicles did not arrive on schedule. We wanted to close the water and oxygen systems; this is really not required for the station, because we are close to the Earth and can resupply expendables in an open system; but it is desirable to develop closed systems that could be used in planetary missions someday. We should have, for the first time, a sizable amount (say 25 kW) of electrical power available to do the many experiments. We wanted to be able to contain damage, to be able to control and repair it, and to have two independent compartments so we do not have to abandon the space station in emergency situations.

One of the key things we put into the guidelines, from your standpoint, was that the station should be able to do an assessment of the impact of artificial gravity. Therefore our designs accommodate both zero-gravity and artificial-gravity conditions. Our premise was that we would have artificial gravity for maybe 2 weeks, 1 month, 2 months, whatever seemed to make the most sense, and then we would go to a zero-gravity mode for the rest of the normal mission. We said that the core module should be potentially a part of a 50-man space base. When we go to the space base, we made a new statement with respect to artificial gravity. We said, "We do not have a minimum configuration any more. We have a very large facility. In that case we want to be able to provide both artificial gravity and zero gravity simultaneously."

Finally, we said the core module, i.e., the basic 12-man module, should be potentially useful as a planetary mission module. We have people in Congress who give us all kinds of trouble on this point. They say whenever we try to get a station started, "All you're really trying to do is get your foot in the door. You're really trying to go to Mars with it." We are not saying that we are planning a Mars mission. What we are saying is that we do not want this to be a dead-ended program.

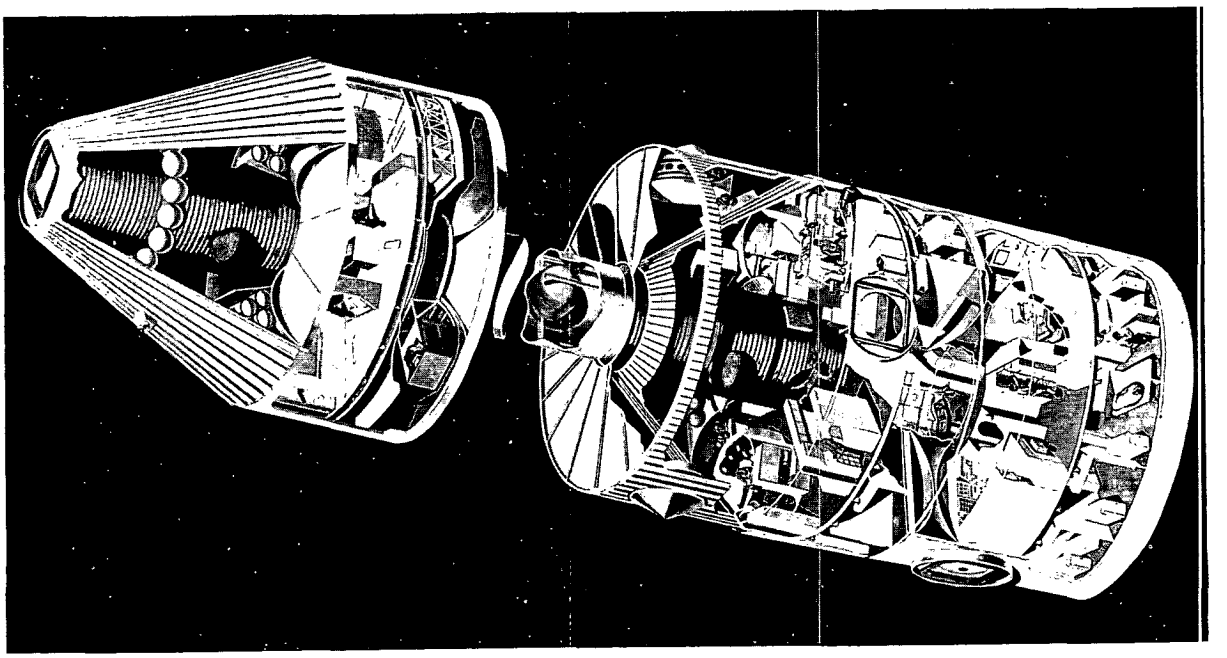


FIGURE 2.—*McDonnell Douglas Corporation 12-man design. Baseline configuration on right; extended module on left.*

I would like to describe now the outputs of the present study effort. Figure 2 shows the 12-man design as developed by the McDonnell Douglas Corporation. They had Martin as a supporting contractor in the experiment area, and IBM in a supporting role on information management. This is their extended-capability configuration. Their baseline configuration was the 10-m cylinder on the right, about 14 m long. Basically it has four decks, each devoted to a particular area of work. Two of them contain crew quarters. One deck is primarily a general-purpose laboratory, and the control station is on another one.

One of the key features of this configuration is that they assumed a radioisotope power supply; therefore you see no large solar arrays such as we have on the Skylab program. Each supply provides 12.5 kW of power, and there are two in the basic module, giving us our 25 kW. Then McDonnell Douglas had an extendible boom which could be attached either to the S–II stage for artificial-gravity assessments or to an extended module such as that shown on the left of figure 2. This concept uses up most of the 50-percent payload margin that I was so careful to point out we were trying to maintain. It cuts us down to about a 10-percent margin, but it gives us a very great increase in capability. They essentially added two additional decks. As the spoke is extended, we have two working decks on the left and four decks on the right. Each deck gives us a different gravity level. In this particular case they took advantage of one deck to put in two centrifuges, one to be used for men and one to be used for plant or animal specimens. All of this could be launched on a single Saturn V.

Figure 3 shows the competitor, Brand X, in this case North American Rockwell. NAR was supported by General Electric. They went the solar panel route, on the feeling that radioisotopes would not be available. They believed that what we should do is develop very large solar arrays. The total area required for this design is 929 m^2 (10 000 ft^2). This area is provided by the four extendible panels shown. NAR's design approach was very similar to that of McDonnell Douglas. They also came up with four decks. The next few figures show details of each deck.

Starting at the bottom of the configuration, figure 4 shows the lower section, containing a

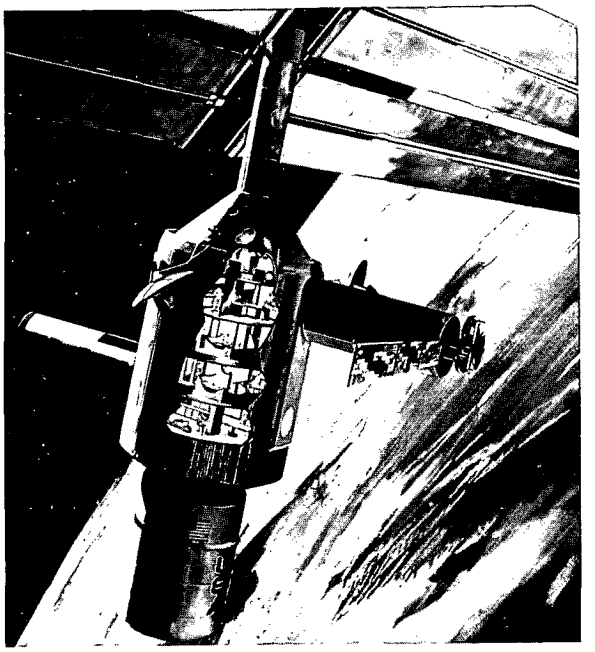


FIGURE 3.—*North American Rockwell space station concept.*

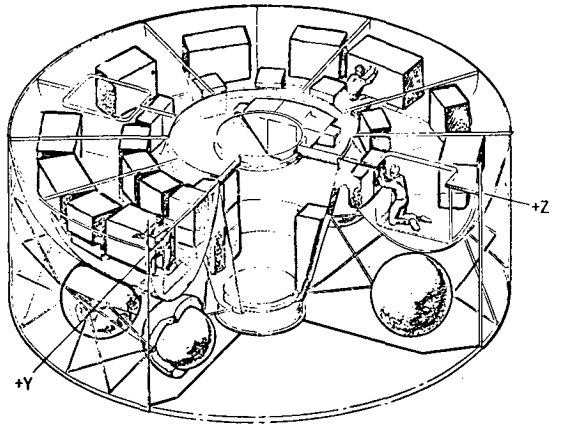


FIGURE 4.—*Lower torus/tunnel/equipment bay arrangement, showing cryogenic tanks in unpressurized portion. Other areas feature shirtsleeve environment.*

toroidal compartment. It is large enough to provide a 1-m circular passageway and the storage of subsystem equipment, bulk storage, and the like. It has two access ports, and there is a docking port at the end of the conical area. Located in the unpressurized volume are tanks for supply of cryogenics.

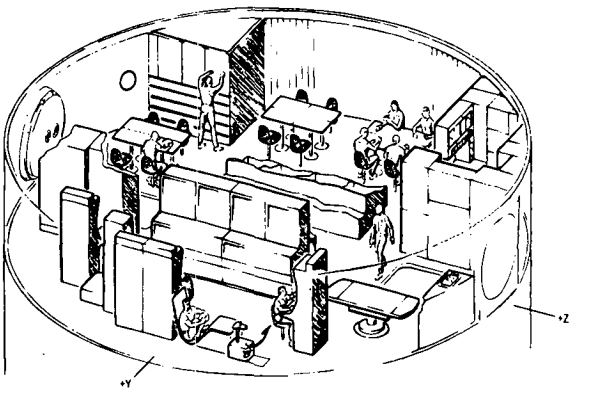


FIGURE 5.—*Deck 1 arrangement, including galley, dining, and wardroom areas and integrated medical and behavioral laboratory measurement system.*

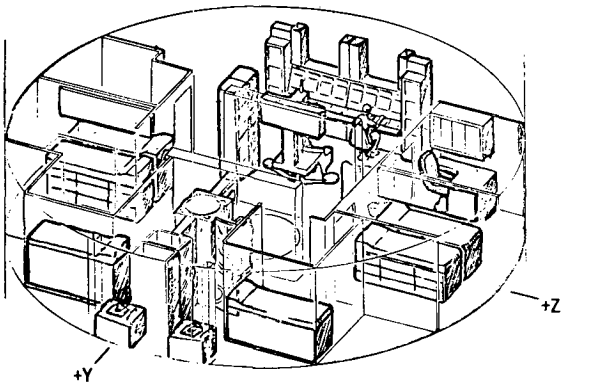


FIGURE 6.—*Deck 2 arrangement, with staterooms for five crewmen plus commander, as well as personal hygiene and primary control rooms.*

Deck 1 (fig. 5) contains the galley, dining, and wardroom areas. It also houses the integrated medical and behavioral laboratory measurement system that Dr. Berry and others will probably have some things to say about later. This deck has two docking ports on it and a clear 1.5-m corridor straight across it so that any large packages of equipment, experimental or otherwise, can be brought in through those ports and then taken up or down through the decks to other ports.

Deck 2 (fig. 6), one of the decks providing living accommodations, has spaces for five of the crew plus the commander. The commander has a little bit larger station; you have to have the right

protocol, you know. This deck also includes a waste-management area and the primary control center for the station.

Decks 1 and 2 are separated from the third and fourth decks by a pressure bulkhead, dividing the station into two separate sections. For emergency purposes we can use either just the first two decks or the third and fourth. Deck 3 (fig. 7) provides another stateroom area, very similar to deck 2. There are staterooms for five of the crew, plus the chief investigator or the chief scientist on board, and another personal-hygiene system. Also the major experiment control is here, and this area can also be used as a backup for the primary station control center. On one side there is an extravehicular airlock that also can be used as an intravehicular emergency access route to transfer between the two sections of the station if necessary.

The fourth deck (fig. 8) is essentially an experiment laboratory, including hard-data labs, photo labs, and bioscience-type equipment. It is essentially a completely open deck for experimenters who want to conduct useful observations and measurements.

At the top of the station is the other toroid (fig. 9), which closes off the second pressure area, and again is used for storage of cryogenics and other bulk-type materials. It also contains the stabilization system, and there is another tunnel area with another docking port at the end; this is normally where the attachment is made to the power supply.

One of the main things we have to remember about a station is that it is not just for the men to live in; it is for them to work in. Table 1 indicates the kinds of disciplines for which we have to provide capabilities on the station. Each area is concerned with certain phenomena. In the astronomy area the main interest is in looking out at solar and stellar phenomena. The space physics area is concerned with either local physics around the area of the station or with taking advantage of its ready access to high-energy cosmic rays. In the space biology area, interest is obviously on low gravity and the absence of normal diurnal changes. In Earth resources, we want to look at the Earth again for obvious reasons. Each of these various phenomena imposes requirements on the station.

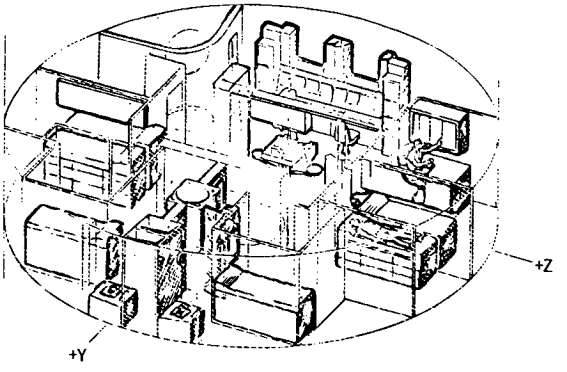


FIGURE 7.—*Deck 3 arrangement, with staterooms for five crewmen and chief investigator, plus personal hygiene and experiment/backup control stations.*

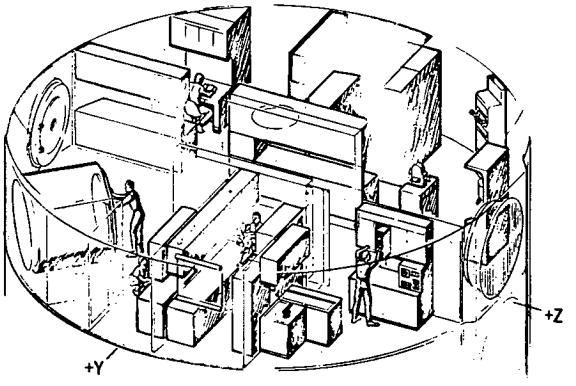


FIGURE 8.—*Deck 4 arrangement, devoted to laboratory facilities.*

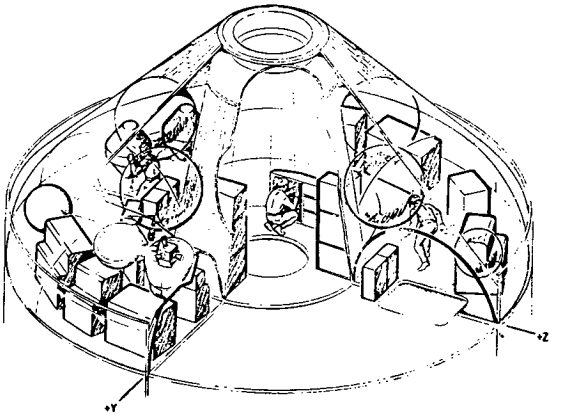


FIGURE 9.—*Upper torus/tunnel/equipment bay, used for bulk storage and stabilization system. Docking port is at top.*

either in terms of stabilization, pointing, or lifetime. What we have tried to do in the station designs is to provide the appropriate capability for each of these phenomena to be observed.

TABLE 1.—*Laboratory Facility Provisions*

Discipline	Phenomena	Activities provided for
Astronomy	Solar and stellar	Observations in radio, IR, visible UV, X-rays and gamma-ray spectra
Space physics	Cosmic ray Plasma physics	High-energy particle sources Hydrogen-helium ratio (relative abundances)
Space biology	Low gravity Circadian rhythm	Plants, small vertebrates, primates, invertebrates & micro-organisms
Earth surveys	Location of earth resources Observation of surface phenomena	Exploratory studies Sensor development
Aerospace medicine	Effects of long-duration zero gravity and artificial gravity	Biomedical Behavioral
Space manufacturing	Zero gravity, vacuum	Precision casting Zone refining Metal forming Large crystal growth
Advanced technology	Natural and induced space environment	Contamination and exposure measurement Large structure assembly Sensor calibration System & component testing

These goals have been pursued by three approaches (fig. 10). The first employs integral laboratory equipment that can be installed before the initial launch or can be brought in at a later time. The 1.5-m hatches and airlocks allow small experiments to be deployed for short periods of time and brought back in for maintenance and replacement. The second approach is the attached mode, where the module is attached full time and is a parasite to the main station. The major purpose of bringing it up separately is so that it can be planned, developed, and assembled separately. When funding is provided and the equipment becomes available, it can be brought up in a separate logistics launch and attached and then remain there until its usefulness is outlived. The third approach is the detached or free-flying module, which is particularly attractive in the astronomy area, primarily because of contamination problems from the station. Even when we go to our closed-loop systems, I am always amazed at how many things like methane gas and other such gasses and leakage of normal atmospheric constituents tend to get out. Therefore, both for contamination reasons and for very precise pointing accuracies, we tend to go to the free-flying or detached modules. In this case they essentially stationkeep with respect to the space station itself, staying within a few hundred or a thousand kilometers of the station, and are periodically returned to the station for maintenance, resupply, etc.

I should like now to describe the artificial-gravity approaches that the two contractors took. NAR took the relatively standard one (fig. 11). Their basic module is attached to the S–II stage, which is the second stage of the Saturn V, and the whole assembly is rotated, producing different gravity

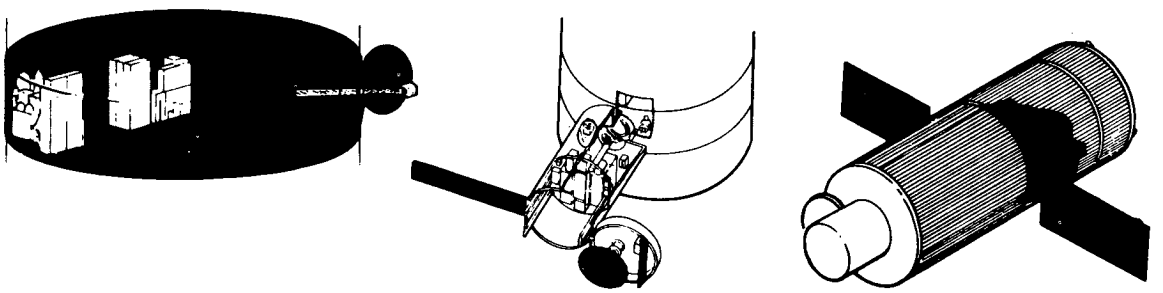


FIGURE 10.—*Equipment operating modes: left, integral; center, attached; right, detached.*

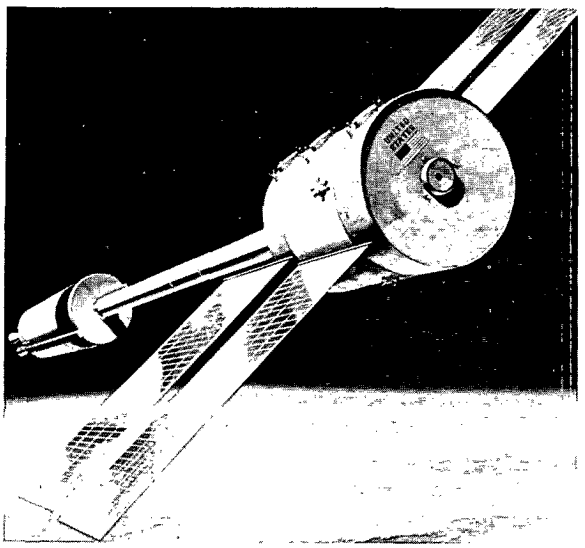


FIGURE 11.—*North American Rockwell artificial-gravity space station concept.*

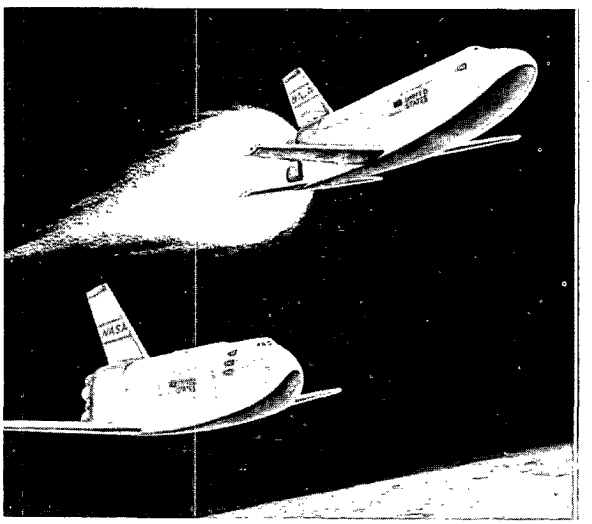


FIGURE 13.—*Two-stage fixed-wing space shuttle.*

levels on each deck. One of the most ticklish problems with this approach is the support of the solar panels during rotation. Therefore they are usually not deployed fully while the station is being rotated.

McDonnell Douglas, in their extended-capability arrangement described earlier, proposed the approach shown in figure 12. At a spin rate of 4 rpm the various decks have *g*-levels from 0.7 down to 0.2 g. Of course they do not have to worry about the solar arrays because they utilized the isotope-Brayton power system.

One of the keys to the station is the matter of logistics. I would be remiss if I did not say something about the space shuttle (fig. 13), because it is our planned method for resupply of the station. It is planned as a two-stage fully reusable system. Figure 13 shows staging, where the first stage, which is about the size of a C–5A but operates at about Mach 10, is returned to Earth by a two-man crew and lands normally, although it is launched vertically. The orbiter stage then would have a cargo bay in the top part of it. We have considered various sizes. The present design is about 4.5 m in diameter by 18 m long. This is large enough to carry any kind of experiment that could be conducted aboard the station, could be kicked out into orbit and operate in a free-flying mode, or could be attached to the station.

For station resupply, figure 14 shows the kind

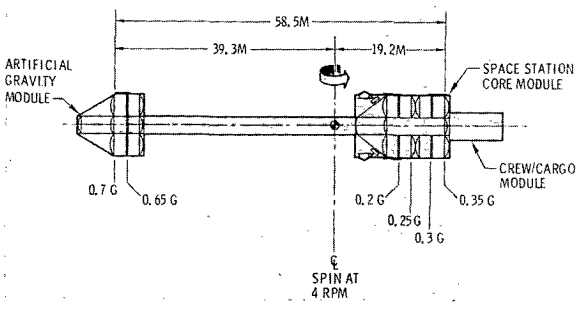
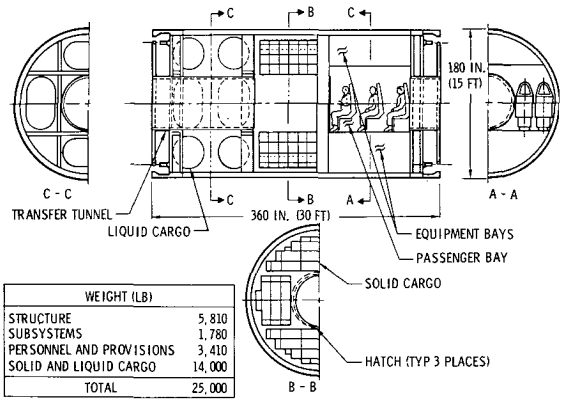


FIGURE 12.—*McDonnell Douglas artificial-gravity test configuration.*



WEIGHT (LB)	
STRUCTURE	5,810
SUBSYSTEMS	1,780
PERSONNEL AND PROVISIONS	3,410
SOLID AND LIQUID CARGO	14,000
TOTAL	25,000

FIGURE 14.—*Cargo/crew shuttle transport module.*

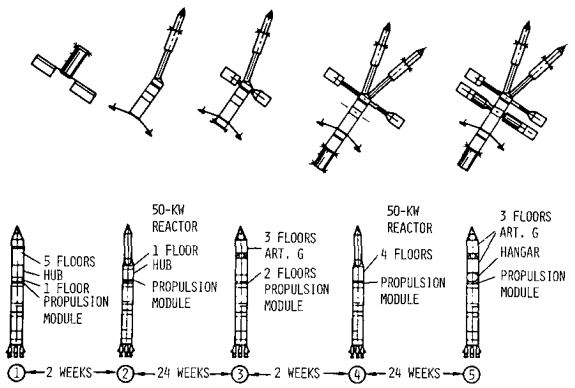


FIGURE 15.—*Space base buildup scheme, showing launch configuration for each step.*

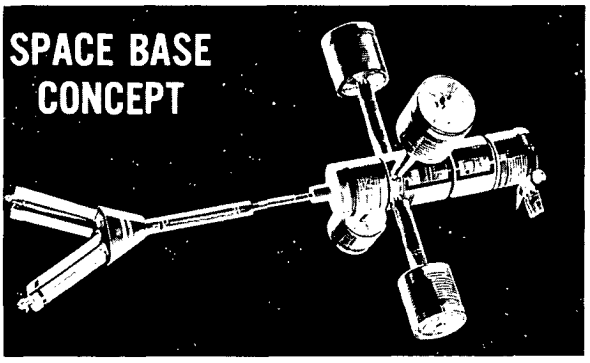


FIGURE 16.—*Rotating space base configuration.*

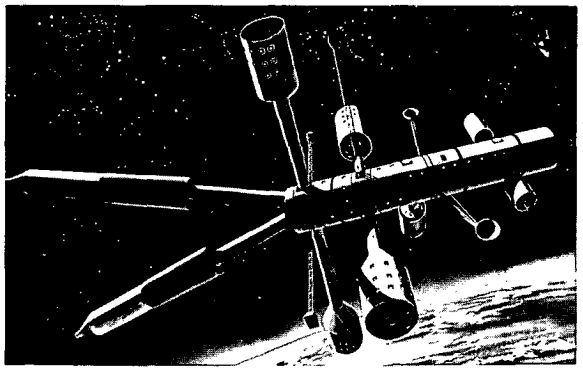


FIGURE 17.—*Rotating space base with counterrotating hub section, providing zero total momentum along with artificial gravity.*

of module or cargo container which would be required. This is a 4.5-by-9-m module which would fit in the cargo bay of the shuttle. Part of the volume would be used for liquid cargo, part of it for solid cargo, and part of it for the 12-man crew that would be taken up to the station. This particular design totals about 11 300 kg, which is approximately the payload capability anticipated for the shuttle.

I said that I would tell you how we plan to go from a station to a base (fig. 15). If we take the initial station that I have been describing, in this case with solar arrays, the question is: How do you use that to build up to a 50-man base? Our design has been planned so that, by adding hub systems and nuclear reactor power supplies, providing 50 to 100 kW of power, then adding rotating modules (again, these modules can be similar to the ones we have designed for the basic station), and using counterrotating systems to reduce the total momentum of the base, we can provide a 50-man capability. Actually, about 50 percent of the usable volume ends up in the zero-gravity central hub, with the rest in the living areas out at the ends of the rotating booms. Two artists' sketches of these configurations are shown in figures 16 and 17. The first (fig. 16) is the counterrotation approach, where each living module accommodates a 12-man crew and rotates to provide the artificial-gravity environment. Part of the hub counterrotates at a rate synchronous with the station's orbital rate, thus providing continuous Earth viewing from one side. The nuclear reactors are placed at the end of the long axial boom to provide a minimum radiation environment to the rest of the base.

The other configuration (fig. 17) is quite similar except that it does not have zero total momentum. A major advantage of the counterrotating system, of course, is that you can change fairly easily the orientation of the total base whenever you want to. In this case it requires considerable force because of the rather strong precession torques.

In closing I would like to say that, from an engineer's standpoint, I think we have the designs in hand for building whatever is desirable in the way of a station or a base. You solve the medical problems and we will solve the engineering problems. It would help if you could get us a little money. Thanks very much.

SESSION I

Chairman: **JAMES W. HUMPHREYS, JR.**
NASA Headquarters

Findings on American Astronauts Bearing on the Issue of Artificial Gravity for Future Manned Space Vehicles

CHARLES A. BERRY
National Aeronautics and Space Administration

SUMMARY

This paper reviews findings for American astronauts that may indicate some alteration in vestibular response related to exposure to zero gravity. Of 25 individuals participating in Apollo missions 7 through 15, nine have experienced symptomatology that could be related to motion sickness. The apparent divergence between these results and those from the Soviet space program, which initially appears great, may reflect the greater emphasis given by Soviet investigators to vestibular aberrations. Presently the incidence of motion sickness, long known as an indicator of vestibular disturbance, seems too low to warrant any positive statement regarding inclusion of an artificial gravity system in future long-term space missions. Where motion sickness has occurred, adaptation to weightlessness has always resulted in abatement of symptoms. In the absence of biomedical justification for incorporating artificial gravity systems in long-term space-flight vehicles, engineering considerations may dictate the manner in which the final ballot is cast.

INTRODUCTION

One of the major purposes of the biomedical effort in support of the Mercury, Gemini, and Apollo manned space flights has been to evaluate the adaptation of astronauts to weightlessness. In general, adaptation has proceeded much more smoothly than some had predicted. For example, concern was expressed about a decreased tolerance to gravity or to increased gravity forces following weightless flight. The Gemini 4 crew sustained a peak of 8.2 *g* during the reentry phase without adverse effects. Apollo reentry forces on return from lunar missions have reached levels near 6.7 *g*. All of these acceleration levels, which had been previously experienced by the crews during centrifuge training, were well tolerated (ref. 1).

Medical and performance data available at the present time do not support a requirement for artificial gravity systems in spacecraft. In the view of spacecrews to the present time, artificial gravity systems are unnecessary for task performance. Crews have learned to live in a weightless state and feel very confident in that state. Many have, in fact, expressed a preference for a 0-*g* environment, since the absence of gravity increases the useful volume of what would otherwise be rather confined work spaces.

On the other side of the coin, the provision of artificial gravity would undoubtedly increase the habitability of spacecraft. Eating would become a simple affair, and locomotion would proceed nearly as it does in the Earthbound environment. With artificial gravity, everyday activities in spacecraft could be carried out as easily and simply as they are in one's own home. Where engineering considerations are concerned, design tasks would be simplified in that restraint systems and locomotion aids would no longer be required. On the other hand, the engineering tasks associated with designing the artificial gravity system itself are formidable and they could be costly.

The issue of the need for artificial gravity in spacecraft of the future is no more clearly resolved among Soviet scientists than it is in the United

States. Valyavski (ref. 2) has summarized the Soviet position by stating that investigations of the problem of weightlessness may indicate that man can form adaptive mechanisms to the weightless state. However, he added that until this is proved, technology would continue in the Soviet Union to attempt to create artificial gravitation. This is a problem which is "much in the forefront of modern cosmonautics." In discussions by the author with various Soviet space medical authorities, it has been indicated that the Soviet medical community finds no basis for requiring artificial gravity, but it is still a question of concern to Soviet space-system designers.

Performance of the vestibular system is one good index of man's adaptive response to the weightless environment, since this sensor system is exquisitely sensitive to gravitational effect. In view of this consideration, close attention has been paid to the function of the vestibular apparatus in weightless space flight. The relationship between the function of the vestibular system and motion sickness is well established; this phenomenon has therefore been regarded as an indicator of vestibular dysfunction.

The object of this article is to review the response of astronauts in the American space program to the weightless environment, using performance of the vestibular system, as indicated principally by the motion-sickness phenomenon, as an index of adaptation to zero gravity, and to attempt to draw inferences from these findings related to the need, or lack of need, for artificial gravity for future space operations.

EARLY EXPECTATIONS CONCERNING VESTIBULAR FUNCTION IN WEIGHTLESSNESS

At the time of the first Mercury mission, there was considerable concern regarding the possibility of inflight motion sickness. Consequently antimotion-sickness drugs have been included in the onboard medical kit since this early manned mission. They were carried in both injectable and oral form. The drugs included were Tigan and Marezine. To the surprise of many, these drugs were not needed. For experimental purposes, attempts were made to provoke vestibular responses by performing head movements in the spacecraft. Crews were instructed to perform head movements cautiously while reaching for or pointing to specific areas in the spacecraft to demonstrate whether perceptual-motor impairment had been provoked. Again, no problem was noted.

THE FIRST INDICATION OF DIFFICULTY

No problems that might be indicative of vestibular disturbance were noted in the brief American flights that preceded the first one-day flight of the Russian cosmonaut G.S. Titov in Vostok 2. Titov was reported to have shown transitory vestibular-autonomic nervous system changes, but his "work fitness" was said to be preserved, and he was reported to have experienced no "essential physiological discomfort" (ref. 3). This first experience of alteration in vestibular function proved to have a rather significant psychological impact on American astronauts who were to follow the Titov flight. One astronaut had become so convinced that he would be ill in flight that he experienced sensations of nausea on the launch pad. However, when this astronaut cautiously performed head movements in flight toward the end of the mission, all went very well and he experienced no real problems.

None of the Project Mercury astronauts experienced any vestibular difficulty that could be linked to exposure to weightlessness. Coincidentally, both astronauts Glenn and Shepard were victims of episodes that produced vestibular disturbances some time after their space-flight exposure, but neither of these incidents was in any way related to the astronaut's weightless experience. Astronaut John Glenn experienced some very marked vestibular disturbances (vertigo, nausea, and nystagmus) after a fall in which he sustained a severe blow to the head in the area of the labyrinth. X-ray examinations indicated that some hemorrhage had probably occurred in the area of the labyrinth. Symptoms, however, cleared over a period of time, and he has had no residual problems and has returned to flying. The case of astronaut Alan Shepard was initially less clear-cut. Several years after his first space flight he reported episodes of vertigo. The presenting symptoms initially indicated viral labyrinthitis. It later became obvious that the astronaut was in fact afflicted with Ménière's disease. As a result, he was grounded for a period of time. His therapy at first included medication, which was

not altogether effective. Twenty-nine months prior to the Apollo 14 flight, the condition was alleviated by surgery that involved the emplacement of an endolymphatic shunt, permitting the endolymph to drain into the cerebrospinal fluid. This operation was completely successful, and the symptoms became totally absent (ref. 4). Astronaut Shepard was evaluated extensively at the Naval Aerospace Medical Institute prior to his participation in the Apollo 14 mission and experienced no difficulty whatever of a vestibular nature prior to, during, or after that mission.

GEMINI RESULTS

Despite the increased internal volume of the Gemini spacecraft, which permitted increased opportunity for movement, no disorientation or motion sickness was reported (ref. 5). Normal central nervous system function, including labyrinthine function, was evidenced by the crewmembers' ability to rendezvous with the Agena vehicle, perform extravehicular activity, and execute accurate reentry maneuvers. During the 8-day Gemini 5 mission and the 14-day Gemini 7 mission, ocular counterrolling was measured and compared with preflight and postflight responses. After flight each pilot maintained his respective preflight level of response, which indicated that no significant change in otolithic sensitivity occurred as a result of the flight (ref. 6).

Another effect that occurred during the Gemini program and has persisted through all missions since that time is an experience of "fullness of the head" as a result of redistribution of the total circulating blood volume. This phenomenon was unfortunately described by some astronauts as a feeling of "hanging upside down on a parallel bar." As a result of this unhappy choice of words, the notion that an inversion illusion had been experienced became rather widespread. This, however, was not the case. Since the earliest observation of this phenomenon, crewmen have looked for this symptom in themselves and in others and have described it as appearing as a roundness of the face, in some cases with red coloration. These observations may yet be verified by photography.

APOLLO RESULTS

Apollo-mission astronauts, like their Soviet counterparts who have reported "space sickness" during nearly every mission (refs. 7 and 8), have experienced motion-sickness symptoms on a number of occasions. The divergence of findings between these later American and Soviet missions and earlier American space missions is difficult to interpret. It should be noted that, since vestibular disturbances appeared early in the Soviet space program, highly trained observers have recorded even very minimal findings and these, in turn, have been interpreted in this country as more significant than they might actually have been. In support of the point that this divergence may be more illusory than real, it should be noted that electrooculograms recorded during the flight of Vostok 3 and 4, sometimes simultaneously with the administration of special tests, revealed no asymmetry of oculomotor reactions or nystagmus (ref. 9). Moreover, vestibulometric studies did not reveal any change in the threshold of sensitivity of the otolith to galvanic current in the Voskhod 1 cosmonauts, and postflight studies did not reveal any substantial changes in the function of the vestibular apparatus. What then accounts for the difference in responses of the Mercury and Gemini astronauts, on the one hand, and the Apollo astronauts on the other? Many factors, including individual variables such as prior space-flight exposure, individual susceptibility to motion sickness, physical condition at the time of flight, and medication taken in flight, may play a part. However, a more obvious difference between the two types of mission should be noted before any attempt is made to evaluate these variables. This is the difference in the relative volumes of the spacecraft employed. The larger the volume of a spacecraft, the greater the opportunity for increased and probably more rapid movement. Excessive head movements, particularly when such movements occur early in the mission, appear from in-flight experience to be directly related to the onset of motion-sickness symptoms.

COMPARISON OF MERCURY, GEMINI, AND APOLLO VEHICLE VOLUMES

The volumes of the three different command modules used to date are compared in figure 1. The Apollo Command Module, in addition to being much larger than the other two vehicles, afforded still further opportunity for movement when the

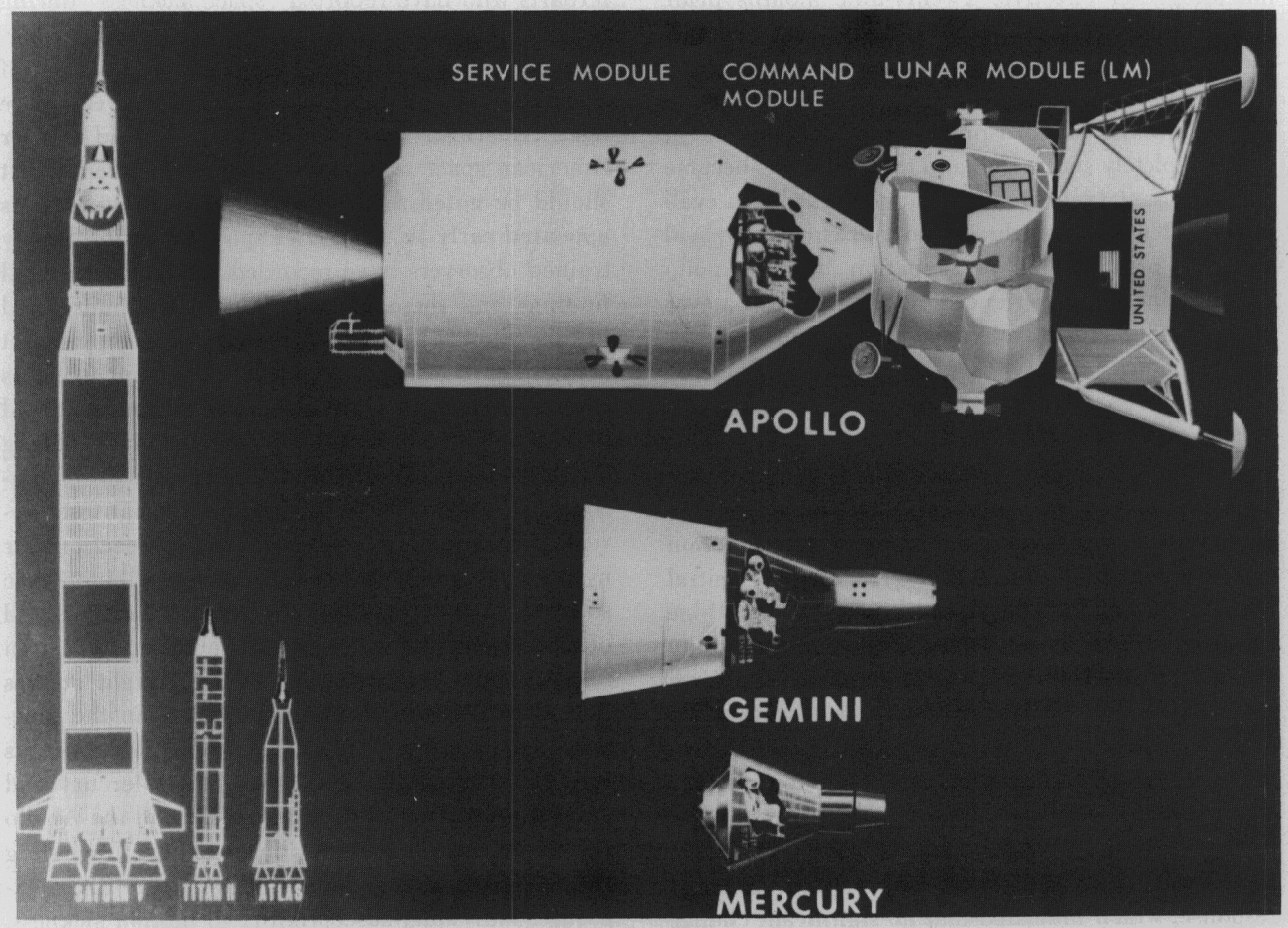


FIGURE 1.—*Comparison of spacecraft and launch vehicle configurations.*

Lunar Module was first mated to the Command Module for the Apollo 9 mission. For the first time, with the Apollo 9 mission, astronauts were able to move about inside two spacecraft by traversing the tunnel back and forth between the Lunar and Command Modules. Because the Apollo spacecraft was so much larger than the earlier craft, astronauts could move freely, using no restraints; they could stand and tumble and exercise. Further, with lunar landings, transitions from 0 *g* to 1/6 *g* and back again were possible. This, too, may not be without influence on a gravity-sensitive system such as the otolith apparatus.

EARLY OBSERVATIONS AND ATTEMPTS AT PREDICTION AND PREVENTION OF MOTION SICKNESS

The first clear-cut case of motion sickness in American space flight was experienced by the Apollo 8 astronauts. All three crewmembers experienced symptoms, ranging from stomach awareness to vomiting, lasting for up to 5 days in the case of one crewman. Motion-sickness drugs were taken with mixed results.

Because of these findings, attempts were made to design a prevention program that was to begin prior to flight, with the aim of increasing resistance to in-flight motion sickness. This program included:

1. Instruction in the use of head movements designed to hasten the process of vestibular adaptation to 0 *g*.

2. Substitution of Dexedrine plus Hyoscine for the treatment of motion sickness. This substitution was based on findings at the Naval Aerospace Medical Institute indicating that the combination of the two drugs was more effective than Marezine for the treatment of motion sickness.

3. The initiation of a more rigorous flying pro-

file, including aerobatic maneuvers prior to participation in weightless space flight.

The above measures were adopted in lieu of more elaborate preflight evaluation because of mission time constraints.

APOLLO SERIES VESTIBULAR EVALUATION

In an effort to understand the significance of in-flight motion sickness episodes, detailed histories were compiled for each astronaut in the Apollo mission series. The data points were gleaned from a variety of sources and cross-checked in order to insure that the information was accurate. These histories indicated whether or not an individual crewman had previously experienced motion sickness in automobiles, boats, and aircraft or while flying 0-*g* parabolas or engaging in egress-type exercises during training. Symptoms experienced both during exposure to the provoking situation and after the exposure were noted. Table 1 lists these findings and indicates motion sickness symptoms and vestibular-related illusions experienced during space flight. In addition, the table indicates aircraft flight experience for each crewmember prior to participation in the Apollo mission noted.

TABLE 1.—*Illusions and Motion-Sickness Symptoms Experienced by Apollo Astronauts*

Mission	Astronaut	Aircraft Flight Hours Prior to Mission	Motion-sickness history			Illusions/motion-sickness symptoms in space flight			
			In land, air and sea vehicles	In 0-*g* parabola	In spacecraft egress or egress training	Tumbling illusions	Stomach awareness	Nausea	Vomiting
7	A	4517	X						
	B*	4107	X	X	X				
	C*	3574	X		X	X			
8	D	5627	X				X	X	X
	E	4358	X	X	X		X	X	
	F*	3399	X	X			X		
9	G	4323							
	H	4266			X	X	X		
	I*	2279	X	X	X		X	X	X
10	J	5221	X						
	K	4747	X						
	L	2787	X				X		
11	M	6400	X	X	X				
	N	4425	X	X	X				
	O	3676	X	X					
12	P	4057	X						
	Q*	3638							
	R	3914			X				
13	S(E)	4282	X	X	X				
	T*	6249					X	X	X
	U*	6135					X		
14	V	5063	X						
	W*	3594	X						
	X*	4276							
15	Y(H)	4780			X				
	Z*	6715		X		X	X		
	AA*	3370							

* Had no prior space flight experience.

All three crewmembers in the Apollo 8 mission experienced motion-sickness symptoms. Each of these individuals had some history of motion sickness and two had prior space-flight experience. The crewmember designated E had participated in the 14-day Gemini mission. He experienced symptoms of nausea on the first day of the Apollo 8 flight. This crewmember first complained of stomach awareness upon arising from his couch, and felt that motion sickness would be precipitated if he continued to move about freely. These symptoms, he reported, lasted for several hours and then ceased. Astronaut F experienced similar, but not identical, symptoms that also abated after several hours. In the case of astronaut D, parallel symptoms were experienced but appeared to clear. The astronaut used a Seconal tablet to aid sleep, but awoke feeling ill again. He complained of headaches and was nauseated. He therefore remained in his couch, attempting to perform chores from the reclining position. After making a valiant effort to control the symptoms, even managing to swallow a small amount of vomitus several times, he finally succumbed and vomited into a fecal bag. This apparently relieved his discomfort, but it appeared to precipitate symptoms once again in crewman F. The crewmen expressed the feeling that the symptoms were related to Seconal administration, in conjunction with which they had experienced symptoms of nausea on the ground. In fact, each man had been afflicted with gastroenteritis when they had been medicated, and it is likely that the illness rather than the drug precipitated the difficulty.

Of the Apollo 9 crew, two were experienced astronauts. Astronaut G alone had no reported history of motion sickness and he, unlike the others, experienced no symptoms whatever during his Apollo mission. Astronaut H had experienced Coriolis effects during the Gemini 8 mission when that spacecraft rotated at a very high rate of speed and he was reaching up to switch circuit breakers to correct the situation. Interestingly, when he became aware of the fact that his symptoms were associated with looking for the controls, he simply reached for them but did not look for them, and the Coriolis effects ceased. In the Apollo flight, he experienced stomach awareness that lasted for 2 to 3 hours at the beginning of the flight. Coupled with this, he expressed a lack of interest in food, which lasted through the second day of the mission. While donning his spacesuit on that day, he experienced a feeling of tumbling and spinning as he bent down to put on the suit. During the performance of extravehicular activity, however, he experienced no symptoms.

Astronaut I, who had had no prior space-flight exposure, had more symptoms of motion sickness than any other crewmember on the Apollo 9 mission. Because he was concerned about the possibility of in-flight difficulties, he was given Marezine before launch and then took it again after about 1.5 hours in orbit. Despite the medication and attempts to restrict movement as much as possible, he experienced stomach awareness and two episodes of nausea, and he vomited. He also lost his appetite for the first 6 to 7 days of the mission. Finally, he adapted to the weightless environment and had no further problems. This individual had been tested before the flight and exhibited no labyrinthine abnormality, nor did he prove to be highly susceptible to motion sickness during ground testing or while performing nine 0-*g* parabolic aircraft flights in a postflight evaluation program. In early parabolic flights, he experienced some symptoms but quickly adapted. Strangely, however, he did not show the same facility for adaptation in space flight.

By the time of the Apollo 10 mission, five of nine Apollo mission astronauts had experienced motion sickness difficulties ranging in degree from mild to severe. Of the Apollo 10 astronauts, the one with markedly fewer aircraft flying hours was the only mission crewmember who felt any symptoms of motion sickness. For the first 2 days of the Apollo 10 flight, he suffered from stomach awareness and a loss of appetite. He employed programed head movements in an attempt to hasten adaptation, but these, he reported, produced nausea to a point near vomiting, at which time he terminated the movements. When the head movements were attempted once again on the second day of flight, exacerbation of the symptoms occurred after 1 minute. After 2 days of flight, during which the astronaut moved cautiously to minimize symptoms, he adapted completely and experienced no further difficulty. On the seventh day of the mission, he experimented with the head movements once again and was this time able to perform them for 5

minutes, but again the movements precipitated feelings of motion sickness.

Among the Apollo 11 crew, all crewmembers had fairly extensive histories of motion-sickness difficulties, but none had any difficulties whatsoever during orbital flight or during lunar surface activities. Many predictions had been made regarding the possibility of experiencing disorientation when stepping out upon the lunar surface. However, no such difficulty has been encountered by any of the astronauts performing lunar surface activity.

Only one member of the Apollo 13 crew had a history of motion sickness and, paradoxically, he alone experienced no symptoms during weightless flight. The other two crewmembers, who suffered in one case stomach awareness lasting for one day and in the other nausea and vomiting as well, had had no prior space-flight exposure.

The Apollo 14 crew had a mixed history of pre-space-flight motion-sickness episodes, and yet no difficulty was noted during weightless flight. None of these crewmembers took any medication at all.

In the Apollo 15 crew, two of the astronauts encountered no difficulty during the mission. One of these was the only crewmember with no history of motion sickness. He also had significantly less flight time than the other two crewmembers.

CONCLUSIONS

Of 25 astronauts who participated in Apollo missions 7 through 15, nine have shown some sort of in-flight symptomatology that could be related to motion sickness. Of the nine, two had entirely negative histories for motion sickness, four had only one positive history finding, and three had markedly fewer flying hours than the other astronauts. Of the 16 astronauts who exhibited no in-flight motion-sickness symptomatology in any flight, 12 had positive histories.

No trends can be seen here. There appears to be no definite relationship between either flying time or prior experience, or any other variable, and the experience of motion-sickness symptoms in the weightless state. Despite individual variables, however, various elements of the space-flight environment appear to precipitate motion-sickness symptoms. The volume of the spacecraft seems to be important. Increased volume, affording additional opportunity to move about freely, exacerbates the motion-sickness problem. This seems to be evidenced by the fact that no astronauts in the smaller Mercury or Gemini capsules experienced any vestibular-related difficulty. It does not appear likely that transitioning from 0-*g* to the 1/6-*g* environment of the lunar surface, or the return transition, has any significant effect. Head movements, programmed to hasten otolithic adaptation to 0 *g*, appear to produce motion-sickness difficulty in flight. The head-movement technique, however, may not have received an altogether fair test because the regimen, which calls for the prior use of medication, has not always been followed explicitly.

Emotional factors cannot be disregarded in the production of motion sickness symptoms. This is clearly demonstrated by the experience of the Apollo 7 astronaut who exhibited symptomatology even before he was airborne. Soviet investigators (ref. 7) believe that the emotional state of the individual bears a definite relationship to motion sickness.

The magnitude of the motion-sickness problem experienced by astronauts to date does not appear to suggest clearly the need for design and incorporation of an artificial gravity system in near-future space vehicles. By and large, adaptation to zero gravity proceeds relatively smoothly. In no case has an individual who has experienced motion-sickness symptoms failed to adapt to the weightless state eventually, and this adaptation usually proceeds rather quickly. In general, adaptation occurs more smoothly when movements are made cautiously during the adaptation period. Once adaptation is complete, even violent movements such as tumbling and spinning are possible without provocation of unusual symptoms. Although there is no plain requirement from a medical standpoint for the provision of artificial gravity, other considerations may ultimately lead us in that direction.

REFERENCES

1. Berry, C. A.: Summary of Medical Experience in the Apollo 7 through 11 Manned Space Flights. Aerospace Med., vol. 41, 1970, pp. 500–519.
2. Valyavski, Y.: Some Compensation Reactions During Extended Weightlessness. Paper presented at the Third International Symposium on the Basic Environmental Problems of Man in Space, Geneva, Switzerland, 15–19 November 1968. NASA TT-F-12094, National Aeronautics and Space Administration, Washington, D.C., January 1969.

3. VOLYNKIN, Y. M.; PARIN, V. V.; AND YAZDOVSKIY, V. I.: Preliminary Data on Physiological Studies During Human Flight in Space. In Problems of Space Biology, Vol. II Joint Publications Research Service, Washington, D.C., 1962, pp. 5–8, NASA TT 63–21437; JPRS 18, 395, 1964.
4. BERRY, C. A.: Medical Results of Apollo 14: Implications for Longer Duration Space Flights. Paper presented at the XXIInd International Astronautical Congress, Brussels, Belgium, 25 September 1971.
5. BERRY, C. A.; AND CATTERSON, A. D.: Pre-Gemini Medical Predictions versus Gemini Flight Results. Gemini Summary Conference, NASA SP–138, 1967, pp. 197–218.
6. MILLER, E.: Experiment M–9, Human Otolith Function. Gemini Midprogram Conference, NASA SP–121, 1966, pp. 431–436.
7. VOLYNKIN, Y. M.; AND VASIL'YEV, P. V.: Some Results of Medical Studies Conducted During the Flight of the "Voskhod". In Problems of Space Biology, VI, NASA TT–F–528, National Aeronautics and Space Administration, Washington, D.C., July 1969, pp. 52–66.
8. YUGANOV, Y. M.: Physiological Reactions in Weightlessness. In Aviation and Space Medicine, 1963, NASA TT–F–228, National Aeronautics and Space Administration, Washington, D.C., December 1964, pp. 431–434.
9. AKULINICHEV, I. T.; BAYEVSKIY, R. M.; BELAY, V. Ye.; VASIL'YEV, P. V.; GAZENKO, O. G.; KAKURIN, L. I.; KOTOVSKAYA, A. R.; MAKSIMOV, D. G.; MIKHAYLOVSKIY, B. P.; AND YAZDOVSKIY, V. I.: Results of Physiological Investigations on the Space Ships Vostok 3 and Vostok 4. In V. V. Parin (Ed.), Aviation and Space Medicine, NASA TT–F–228, National Aeronautics and Space Administration, Washington, D.C., December 1964, pp. 3–5.

An Overview of Artificial Gravity

RALPH W. STONE, JR.
Langley Research Center, NASA

SUMMARY

The unique characteristics of artificial gravity that can affect human performance and physiology in an artificial-gravity environment are reviewed. The rate at which these unique characteristics change decreases very rapidly with increasing radius of a rotating vehicle used to produce artificial gravity. Reducing their influence on human performance or physiology by increasing radius becomes a situation of very rapidly diminishing returns. The cost of a rotating vehicle, from the standpoint of weight, angular momentum, rotation energy, fuel use, etc., will increase with increasing radius; therefore, we are faced with a major tradeoff between increasing cost and decreasing effect. A search for the minimum radius of rotation acceptable to man and amenable to effective performance is therefore necessary.

A review of several elements of human performance has developed criteria relative to the sundry characteristics of artificial gravity. A compilation of these criteria indicates that the maximum acceptable rate of rotation, leg heaviness while walking, and material handling are the factors that may define the minimum acceptable radius. The ratio of Coriolis force to artificial weight may also be significant. Based on our current limited knowledge and assumptions for the various criteria, a minimum radius between 15.2 and 16.8 m seems desirable. Validation of the form and numerical value of the criteria by ground-based simulation studies and ultimately by space flight is required.

INTRODUCTION

United States and Soviet astronauts have experienced weightlessness for periods of several days and apparently have not been seriously affected by the exposure. There is still concern, however, that for much longer periods than 18 days the astronauts may not be able to sustain adequate performance in the weightless state and may become so weary of the unique environment that the condition becomes unbearable to them. Further, although only slight physiological change has been noted following their exposure to the weightless state and the astronauts have recovered to a normal state in a few days, what physiological changes may occur after much longer periods is unknown, and these may be much more subtle than those noted so far. For these reasons, then, creation of artificial gravity is being considered for space flights of long duration. Maintenance of performance and habitability have been expressed as major factors of concern, with physiological effects being of only secondary concern. There are, however, no real data to support such concerns, or at least the priorities given to them.

An artificial-gravity environment and that of other alternatives to weightlessness have unique characteristics which, in themselves, have or may have a debilitating effect on man and his performance. This paper reviews current knowledge about artificial gravity and examines the unique characteristics of such an environment as to how they may affect performance and habitability and determine the design of rotating space vehicles.

SYMBOLS

a	acceleration, m/s^2
α	angular acceleration, rad/s^2
r	radius, m
ω	rate of rotation, rad/s
W	artificial weight, kg

ΔW	change in artificial weight due to radius change or relative velocity, kg
g	acceleration of Earth's gravity
h	distance or height along body, m
x	instantaneous radial distance, m
y	instantaneous tangential distance, m
θ, ϕ, ψ	Euler angles, rad
P_{Hs}	hydrostatic pressure, kg/m^2
d	tangential distance, m
ρ	density, kg/m^3
Subscripts:	
cg	center of gravity
v	vehicle
1 and 2	radial positions
i	incremental
h_x, h_y, h_z	head or object relative to inertial space
h_θ, h_ϕ, h_ψ	head or object relative to vehicle axis
F	floor
cc	cross coupling

A dot over a symbol indicates its first derivative with respect to time; double dot indicates the second derivative with respect to time.

THE UNIQUE CHARACTERISTICS OF ARTIFICIAL GRAVITY

Our concern about a new and strange environment is with those aspects of it that are different from the normal environment. These differences or unique characteristics are the features of the environment that can alter human performance. In artificial gravity such characteristics fall into two categories, static and dynamic, where the distinction lies with the state of the person or the object relative to the artificial-gravity environment. The static characteristics are the artificial-gravity level, the gradient of the artificial gravity in the radial direction, and the unique variation of the hydrostatic pressures in fluid systems caused by the gravity gradient. The dynamic characteristics are the Coriolis accelerations acting when a person moves linearly relative to the rotating environment and the cross-coupled angular accelerations acting when he rotates relative to the rotating environment.

Mathematical expressions for these characteristics and associated derivatives of them are listed in table 1. Listed there also are expressions for changes in weight of objects when repositioned in the radial direction and variation in gravity level along the body of a standing man, which has the peculiar feature of lowering his center of gravity. Also given is the distance that objects will fall beyond where one would normally expect them to fall.

TABLE 1.—*Mathematical Expressions of the Characteristics of Artificial Gravity*

Static characteristics
g level, $a_{cg}=r_{cg}\omega_v^2$
g gradient, $da_{cg}/dr=\omega_v^2+a_{cg}/r_{cg}$
g ratios, $a_1/a_2=r_1/r_2$
Object weight change, $\Delta W/W=(r_1-r_2)/r_2$
Gravity variations along body, $h\omega_v^2=(h/r)a_{cg}$
Hydrostatic pressure variation, $P_{Hs}=\rho/2(r_2^2-r_1^2)\omega_v^2$
Dynamic characteristics
Coriolis acceleration (radial), $\ddot{x}=\ddot{r}-r(\omega_1^2+2\omega_1\omega_v+\omega_v^2)$
Coriolis acceleration (tangential), $\ddot{y}=2\dot{r}(\omega_1+\omega_v)+r\omega_1$
Angular cross-coupling,
$\dot{\omega}_{h_x}=\dot{\omega}_{h_\phi}-\omega_v(\omega_{h_\theta}\sin\theta+\omega_{h_\psi}\cos\theta\sin\psi)$
$\dot{\omega}_{h_y}=\dot{\omega}_{h_\theta}-\omega_v(\omega_{h_\psi}\cos\theta\cos\psi-\omega_{h_\phi}\sin\theta)$
$\dot{\omega}_{h_z}=\dot{\omega}_{h_\psi}+\omega_v(\omega_{h_\theta}\cos\theta\cos\psi+\omega_{h_\phi}\cos\theta\sin\psi)$
Distance objects fall from expected position,
$d=r_F(\sqrt{r_F^2-r_1^2/r_1}-\tan^{-1}\sqrt{r_F^2-r_1^2/r_1})$

The variation in rate of rotation of the spacecraft versus vehicle radius for several g-levels is shown in figure 1. One very significant factor shown in this figure is the flattening of rate curves with increasing radius. For example, assume a 0.4-g level is planned. Adding 6 m to a 6-m vehicle radius will

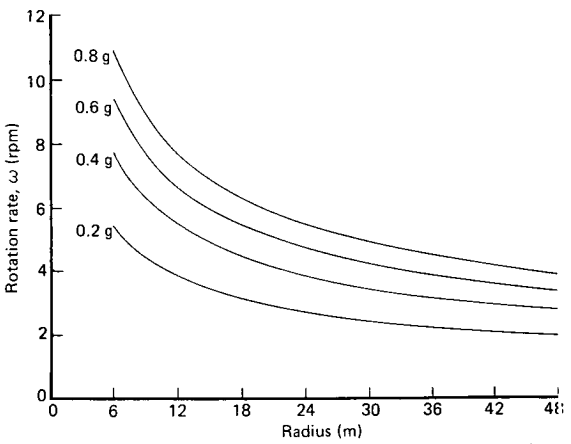


FIGURE 1.—*Rate of vehicle rotation versus radius of rotation for various artificial-gravity levels.*

reduce the required speed by over 2 rpm. However, adding 6 m to a 30-m spacecraft radius will reduce the required speed by only 0.3 rpm. Increasing radius brings about increasing weight, inertia, momentum, and energy, along with increasing difficulties in control, stabilization, fuel requirements, launch weight, etc. Thus a very critical tradeoff exists between these factors and the degree of acceptability of the environment to man.

Figure 2 shows the variation of the gravity gradient versus vehicle radius for several g-levels. Again we see the very rapid change in gradient with short radii, but flattening very rapidly as radius increases. At the 0.4-g level, adding 6 m to a 6-m vehicle radius reduces the gravity gradient by 0.3 g/m, whereas adding 6 m to a 30-m vehicle radius reduces the gravity gradient by less than 0.002 g/m.

Figure 3 depicts the ratio of the weight of an object on a 2-m-high shelf to that of the object on the floor. It also may be considered to represent the ratio of the artificial gravity at the head of a standing man 2 m tall to that at his feet. There is again a rapid reduction in weight ratio with increasing radius. A 15-percent reduction in weight change results from adding 6 m to a 6-m vehicle radius, but there is only a 1-percent reduction if 6 m is added to a 30-m vehicle radius. Again, the advantage of increasing radius diminishes rapidly as the radius increases.

Ratios of hydrostatic pressure along the body length of a man standing in artificial gravity to the hydrostatic pressure of a man standing on Earth are presented in figure 4. The significant aspect is probably the effect of gravity level, since the hydrostatic pressure, and therefore the influence of this direct stressor on the cardiovascular system, for example, are directly proportional to the g-level. Clearly there is little gain with increasing radius.

Objects when moved within the rotating environment react relative to that environment in a manner determined by Coriolis forces. Here an object, which by natural law will move linearly relative to inertial space, must be forced to move linearly relative to the rotating environment. Figure 5 illustrates the radial forces required for moving objects in a tangential direction. The forces are shown as ratios to the artificial weight nominally existing and are for motions of 1 m/s in the direction of rotation. As with the other changes just mentioned, there is a rapid reduction in the change of this ratio with increasing radius. At 0.4 g, for example, adding 6 m to a 6-m vehicle radius reduces this ratio by about one-third, whereas adding 6 m to a 30-m vehicle radius reduces the ratio by less than one-tenth.

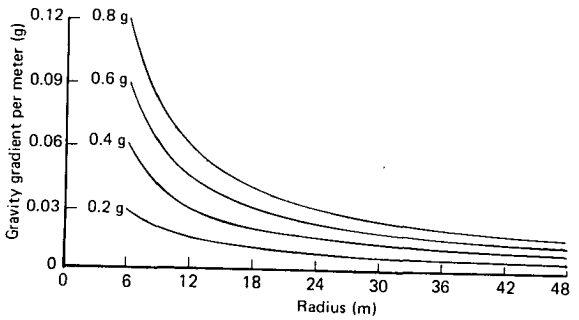


FIGURE 2.—*Gravity gradient versus vehicle radius for various artificial-gravity levels.*

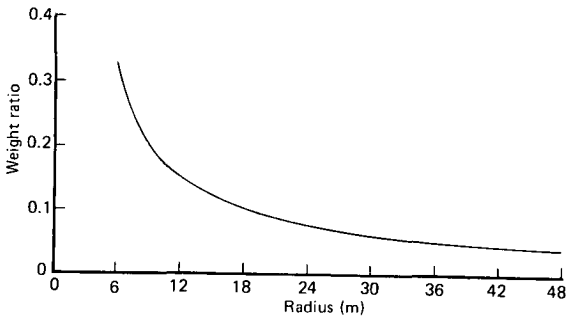


FIGURE 3.—*Artificial weight change versus vehicle radius for a 2-m difference in radial position of objects.*

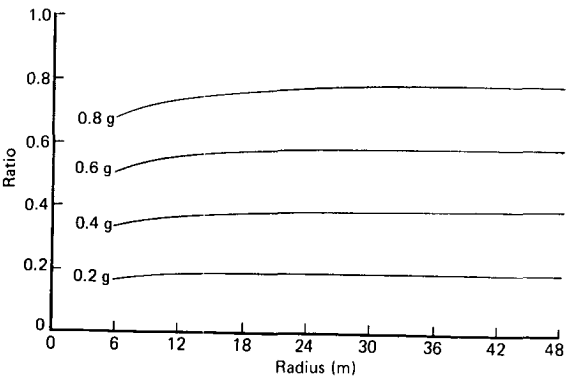


FIGURE 4.—*Ratios of hydrostatic pressure along the body in artificial gravity to that in Earth gravity for various artificial-gravity levels.*

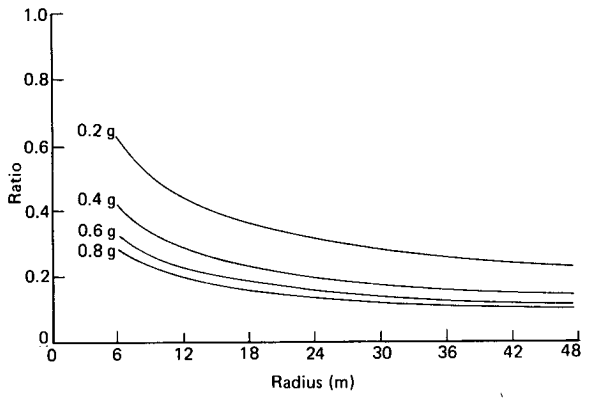


FIGURE 5.—*Ratio of radial Coriolis acceleration to artificial gravity versus radius of vehicle rotation. Curves shown are for radial Coriolis acceleration caused by tangential motions at 1 m/s in the rotating vehicle.*

Figure 6 illustrates the tangential forces required for moving objects in a radial direction. Again the forces are shown as ratios to the artificial weight nominally existing, but they are for motions of 0.6 m/s in the radial direction. Here again there is a rapid reduction of the change in this ratio with increasing radius. At 0.4 *g*, adding 6 m to a 6-m vehicle radius reduces the ratio by more than one-fourth, whereas increasing a 30-m vehicle radius to 36 m reduces the ratio by less than one-tenth.

The other significant aspect of a rotating environment is related to what occurs when objects are rotated relative to the environment. This is expressed in table 1 as angular cross-coupling and is a function of the orientation of the object, the axis about which it is rotated, and the rate at which it is rotated. The angular accelerations expressed in table 1 must be applied to the rotated object in addition to the desired basic rotational acceleration so that the object will rotate as expected within the rotating environment. These moments are to the first order independent of the radius of rotation, but are directly proportional to the rate of rotation. Rotating the object about an axis not through its center of gravity will bring about the influence of the Coriolis forces. The gravity gradient, which shifts the center of gravity radially outward, will impose additional but secondary effects when objects are rotated. This effect, of course, would be radius dependent. The mass distribution (moments of inertia) of the objects rotated and the angular accelerations expressed in table 1 can be used to determine the specific torques required to rotate any specific object.

One of the peculiar characteristics of artificial gravity is that objects thrown or dropped will always fall in a direction counter to the direction of rotation from where normally expected. Figure 7 depicts where objects will strike the floor when dropped from 1 m above the floor. It is not clear how disturbing this factor might be; very possibly man will adapt to it readily. At any rate, we see again the very marked reduction in this effect with increasing radius. Adding 6 m to a 6-m radius reduces this distance by more than 12 cm, whereas adding 6 m to a 30-m radius reduces this distance by only about 1.5 cm. Relative to dropped, thrown, or rolled objects, interesting phenomena are seen in Earth-based rotating simulators because of the presence of Earth's gravity and the orientation of the floor relative to the axis of rotation. As an example, the

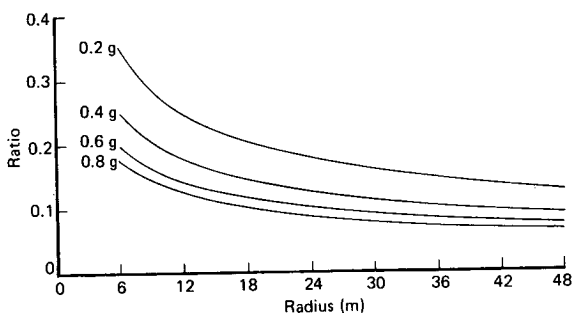


FIGURE 6.—*Ratio of tangential Coriolis acceleration to artificial gravity versus radius of vehicle rotation. Curves shown are for tangential Coriolis accelerations caused by radial motions at 0.6 m/s in the rotating vehicle.*

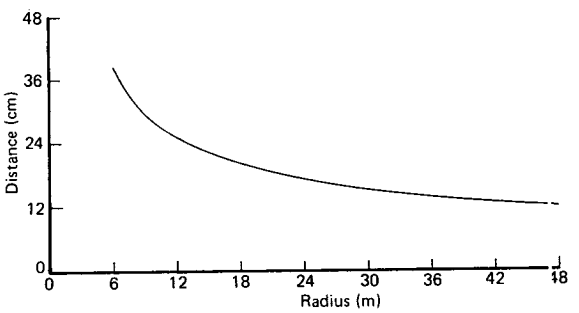


FIGURE 7.—*Distance from expected position an object falls when dropped from 1 m above the floor, plotted against radius of vehicle rotation.*

lateral movement of a ball, when it is rolled in the tangential direction in a room tilted so that the resultant of the gravity and centrifugal force vectors are normal to the floor, is caused by the presence of Earth's gravity and will not occur in pure artificial gravity.

HUMAN PERFORMANCE IN ARTIFICIAL GRAVITY

We have examined briefly the unique features of artificial gravity and how they will change with varying radii of vericle rotation. Since our goal is to establish an environment that will be acceptable and provide a pleasant habitation, and one that will promote maximum required performance, we must now consider the influence of the sundry unique characteristics on specific elements of performance. Because the peculiar elements of the new environment will alter performance, we cannot expect that man will function in the new environment as well as in his normal earthly environment. With a period of adaptation, performance may improve, but man in artificial gravity may never perform as effectively as in Earth's gravity. We are concerned then with two elements: (1) the radius and rate of rotation that will lead to effective performance and acceptable habitable conditions, and (2) the cost of attaining this desired condition. Cost here is intended to encompass not only dollars but also weight, moments of inertia, angular momentum, rotational energy, fuel use, and stability. These factors will naturally increase with vehicle size and rate of rotation. A tradeoff thus exists between costs and gains in performance and habitability with increasing size. We have already pointed out that changes in the peculiar characteristics of artificial gravity diminish rapidly with increasing radius, implying that incremental reductions in a given characteristic of artificial gravity require greater and greater increments of radius. If gains in human performance are associated directly with changes in the unique features of artificial gravity, incremental gains in performance will also require greater and greater increments of radius. It is therefore desirable to determine the smallest radius and rate of rotation at which acceptable performance and habitability may be attained.

There is no past experience that can serve as a basis for establishing these conditions. Through knowledge of the unique characteristics of artificial gravity, we must estimate what influence these characteristics may have on specific performance factors. The remainder of this paper sets down criteria based on such estimates. It is of course necessary to verify these criteria and to establish appropriate magnitudes for them by ground-based simulation.

Various aspects of human performance may be influenced by artificial gravity (table 2). Gross motor performance includes self-locomotion, material handling, and force and torque applications. Fine motor performance involves dexterity and eye-hand coordination with head motions. Dynamic elements of performance are expected to be influenced by the gravity level and Coriolis forces and possibly by the gravity gradients that cause changes in effective weight with radial position. Postural balance may be influenced by reduced gravity and by the Coriolis forces due to body motions necessary in the act of balancing or moving. Transition from artificial gravity to zero gravity and return, as may be required in space-base operations, can possibly degrade performance because of prior adaptation to one or the other condition. Passive radial locomotion, as with an elevator on a space base, may be influenced by the Coriolis forces, depending on the rate of elevator movement. In addition, the potential rapid increase in gravity when radius is increased could have an acute influence on orthostatic tolerance.

TABLE 2.—*Potential Criteria for Artificial Gravity*

Gravity level
$9.8 \geq r\omega_1^2 + 2r\omega_1\omega_v + r\omega_v^2 \geq 0.98$
Coriolis forces
Tangential movement, $(1+C_1)r\omega_v^2 \geq r\omega_1^2 \pm 2r\omega_1\omega_v + r\omega_v^2 \geq (1-C_1)r\omega_v^2$
Radial movement, $\frac{2\dot{r}}{r\omega_v} \geq C_2$
Dropped objects, $r(\sqrt{r^2-r_1^2/r_1} - \tan^{-1}\sqrt{r^2-r_1^2/r_1}) \leq C_3$
Gravity gradient
Change in object weight, $(r-r_1)/r \leq C_4$
Along body, $(r-r_h)/r \leq C_5$
Hydrostatic pressure, $(r_{heart}+r_h)/(r+r_h) \geq C_6$
Cross-coupled angular accelerations
$\omega_v \leq C_7/\omega_1$
$\omega_v \leq C_8/\omega_h$

Self-locomotion

Self-locomotion within the rotating vehicle will certainly be a very significant element of performance. Habitability, with regard to orientation and arrangement of facilities within the vehicle, will depend directly on this capability. Figure 8 is a graphic representation of the influence of the characteristics of artificial gravity on a man moving within a rotating spacecraft. With radial movement toward and away from the center of rotation, the tangential components of Coriolis forces operate. During tangential movement with and against the direction of rotation, alterations in the radial forces (weight) occur. Axial motion causes no Coriolis effects except for those created by lateral movement of limbs or lateral body sway. The influence of the characteristics of artificial gravity on human mobility is probably least during axial motion.

Figure 9 shows four boundaries expected to influence walking on the floors of rotating vehicles. The 1-g limit is one below which the forces on the body, when walking at 1 m/s in any direction, will never exceed 1 g. Other walking speeds will of course cause shifting of this boundary. The speed of 1 m/s, however, is considered to be a reasonable maximum for moving about within a vehicle. Early simulations (refs. 1 and 2) have indicated that leg heaviness caused by this factor was readily recognized and could become objectionable. The level of artificial leg weight at which it becomes objectionable is not established and may vary with artificial gravity level. Below the leg weight boundary the forces on the feet, during walking at 1 m/s, will not exceed 1 g.

Shown along the 6-m radius line of figure 9 are points at which preliminary studies of simulated walking (refs. 1 and 2) have been made at the Langley Research Center. The upper point is for walking at 0.5 g; it was at this point that leg heaviness was noted and considered undesirable. The boundary proposed here may not be adequate. Above the 0.1-g traction limit, traction is expected to be adequate. During the simulation studies (refs. 1 and 2) traction became difficult during walking against the rotation at 1/6 g (the lowest point on the 6-m radius line of figure 9), which tends to

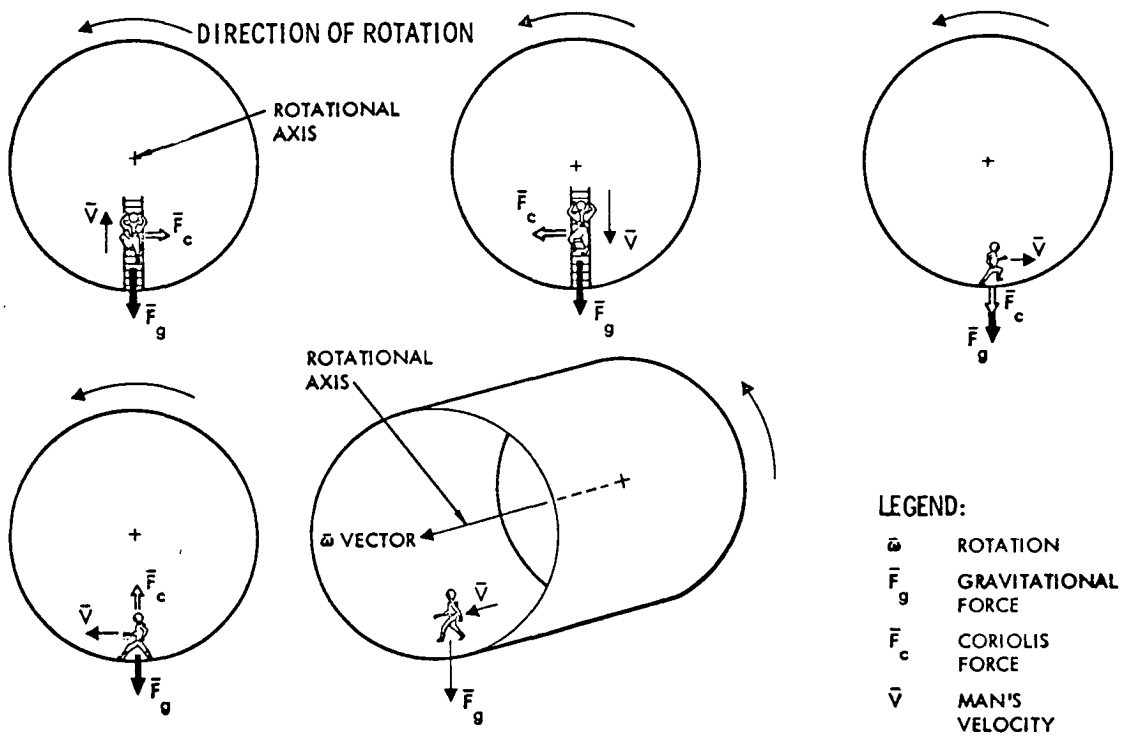


FIGURE 8.—*Influences of artificial gravity on locomotion.*

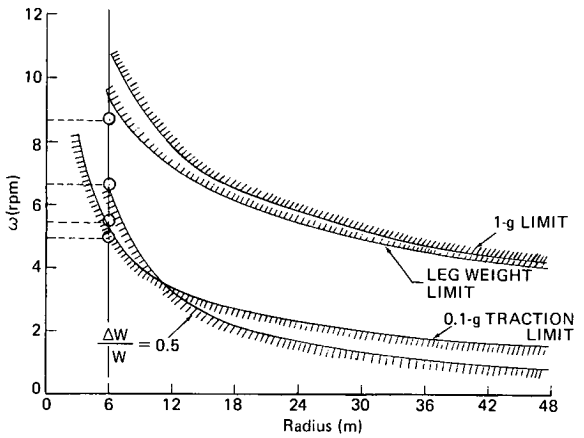


FIGURE 9.—*Rotation rate and radius boundaries for acceptable tangential locomotion at a rate of 1 m/s.*

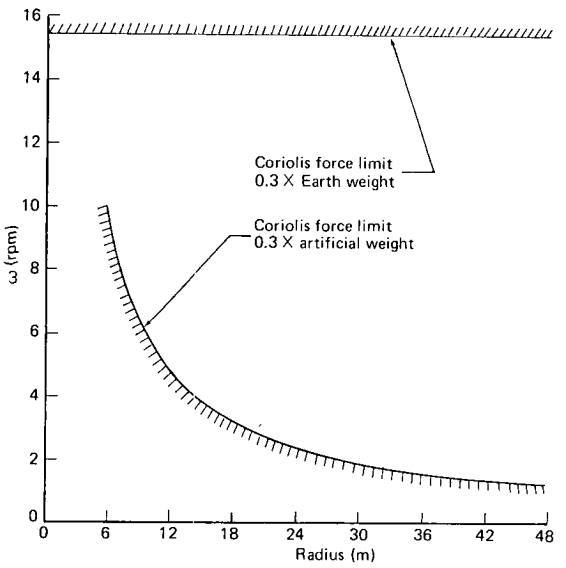


FIGURE 10.—*Rotation rate and radius boundaries for acceptable radial locomotion at 0.6 m/s.*

verify this boundary. The fourth boundary, $\Delta W/W$, often referred to as the ratio of Coriolis force to artificial weight, is one above which the change in weight due to the relative motion during walking at 1 m/s will not exceed half the artificial weight. Of the four conditions shown on figure 9, that for 0.3 *g* seemed to be the most amenable condition studied (ref. 1). This does not mean that 0.3 *g* is the desired design level for artificial gravity. It implies only that, at a 6-m radius, with all the factors involved, it was subjectively the best of four gravity levels tested for a very limited number of subjects. The ratio of Coriolis force to artificial weight of 0.5 tends to be confirmed by these data; however, this ratio has often been considered to have a maximum desirable value of 0.25 or less. It should be noted that figure 9 and the data of reference 1 were obtained for curved floors, of constant radius. Flat floors can impose additional complexity, since the local artificial gravity vector is not perpendicular to the floor except at its center. This situation would involve leaning relative to the floor as on a sloping walkway.

Figure 10 shows boundaries for radial motion of the astronaut, as when climbing toward or away from the center of rotation. Two boundaries are shown, one for which the Coriolis force due to radial motion at 0.6 m/s does not exceed 0.3 times the artificial weight, and the other where it does not exceed 0.3 times the Earth weight. There are no data to support either criterion or the magnitude of 0.3. Coriolis forces act in a tangential direction during these radial motions. A man on a ladder, depending on the orientation of the ladder, will be pushed into or out from the ladder or sideways on the ladder. Such forces require arm reactions rather than leg reactions. Thus the actual force, rather than its ratio to the artificial weight, may be more significant. The boundary shown on figure 10 for 0.3 times Earth weight may be too large, particularly since muscle tone and strength may be degraded by continued exposure to reduced gravity.

Material Handling

Moving materials in the artificial gravity environment will be different from moving things on Earth. Objects will change weight with radial position; they will experience Coriolis accelerations when moved about and cross-coupled angular accelerations when rotated. Boundaries for material handling based on these factors are shown in figure 11. One boundary is for tangential movement above which the change in weight due to motion at 1.2 m/s will not exceed 0.25 times the artificial weight. A second is for radial motion where the tangential Coriolis forces do not exceed 0.25 times the artificial weight. Beyond the third boundary the weight of an object when raised 2 m does not decrease below 0.5 times its original artificial weight. A fourth possible boundary (not shown) would relate to the

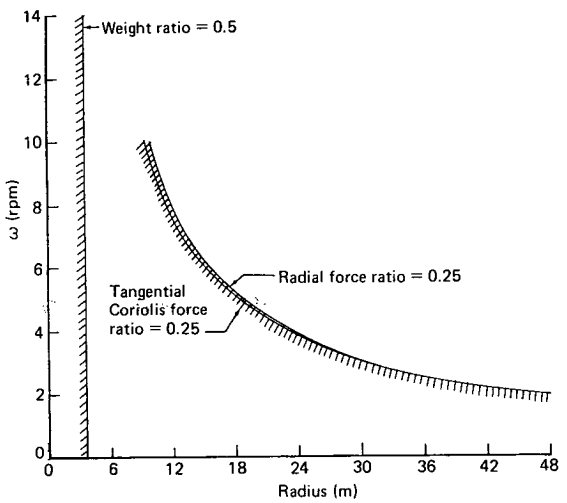


FIGURE 11.—*Rotation rate and radius boundaries for acceptable material handling at a material movement rate of 1.2 m/s.*

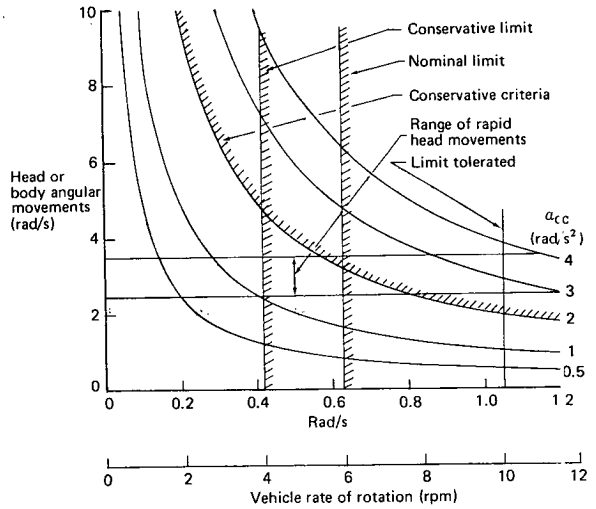


FIGURE 12.—*Possible criteria for acceptability of cross-coupled angular accelerations, a_{cc}, in vehicles with artificial gravity.*

rotation of an object out of the plane of rotation. It has been assumed that a torque caused by angular cross-coupling probably can be 1/2 of the applied torque. Rate of vehicle rotation for this value could be rather high, greater than 10 rpm.

Transition from Artificial Gravity to Weightlessness

Adaptation to the rotating environment has received considerable attention in recent years (e.g., refs. 3 to 6). The primary problem regarding adaptation is the angular motion of the head within the rotating environment, wherein the cross-coupled angular accelerations (see table 1) cause vestibular stimulations that are essentially in conflict with the visual cues from a stationary environment. The endpoint of such disturbing conflicts is often nausea. The maximum cross-coupled angular acceleration is the product of the angular velocity of the vehicle and the angular velocity of the head (ref. 7). There have been a number of studies in which angular velocities up to about 10 rpm have been experienced. In some cases a considerable period of adaptation was used to reach this level. Experience on stopping rotation has ranged from little effect to seriously disquieting effects such that performance was degraded. In large space vehicles of the future, such as the currently planned space base, it is possible that there will be an area of weightlessness and an area of artificial gravity in the vehicle, the former for experimental studies and the latter for living. Therefore it will be necessary to pass back and forth frequently between the two areas. The influence of such transitions upon performance is of major concern.

Figure 12 relates angular head motions and vehicle rate of rotation. The stimulus that causes disturbances in a rotating environment is the cross-coupling that exists between these two angular velocities, and the product of the two values gives the maximum stimulus from the cross-coupled angular acceleration. Shown in figure 12 are curves of constant values of this cross-coupling, ranging from 0.5 to 4 rad/s². Based on current experience, a value of 2 rad/s² ($\approx$115°/s²) for the cross-coupling seems to be a conservative criterion of tolerance. A range of rapid head motions possible for a man is from 2.5 to 3.5 rad/s ($\approx$140 to 200°/s). Vehicle rotation rate boundaries of 4 rpm (a conservative value), 6 rpm (a nominally acceptable value), and 10 rpm (a tolerable value) are shown. Adaptation to such rotational rates will be required. Therefore adaptation, retention of adaptation, and simultaneous adaptation to both rotating and nonrotating environments are problems of critical concern. Adaptation to any given environment is possible, but the influence of rapid transition back and forth between different environments remains relatively unexplored.

Postural Balance in Artificial Gravity

In reduced gravity and with the peculiar forces encountered in a rotating environment, postural balance may be more difficult than in Earth gravity. Figure 13 indicates boundaries for postural balance. The first is a static or neutral stability boundary beyond which the vestibular mechanism would sense falling before an unbalance angle for a stationary man would be exceeded. Experience at 1/6 *g* on the Moon by four American astronauts certainly indicates that this boundary is not significant for a normal man. Two dynamic boundaries are shown, one for the vertical motions and the other for the lateral motions of walking. In both instances, these could be more critical when walking axially, since the unusual Coriolis accelerations would act in the lateral plane, in which a man is least stable. When a man is walking tangentially, the accelerations act in the sagittal plane. It is assumed that these Coriolis accelerations should not exceed 0.25 times the artificial weight.

Other Motor Performance

No criteria have yet been established for gross motor performance other than those previously discussed, i.e., walking, climbing, and material handling. The use of tools, opening and closing of doors and hatches, etc. will certainly be influenced by Coriolis forces. It is not clear what magnitudes of these forces may influence these actions. Experiments are required through simulation to determine these magnitudes as well as the influence of orientation within the vehicle. Studies of this nature may have considerable influence on the interior design, e.g., wall, door, and hatch arrangements.

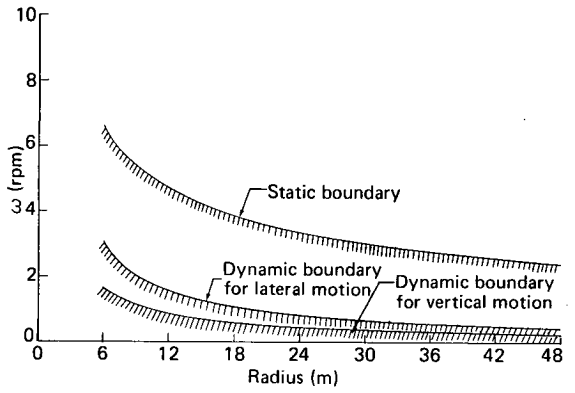


FIGURE 13.—*Boundaries of rotation rate versus radius for acceptable postural balance in artificial gravity.*

Fine motor performance probably will not be influenced appreciably by the Coriolis forces, since the motions involved will generally be small. However, if head motions are involved, the tolerance and adaptation to cross-coupled acceleration may be of significant influence. The previous discussion of cross-coupling and the data of figure 12 therefore may have a bearing on potential problems of fine motor performance. Simulation studies should bring some insight into this problem.

Passive Radial Locomotion

Elevators may be used to move people and materials from the hub of a large rotating base to the living areas. There will undoubtedly be considerable experience with vehicles of smaller size before the need for elevators or, for that matter, for artificial gravity is established. However, if elevators are used, relatively rapid changes in *g*-level and large Coriolis forces will be experienced. Proper orientation in the elevator and possibly restraints may be required. Objects carried become progressively heavier or lighter as the elevator moves, and tangential components of Coriolis accelerations may result in interesting phenomena. The criteria for material handling, previously discussed and shown on figure 11, may apply here as well but, of course, must be adjusted to the velocity of the elevator, which could be much larger than the 1.2 m/s assumed for figure 11.

It has been stated that rapid elevator movement will cause a rapid increase in *g*-forces on the body which, like other passive situations such as tilting in the gravity environment or lower body negative pressure, may cause orthostatic intolerance. Several minutes in the passive state are normally required, however, before orthostatic intolerance is manifested, and this is very likely much longer than would be involved in a space vehicle elevator ride.

The consequences of transferring from the weightless state to artificial gravity, regardless of the procedure, would be rather complex. The artificial-gravity level, the degree of habitation to the weightless state, and the general condition of the individual would be involved. Since susceptibility is aggravated by exposure to weightlessness (unless possibly some special therapeutic measures are taken) and since the degree and character of change with

exposure are not thoroughly understood, further consideration of this problem seems warranted.

POTENTIAL CRITERIA FOR ARTIFICIAL GRAVITY

We have discussed the various elements of performance and boundaries of the two independent variables, radius and rate of rotation, of artificial gravity. These boundaries may therefore be characterized by criteria expressing the dependent variable, or particular characteristic of the rotating environment, in terms of the independent variables. In some instances only one or the other independent variable may be involved. Table 2 lists these criteria.

The criterion for gravity level is associated basically with human tangential mobility and assumes that the g-level should not exceed 9.8 m/s^2 (1 g) or be less than 0.98 m/s^2 (0.1 g). The 1-g level may apply to the whole body motion or the leg motion, where the product of r_1 and ω_1 represents the velocity of the object relative to the rotating environment. The leg motion may be the more critical element. The lower criterion (0.1 g), assumed to be a minimum level for adequate traction, requires substantiation. If the maintenance of some specific degree of physiological tone is a requirement, this lower limit will probably be raised.

The criteria for Coriolis forces involve basically the movement of materials as well as the movement of man. The value of C_1 for the tangential movement of man has been assumed as 0.5 and for the movement of materials as 0.25. The value of C_2 for the radial movement of man has been assumed to be 0.3 and for materials 0.25. These criteria are expressed as ratios of the force involved to the local artificial-gravity level. In the discussion of the radial movement of man, it was noted that this ratio possibly should be related to man's Earth weight rather than his artificial weight. The value of C_3, the distance dropped objects fall from their expected position, has been assumed to be 0.3 m when they are dropped from 1 m.

The criteria for gravity gradients relate to the changes in weight of objects with radial position and the changes along the body of a standing man. The assumed value for C_4 here is 0.5 for objects raised 2 m. The C_4 and C_5 criteria also relate to man. No values for these two criteria are presented here because current evidence indicates that the gradient probably does not intrinsically influence performance. There may be some effect that may alter the basic influence of reduced gravity on human physiology. The variations in gradient and hydrostatic pressures presented in figures 2 and 4 indicate that their influence will more likely be felt, if at all, at relatively small radii. The dominant influence on human physiology will undoubtedly be from the reduced gravity, with minimal effects from the gradient.

Criteria for cross-coupled angular acceleration relate to the rotation of objects and of man within the rotating environment. The value of C_7 for the rotation of objects is not presented in this paper. As previously noted, rotation of objects in a vehicle rotating at a rate as high as 10 rpm may not be a problem. The mass distribution of objects may critically influence this opinion; e.g., long, slender objects may be difficult to handle when rotated in certain directions relative to the axis of vehicle rotation. A value of 2 rad/s^2 has been selected for C_8. This rate, when associated with maximum average head rates of rotation (ω_h) of 3 rad/s, gives a vehicle rate of rotation (ω_v) of 0.67 rad/s (see fig. 12), or about 6 rpm, a value at which adaptation has already been demonstrated in ground-based experiments. Concern over this value remains, however, since the problem of tolerance is not totally resolved. A conservative value of 4 rpm or less is under consideration.

These criteria were considered in essentially the

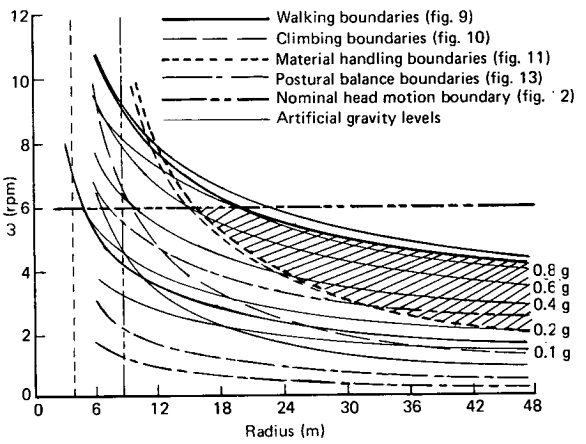


FIGURE 14.—*Compilation of boundaries for acceptable human performance in artificial gravity.*

same form in reference 1, but were considered in that report for fewer performance elements than are considered herein. Some differences in values are also proposed in this paper. Figure 14 is a compilation of all the previous boundaries shown, superposed on plots of artificial gravity. The area encompassed by all boundaries shows a minimum radius between 15.2 and 16.8 m. The boundaries for material handling are the critical elements. These boundaries are not well established and must be validated numerically and analytically by experiments. If these are not valid, a much smaller radius may be possible. It must be noted that all other boundaries require similar verification. The ratio of Coriolis force to artificial gravity ($\Delta W/W$) has often been considered to have a maximum desirable value of 0.25. Such a value would impose a radius on the order of 15.2 m, as was noted in reference 1.

REFERENCES

1. HEWES, D. E.: Status Report on Recent Langley Studies of Lunar and Space Station Self-locomotion. AGARD Behavioral Problems in Aerospace Medicine, Oct. 1967.
2. STONE, RALPH W., JR.; PILAND, W. M.; AND LETKO, WILLIAM: Certain Aspects of Onboard Centrifuges and Artificial Gravity. Fourth Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–187, 1970, pp. 331–343.
3. GUEDRY, F. E., JR.; COLLINS, W. E.; AND GRAYBIEL, A.: Vestibular Habituation During Repetitive Complex Stimulation: A Study of Transfer Effects. J. Appl. Physiol., vol. 19, 1964, pp. 1005–1015.
4. NEWSOM, B. D.; BRADY, J. F.; SHAFER, W. A.; AND FRENCH, R. S.: Adaptation to Prolonged Exposures in the Revolving Space Station Simulator. Aerospace Med., vol. 37, 1966, pp. 778–783.
5. GRAYBIEL, A.; DEANE, F. R.; AND COLEHOUR, J. K.: Prevention of Overt Motion Sickness by Incremental Exposure to Otherwise Highly Stressful Coriolis Accelerations. Aerospace Med., vol. 40, 1969, pp. 142–148.
6. GRAYBIEL, A.; AND WOOD, C. D.: Rapid Vestibular Adaptation in a Rotating Environment By Means of Controlled Head Movements. Aerospace Med., vol. 40, 1969, pp. 638–643.
7. STONE, RALPH W., JR.; AND LETKO, WILLIAM: Some Observations on the Stimulation of the Vestibular System of Man in a Rotating Environment. The Role of the Vestibular Organs in the Exploration of Space, NASA SP–77, 1965, pp. 263–278.

Vestibular Mechanisms Underlying Certain Problems in a Rotating Spacecraft*

ASHTON GRAYBIEL
Naval Aerospace Medical Research Laboratory

SUMMARY

Vestibular side effects are discussed with the aid of a conceptual framework based on an analysis of vestibular input-output relations. These side effects tend to fall into two main categories: (1) reflex phenomena and (2) motion sickness, a delayed epiphenomenon. Although the symptomatology of motion sickness is similar wherever experienced, both the eliciting stimuli and the opportunity to adapt may differ in different motion environments. These differences not only are exemplified when motion sickness is compared in a weightless and in a rotating environment, but they also point to important differences in the problem of preventing (or treating) motion sickness in these two very different environments.

INTRODUCTION

Prolonged space missions involving the generation of artificial gravity by rotation of a part of the spacecraft pose problems, some of which are mainly of vestibular origin, when astronauts are exposed to weightlessness, rotation, and transitions between these two very different environments. The main problem is motion sickness, which has been experienced in orbital flight (refs. 1 and 2), in the weightless phase of parabolic flight (refs. 3, 4, and 5), and in rotating environments (refs. 6 and 7). The fact that persons with bilateral labyrinthine defects do not experience motion sickness in parabolic flight (ref. 5) strongly suggests the essentiality of the vestibular system in the elicitation of symptoms not only in the weightless phase of parabolic flight but also in orbital flight. Stated differently, in orbital flight we are dealing with a vestibular side effect—motion sickness—and not with symptoms characteristic of motion sickness but of nonvestibular origin.

Vestibular side effects other than motion sickness will be encountered on space missions involving the generation of artificial gravity. Nystagmus, illusions, and postural disturbances (partly of vestibular origin) need to be taken into account but, in all likelihood, pose only minor problems.

In the discussion that follows, mechanisms underlying vestibular side effects—especially motion sickness—are inferred from an analysis of input-output relations.

CONCEPTUAL FRAMEWORK

The schema shown in figure 1 represents an attempt to fit important elements concerned with vestibular input-output relations into a conceptual framework (ref. 8).

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

Block II indicates the transducer functions of the end organs. Although a feature common to both end organs is the conversion of the accelerative stimuli to electrical energy, thus altering the temporal and

* This study was supported by NASA Order No. W13433.

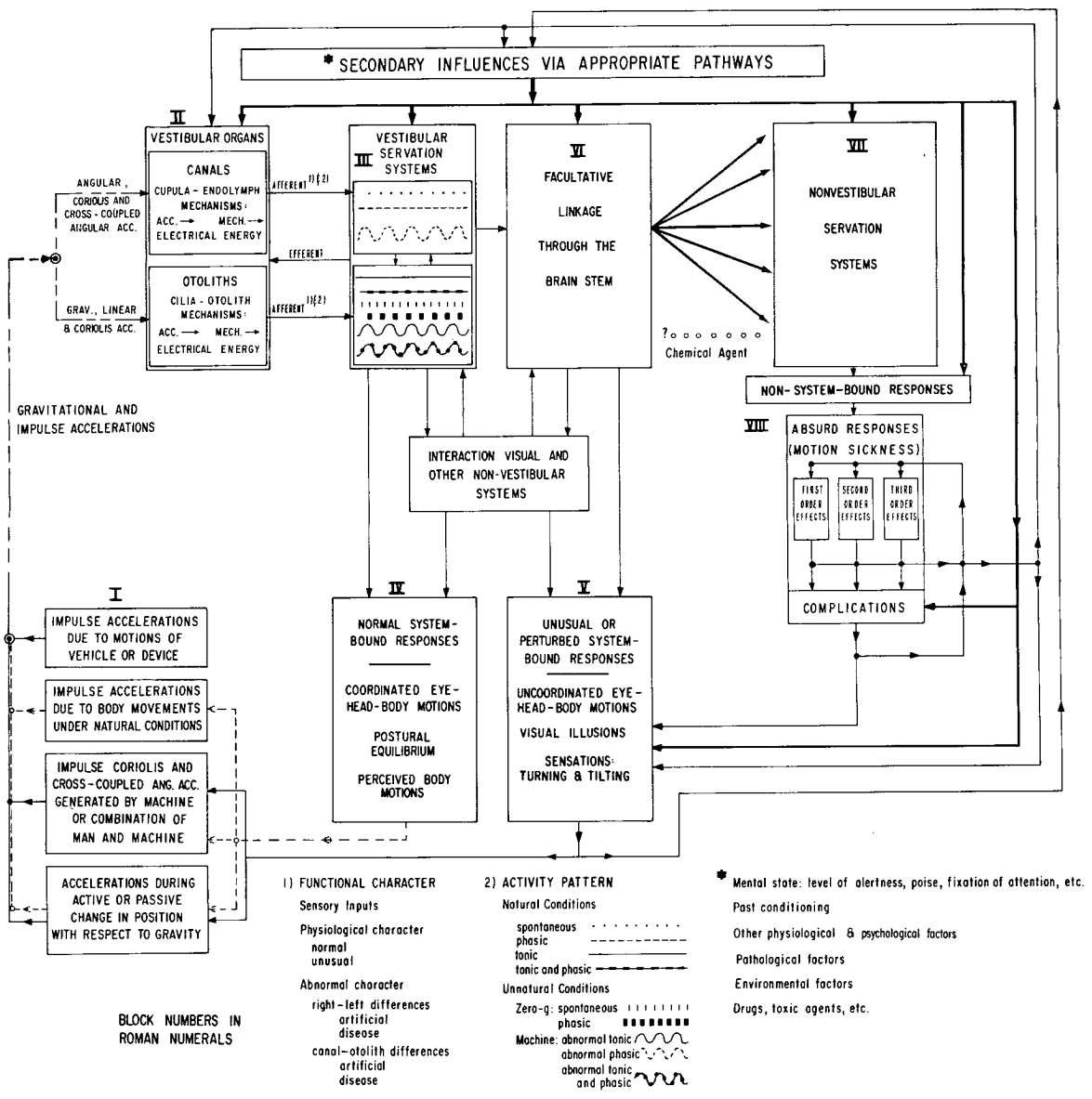


FIGURE 1.—*A conceptual framework showing important elements and their interactions underlying reflex phenomena and motion sickness—a delayed phenomenon.*

spatial patterns constituting the propagated discharge, there are well-known differences between the two end organs, which must be taken into account.

The cupula-endolymph mechanism in the six semicircular canals responds to impulse angular and cross-coupled angular accelerations and, for practical purposes, is gravity independent. Under nearly all natural conditions, the canals are stimulated only by the motions of man which involve rotations of the head. Under artificial conditions, of course, the canals respond to the same types of accelerations generated by a machine. In the absence of head motions (active or passive) there is no accelerative canalicular stimulus; but a resting discharge, presumably of chemical origin, is present. Its precise role, however, has not been determined.

The cilia-otolith mechanism in the four otolith organs is activated by gravity and by impulse linear

and Coriolis accelerations so directed as to cause a shearing displacement of the otolithic membrane supporting the hair cells. The result is mechanical deformation of the kinocilia, which in turn results in chemical changes affecting the generation of bioelectricity (nerve-action potentials). The stimulus due to gravity deserves additional comment. There is general agreement that if the head is rotated in the gravitational field in such a manner as to cause relative motion between otolithic and cupular membranes, this constitutes an effective stimulus in addition to whatever suprathreshold linear accelerations are generated. When the head is fixed in the gravitational field, there is no question but that the otolith organs are stimulated, as indicated not only by the persistence of ocular counterrolling (ref. 9) and the oculogravic illusion (ref. 10), but also by the fact that these responses vary with hypogravity and supragravity *g*-loadings (refs. 11 and 12). Whether the effective stimulus is due to a constant weight (or pressure) or due to slight unavoidable changes in position of the macular plates with respect to gravity (even though the head is presumably fixed) is not clear. In either event, the stimulus gives rise to a tonic sensory input over and above the resting discharge.

In block III are shown the vestibular servation system and its two components (canalicular and otolithic) which have reciprocal modulating influences. An attempt has been made to indicate typical normal and abnormal canalicular and otolithic activity patterns. Also shown are major acting or interacting influences, including vestibular efferent fibers ensuring a return flow of impulses to the end organs, thus closing one loop. This efferent vestibular activity is under intensive study; the morphological evidence is on a firm basis (refs. 13, 14, and 15), and the functional role in man, while uncertain, is likely to prove to be inhibitory.

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

The vestibular responses under abnormal stimulus conditions fall mainly into two categories, system-bound and nonsystem-bound responses. The main chain of events in system-bound responses involves blocks I, II, III, V, and VI.

Some but not all system-bound responses reflect instability of the vestibular system, and they will be referred to as reflex vestibular disturbances. Typical manifestations in normal persons include nystagmus, "sensations," the oculogyral illusion, and eye-head-body incoordination. Systematic studies of reflex manifestations reveal characteristics of the various responses which may be observed or inferred. In general, they have the following in common: (1) short latencies, (2) maximal response to the initial stimulus, (3) no perseverance of responses unless explicable by continuation of stimulation, and (4) adaptation.

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

Little is known concerning the facultative linkage (block VI). The fact that "irradiating" vestibular activity is demonstrably open to modulating influences points to the use of common pathways in the brain stem formation. Mild symptoms of motion sickness have disappeared under the influence of experimenter-directed tasks which may have preempted neural pathways used by irradiating vestibular activity. What makes the vestibular facultative

linkage unusual but not unique is the readiness with which vestibular activity may get "out of bounds" and elicit the widespread responses which include the typical symptoms of motion sickness. The sometimes long delay between the onset of stimulation and appearance of motion sickness suggests that a chemical linkage also may be involved.

Certain secondary etiologic influences are listed in the lower right corner of figure 1. Some of these influences, e.g., eyes open or closed, are always present, tending to increase or decrease susceptibility to motion sickness. Also, it may be assumed that any factor tending either to evoke or inhibit a response characteristic of motion sickness will affect susceptibility accordingly.

Although the typical symptoms of motion sickness are well known, a list of first-order responses (let alone the precise sites of origin) has not been compiled (block VIII). At least some first-order responses also act as stimuli, and so on, until the disturbances involve the organism as a whole. It is apparent that there are great gaps in our knowledge regarding mechanisms underlying the symptomatology of motion sickness. It would seem that the starting point in conducting studies would not be the full constellation of symptoms and syndromes but rather the first-order responses. Although there is general agreement as to what constitutes frank motion sickness, this agreement dwindles with the reduction in number and kind of responses. It is possible, for example, to elicit either sweating (probably a first-order response) and drowsiness (undoubtedly a second- or higher-order response) as the only definite overt symptom.

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

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

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

IMPLICATIONS FOR SPACE FLIGHT

Even a fragmentary knowledge of vestibular mechanisms can be usefully applied to the solution of vestibular problems in space missions involving the generation of artificial gravity.

The fact that the symptoms of acute motion sickness are similar in weightlessness and in rotating environments (indeed, in any conveyance or device) has important implications. First, the treatment of symptoms (apart from prevention) would be much the same in persons with similar symptomatology. Second, similarity of symptoms implies the operation of a common mechanism immediately prior to their elicitation. It would seem, therefore, that investigations in the laboratory dealing with the symptomatology of motion sickness would have relevance for operational conditions.

The initiating mechanisms eventually resulting in motion sickness are quite different in weightlessness and in a rotating environment, although the execution of head movements is essential in both circum-

stances. In weightlessness the head movements (rotations) generate normal angular accelerative stimuli, but the resulting sensory input encounters unusual central vestibular patterning due to the absence of the constant stimulus of gravity. The susceptible astronaut is immediately confronted with the maximum stressful effect possible; the analogous situation in a rotating environment would be immediate transition from zero velocity to terminal velocity. To achieve adaptation, head movements must be made, and a program based on a knowledge of the characteristics of motion sickness, the means of fixing the head relative to the thorax, the spacing and excursion of head movements, and the use of anti-motion-sickness drugs should be helpful.

Head movements out of the plane of rotation of the spacecraft generate abnormal accelerative stimuli and, at terminal velocity, the astronaut would be exposed to the maximal accelerative stimuli. There is one means of achieving rapid adaptation to rotation if a Bárány-type chair is available. Rotating the chair opposite to the rotation of the spacecraft would permit manipulation of the angular velocity between zero and terminal velocity, thus providing the opportunity to use incremental adaptation schedules (ref. 16).

REFERENCES

1. Berry, C. A.: Summary of Medical Experience in the Apollo 7 Through 11 Manned Spaceflights. Aerospace Med., vol. 41, 1970, pp. 500–519.
2. Yuganov, E. M.; Gorshkov, A. I.; Kas'yan, I. I.; Bryanov, I. I.; Kolosov, I. A.; Kopanev, V. I.; Levedev, V. I.; Popov, N. I.; and Solodovnik, F. A.: Vestibular Reactions of Cosmonauts During the Flight in "Voskhod" Spaceship. Aerospace Med., vol. 37, 1966, pp. 691–694.
3. Kas'yan, I. I.: Some Human Physiological Reactions Under Conditions of Alternating Overloads and Weightlessness. Izv. Akad. Nauk SSSR (Biol.) No. 6, 1962, pp. 896–908. (JPRS Translation)
4. Kas'yan, I. I.; Kolosov, I. A.; Levedev, V. I.; and Yurov, B. N.: Reaction of Cosmonauts During Parabolic Flight in Aircraft. Izv. Nauk SSSR (Biol.) Series B, No. 2, 1965. (Transl.)
5. Miller, E. F. II; Graybiel, A.; Kellogg, R. S.; and O'Donnell, R. D.; Motion Sickness Susceptibility Under Weightless and Hypergravity Conditions Generated by Parabolic Flight. Aerospace Med., vol. 40, 1969, pp. 862–868.
6. 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., vol. 3, 1960, pp. 55–73.
7. Clark, B.; and Graybiel, A.: Human Performance During Adaptation to Stress in the Pensacola Slow Rotation Room. Aerospace Med., vol. 32, 1961, pp. 93–106.
8. Graybiel, A.: Structural Elements in the Concept of Motion Sickness. Aerospace Med., vol. 40, 1969, pp. 351–367.
9. Miller, E. F. II; and Graybiel, A.: Ocular Counterrolling Measured During Eight Hours of Sustained Body Tilt. NAMRL–1154 and NASA Orders T–81633 and L–43518. Naval Aerospace Medical Research Laboratory, Pensacola, Fla., 1972.
10. Clark, B.; and Graybiel, A.: Perception of the Visual Horizontal in Normal and Labyrinthine Defective Observers During Prolonged Rotation. Amer. J. Psychol., vol. 79, 1966, pp. 608–612.
11. Miller, E. F. II; Graybiel, A.; and Kellogg, R. S.: Otolith Organ Activity Within Earth Standard, One-Half Standard, and Zero Gravity Environments. Aerospace Med., vol. 37, 1966, pp. 399–403.
12. Miller, E. F. II; and Graybiel, A.: The Effect of Gravitoinertial Force on Ocular Counterrolling. J. Appl. Physiol., vol. 31, 1971, pp. 697–700.
13. Rasmussen, G. L.: Efferent Fibers of the Cochlear Nerve and Cochlear Nucleus. Neural Mechanisms of the Auditory and Vestibular Systems, G. L. Rasmussen and W. F. Windle, eds., Charles C. Thomas, 1960, pp. 105–115.
14. Rasmussen, G. L.; and Gacek, R.: Concerning the Question of an Efferent Fiber Component of the Vestibular Nerve of the Cat. Anat. Record, vol. 130, 1958, pp. 361–362.
15. Rossi, G.: Central Projections to the Vestibular Receptors. Third Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–152, 1968, pp. 213–224.
16. Reason, J. T.; and Graybiel, A.: Progressive Adaptation to Coriolis Accelerations Associated With 1-RPM Increments in the Velocity of the Slow Rotation Room. Aerospace Med., vol. 41, 1970, pp. 73–79.

SESSION II

Chairman: **CHARLES A. BERRY**
Manned Spacecraft Center, NASA

Space Station Capability for Research in Rotational Hypogravity

GEORGE KELLER
George C. Marshall Space Flight Center, NASA

SUMMARY

This paper outlines certain capabilities provided in preliminary designs of obital space stations for research in rotational hypogravity. It also indicates alternative configurations that are being considered. Principal addressees are members of an international community of physiologists whose work in Earth-oriented, as well as space-oriented, physiology can be supported through observation under the background environment of null gravity. Their participation in originating and devising advanced experiments and in developing requirements is encouraged. Such participation is expected to enhance final design of the selected space station and to make the research program more meaningful.

INTRODUCTION

The purposes of this paper are to provide information for an audience of physiologists and to encourage their participation in certain research opportunities of the future. This information includes the description of prospective orbital space stations and their capabilities. The research opportunities relate to the unique rotational hypogravity that can be provided within a space station. Such research can lead to further knowledge of the physiological functioning and underlying mechanisms in man within his Earth environment, and the observations can be more meaningful as the range of conditions is increased. Experimental observations under normal Earth gravity and hypergravity have been rather extensive, of course, whereas the condition of hypogravity is relatively unexplored. Thus, orbital space stations offer significant opportunity, not so much for investigating man's performance in space, but rather as a means for learning more about man in his normal habitat and, more significantly to some, more about the role of gravitational fields in the origin and evolution of man.

Concepts for manned orbital platforms have been advanced over many years, centuries, or longer. They are, perhaps, as old as man's imagination itself. Such platforms in the sky would afford closer views of the Moon, the stars, and the Earth; act as way stations on journeys beyond; provide a means for surveillance of enemy forces, for launching bombs, or for shooting ray guns; serve as communication relays or collectors of solar energy; or even be merely places away from the ordinary.

Reports on rotating space stations are numerous and include some in the proceedings of the first four symposiums like the present one (NASA SP–77, SP–115, SP–152, and SP–187). The bibliography in this report, however, is limited to documentation under present contracts by the National Aeronautics and Space Administration (NASA) with McDonnell Douglas Astronautics Company (MDAC) and with North American Rockwell (NAR).

It is the place away from the ordinary that is of interest here, and it is extraordinary hypogravity that has received much of the attention in recent focus on orbital space stations. Two extensive space station definition studies have been completed this month; each of these efforts centered about a respective baseline space station as a research facility

in orbit. Both accommodate experiments in plant and animal physiology under conditions of partial gravity, and both provide for operational assessment of artificial gravity as an environmental condition of habitation. One of the studies resulted in an expanded capability configuration, with emphasis on research in partial gravity. Other concepts have been proposed and have been successful to lesser extents; these include the so-called modular stations and, receiving attention after years of dormancy, the space wheel. Both of these are devised to be launchable as a payload of the Earth-to-orbit reusable shuttle. Further definition studies are in progress. Each of the configurations just mentioned is described in the following sections of this report. The descriptions should provide an understanding of the capabilities afforded for research in certain areas of physiology.

SPACE STATION CONFIGURATIONS

Baseline Space Stations

McDonnell Douglas Astronautics Company (MDAC)

Figure 1 shows the inboard profile of the vehicle in launch configuration. Note the artificial-gravity module, which contains cables and mechanisms for controlled separation of the S–II stage and the station. Also, note the extendable artificial-gravity tunnel, within which is provided a counterrotating cab. Figure 2 shows the station and S–II stage (counterweight) deployed for artificial-gravity spinning; note here that a turnaround maneuver was required to mate the tunnel with the artificial-gravity module. Figures 3, 4, and 5 show decks 1 through 4 of this configuration. Figure 6 shows radii of rotation and *g*-level versus rotation speed for each of the four decks. Figure 7 shows the artificial-gravity configuration and the 0-*g* configuration. The transformation is made by jettisoning the S–II counterweight. Note that experiment modules are then dockable to the basic station under 0-*g* conditions.

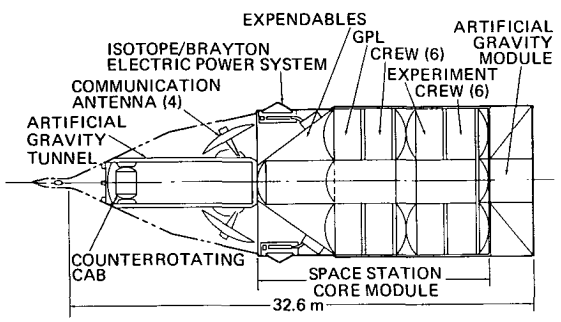


FIGURE 1.—*Baseline space station with reference artificial-gravity provisions (MDAC).*

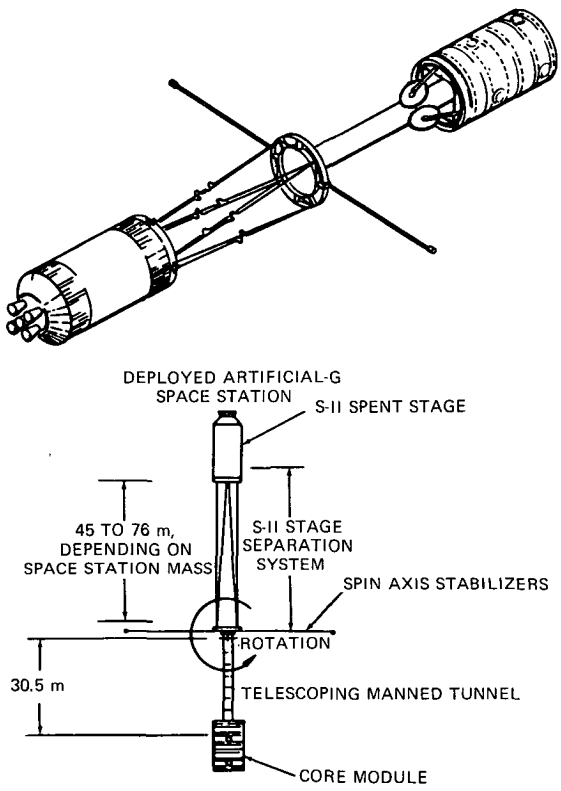


FIGURE 2.—*Space station artificial-gravity configuration (MDAC).*

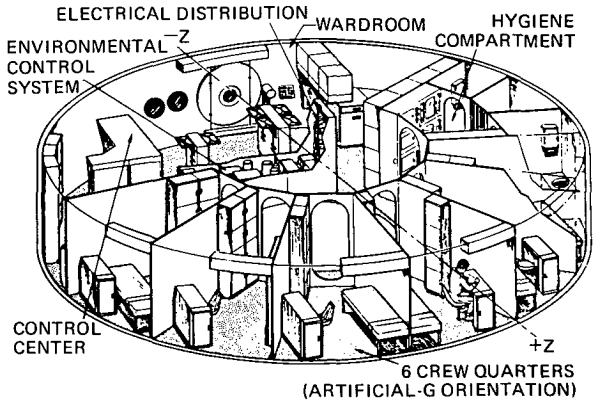


FIGURE 3. *Crew facilities and operations (decks 1 and 3) (MDAC).*

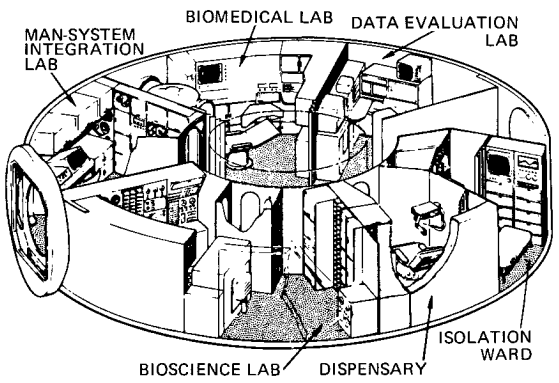


FIGURE 4.—*Experiments (deck 2) (MDAC).*

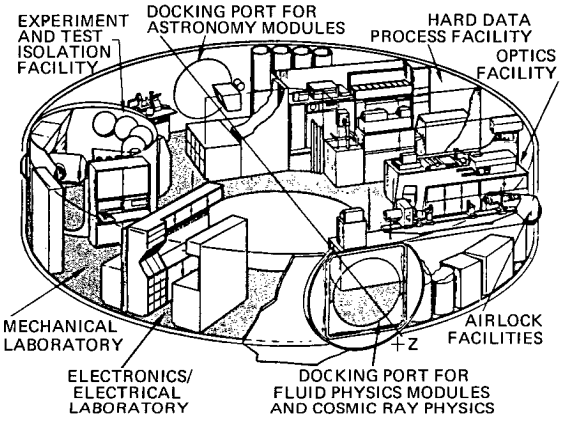


FIGURE 5.—*General-purpose laboratory (deck 4) (MDAC).*

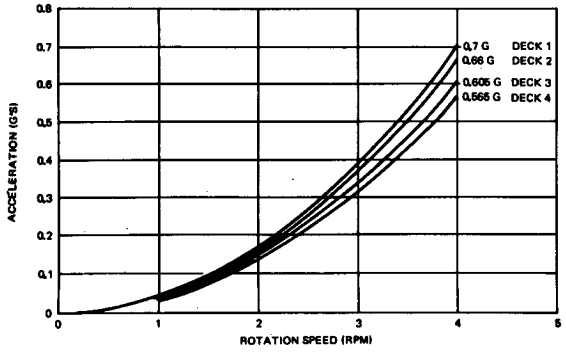


FIGURE 6.—*Artificial-gravity level versus rotation rate for each deck of core module in MDAC test configuration.*

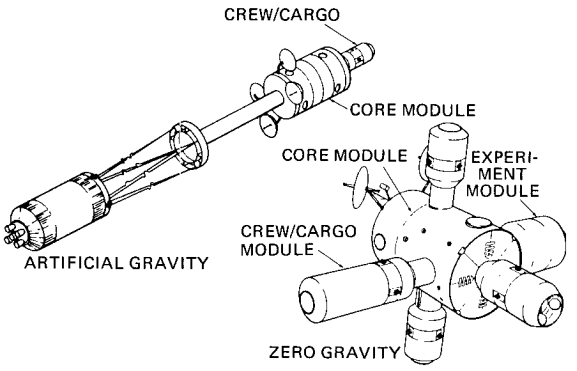


FIGURE 7.—*Baseline space station configurations (MDAC).*

North American Rockwell (NAR)

The docked S–II and extended S–II configurations are shown in figure 8. Note the differences from the MDAC design in: (1) solar array versus isotope-power supply, and (2) extendable boom versus cable deployment for separation of the S–II counterweight. Figure 9 shows a layout of deck 1 within this design. Of particular interest for comparison is the orthogonal versus radial array of the spaces within the two baseline stations. Figure 10 shows some details of facilities provided within deck 1.

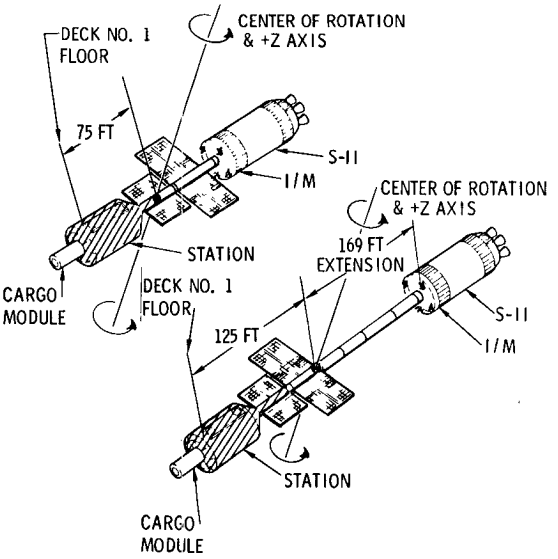


FIGURE 8.—*Artificial-gravity mode (NAR). Left, docked S-II; right, extended S-II.*

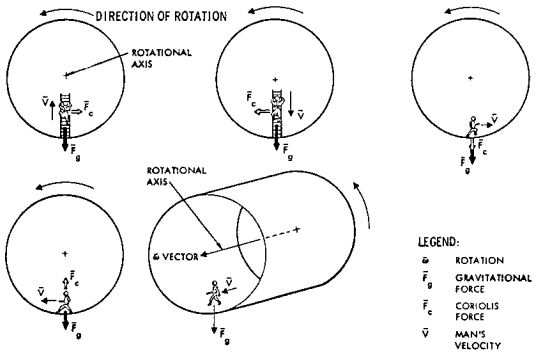


FIGURE 9.—*Space station deck 1 arrangement (NAR).*

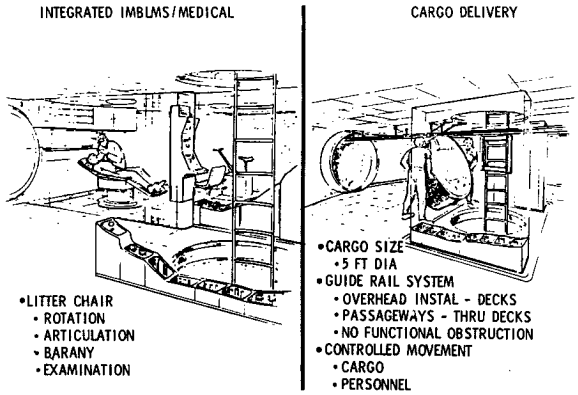


FIGURE 10.—*Deck 1 features (NAR).*

Space Base Concepts

Space Wheel

Figure 11 depicts an octagonal space wheel with spokes and central hub. At the end of the hub structure is shown a reactor power system. A portion of this central hub could be counterrotated to yield a null-gravity environment. Figure 12 shows the internal arrangement of two decks, each of constant radius. Figure 13 shows a basic shuttle-launchable module from which the annulus of the wheel is assembled. Note the floor plan for upper and lower floors at left and center, respectively, and the hatchways provided between modules at the lower floor level. Figure 14 shows sample floor plans for a hospital/dispensary module. Other modules would be equipped for functions such as operational control of the station, general-purpose laboratory for physical sciences experiments, electronics and optics

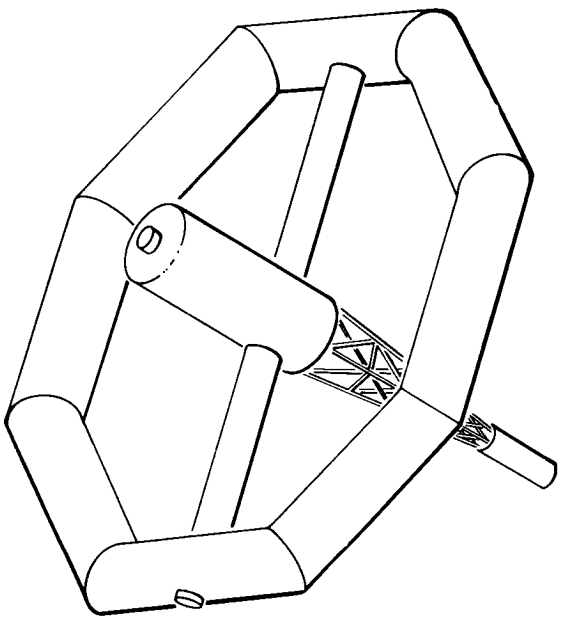


FIGURE 11.—*Operational station: space wheel.*

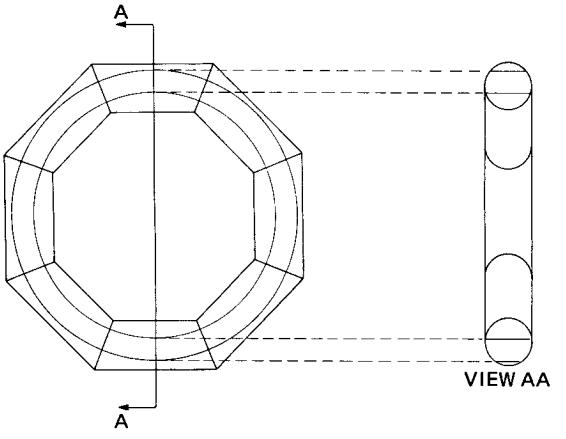


FIGURE 12.—*General internal arrangement: space wheel.*

laboratory, food preparation and eating, sleeping, rest and recreation, inspection and repairs. Figure 15 shows a general experiment floor. Figure 16 shows pertinent design interrelationships for space stations of the wheel type.

Modular Space Station

Figure 17 shows an artificial-gravity configuration for one of the modular concepts now being

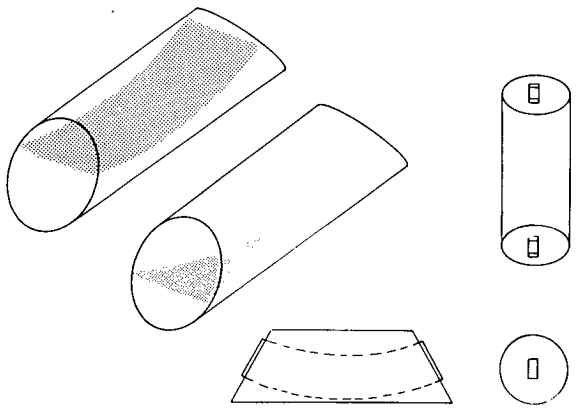


FIGURE 13.—*The basic shuttle-launchable module: space wheel.*

investigated. The *g*-levels that could be obtained with such a design are shown in table 1.

Expanded-Capability Space Station

The name for this design stems from the significant increase in experimental capability, including in particular increased capability for work in artificial gravity. Figure 18 illustrates the launch configuration of the station atop the first two stages of a Saturn V derivative, and figure 19 shows the orbital configuration prior to manning and activation. Note that the intermodule skin has been separated and antennas have been deployed. Figure 20 shows internal details of the core and artificial-gravity modules. The interconnecting tunnel is shown foreshortened for convenience of illustration. Note the counterrotating cab in the lower portion of the tunnel (also shown in fig. 1). Figure 21 shows a crew/cargo module enroute from the shuttle to the station for initial manning. Figure 22 shows the station, with crew/cargo module docked, activated and spinning. Figure 23 shows the station in a null-gravity mode with experiment modules attached and in free flight. Figures 24 and 25 show internal arrangement of the core and artificial-gravity modules. Figure 26 shows dimensions of the station in rotational configuration. Table 1 gives gravity level versus spin rate for each of the six decks.

DISCUSSION

Apart from these outline descriptions of space station configurations, it should be noted that two categories of research activities are involved. One is the more obvious, relating directly to the subject matter of this symposium; that is, the qualification of man for space flight. The other relates to the extraordinary environment of null and partial gravity as a significant experimental condition—not simply for research under these conditions, but as a means to a better understanding of the fundamental mechanisms operating in the gravity field of Earth life.

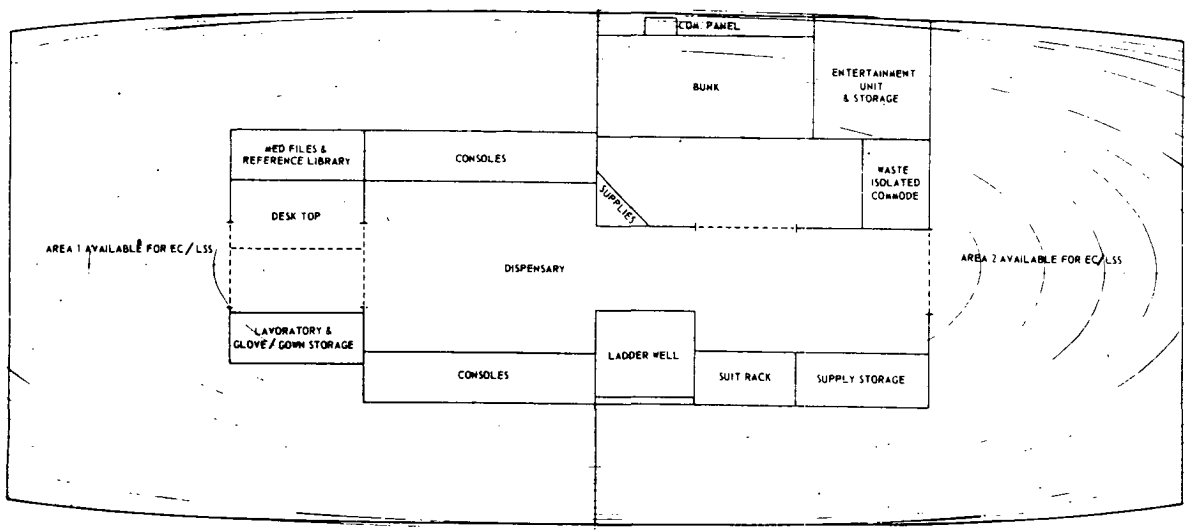
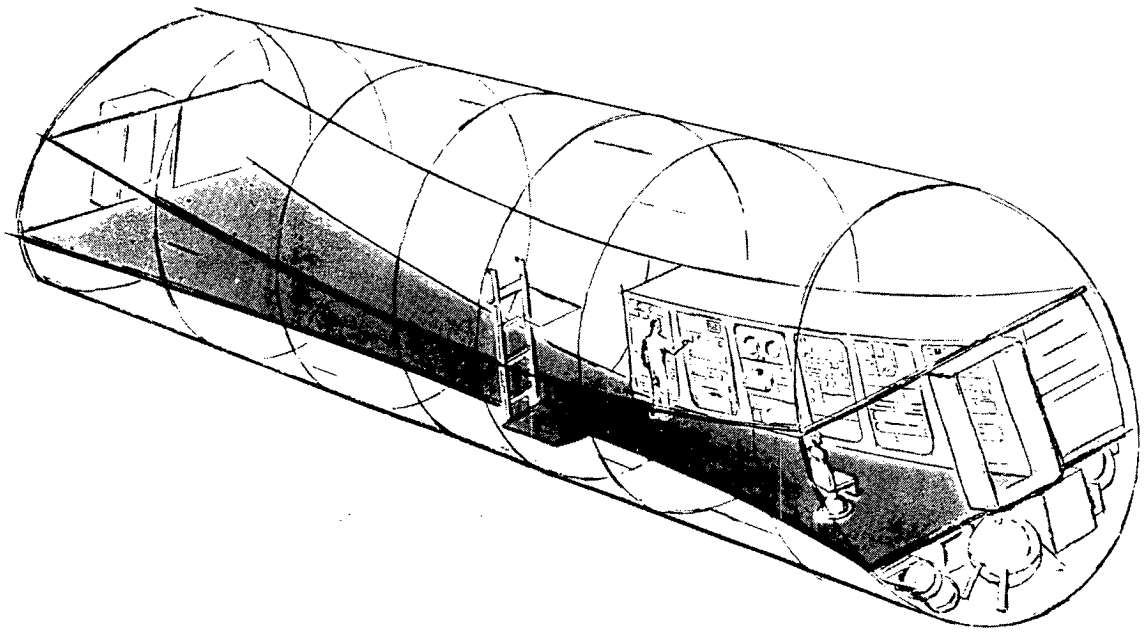


FIGURE 14.—*Proposed floor plan for hospital/dispensary module: space wheel.*



PROJECTED EQUIVALENT FLOORSPACE

FUNCTION

- EXPERIMENT AREA
- INTERMODULE TRANSFER ROUTE

HEADROOM 6.5' OVER ENTIRE FLOOR

FLOORSPACE APPROXIMATELY 368 SQ. FT.

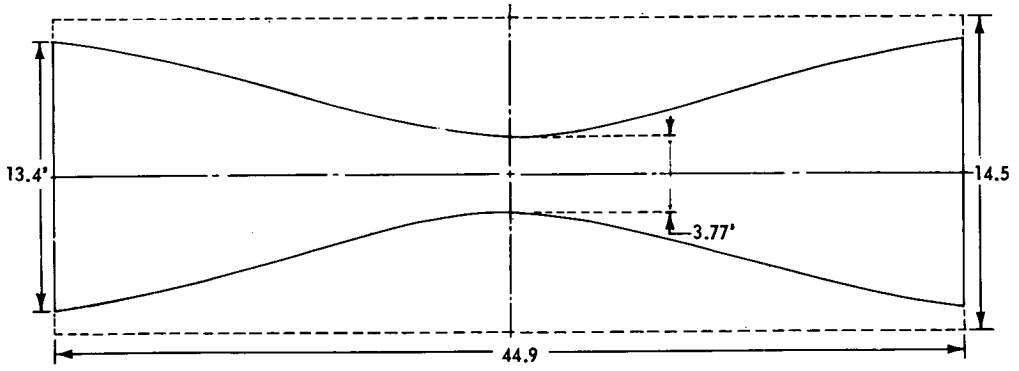


FIGURE 15.—*General experiment floor: space wheel.*

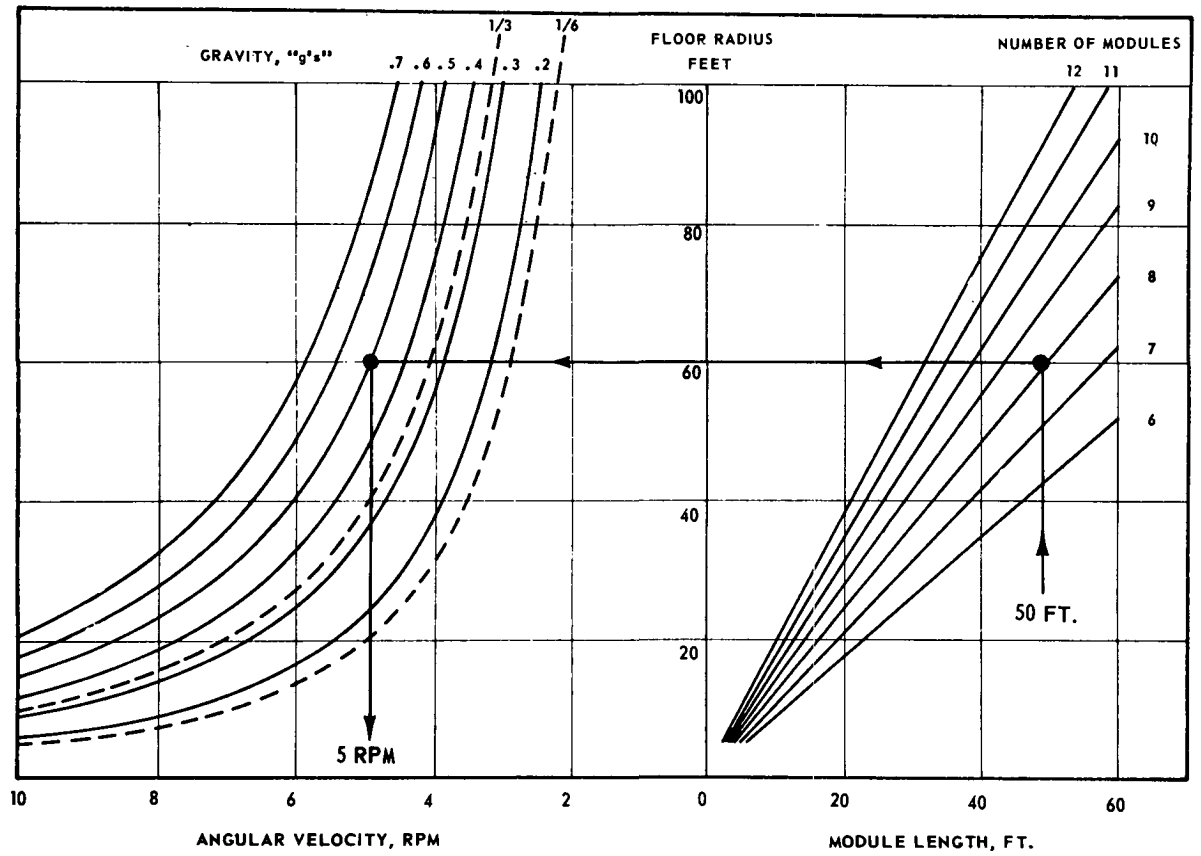


FIGURE 16.—*Design interrelationships: space wheel.*

Most studies concerning space stations thus far have been directed toward providing an environment in orbit to which the average healthy person could readily adapt without discomfort or loss of performance. A report by the Artificial Gravity Experiment Definition Study Group will soon be ready for limited distribution by NASA. This group has defined a program to study man's response to rotational environments within a null-gravity field such that his tolerances and adaptations are clearly known or can be predicted. As mentioned before, both the basic space station designs accommodate this objective. Through differences in interpretation of requirements, the NAR design provides for a single operational assessment with one crew, whereas the MDAC design provides for repeated assessments with each of several crews over a period of 15 months. Except for numbers of subjects, these experimental capabilities are similar. The expanded capability configuration, however, offers broader opportunities which may be of interest to you and your associates in similar and related fields of investigation and application. Prospects for a vehicle like the modular station or the space wheel are left to your individual considerations.

For purposes of tradeoffs and analyses within the space station definition studies, NASA provided a candidate experiment program. It was intended to be typical or representative of a real experiment program to be implemented during the 10-year life

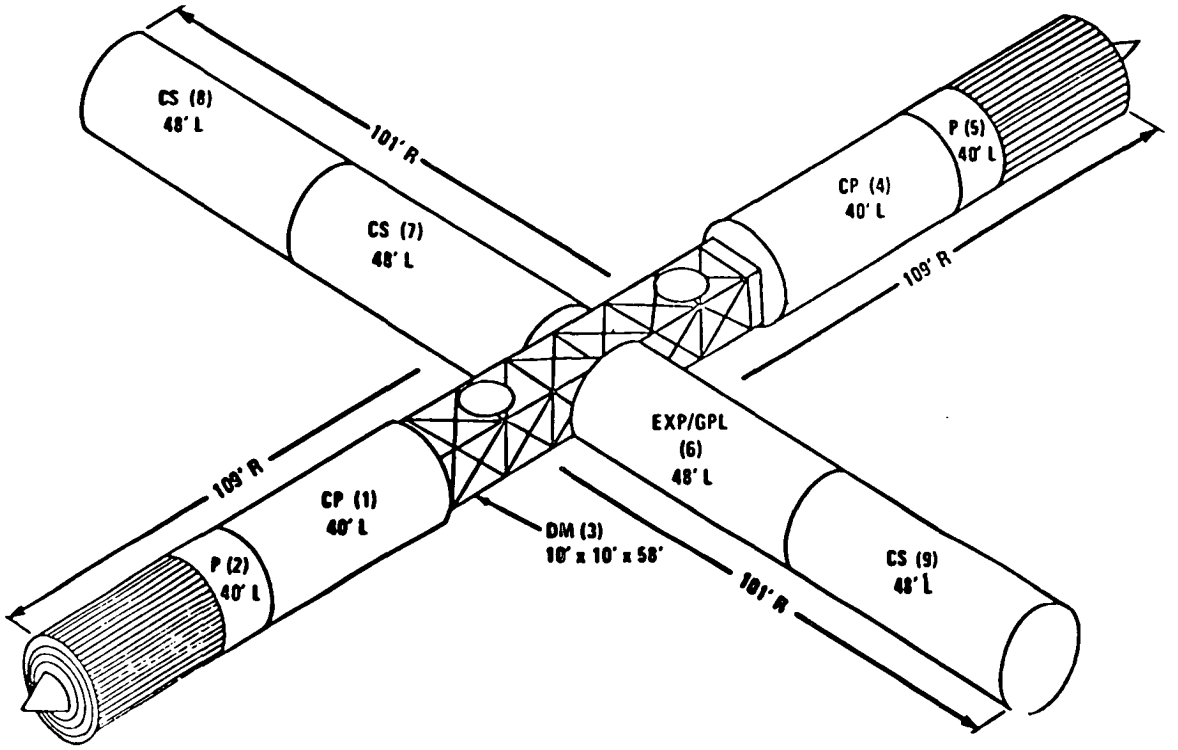


FIGURE 17.—*Modular space station artificial-gravity configuration.*

TABLE 1.—*Artificial-Gravity Parameters*

Deck	6	5	4	3	2	1
Spin radius (m)	39.3	37.2	9.4	11.6	15.0	17.1
Spin rate (rpm)	Acceleration (g)					
10	4.38	4.15	1.06	1.30	1.66	1.91
9	3.56	3.36	0.85	1.05	1.35	1.55
8	2.81	2.66	0.67	0.83	1.07	1.22
7	2.17	2.03	0.51	0.63	0.81	0.94
6	1.58	1.49	0.37	0.47	0.60	0.69
5	1.10	1.04	0.26	0.32	0.42	0.48
4	0.70	0.66	0.17	0.21	0.27	0.30
3	0.40	0.37	0.09	0.12	0.15	0.17
2	0.18	0.17	0.04	0.05	0.07	0.08
1	0.04	0.04	0.01	0.01	0.02	0.02

Enclosed area indicates acceleration range of interest (0.1 to 1.0 g).

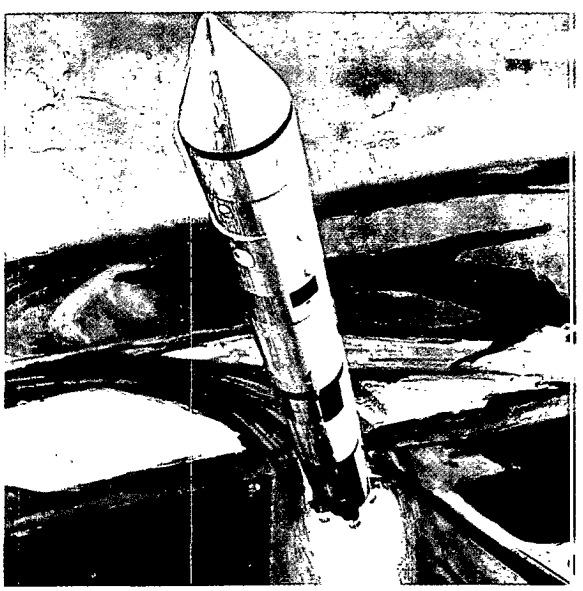


FIGURE 18.—*Space station launch: expanded-capability space station.*

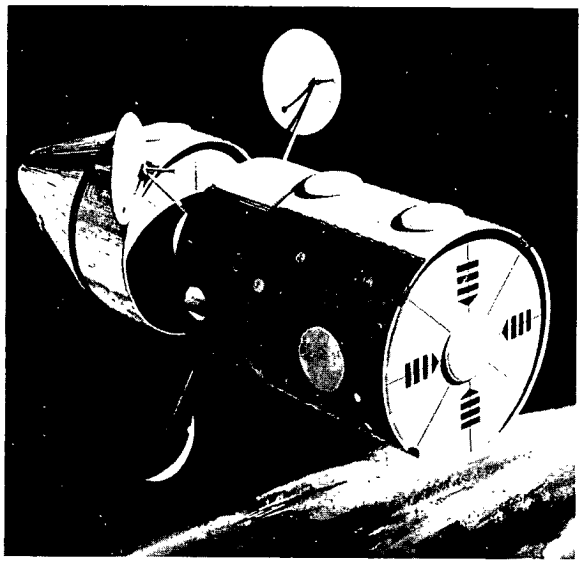


FIGURE 19.—*Unmanned operations: expanded-capability space station.*

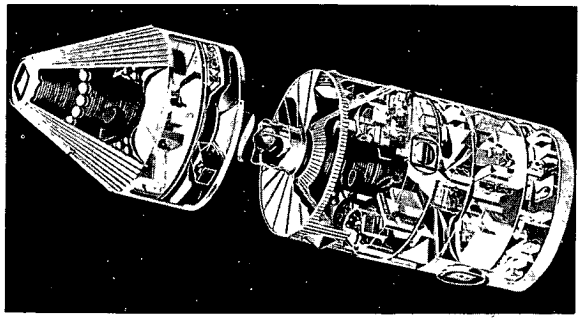


FIGURE 20.—*Expanded-capability space station showing internal details of the core and artificial-gravity modules.*

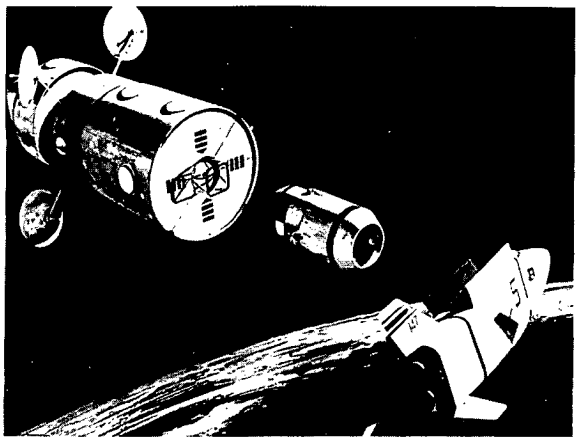


FIGURE 21.—*Manning operations: crew/cargo module enroute from shuttle to station (expanded-capability station).*

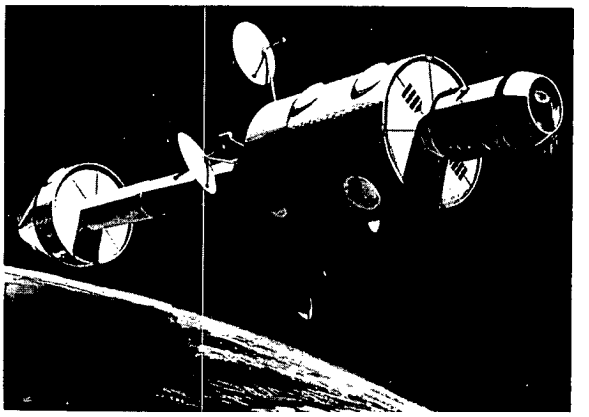


FIGURE 22.—*Artificial-gravity experiment: station with crew/cargo docked.*

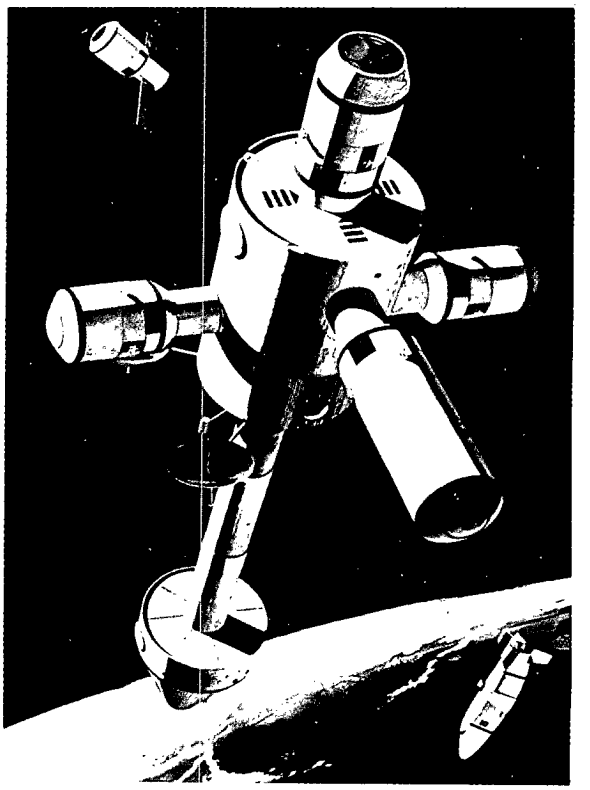


FIGURE 23.—*Expanded-capability space station with experiment modules attached and in free flight.*

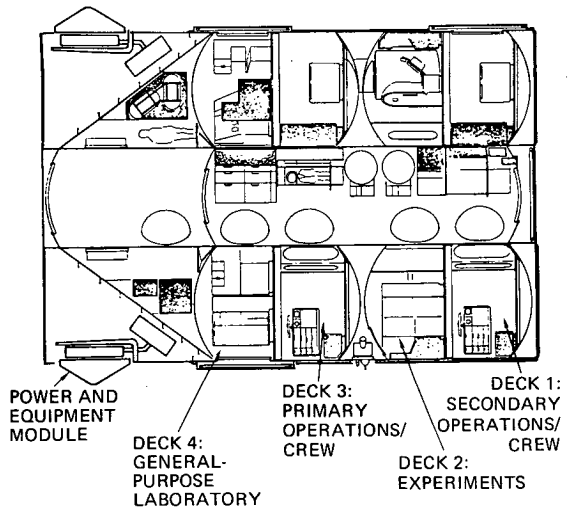


FIGURE 24.—*Core module: expanded-capability space station.*

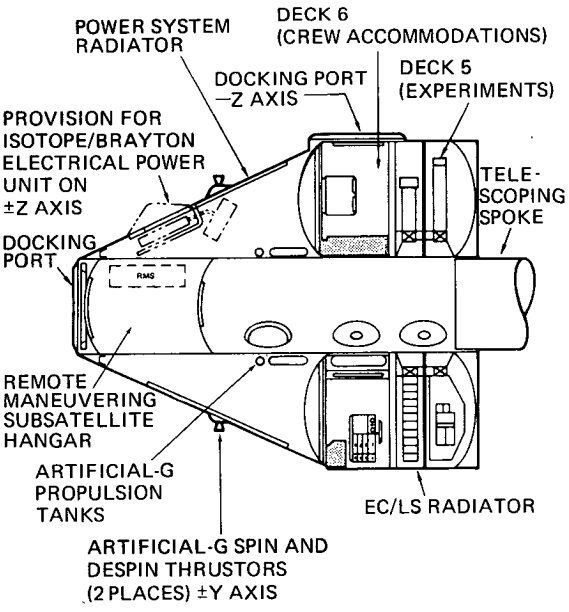


FIGURE 25.—*Artificial-gravity module: expanded-capability space station.*

span projected for the station. It includes elements of plant and animal physiology, some of which are in the form of centrifuged experiments. Except for elements of advanced subsystems or technology experiments, however, it does not include very many fundamentals in human physiology related to either

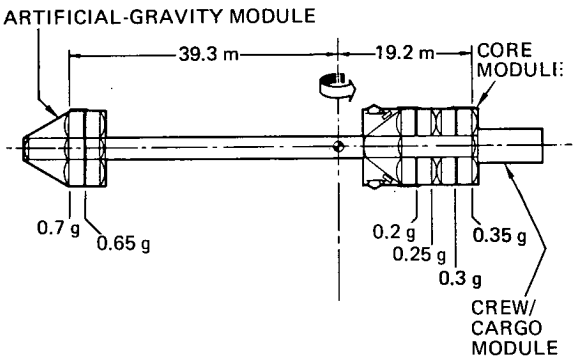


FIGURE 26.—*Dimensions and artificial-gravity levels of expanded-capability space station rotating at 4 rpm.*

long-term habitation in hypogravity, or basic effects of gravitational fields. Presently, on the other hand, additional coverage along these lines is being considered within NASA.

Additionally, it is important, I believe, that investigators throughout the fields of physiology take a close look at the opportunities afforded by an orbital space station, that they evaluate those opportunities in terms of their individual needs and interests, and that they assert their views and conclusions. How all these investigators might become aware of the opportunity is not clear. Certainly the group at this meeting already knows of prospects for research in fundamentals related to the disorientation and confusion of acceleration environments. Others surely know of some of the prospects for measurement of physiological performances. Still others have devoted some attention to techniques of surgery, wet chemistry, electrophoresis, and the like, performed in orbit or in planetary flight. On the other hand, overall definitions of what can and ought to be done, and how it should be done, are still incomplete. Better concepts and improved designs of experiments are certain to come. As soon as these are available, they can be translated into requirements so that improvements in station configuration, subsystems, and research facilities can be considered. In this way the space station operation can become more useful and more valuable.

I should like to encourage you to view the space station as a facility with experimental conditions for Earth-oriented as well as space-oriented research of rather unique dimensions, and to call upon your

imagination and your professional and creative skills to devise and develop new ideas and paths toward further knowledge. Also, I should like to encourage you to communicate with associates and researchers in other areas of your fields and to participate in the fundamental work underlying the definition of the experiment program.

ACKNOWLEDGMENTS

Credits are due North American Rockwell and General Electric Company for the designs and illustrations of the baseline stations and to McDonnell Douglas Astronautics Company and Martin Marietta Corporation for those of the expanded-capability station. Credits are due Program Development, Marshall Space Flight Center, for the designs and illustrations of the modular station and the wheel. Further details may be requested from William A. Brooksbank, Jr. at Marshall Space Flight Center or Rene Berglund at Manned Spacecraft Center for work done at the respective centers or by contractors. John Hammersmith of NASA Headquarters is chairman of the study on artificial gravity experiment definition, and Dr. Rodney Johnson, also of NASA Headquarters, is manager of the experiment program for these purposes. Any one of us will be pleased to provide further detail or clarification, or to correspond with you as desired.

BIBLIOGRAPHY

Contract NAS8–25140 (McDonnell Douglas Astronautics Company/Martin Marietta Corporation/International Business Machines Corporation) reports:

Experiment Support Requirements: DRL 8 Vol. I

Payload Accommodation and Integration: DRL 8 Vol. VI

Summary, Space Station Definition: DRL 8 Summary

Expanded Capability Study: DRL 8 Vol. VIII

Contract NAS9–9953 (North American Rockwell) report:

Solar-Powered Space Station Artificial Gravity System Preliminary Performance Specification

Design of Experimental Studies of Human Performance Under Influences of Simulated Artificial Gravity

WILLIAM M. PILAND, H. GEORGE HAUSCH, and GRADY V. MARAMAN
NASA Langley Research Center

JAMES A. GREEN
North American Rockwell Corporation

SUMMARY

Future manned space vehicles, such as Earth-orbital space stations and interplanetary vehicles, may require artificial gravity for the maintenance of adequate crew performance and physiological condition. Before final designs of these vehicles can be undertaken, data must be made available on the level of gravity required. Also needed is a better understanding of what combinations of radius and rotation rate (for the required gravity level) will provide a habitable environment for the crew.

A ground-based research program is now being undertaken to provide data concerning the effects of a rotating environment on man's ability to adequately perform gross and fine psychomotor tasks. Emphasis is being placed on establishing the levels of artificial gravity and rates and radii of rotation required in future space systems for preservation of crew performance and comfort. This paper reports on an experimental study utilizing the North American Rotational Facility to investigate crew mobility, cargo transfer and handling, and fine motor coordination at radii up to 24 meters and at rotational rates up to 5 rpm.

INTRODUCTION

NASA and the aerospace industry are currently involved in a cooperative planning activity to define requirements for and configurations of post-Apollo spacecraft. These spacecraft will have the primary function of serving as space laboratories in which research can be conducted in such disciplines as physics, astronomy, biotechnology, advanced spacecraft technology, and Earth resources. Since the spacecraft must provide the scientist-astronaut with an environment for conducting his research without undue hardship and stress, many spacecraft configurations being derived evolve around the use of artificial gravity by rotation of either all or part of the vehicle. The idea is that artificial gravity would provide a laboratory setting similar to that which the researcher is accustomed to on Earth and that artificial gravity would have the potential of averting any adverse effects of weightlessness on the human physiological state.

It should be noted that weightless flights to date have not substantiated whether the use of artificial gravity is pertinent to the successful accomplishment of future space missions. Whether artificial gravity is required will be determined only after missions of longer duration have been undertaken in weightlessness. However, since the possibility exists that artificial gravity may enhance mission performance and that future spacecraft may be required to rotate, it is important that the effects of rotation on man be thoroughly understood. A technological base for designing artificial-gravity spacecraft must be established so that mission planning and spacecraft development will not be delayed because of a lack of design criteria.

The objective of the ground-based research program presented herein is to provide some of the needed information concerning man's performance in an artificial-gravity environment. This program will not answer the question of whether artificial gravity is needed. It is, however, designed to provide

some insight into the aspects of a rotating spacecraft that are critical in the preservation of crew performance and in the maintenance of a habitable environment.

POSSIBLE EFFECTS OF ARTIFICIAL-GRAVITY ENVIRONMENT ON HUMAN PERFORMANCE

Although artificial gravity does have the potential for providing the astronaut an environment which is similar to that experienced on Earth (i.e., a directional-gravity environment), there are certain aspects of rotation that make this environment somewhat unusual. The adverse effects of rotation have been expounded upon in references 1, 2, and 3 and in many other reports. Basically, the complexity of the environment is caused by the presence of Coriolis forces, gravity gradients, varying gravity levels, and cross-coupled angular accelerations. Coriolis forces will be experienced by a man when he moves within the rotating vehicle or will be exerted on objects when they are moved. The magnitude and direction of these forces are dependent upon the rate at which the man or object moves, the direction of movement, the relative location at the point of movement within the vehicle, and the rate of vehicle rotation. These forces may cause an astronaut to have difficulty in walking, climbing, and performing routine tasks involving the handling of masses of various sizes.

Gravity gradients and changing gravity levels within the rotating spacecraft exist because the gravity environment is a function not only of rate of rotation but also of the radius. Thus, because of differences in radius, objects on shelves weigh less on "higher" shelves than on "lower" ones, a person weighs less while standing than while sitting or squatting, and the astronuat passing from one floor level to another will experience a different gravity environment at each level. This last fact is typified in table 1 where it is shown that an astronaut or scientist will be required to move from one level to another during his daily activity; at each level he will be required to perform various tasks in an environment sometimes significantly different from that which he has just left.

There are two important aspects of the effects of cross-coupled angular acceleration on man in a rotating environment. These effects are experienced because of the gyroscopic coupling of the angular velocity of the rotating vehicle with the angular velocity of objects being rotated within the environment. One result of this coupling is the unusual reaction of masses (such as cargo packages) when they are handled. Because of the cross-coupling effect, the mass is angularly accelerated about an axis perpendicular to the axis of manually induced rotation. The result of this reaction may be a degree of difficulty in working with objects of significant size.

The second effect of cross-coupled angular acceleration is related to the stimulation of the human vestibular system. Numerous previous publications (refs. 1, 2, 4, and 5, for example) have reported on these effects. Briefly, since the vestibular system is an acceleration-sensing mechanism, the cross-coupled accelerations caused by head movements in a rotating environment sometimes create bizarre stimulations to the system which result in disorientation and possibly in varying degrees of motion sickness.

Singular and combined results of all the adverse effects of rotation discussed above have been predicted to influence an individual's performance capabilities in artificial gravity. Of course, human adaptation to the rotating environment has been found to occur by many investigators (refs. 3 and 5, and others). However, the degree of accommodation to the environment for effective performance of specific tasks is relatively unknown. These unknowns are related primarily to possible variations in performance as functions of changing gravity environments, variations from one individual to another, and changes with the degree of adaptation to rotation. The ground-based experimental program described below deals with the study of specific human tasks that may be adversely affected by a rotating environment and that are of concern during routine manned space mission operations.

GROUND-BASED TEST PROGRAM

In designing an experimental program to study the effects of artificial gravity on human performance, a guideline mission was needed to insure that the results of the program would be applicable to the development of a reasonably well-defined mission and would be complementary to current space-planning activity. For this purpose, the presently planned space-base configuration (fig. 1) was used to formulate the program and to focus on critical problem areas being uncovered in the prelim-

TABLE 1.—*Typical Time Lines of Space-Base Activities (assuming six working levels between 18 m and 40 m at 4 rpm)*

Time	Level	Activity	Approximate g's
		Crewman	
0630	1	Arise	0.3
0630 to 0700	2	Personal hygiene	0.41
0700 to 0800	3	Mess	0.48
0800 to 1030	6	Calibration shops	0.7
1030 to 1200	6	Wet laboratory	0.7
1200 to 1300	3	Mess	0.48
1300 to 1400	4	Sick bay	0.59
1400 to 1800	5	Control station	0.63
1800 to 1830	2	Personal hygiene	0.41
1830 to 1930	3	Mess	0.48
1930 to 2200	3	Recreation and exercise	0.48
2200 to 0630	1	Quarters	0.3
		Scientist	
0630	1	Arise	0.3
0630 to 0700	2	Personal hygiene	0.41
0700 to 0800	3	Mess	0.48
0800 to 1200	Hub	Research	0
1200 to 1300	3	Mess	0.48
1300 to 1600	4	Data reduction	0.59
1600 to 1800	6	Wet laboratory	0.7
1800 to 1830	2	Personal hygiene	0.41
1830 to 1930	3	Mess	0.48
1930 to 2200	3	Recreation and exercise	0.48
2200 to 0630	1	Quarters	0.3

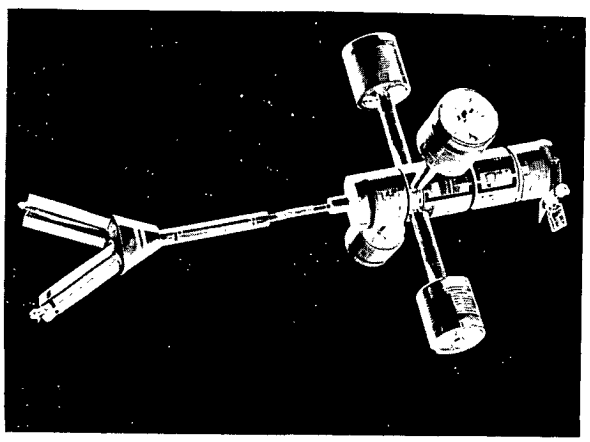


FIGURE 1.—*Typical space-base configuration.*

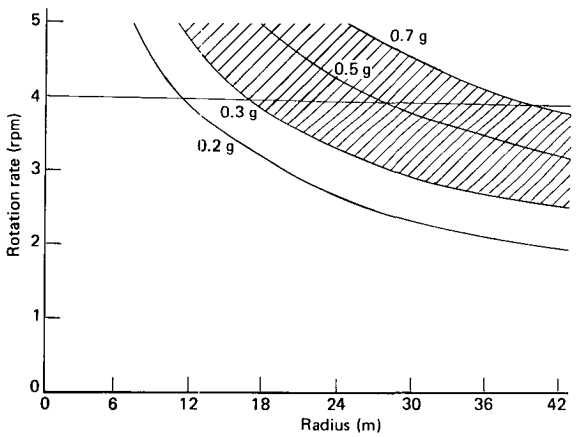


FIGURE 2.—*Currently proposed space-base environment.*

inary design of this spacecraft. Figure 2 illustrates the artificial-gravity characteristics of this vehicle. Acceleration in the main operating areas of the spacecraft is between 0.3 and 0.7 times the Earth's gravity, and the nominal rate of rotation is 4 rpm. This environment limits the operating radius of the spacecraft to between about 18 and 40 m. In searching for a ground-based facility with which to investigate the related problems of artificial gravity, it was determined that only one facility was readily avail-

able and adaptable for the conduct of this research and would closely simulate the environment of the space base. This facility is the North American Rotational Test Facility, located in Downey, California (fig. 3).

The North American Rotational Test Facility has a radius of 24 m and the capability of rotating up to 6 rpm. The facility has been designed with the capability of being expanded in radius to 38 m for future research at simulated space-base levels farther from the rotational center. A habitable module is located at one end of the facility (at a 23-m radius) and is capable of supporting a crew of four men for a period of 30 days. The facility shown in figure 3 has undergone a number of structural modifications for the accommodation of the experiments to be conducted (fig. 4). In addition to the living module located at one end, a number of test stations have been added for studying performance at various radii, a movable enclosure is included for studies of walking while the subject's body is alined with the artificial-gravity vector, ladders are provided for examining the subject's ability to move radially, and there is a simulated elevator for passively traversing toward and away from the center of rotation. The use of each of these experimental apparatuses is described in the following paragraphs.

Research Program

The present ground-based research program is divided into two parts. Initial experimentation involves the exposure of four test subjects to a series of 12 days of testing, during which the subjects will be required to perform various gross and fine psychomotor tasks and general operational tasks while on the rotational facility. A total of 8 hours of testing will be conducted during each of the 12 days.

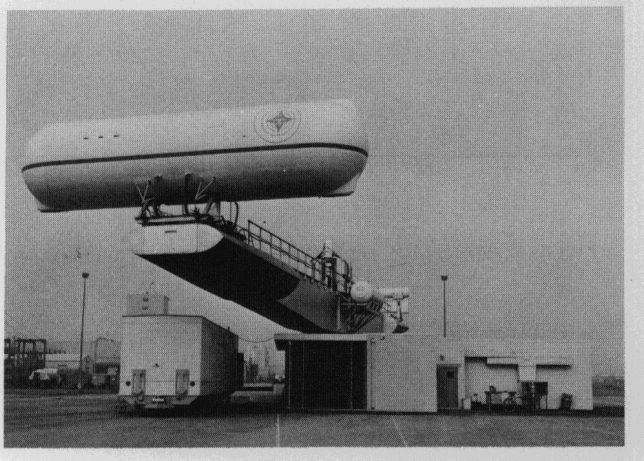


FIGURE 3.—*North American Rockwell rotational test facility.*

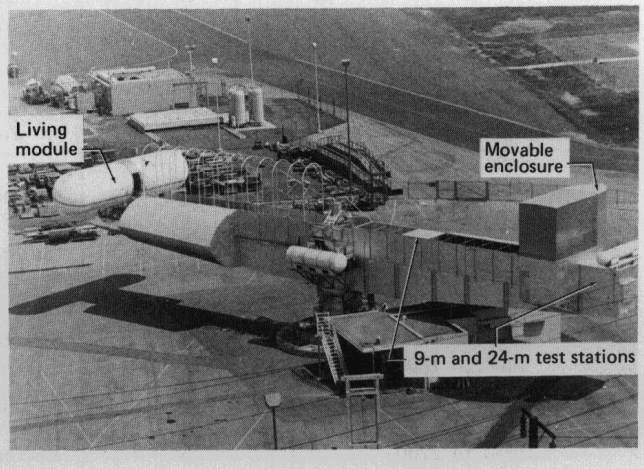


FIGURE 4.—*Modified rotational test facility.*

The second part of this program involves the exposure of four subjects (two participants of the 8-hour tests plus two naive subjects) to 7 continuous days of rotation, during which tests similiar to those of the 8-hour tests will be conducted. The present discussion is limited to the 8-hour tests, since the design of the 7-day test will not be completed until results of the 8-hour tests (now underway) have been analyzed.

The subjects selected for these experiments are employees of the North American Rockwell Corporation, and they are either test pilots or they are engineering personnel with prior experience in human factors research. Each subject has been selected on the basis of his motivation, his medical history and present physical condition, and his degree of susceptibility to motion sickness. The last characteristic was determined at the Naval Aerospace Medical Research Laboratory, using methods established by that laboratory.

Each of the following experiments will be conducted at rotation rates of 3, 4, and 5 rpm to determine variations in performance with rotational rate. Changes in performance as a function of radius will be studied by conducting each experiment at more than one location on the facility. In addition to the experimental measurements dis-

cussed, each subject will wear an electrocardiogram (ECG) telemetry system throughout the tests to monitor that related aspect of the physiological system during the performance of each task.

Crew Mobility

Some of the concerns for crew mobility in artificial gravity are illustrated in figure 5. For example, when a crewmember moves radially in the space base (as in climbing a ladder from one floor level to another), the Coriolis force acts on the body tangentially in the prospin direction for motion toward the spin axis and in the antispin direction for motion away from the spin axis. The crewmember moving radially with a constant velocity will therefore experience a constant tangential force superimposed upon a varying gravity force. The resultant force experienced will continually change in both magnitude and direction. The problem becomes further complicated because his limbs will move at different speeds from his gross climbing speed, and they will be subjected to different and varying Coriolis forces.

A crewmember moving tangentially within the rotating spacecraft will experience changes in his local weight. Increases and decreases in weight will depend on the rate and direction of movement, and the changes are due to varying centrifugal acceleration and Coriolis forces acting on the body in motion. Thus, as illustrated in figure 5, locomotion in the direction of vehicle rotation results in the crewmember feeling heavier, and locomotion against the rotational direction results in a feeling of being lighter. Pure axial motion in either direction would result in no change in weight or extraneous forces, since Coriolis forces are nonexistent. Of course, if the crewmember does not move at all, he will experience only a constant gravity environment.

To study the ability of an astronaut to transfer from one floor level to another in the space base, experiments are being conducted that involve the simulation of climbing a ladder toward and away from the center of rotation. Figure 6 illustrates this simulation. During these experiments the subject is supported by a sling apparatus which is similar to that which has been successfully used in the past for

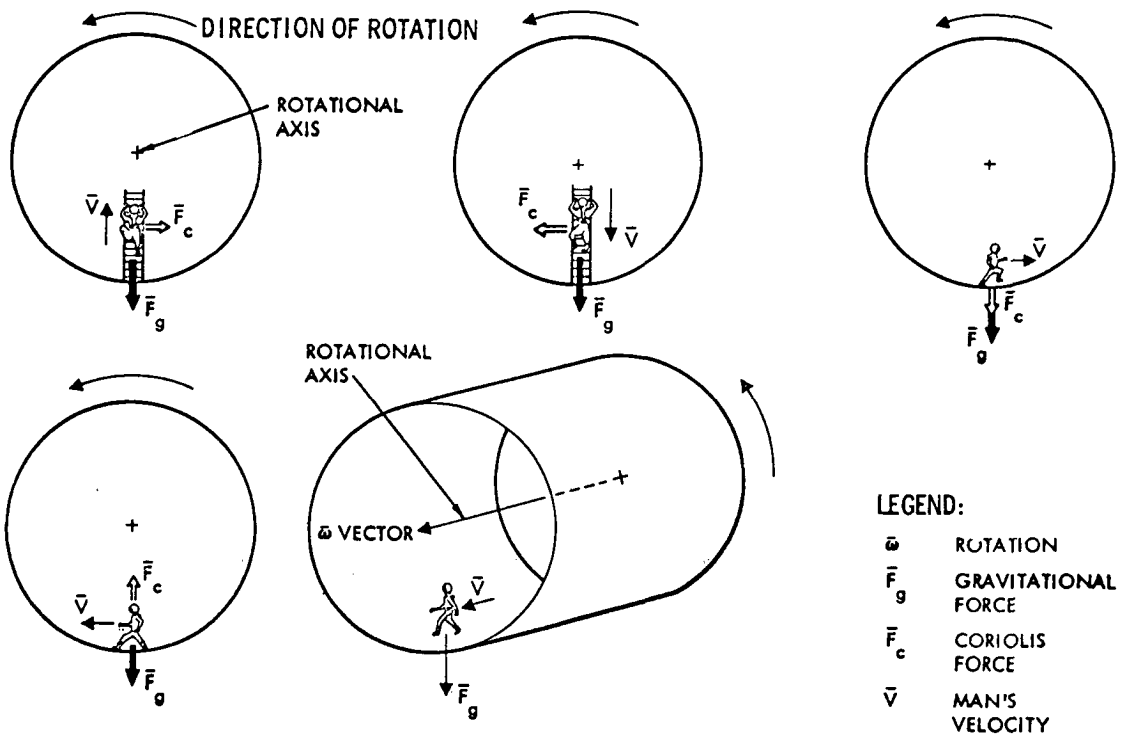


FIGURE 5.—*Inertial reaction forces experienced by man moving in a rotating environment.*

FIGURE 6.—*Ladder-climbing experiment.*

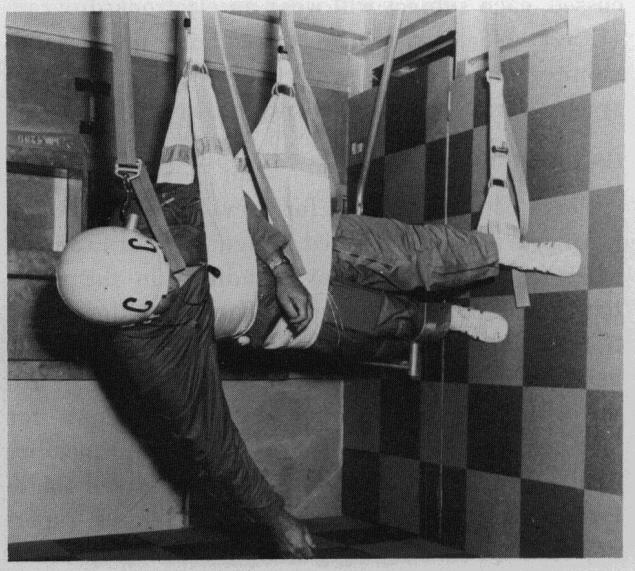


FIGURE 7.—*Tangential walking experiment.*

lunar walking studies. This technique deletes most of the effects of Earth's 1-*g* force from the simulation, and the effects of changing gravity levels and Coriolis forces resulting from radial locomotion are effectively studied.

The ladders have a length of 18 m and are positioned on the rotational facility between radii of 3 m and 21 m. One ladder is used to study radial climbing with the subject facing the direction of rotation, and the other is used for locomotion facing opposite the rotation. Each subject's comments are recorded via a radio transmitter in his helmet, and motion pictures and closed-circuit television record the subject's movement patterns, while he executes a number of excursions up and down each ladder. Each test is repeated while the facility is rotated at 3, 4, and 5 rpm.

The experiment shown in figure 7 serves the purpose of studying the effects of rotation on man's ability to walk tangentially within a rotating spacecraft. The movable enclosure shown in figure 4 is positioned at radii of 6, 12, 18, and 21 m for these experiments, and the facility is rotated at 3, 4, and 5 rpm. In a manner similar to that of the ladder-climbing experiment, each subject is suspended by a sling assembly to nullify the 1-*g* force and to allow the subject's longitudinal body axis to be alined with the artificial-gravity vector. While subjective comments are recorded and while movement is recorded with motion pictures and closed-circuit television, each subject is required to walk the 4.5-m length of the enclosure both with and against the rotational direction. In this way the ease of mobility in each environment is determined. Changes in walking performance are also indicated by the total time required to traverse the 4.5-m distance.

Cargo Handling

To examine the ease with which cargo of significant size and mass may be handled in the rotating environment, the experiment illustrated in figure 8 is performed. Each subject is positioned in the sling apparatus located within the movable enclosure. While subjected to various rotating environments, he moves masses of 1.459 kg (1 slug) from top shelves to lower shelves (and vice versa). During the movement of each mass, it is rotated according to a preprogramed sequence to study the effects of cross-coupled angular acceleration on handling characteristics. In another test, to study the effects of rotation on cargo transport, each subject walks the length of the enclosure while carrying a 1-slug mass. Voice and motion-picture recordings are made

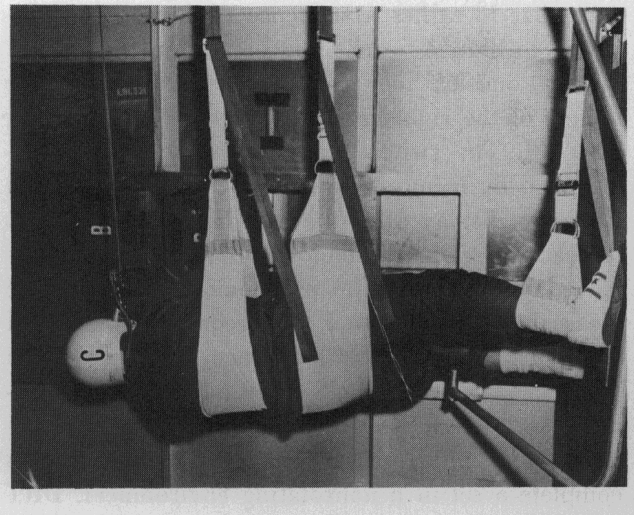


FIGURE 8.—*Cargo-handling experiment.*

of each task, and variations in ease of handling are indicated by the total time required to perform the task. During each experiment the effects of the Earth's gravity on the simulation are deleted by suspending each element of mass by a cable.

Crew Passive Radial Transport

Figure 9 shows the simulated passive radial transport system (or elevator) that is presently being used to study problems relating to the space-base operation of such a device. One potential problem associated with the use of an elevator in a rotating spacecraft is related to rapid changes in hydrostatic pressure within the cardiovascular system when the astronaut is transported at a fast rate. As he is moved, the gravity acting along his body axis changes rapidly, giving an effect similar to that of being subjected to a tilt-table test. The purpose of this experiment is to determine acceptable rates of radial transfer in rotational environments of 3, 4, and 5 rpm.

The procedure for this experiment involves radial movements of the system (with subject) at 0.6, 1.2, and 1.8 m/s. The total distance of transport is 18 m between radii of 21 m and 3 m. Subjective comments made by each subject are recorded, and ECG recordings are monitored during each test. Later tests will be conducted to study the compound effects resulting from programmed head motions of each subject while being transported.

FIGURE 9.—*Crew passive radial transport system experiment.*

Psychomoter Tests

The effects of various rotational environments on fine psychomotor performance are studied by use of three test devices. The first device is the Langley Complex Coordinator (LCC), which has been used successfully during Projects Tektite and Ben Franklin and which is presently being used during the Langley-supported 90-day environmental control and life support system test. This device is shown in figure 10. The test involves the measurement of fine motor control of all four limbs by simultaneously matching a preprogramed sequence of activated lights with corresponding lights controlled by the hands and feet. The time required to perform a series of tests while under the influence of unusual stress is recorded and compared with times of previously performed baseline tests that were conducted under ideal circumstances. In this manner psychomotor performance changes due to the stressful environment may be established. For the purpose of the present experiment, the LCC device is located on the rotational facility and used in the crew living module to study the effects of exposure to rotation on overall psychomotor performance

FIGURE 10.—*Langley complex coordinator.*

and to indicate any degree of adaptation to the environment whereby performance is expected to improve with exposure time.

The other two psychomotor performance measurement devices are a modified Stromberg Dexterity Test and the Decision Response Time (DRT) test. The well-known Stromberg Dexterity Test involves the placing of cylindrical blocks of three different colors into correspondingly colored holes while following an established procedure. The measurement of performance is the time required to complete the test, and these times are compared with baseline times derived from previous trials performed in a nonrotating environment. Since hand and arm motions are required, the use of the Stromberg Dexterity Test permits the study of the combined effects of the environment on psychomotor behavior and on manipulation of small objects under the influence of Coriolis forces. There are two Stormberg Dexterity Test devices located on the facility, one at the crew living module and the other at the nonrotating hub.

The DRT test is an electronically controlled test in which the subject is presented a numbered code that identifies a sequence of four lights presented to him. As each light comes on, the subject presses one of four switches controlled by the fingers of the right hand to turn the corresponding light off. The switch that must be pushed is determined by memorizing the code, which is deleted from the display after the first light is turned off. As each light is turned off, another light is displayed, and so on. The psychomotor performance measurement is the total time required to turn off 96 lights presented in sequence, with the code changing after every 12 lights. Changes in performance are determined by comparing the time required to complete a problem set with the time to complete a set in a nonrotating environment. DRT devices are located at the performance test stations at radii of 9 and 24 m (figs. 4 and 11). There is also a DRT device located at the nonrotating hub, along with a Stromberg Dexterity Test for performance measurements at that station (fig. 12). These devices are used primarily to study the performance changes that occur when a subject moves from a rotating to a nonrotating environment. Concern is for the astronaut's ability to perform required tasks when he moves to the zero-gravity hub of the space base. This simulation is expected to give some indication of whether significant changes in performance do occur.

All three psychomotor performance measurement tests are being used throughout the series of 8-hour

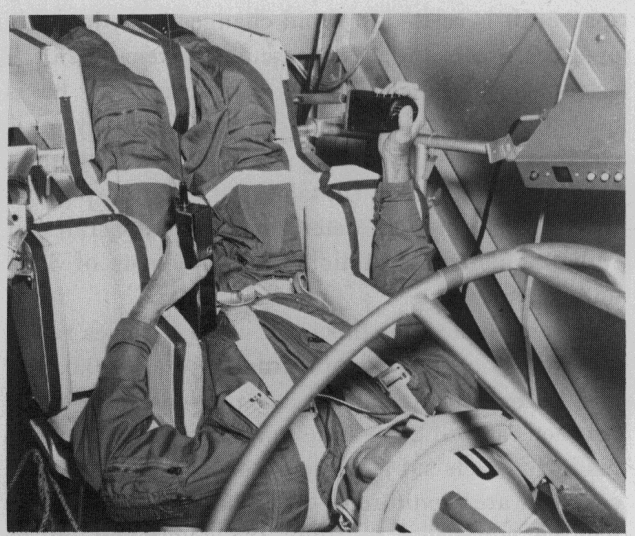


FIGURE 11.—*Decision Response Time device (9-m and 24-m test stations).*

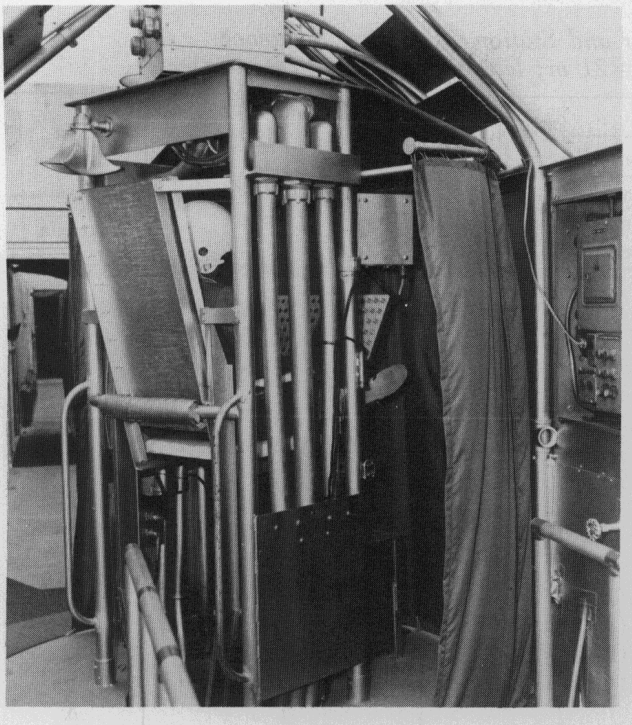


FIGURE 12.—*Nonrotating hub test station.*

experiments to monitor changes in performance due to increases in exposure to rotation and due to changes in rate and radius of rotation.

Ataxia Tests

In addition to the experiments discussed above, at the end of each day's testing a battery of ataxia tests (ref. 6) is performed on each subject. The purpose of these tests is to obtain some indication of the degree of adaptation of each subject to the rotating environment. If the results show that the subject is ataxic, it can be assumed that he is adapting to rotation, and the degree of ataxia can be related to the degree of adaptation.

Test Procedure

The experimental procedure for the series of 8-hour tests is typified in tables 2 and 3. The subjects participating in each test are identified by the letters A, B, C, and D, and the test conditions (rate and radius of rotation) for each day are indicated. Letters in parentheses indicate a subject who is an assistant for the participating subject in that particular experiment.

Testing is initiated in the morning with the reestablishment of baselines on the Langley Complex Coordinator (LCC). The LCC scores during the latter part of the day indicate not only the effects of the environment on psychomotor performance but also the accumulative effects of adaptation when scores are compared on a day-to-day basis. The last test performed at the end of each day is the ataxia test. This test is conducted immediately after the facility comes to rest and before the subjects are allowed to move about excessively in the nonrotating environment.

By comparing the test sequence of one day with another, it can be seen that the psychomotor tests, walking and cargo-handling tests, ladder-climbing tests, and the elevator experiment are randomized from day to day. Tests performed during the morning hours of one day are performed in the afternoon of another day. In this way, the diurnal effects on the experimental results may be eliminated by statistical analysis.

Although not shown in tables 2 and 3, the psychomotor tests at the 9-m and 24-m test stations are performed while the subject is in three different orientations: facing axially, facing in the rotational direction, and facing against rotation. By conducting these experiments in this manner, the significance of relative orientation of an astronaut in a rotating spacecraft is determined. The horizontally situated couches at the 9-m and 24-m test stations are rotated to these positions for this purpose.

CONCLUDING REMARKS

The design of an experimental study to investigate the effectiveness of man in performing everyday tasks in a rotating environment is presented. The 8-hour test series discussed herein is expected to provide sufficient information about the effects on human performance of short-term exposure to rotation so that a more comprehensive experiment can be conducted in which subjects will be exposed to 1 week of continuous rotation. During this future experiment, efforts will be made to duplicate as closely as possible a routine of performance tasks similar to those expected of a crew within a rotating spacecraft. Emphasis will be placed on the determination of effects of long exposure on adaptation to the environment, the habitability aspects of the environment, and the general performance capabilities of the subjects.

TABLE 2.—*Test Run 1: Subject Order and Station Allocation Sequence (Rotation rate=4 rpm; walking-wall radius=21 m; letters A to D indicate subjects)*

Prerotation	Psychomotor tests					Walking and cargo handling	Ladder climbing	Elevator	Post-rotation
LCC baseline	Hub	9 m	24 m	Cabin	Cabin				
Cabin	DRT LCC	DRT Dext.	DRT Dext.	LCC	Stand. Stromberg Dext.				Ataxia
C D A B									
	A	B	C	D					
	B	C	D	A					
	D	A	B	C					
	C	D	A	B					
						A (B)	C (D)		
						B (A)	D (C)		
						C (D)	A (B)		
						D (C)	B (A)		
					A (B)			C (D)	
					B (A)			D (C)	
					C (D)			A (B)	
					D (C)			B (A)	
									A
									B
									C
									D

TABLE 3.—*Test Run 6: Subject Order and Station Allocation Sequence (Rotation rate=3 rpm; walking-wall radius=6 m)*

Prerotation	Psychomotor tests					Walking and cargo handling	Ladder climbing	Elevator	Post-rotation
LCC baseline	Hub	9 m	24 m	Cabin	Cabin				
Cabin	DRT LCC	DRT Dext.	DRT Dext.	LCC	Stand. Stromberg Dext.				Ataxia
A B C D									
					A (D)			B (C)	
					D (A)			C (B)	
					B (C)			A (D)	
					C (B)			D (A)	
						A (D)	B (C)		
						D (A)	C (B)		
						B (C)	A (D)		
						C (B)	D (A)		
	C	D	A	B					
	D	A	B	C					
	B	C	D	A					
	A	B	C	D					
									C
									D
									A
									B

REFERENCES

1. STONE, RALPH W., JR.; AND PILAND, WILLIAM M.: Potential Problems Related to Weightlessness and Artificial Gravity. NASA TN D–4980, 1969.
2. STONE, R. W., JR.; PILAND, W. M.; AND LETKO, WILLIAM: Certain Aspects of Onboard Centrifuges and Artificial Gravity. Fourth Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–187, 1970, pp. 331–343.
3. BEAUCHAMP, G. T.: Adverse Effects Due to Space Vehicle Rotation. Advances in the Astronautical Sciences, vol. 8, Horace Jacobs, ed., Plenum Press, 1963, pp. 122–125.
4. STONE, RALPH W., JR.; AND LETKO, WILLIAM: Some Observations on the Stimulation of the Vestibular System of Man in a Rotating Environment. The Role of the Vestibular Organs in the Exploration of Space, NASA SP–77, 1965, pp. 263–278.
5. GRAYBIEL, ASHTON; KENNEDY, ROBERT S.; GUEDRY, FRED E., JR.; MCLEOD, MICHAEL E.; COLEHOUR, JAMES K.; MILLER, EARL F. II; KNOBLOCK, EDWARD C.; AND MERTZ, WALTER: The Effects of Exposure to a Rotating Environment (10 rpm) on Four Aviators for a Period of 12 Days. The Role of the Vestibular Organs in the Exploration of Space, NASA SP–77, 1965, pp. 295–337.
6. GRAYBIEL, ASHTON; AND FREGLY, ALFRED R.: A New Quantitative Ataxia Test Battery. Acta Oto-Laryngol., vol. 61, 1966, pp. 292–312.

Locomotion in a Rotating Environment

WILLIAM LETKO*
NASA Langley Research Center

SUMMARY

Plans are being formulated for establishing a manned space station in Earth orbit in the near future. Its purpose is to provide a test facility for a large number of experiments that can best be performed in space or that can only be performed in space. The space station is to have a lifetime of several years and will be occupied by different crews, each of which will spend relatively long periods of time in the station before a different crew is brought aboard. The configuration of the space station, therefore, needs to be such as to best utilize and maintain the capabilities of the crew. The configuration of the station should also allow for the performance of the large variety of experiments contemplated.

In order to provide the best possible space station configuration, a number of space and ground-based experiments are to be undertaken to provide design data for the operation of space stations. In this connection, the initial experiments in space planned for Skylab I will include experiments on man's habitability requirements and performance capabilities in zero gravity. Also, ground-based experiments are to be performed and later space experiments are being considered to examine the efficacy of providing artificial gravity by rotation of the space station.

The Langley Rotating Space Station Simulator is one of the ground-based facilities that is being used to obtain data on man's performance in a rotating environment. At present the facility is being used to assess the effects of rotation on man's walking capabilities on circular and flat walls at g-levels from 0.05 to 0.75 g.

This paper reviews some of the preliminary results.

SIMULATOR DESCRIPTION AND TESTS

A photograph of the Langley Rotating Space Station Simulator is shown in figure 1. The facility consists of a circular platform of 12-m diameter with a 1.8-m vertical wall at the periphery. The wall simulates the floor of a rotating vehicle upon which subjects suspended horizontally can walk and otherwise perform as they would in a rotating vehicle. The subjects, as can be seen in the figure, are suspended by a system of cables and harnesses attached to a trolley that is free to move radially along an overhead boom. The boom is servo-mechanized so that it tracks the subject and allows him to move relative to the wall, free of the effects of boom inertia and wind drag.

In the walking experiments the subjects were

FIGURE 1.—*Langley rotating space station simulator.*

* In Mr. Letko's absence the paper was presented by Randall Harris, Langley Research Center, NASA.

asked to walk at their own pace as smoothly as possible. The simulator rotation as viewed from above was always in the counterclockwise sense. The subjects walked both in and opposite to the direction of simulator rotation, thus increasing or decreasing the nominal levels of gravity by an amount that depended on their walking rate. The subjects were required to walk on both the circular floor and a flat segmented floor. The rotational rate of the simulator was varied from about 3 to 10.5 rpm, corresponding to g-levels from 0.05 to 0.75 g at the subject's feet.

In order to assess the subject's walking performance, measurements were made of the vertical floor reactions and fore-and-aft shear forces generated by the subject's feet. These floor reactions or forces were measured using two 15-by-30-cm rectangular plates attached to strain-gage balances mounted on the walls of the simulator. Because of strain-gage balance limitations, the vertical reaction force and the fore-and-aft shear force had to be determined individually in separate tests. The force plates were located tangentially along the wall so that the distance between centers was 66 cm while the lateral displacement of the centers was 15 cm, as can be seen in figure 2.

The distance between the plate centers was the same on both the circular floor and the flat segmented floor. The flat segmented floor was made by mounting three sheets of plywood, each 2.4 by 1.2 m, on the circular floor as indicated in figure 3. The orientation of the force plates for clockwise and counterclockwise walking on the flat segmented floor is also shown in figure 3.

In addition to the force measurements, subjective comments were elicited and motion pictures were obtained to provide additional information on walking in a rotating environment. Subjective opinions were obtained from the four subjects for whom floor reactions were measured and for two additional subjects who had considerable experience walking in reduced-gravity simulators.

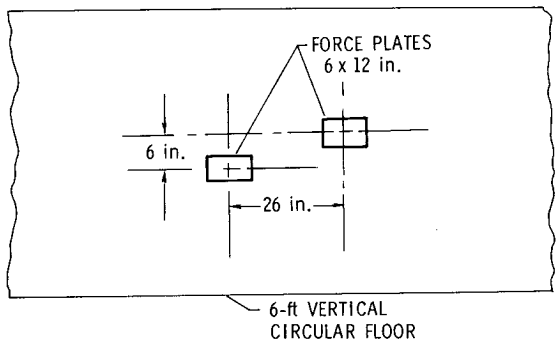


FIGURE 2.—*Sketch showing displacement of force plates on simulator floor.*

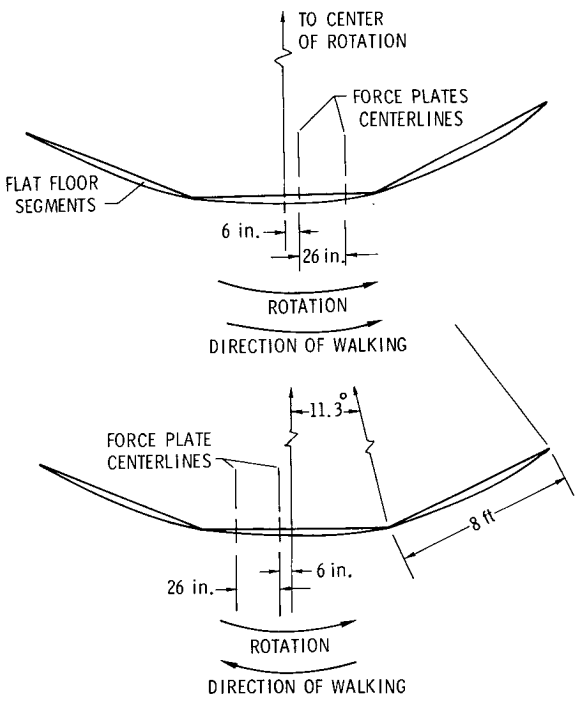


FIGURE 3.—*Force plate orientation for walking in and opposite to the direction of simulator rotation on the segmented flat floor.*

RESULTS AND DISCUSSION

The subjective results of this investigation confirm those of an earlier investigation in the same simulator and reported in references 1 and 2. The aforementioned walking experiments were performed in the same simulator, using the circular floor only. They were repeated herein because, in the earlier tests, the subject support boom was not servomechanized, and there was some uncertainty as to the extent to which the walking performance was affected by boom friction and inertia.

The following discussion describes the general results of the present investigation. At an artificial g-level of about 0.1 g, it was difficult to initiate

and terminate the walk, and thus the time to reach a normal walking rate was increased. It was possible to walk counterclockwise when the station rate of rotation provided a nominal 0.05 g. However, at this g-level, when walking opposite to the direction of station rotation, the subjects would tend to rise and float free of the floor. At g-levels of 0.3 and above, the subjects reported sensations of leg and body heaviness; these sensations were quite disturbing at g-levels of 0.5 and 0.75. The subject reported that walking at g-levels of 0.167 and 0.3 was easier than at the other g-levels tested.

Generally, it was found that walking on the flat segmented floor was quite similar to walking on the curved floor. However, at g-levels near 0.1 g it appeared more difficult to initiate the walk and stop than was found for the circular floor. This tendency was reduced as the g-level was increased. The difficulty of initiating and stopping the walk on the flat floor may be due to the fact that the local gravity vector is not perpendicular to the floor except at the center of the flat floor segments, as shown in figure 4. As can be seen in the figure, a subject standing on the floor would be subjected to a component of gravity acting opposite to the direction of walking when on one side of the perpendicular and acting in the direction of walking when on the other side of the perpendicular. From vestibular and proprioceptive cues it appears the subjects should have felt that they were walking uphill and then downhill. Motion pictures showed them walking alined with the local gravity vector. Actually, however, the subjects reported that they felt as if they were walking on the level. The reason for this finding is not completely understood at this time.

As mentioned earlier, in addition to the subjective data, floor reaction forces were also obtained to help assess the effects of artificial gravity on walking performance. During walking, vertical fore-and-aft shear and lateral forces, as well as lateral torques, are generated by the feet. For the purpose of this investigation, only the vertical forces and fore-and-aft shear forces are considered.

The vertical forces and fore-and-aft shear forces generated by one foot while walking in the normal 1-g environment are shown in figure 5. The figure shows the forces in percentage of subject weight plotted against time from the moment of heel con-

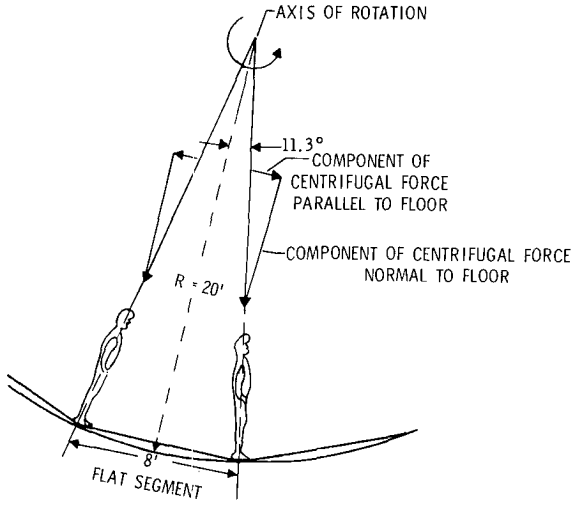


FIGURE 4.—*Sketch showing components of the local gravity vector (centrifugal force) parallel and perpendicular to one of the flat floor segments.*

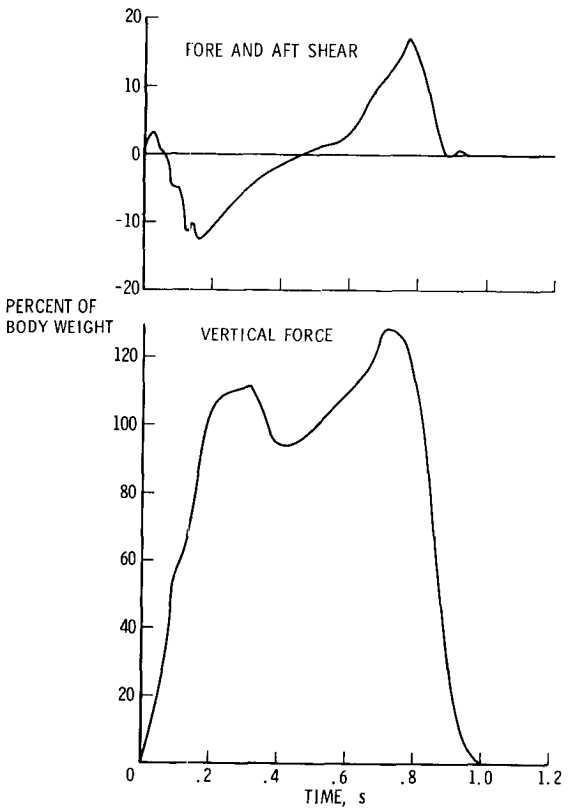


FIGURE 5.—*Time history of vertical force and fore-and-aft shear generated by one foot of a subject walking in the normal 1-g environment. Note that the vertical scale for the shear force is twice that for the vertical force.*

tact to toe liftoff. A positive vertical force is one acting upward on the foot, while a positive shear force is one acting on the foot in the direction of walking. The data of figure 5 are similar to those obtained in references 3 and 4, for example. The vertical force profile has the characteristic double hump, while the lateral shear is initially negative because of deceleration of the striking foot and then becomes positive as the foot pushes backward. It should be pointed out that the data presented herein are for one subject. Consideration should be given the fact, as indicated in reference 3, that although the force profiles for different individuals are generally the same, the magnitude of the forces varies with weight, body build, and ratio of leg length to length of stride, as well as walking cadence. Nevertheless, an examination of the data for all subjects for whom force measurements were taken indicated that the trends in the data caused by varying the test parameters can be determined from data for a single subject.

Figures 6 and 7 show the effect of varying the artificial g-level on the vertical and shear forces generated by one foot of a subject walking on the circular floor. The data on figure 6 are for walking opposite to the direction of rotation (clockwise) and on figure 7 for walking in the direction of rotation (counterclockwise). Both figures 6 and 7 show that the vertical force and fore-and-aft shear forces decrease with a decrease in g-level, as expected. Comparison of the vertical floor reaction data of reference 4, shown in figure 8, obtained for one subject in an airplane flying trajectories to produce g-levels from 0.17 to 1.0 g indicates the same general effects of reducing g-levels as found in the present investigation.

The consequences of the direction of walking in the rotating simulator can be seen by comparing the vertical force and fore-and-aft shear data of figures 6 and 7. The vertical force and fore-and-aft shear force for clockwise walking (opposite the direction of simulator rotation) are considerably lower than for counterclockwise walking. The fore-and-aft shear forces are reduced to nearly zero for walking opposite to the direction of rotation at

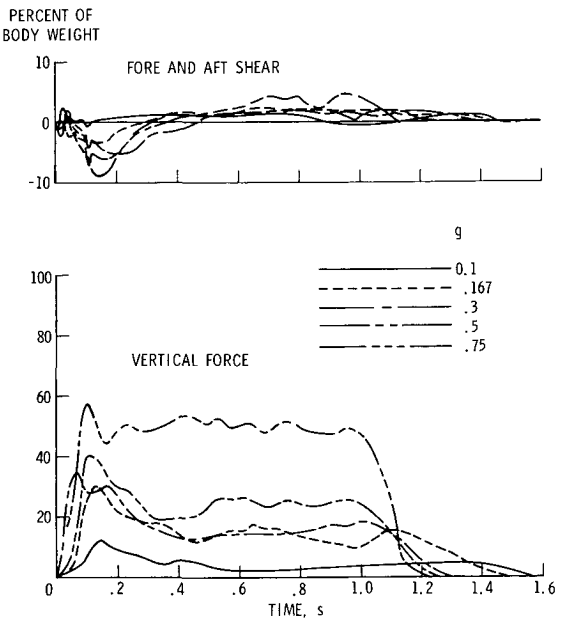


FIGURE 6.—*Time history of vertical force and fore-and-aft shear generated by one foot of a subject walking on the circular floor of the rotating simulator, opposite the direction of rotation. Note that the vertical scale for the shear force is twice that for the vertical force.*

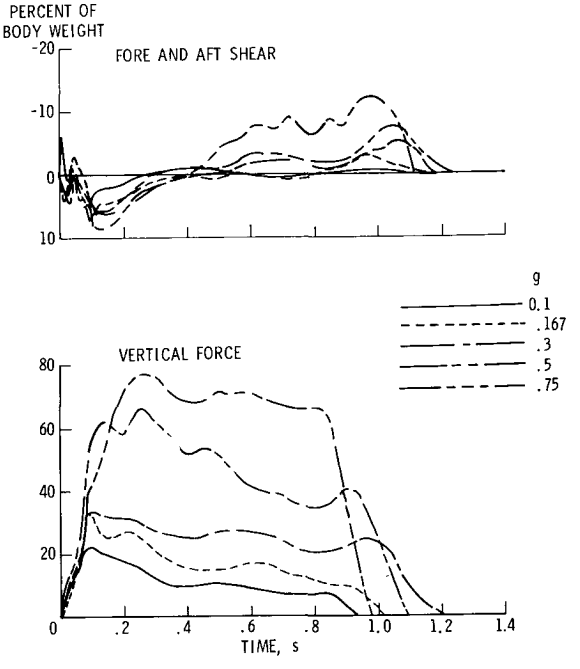


FIGURE 7.—*Time history of vertical force and fore-and-aft shear generated by one foot of a subject walking on the circular floor of the rotating simulator, in the direction of rotation. Note that the vertical scale for the shear force is twice that for the vertical force.*

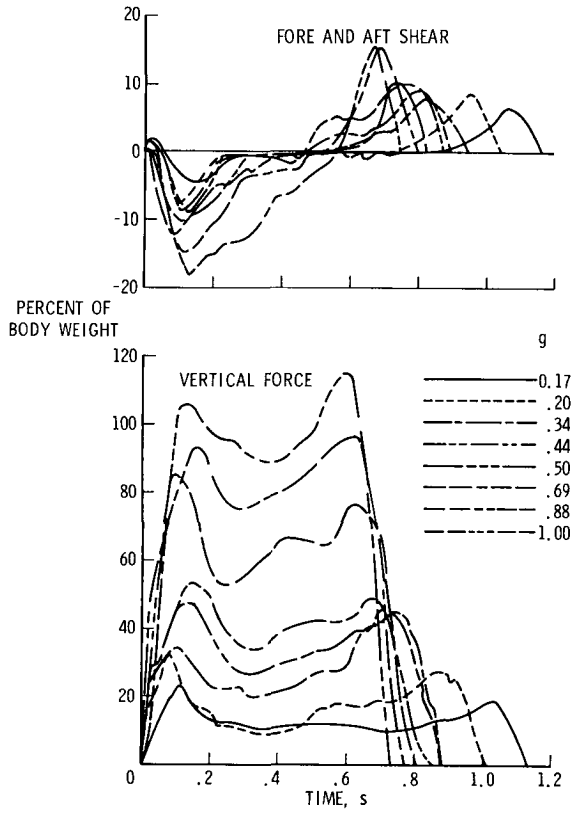


FIGURE 8.—*Time history of vertical force and fore-and-aft shear generated by one foot of a subject walking in an aircraft flying trajectories producing various g-levels. Note that the vertical scale for the shear force is twice that for the vertical force. Data reproduced from reference 4.*

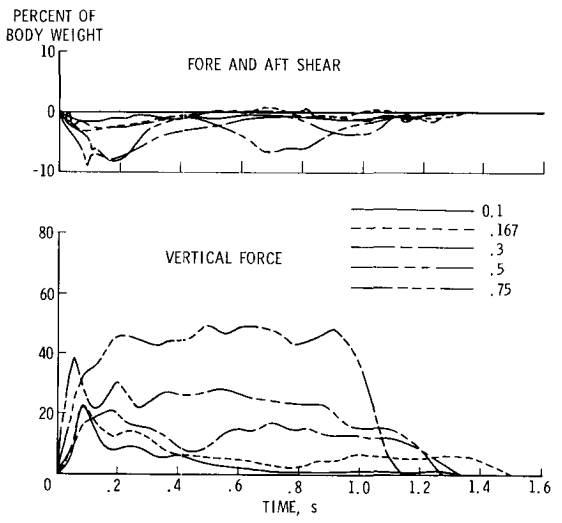


FIGURE 9.—*Time history of vertical force and fore-and-aft shear generated by one foot of a subject walking on the segmented flat floor of the rotating simulator, opposite the direction of rotation. Note that the vertical scale for the shear force is twice that for the vertical force.*

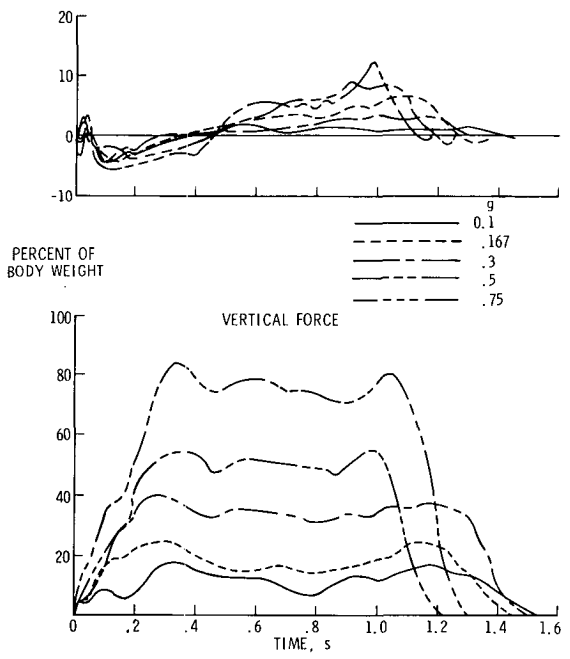


FIGURE 10.—*Time history of vertical force and fore-and-aft shear generated by one foot of a subject walking on the segmented flat floor of the rotating simulator, in the direction of rotation. Note that the vertical scale for the shear force is twice that for the vertical force.*

0.1 *g*, substantiating the subjective opinion that walking is difficult at this *g*-level, including the tendency to float free of the floor, especially when walking opposite the direction of rotation.

Figures 9 and 10 present the effects of changing the artificial *g*-level on the vertical force and fore-and-aft shear force generated while walking on the flat segmented floor. The effects of reducing *g*-level and the effects of direction of walking are generally similar to those obtained for the circular floor.

CONCLUDING REMARKS

On the basis of the results of this investigation of the effects of artificial *g*-levels on walking performance, the following remarks are indicated. The subjects found that they were able to walk at levels as low as 0.1 *g*. At 0.05 *g*, walking opposite to the direction of rotation caused the subjects to lose

traction, and they floated free of the floor. At low g-levels there were some indications that the initiation of walking on the flat floor was more difficult than on a circular floor. A more detailed examination of the data is required to confirm this opinion.

In general, the subjects felt that, for the simulator used (12-m diameter), walking at g-levels of 0.167 and 0.3 was easier than walking at the other g-levels tested.

REFERENCES

1. Stone, Ralph W., Jr.; Piland, William M.; and Letko, William: Certain Aspects of Onboard Centrifuges and Artificial Gravity. Fourth Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–187, 1970, pp. 331–343.
2. Hewes, Donald E.: Status Report on Recent Langley Studies of Lunar and Space Station Self-Locomotion. Paper presented at the 24th Aerospace Medical Panel Meeting, AGARD, Brussels, Belgium, Oct. 24–27, 1967.
3. Cunningham, D. M.: Components of Floor Reactions During Walking. Prosthetic Devices Research Project, Institute of Engineering Research, University of California, Berkeley, California, Nov. 1950.
4. Beebe, Dennis Edward: Force Analysis of Walking at Reduced Gravity. Thesis, School of Engineering, Institute of Technology, Wright-Patterson Air Force Base, Dayton, Ohio, Aug. 1964.

SESSION III

Chairman: **RALPH W. STONE, JR.**
NASA Langley Research Center

Some Physiological Aspects of Artificial Gravity

D. B. CRAMER and ASHTON GRAYBIEL
Naval Aerospace Medical Research Laboratory

SUMMARY

On the assumption that man is a gravity-sensitive creature, lengthy exposure to weightlessness is likely to produce prominent alterations in some aspects of his bodily functions. Artificial gravity represents a logical solution to this problem, but due to the engineering complexity and substantial cost of producing artificial gravity, ground-based studies are needed to define its potential usefulness aloft. The effects of increasing artificial-gravity exposure on four aspects of physiological fitness are examined in four young men who, prior to exposure, were deconditioned with bed rest and water immersion. The four aspects of physiological fitness are orthostatic tolerance, exercise tolerance, forearm endurance, and maximum strength. Orthostatic tolerance was sharply reduced by deconditioning and was substantially improved by walking in simulated lunar gravity (1/6 *g*) for 2.5 hours daily for 7 days or by walking in 1/2 *g* and 1 *g* for 1 hour daily for 3 days. Exercise tolerance was also sharply reduced by deconditioning but did not significantly improve with increasing *g*-exposure. Walking in 1 *g* for 1 hour daily for 3 days raised exercise tolerance only a little above the low produced by deconditioning. Forearm endurance and maximum strength were relatively unaffected by deconditioning and subsequent *g*-exposure. Explanations for these results are proposed, including a discussion of ground-based experimentation to estimate the usefulness of artificial gravity aloft.

INTRODUCTION

Man, in all likelihood, is a gravity-sensitive creature; therefore, lengthy exposure to accelerations substantially greater or less than that of Earth gravity will produce measurable alterations in some aspects of his bodily functions. This assertion is supported by some existing evidence. It has often been shown in laboratory animals that exposure to acceleration levels greater than 1 *g* produces significant physiological alterations (refs. 1 to 3). Similarly, simulations of weightlessness have also produced readily detectible alterations of physiological function (refs. 4 to 6), and in the brief exposures to weightlessness encountered thus far in our space program, transient alterations have been documented (refs. 7 and 8). If exposures to both weightlessness and hypergravity are capable of producing readily detectible alterations in bodily form and function, then a high probability exists that man is indeed gravity sensitive.

The physiological alterations related to 0-*g* exposure to date have all been transient. However, if the exposure to weightlessness were long enough, readaptation to the 1-*g* environment upon returning to Earth would perhaps not be so quickly accomplished. To keep the astronaut fit in space, capable of safe reentry and of readily reconditioning to 1 *g*, longer space missions will employ a variable combination of countermeasures and reconditioning techniques. Artificial gravity is clearly a logical antidote for man's sensitivity. However, due to the engineering complexity and overall expense of creating artificial gravity in any part of the spacecraft, substantial ground-based studies are needed to define and justify its use.

The present experiment was designed to examine the effects of increasing artificial-gravity exposure on four aspects of physiological fitness: orthostatic tolerance, exercise tolerance, forearm endurance, and maximum strength. The subjects were healthy young men who, prior to exposure, were decondi-

tioned by a combination of bed rest and water immersion. Artificial-gravity magnitudes were 1/6 *g*, ½ *g*, and 1 *g*, with exposures ranging from 30 minutes to 8 hours per day for up to 7 days. It was hypothesized that deconditioning would have substantially different effects on the four selected parameters of physiological fitness; likewise, artificial-gravity exposure in the deconditioned subjects should also have substantially different effects on the same fitness parameters, probably varying both in time course and magnitude.

METHODS

Subjects

Four healthy male college students were selected on the basis of a rigorous physical examination and interviews with the research staff. To be considered to have acceptable cardiovascular function, each subject had to display:

1. A negative medical history for any prior significant cardiorespiratory illness.
2. A normal physical examination.
3. A normal resting 12-lead electrocardiogram (ECG).
4. A normal radiographic cardiac series.
5. A normal exercise-tolerance test in which the subject must run for at least 5 minutes on a level treadmill traveling at or greater than 9.6 km/h without his heart rate reaching 180 beats per minute.
6. A normal postexercise ECG with no ischemic changes.
7. Normal orthostatic tolerance as measured with lower-body-negative pressure (LBNP), in which the subject must show the typical blood-pressure and pulse changes with a grayout endpoint at greater than 5 minutes at 70 mm Hg subatmospheric.

Inasmuch as the artificial gravity was generated on a centrifuge at a 3-m radius with angular velocities ranging from 8 rpm to 15 rpm, the incidence of motion sickness was a considerable likelihood. In the interviews, only those subjects were selected who had gained some previous experience with motion sickness in a rotating environment; by using experienced subjects, the incidence of motion sickness could hopefully be reduced. The subjects lived inside the laboratory for the entire experiment and were paid $35 per day for their participation.

Ambulatory Control Periods

The experiment began with a 5-day ambulatory control period. During this time the four subjects were not permitted to leave the laboratory but were encouraged to move about in the building, spending much of their day either standing or sitting. On the first day the subjects were briefed on all of the experimental equipment and each was given an opportunity to familiarize himself with the experimental tests and procedures. For the next 4 days, control data were collected on each of the four fitness tests. Each subject received a second physical examination during the control period, and throughout the experiment routine clinical tests and vital signs were recorded on a daily basis to detect any insidious illness.

Deconditioning

The deconditioning period lasted 7 days and consisted of a combination of bed rest and water immersion. The subjects spent 8 hours per day immersed in water and the remaining 16 hours of the day at strict bed rest. The water-immersion deconditioning consisted of suspending the subject on a nylon litter in about 2.5 m^3 of normal saline, so that only his head was above the surface. The water temperature is critical (ref. 9) and was held to 35.28±0.55 °C as measured 5 cm under the subject's back. To minimize the hydrostatic pressure on the subject's chest, the nylon litter was adjusted to keep no more than 1 cm of water above the subject's chest at full inspiration. While at bed rest each subject was allowed the use of a small pillow and to lift himself on one elbow to eat. Micturition and defecation were accomplished with a bedpan, and constipation, when it did occur, was treated with stool softeners. The diet was not controlled and the subjects were allowed to select whatever they desired from the hospital kitchen. Intake and output estimates were kept, but, in view of the uncontrolled diet, any metabolic conclusions would be tenuous. Movement about the laboratory was accomplished with a cart capable of mechanically lifting the subject on and off the centrifuge while always keeping him horizontal.

Artificial Gravity

The artificial gravity was generated on a centrifuge. In this process the subject was kept horizontal in a fiberglass body mold so that the long axis of the cardiovascular system remained null to the gravity vector but always remained alined with the centripetal vector produced by rotation. The fiberglass mold was constructed to support the subject's weight vertically, and simultaneously permit free movement in the horizontal plane at the knees and hips. The mold was then supported on air bearings and counterbalanced so that the subject could readily walk on the centrifuge wall, which was located at a 3-m radius. A given level of centripetal acceleration equal to the desired level of artificial gravity was then imparted to the subject's center of mass by rotating the centrifuge. Due to the short radius, there was a substantial *g*-gradient along the man. The centrifugal force at his feet was about 40 percent greater than the force applied to his center of mass; similarly, the centrifugal force at his head was about 40 percent less than that of his center of mass. Any subtle effects of *g*-gradients are not presently known, but rather large *g*-gradients have been reported to be well tolerated for at least short periods of time (ref. 10). To generate the 1-*g* level on the centrifuge would have involved rotation at 21 rpm, and at this angular velocity the incidence of motion sickness is quite likely. For this reason the 1-*g* level was obtained simply by having the subject remain erect and walk slowly on a level treadmill.

Fitness Parameters

Orthostatic Tolerance

A lower-body-negative-pressure tank was used to measure orthostatic tolerance. In this device an airtight, soft-rubber seal was made at the level of the diaphragm, and a subatmospheric pressure of 70 mm Hg was maintained on the lower extremities until the subject reported grayout. During this time blood pressure, pulse rate, and the precordial ECG were continuously monitored by an attendant physician. The time in minutes until the subject reported a dimming or reduction in his peripheral vision was taken as the measurement of orthostatic tolerance.

Exercise Tolerance

During the ambulatory-control period the speed of the treadmill was adjusted so that the subject's heart rate reached a steady 180 beats per minute in about 7 minutes. This same speed was then used for all subsequent exercise-tolerance tests, and the time in minutes to reach the steady heart rate of 180 beats per minute was taken as the measurement of exercise tolerance.

Forearm Endurance

The aim of the forearm-endurance test was to determine if the capability of a given muscle group to perform anaerobic work would in any way be altered by the deconditioning and subsequent artificial-gravity exposure. After the maximum strength determination was made, a blood-pressure cuff was then placed around the dominant arm and inflated to 200 mm Hg. Once the cuff was inflated, the subject squeezed the dynamometer every 2 seconds to 50 percent of his maximum strength until the ischemic pain became unbearable. The length of time in minutes the subject could squeeze the dynamometer every 2 seconds to 50 percent of his maximum was taken as the measurement of forearm endurance.

Maximum Strength

In the maximum strength test the subject was asked to squeeze a hand dynamometer as hard as he could with his dominant hand. The maximum of three attempts was chosen as his maximum strength, expressed in kilograms.

Procedure

The experimental procedure is outlined in table 1. During the ambulatory control period, a determination of the four fitness tests was made on each of four days subsequent to the first. The control period was followed by 7 days of water immersion and bed rest. It was essential to document the degree of this deconditioning, and the four tests were performed once at the end of the deconditioning period. Their degree of deconditioning having been established, the four subjects were divided into two groups of two subjects each. The subjects in group I were kept actively walking in 1/6 *g* for 2.5 hours per day, while the group II subjects were exposed to 1/6 *g* for 2.5 hours per day, dur-

TABLE 1.—*Length and Order of Experimental Periods*

Day	Group I	Group II
1–5	Ambulatory control period	Ambulatory control period
6–13	Deconditioning	Deconditioning
14	Test period	Test period
15–21	Walking in 1/6 *g* for 2.5 hours daily	Motionless exposure to 1/6 *g* for 2.5 hours daily
22–24	Test period	Test period
25–27	Deconditioning	Walking in 1/2 *g* for 1/2 hour daily
28	Test period	Test period
29–31	Walking in 1/2 *g* for 1 hour daily	Walking in 1 *g* for 1 hour daily
32	Test period	Test period
33–35	Gradual ambulation	Gradual ambulation
36	Test period	Test period
37–50	Terminal ambulatory control	Terminal ambulatory control
51	Terminal test period	Terminal test period

ing which they rested quietly or slept. At the end of the 7 days the tests were repeated on the subjects to determine any alterations produced by 1/6 *g*. Following these determinations, the subjects passively exposed to 1/6 *g* were exposed to 1/2 *g* for 30 minutes per day for 3 days, during which time they spent the entire half hour walking on the wall of the centrifuge. Group I underwent further deconditioning during the same 3-day period. The results of further deconditioning of group I and of the 1/2-*g* exposure of group II were then measured. Group I then began walking in 1/2 *g*. Since group II had failed to display significant reconditioning in their 1/2-*g* exposure, the gravity level was increased to 1 *g* and the walking time to 1 hour daily for 3 days. At the end of this time only two of the tests were performed because forearm endurance and maximum strength were both at or above earlier control levels. Both groups then started a period of gradual ambulation, after which test results were again noted. The subjects were then released to return to their routine activities at a local university. After 2 weeks they returned for one final determination of the four selected parameters of physiological fitness.

RESULTS

In the experimental hypothesis two things were held likely to occur. In the first case it was expected that the water immersion and bed rest would have substantially different effects on the four selected parameters of physiological fitness. In the second case it was similarly supposed that artificial gravity would also have substantially different effects on the same parameters, probably varying both in time course and magnitude. The data obtained in this probe largely support this hypothesis. Table 2 displays the actual endpoints as they were collected. Since the endpoints of the four physiological parameters were not all expressed in the same units and since the subjects all had different control values, the changes induced by various *g*-exposures were not readily comparable. If, however, the individual endpoints were all expressed as percentages of their particular control values, then the changes induced by artificial gravity would be more apparent. Figure 1 displays the four physiological parameters expressed in terms of their control values and plotted against increasing *g*-exposure.

Control

The mean control time in minutes until the subject reported grayout in the LBNP tank for the four subjects on the first day of testing (day 2) was 14.5 minutes, and this rose gradually to a mean of 16.0 minutes on day 5 of the ambulatory-control period. Earlier experience with the LBNP technique in this laboratory has established that LBNP exposure per se is a conditioning stimulus capable of increasing orthostatic tolerance when used on a daily basis. The need, however, to establish accurate ambulatory-control baseline data necessitated several repeated measurements, so that four determinations were chosen to provide accurate control data, with minimal conditioning having taken place.

The mean time to endpoint in the exercise-tolerance test for the subjects during the 4-day ambulatory-control-test period was 5.52 minutes. This mean fell from 6.89 minutes on day 2 (3 subjects*) to 5.21 minutes on day 5 (4 subjects), the first day having been used for familiarization. For this test this is a common pattern; subjects usually do slightly better initially and then plateau before the effects of training become evident.

*Data for subject Da on second and third days not available because of technical failure of the treadmill.

TABLE 2.—*Endpoint Physiological Fitness Data for Each Subject*

Group I										Group II								
Test		Orthostatic Tolerance (LBNP*)		Exercise Tolerance*		Forearm Endurance*		Maximum Strength (kg)			Orthostatic Tolerance (LBNP*)		Exercise Tolerance*		Forearm Endurance*		Maximum Strength (kg)	
Day	Subject Mode	R	Du	S	Du	S	Du	S	Du	Mode	S	Da	S	Da	S	Da	S	Da
1	Ambulatory control			familiarization runs						Ambulatory control			familiarization runs					
2		8	10	7.00	5.00	1.17	1.90	70	46		20	20	8.67	.....	1.83	1.90	60	64
3		9	6	5.83	5.00	1.83	2.07	54	44		16	20	4.00	.....	1.93	2.47	62	50
4		10	9	5.42	3.00	1.73	1.93	62	44		20	20	6.25	6.00	2.40	2.43	64	64
5		12	12	5.33	3.42	1.87	2.07	54	44		20	20	6.75	5.33	1.97	2.53	66	62
6	Deconditioning									Deconditioning								
14	Testing	6	5	2.33	1.00	1.47	1.70	62	50	Testing	10	12	3.00	2.75	1.87	2.23	70	64
15	Walking in 1/6 *g* for 2.5 hours daily									Motionless exposure to 1/6 *g* for 2.5 hours daily								
22	Testing	7	11	2.83	1.75	1.80	1.97	70	44	Testing day	5	17	1.67	2.42	2.20	2.57	66	72
23		7	8								9	13						
24		8	10															
25	Deconditioning									Walking in 1/2 *g* for 1/2 hour daily								
26																		
27																		
28	Testing day	6	6	2.00	0.83	1.70	2.00	66	44	Testing	11	10	1.67	1.33	2.57	2.83	64	60
29	Walking in 1/2 *g* for									Walking in 1 *g* for								
30	1 hour daily									1 hour daily								
31																		
32	Testing day	8	11	1.83	1.00					Testing day	20	20	3.12	2.17				
33	Gradual ambulation									Gradual ambulation								
34																		
35																		
36		12	13	3.75	2.00	2.67	2.43	74	48		20	20	2.13	2.08	2.73	2.43	70	74
37	Terminal ambulatory control									Terminal ambulatory control								
51		14	9	5.92	3.25	2.30	2.43	70	50		20	20	4.37	1.58	2.53	2.93	72	64

*Minutes to prescribed endpoint

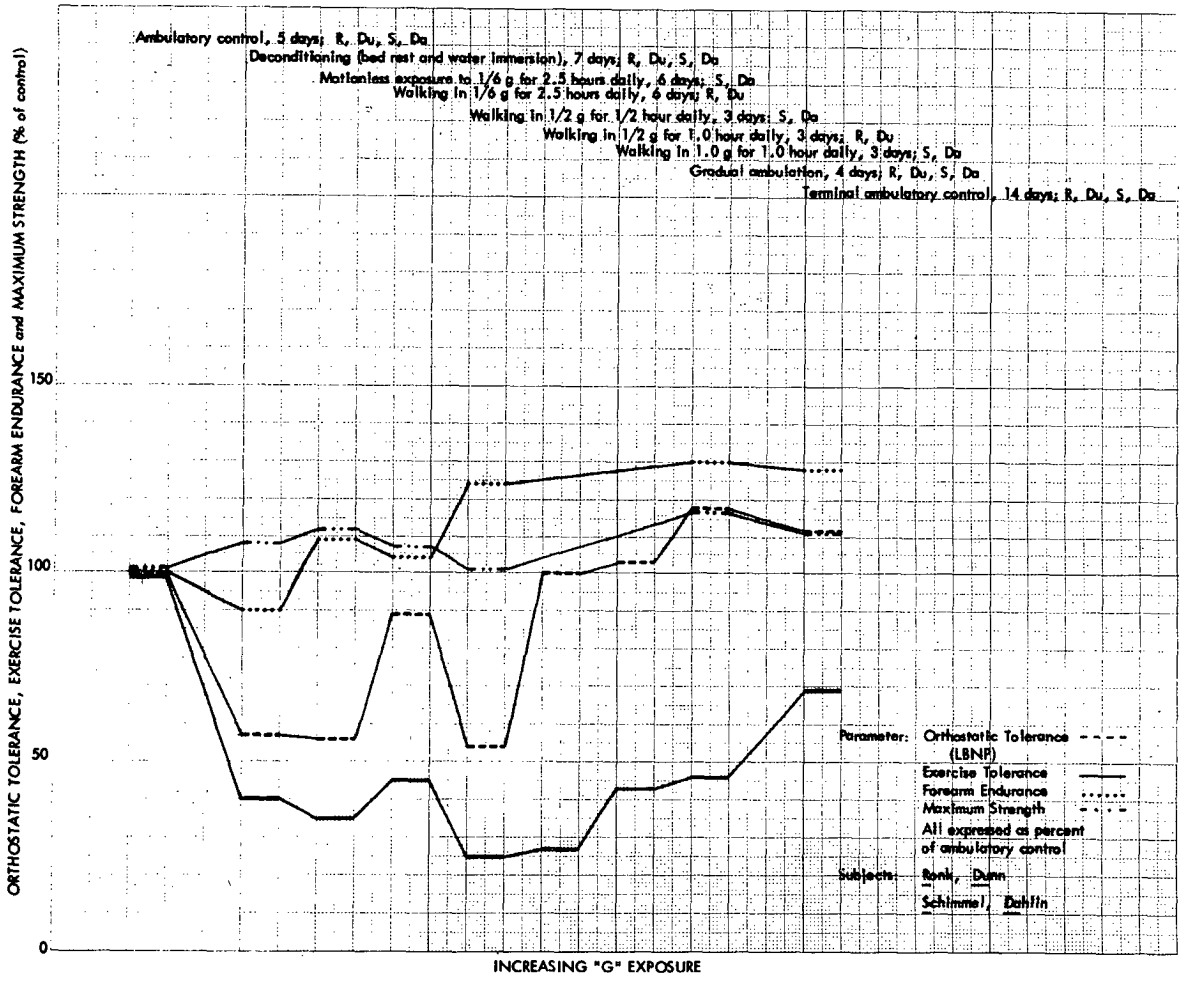


FIGURE 1.—*Composite plots of physiological fitness parameters versus exposure to gravitational force.*

Although the endpoint of forearm endurance was subjective, it was reasonably reproducible but tended to increase readily with repeated determinations. The mean of all four subjects for the 4-day ambulatory-control-test period was 2 minutes. The mean for the 4 subjects on day 2 (first day of testing) was 1.70 minutes, and on day 5 it was 2.11 minutes.

The mean maximum strength of the four subjects for the 4-day ambulatory-control-test period was 57 kg. On the first test day the mean was 60 kg, and it fell to 56.5 kg by the fourth test day. These small differences do not represent a particular trend but reflect more the basic variation of the parameter.

Post-Deconditioning

Seven days of water immersion and bed rest constituted the deconditioning period, and at the end of this period there were variable alterations in the four parameters of physiological fitness. In all four subjects orthostatic tolerance was sharply reduced to about one half of the control value, with a mean reduction of 57 percent. Previous experience in this laboratory has established that this is near the expected degree of deconditioning for a 7-day exposure to this particular schedule of bed rest and water immersion. (A 3-day exposure will typically result in a modest reduction in orthostatic tolerance

to 80 to 85 percent of control, while a 5-day deconditioning period will typically reduce orthostatic tolerance to 60 to 70 percent of control. A 7-day deconditioning period nearly guarantees a reduction of orthostatic tolerance to below 60 percent of control.)

Exercise tolerance was sharply reduced in all four subjects. The individual reductions ranged from 24 to 48 percent of control values, with a mean reduction of 40 percent of the initial ambulatory control value.

Forearm endurance was slightly decreased in all four subjects, with the mean of the four subjects being 90 percent of the mean control value.

Maximum strength was in no way reduced by the deconditioning period. The mean of the four subjects was 108 percent of the mean control value.

Artificial-Gravity Exposure

The results from the two different experimental procedures under the 1/6-*g* condition show that there were apparent differences between the motionless and walking exposures. As compared to the deconditioned level, orthostatic tolerance of the motionless subjects was not increased by artificial-gravity exposure alone; yet when such exposure was combined with slow walking, a substantial increase was noted. One motionless subject displayed somewhat increased orthostatic tolerance while the other showed decreased orthostatic tolerance, the resultant mean being virtually equal to that at the end of the deconditioning period. In contrast, both walking subjects displayed increased orthostatic tolerance so that the resultant mean rose to near the control value. The same pattern to a lesser degree was seen in the exercise-tolerance data, where both motionless subjects displayed modest decreases in exercise tolerance while both walking subjects displayed modest increases. Forearm endurance and maximum strength remained at control levels and were seemingly unaffected by the artificial-gravity exposure.

Since the two subjects who were motionless in the 1/6-*g* exposure (group II) displayed a degree of deconditioning equal to that at the end of their first deconditioning period, no further deconditioning was necessary for them.

Inasmuch as the orthostatic tolerance of the two subjects walking in 1/6 *g* had returned to near control values, it was necessary to decondition them further. Following the measurements related to the 1/6-*g* exposure, these two subjects (group I) were returned to water immersion and bed rest for an additional 3 days. Tests at the end of this period indicated a degree of deconditioning almost equal to that at the end of the first deconditioning period.

The next level of artificial gravity to be studied was 1/2 *g*. The aim here was to explore two brief exposures to determine the minimum exposure period necessary to develop clearly detectable improvements in the deconditioned state. During the second deconditioning period of group I, subjects S and Da were engaged in walking for 1/2 hour daily for the 3-day period. There was no improvement in their deconditioning after such exposure. By extending the same 1/2-*g* exposure to 1 hour daily for subjects R and Du, a significant improvement in orthostatic tolerance was obtained, but exercise tolerance clearly declined. In subjects S and Da who were exposed for 1/2 hour daily in 1/2 *g*, orthostatic tolerance remained essentially the same as that displayed at the end of the deconditioning period. Exercise tolerance in the same two subjects decreased sharply in one and remained substantially below the deconditioning value in the other. The mean of the two was now approximately 1/4 that of ambulatory control. In subjects R and Du, exposed to 1/2 *g* for 1 hour daily, orthostatic tolerance increased sharply in both from about 60 percent of the control values to almost 100 percent of the control values. In contrast, exercise tolerance in the same two subjects decayed to about 1/4 of ambulatory control values, as in the two subjects exposed for 1/2 hour daily. Forearm endurance and maximum strength again remained at or over initial ambulatory controls.

The next level of exposure was walking in 1 *g* for 1 hour daily for 3 days for the group II subjects only. This exposure produced significant improvement in their orthostatic and exercise tolerance. Orthostatic tolerance increased in both subjects to a mean that was 3 percent higher than the control mean. Mean exercise tolerance increased in both subjects to 46 percent of the control mean. Forearm endurance and maximum strength were not

tested following 1-*g* exposure, since these values had already reached control levels.

At this point in the experiment orthostatic tolerance, forearm endurance, and maximum strength were all at or somewhat above control values; exercise tolerance, however, was still significantly below the controls. To estimate how much ambulation would be necessary to return exercise tolerance to near control levels, all four subjects were gradually ambulated over the next 3 days. This procedure increased the exercise tolerance in subjects R and Du to 56 percent of control tolerance. In the other two subjects, whose exercise tolerance was already at 46 percent of the control after the 1-*g* exposure, the 3 days of gradual ambulation failed to increase this parameter; in fact, there was a decrease. Orthostatic tolerance, forearm endurance, and maximum strength, already at control levels, rose somewhat over the 3-day gradual-ambulation period.

All four subjects were then released and asked to return to the laboratory in 2 weeks for further testing. No specific instructions were given as to the level of their activity, but since all were nonathletic college students, their activity levels were roughly similar. At the end of 2 weeks of routine activity, orthostatic tolerance, forearm endurance, and maximum strength were all up to control levels, but exercise tolerance failed to return to the control values. Mean orthostatic tolerance and maximum strength were at 112 percent of the initial controls. Forearm endurance had risen steadily throughout the experiment and had a final value that was 128 percent of control. Two weeks of routine activity increased exercise tolerance somewhat in three of the four subjects, and the final mean tolerance of all four subjects was 69 percent of the control mean.

To summarize the results, forearm endurance and maximum strength were relatively unaffected by deconditioning and by the three levels of *g*-exposure. Orthostatic tolerance was reduced to approximately 50 percent of control values by deconditioning and was substantially improved by walking in simulated lunar gravity for 2.5 hours daily for 7 days or by walking at 1/2 *g* and 1 *g* for 1 hour daily for 3 days. Exercise tolerance was also reduced to approximately 50 percent of control values by deconditioning but did not significantly improve with increasing *g*-exposure. Walking in 1 *g* for 1 hour daily for 3 days raised exercise tolerance only a little above the low produced by deconditioning. Two weeks of routine activity after the artificial-gravity exposure increased exercise tolerance to approximately 70 percent of the control value.

DISCUSSION

In the experimental hypothesis it was supposed that the four parameters of physical fitness (orthostatic tolerance, exercise tolerance, forearm endurance, and maximum strength) would show different effects of the deconditioning and subsequent artificial-gravity exposures. The results of this experiment qualitatively support this hypothesis; however, larger experiments will be necessary to quantitatively define these differences.

It was quite apparent that forearm endurance and maximum strength were unaffected by the deconditioning regimen under the conditions of this experiment. The additional amount of deconditioning necessary to reduce these variables significantly is presently unknown, and the reason they should remain unaffected is not clear. It is reasonable to assume that these represented tasks utilizing rather simple central-nervous-system involvement and that these simple mechanisms remained intact for the limited deconditioning period of this experiment. It is also possible that the subjects' movements in bed could have provided an adequate conditioning stimulus for these muscles.

Orthostatic tolerance as measured in a lower-body-negative-pressure tank was as predicted. The reduction of orthostatic tolerance through deconditioning techniques similar to that described in this experiment has been previously reported (refs. 11 to 14). To our knowledge, however, this is the first report on the use of artificial hypogravity to restore orthostatic tolerance. In view of the primary importance of gravity in stimulating orthostatic mechanisms, it is not surprising that, after deconditioning, orthostatic tolerance was readily returned to control levels by modest artificial-gravity exposures. Walking in 1/6 *g* was more potent in restoring orthostatic tolerance than motionless exposure to 1/6 *g*. Other investigators (refs. 6 and 15 to 17) have found that exercise was not protective. The nature and degree to which exercise augments artificial gravity in restoring orthostatic tolerance remain important

practical questions to investigate. Another interesting aspect of the data concerns the clear increase in orthostatic tolerance with repeated LBNP determinations. This result suggests that LBNP could be used on a repetitive basis as a means of restoring such tolerance to some desired level. The reconditioning potential of negative 40 mm Hg LBNP has been reported (refs. 18 and 19), and experience in this laboratory with various pressures up to negative 70 mm Hg suggests that this is indeed a technique capable of restoring orthostatic tolerance to levels well past ordinary 1-*g* controls.

Exercise tolerance as measured in this experiment was sharply reduced by deconditioning. With increasing *g*-exposure and even with ambulation in 1 *g*, it never returned to control values. The task of efficiently running on a treadmill requires a complex nervous-system response. The reduction in exercise tolerance would appear to be central in origin, since simple strength and endurance were repeatedly shown to be at or above control values while exercise tolerance continued to remain low. The exercise-tolerance-test endpoint was a steady heart rate of 180 beats per minute, and ordinarily this is a measure of the ability to perform hard physical work. If, however the deconditioning process would centrally alter heart rate, then an apparent endpoint might be reached long before the subject had truly reached his maximal work capacity. It is also reasonable that, since gravity has no proven primary role in the exercise response, reduced exercise tolerance should not readily be affected by increasing *g*-exposure alone. Rather, the reduced exercise tolerance would be efficaciously restored by exercise exposure of the magnitude employed in the exercise-tolerance test. In this experiment, walking as a form of light exercise did not appear very effective in restoring the tolerance to hard exercise as in running on a treadmill. A related interfering factor is the hypodynamic environment accompanying water immersion and bed rest. In this design there is no way of determining whether the reduction in exercise tolerance is due more to the hypodynamic state or to the simulated weightless state. There is some evidence that the hypodynamic state may be more responsible for the loss of exercise tolerance (ref. 20).

Answers to the questions raised by this experiment would constitute a first estimate of what we might realistically expect from artificial gravity in space. To look at this more generally, the problem consists basically of two elements. Initially it will be necessary to define the phenomenon of gravity-sensitivity in physiological terms. Those aspects of bodily functions that are altered by brief or prolonged exposure to weightlessness must be identified. Once they are identified, specific high-resolution measurement techniques must be developed to accurately establish the alterations produced by increasing weightlessness exposure. Once the physiological alterations of such exposure and the related measurement techniques have been defined, the second element consists of developing methods to restore the gravity-sensitive systems to some desired level. Artificial gravity in some cases may prove to be specific and quite efficacious in its restoration of a parameter such as orthostatic tolerance; on the other hand, it may prove more nonspecific and less effective in the restoration of a parameter such as exercise tolerance. Thus, many other reconditioning techniques and countermeasures will have to be studied so that intelligent tradeoffs may be made. Perhaps the most important consideration regarding the combination of countermeasures and reconditioning techniques is the duration of the exposure to weightlessness. For short trips in 0 *g*, such as shuttle runs to an orbital lab or similar flights of hours' or days' duration, no countermeasures will likely be necessary and deconditioning will be allowed to proceed throughout the flight. Singular reliance will be placed on the rapid reconditioning occurring upon return to Earth. For 0-*g* exposure in weeks, countermeasures will probably be employed. These will be aimed primarily at those aspects of 0-*g* adaptation that occur in the first few days of exposure. However, again there will be substantial reliance on the rapid reconditioning upon return to Earth. For flights involving 0-*g* exposure in months, more elaborate countermeasures will be necessary and less reliance will be placed on reconditioning in the 1-*g* environment upon return. To consider orthostatic tolerance, for example, LBNP could be used as an in-flight conditioning technique to keep such tolerance at a level lower than that displayed in a 1-*g* environment but adequate to prevent any irreversible alteration in orthostatic mechanisms. For reentry, a *g*-suit could be used as a simple,

effective countermeasure, and finally, the rapid reconditioning upon return to the 1-*g* environment could be used to boost the orthostatic tolerance from the lower level maintained in space by LBNP to "normal" levels appropriate for the 1-*g* field. For flights involving weightlessness exposure of many months' duration, little reliance can be put on the reconditioning upon return to a 1-*g* environment. As the number of elaborate countermeasures and conditioning techniques expands to meet the increasing duration of exposure to weightlessness, they will consume an increasing amount of the astronauts' expensive time, and there will be a point where the addition of artificial gravity will represent the cost-effective solution. It is also here that the difference in countermeasures and conditioning techniques becomes critical. Countermeasures provide immediate protection but do not alter the physiological state of the involved parameter. Conditioning techniques, however, fundamentally alter the physiological state in the direction of the normal 1-*g* functional state. For example, a leotard or *g*-suit is an effective countermeasure for orthostatic intolerance but does not fundamentally modify the orthostatically intolerant state. In contrast, LBNP is an effective reconditioning technique capable of restoring the orthostatic tolerance to where countermeasures might not ordinarily be necessary. Finally, for flights of years' duration, the spacecraft will have to contain all of the means to condition the crew to whatever levels are deemed desirable, since there can be at best minimal reliance on the reconditioning on return to the 1-*g* environment.

The large amount of information needed for intelligent design of the various countermeasures and reconditioning systems is currently not available and would be clearly useful to the engineering community. To fail to begin to gather this information will not significantly alter our immediate space plans, but it could ultimately have the effect of seriously limiting the duration of our space missions. Although on Earth we cannot truly escape gravity for any significant length of time, simulations of weightlessness and artificial gravity could be useful in generating much of this information.

REFERENCES

1. Tulloh, N. M.; and Romberg, B.: An Effect of Gravity on Bone Development in Lambs. Nature, vol. 200, 1964, pp. 438–439.
2. Wunder, C. C.; Briney, S. R.; Kral, M.; and Skungstad, C.: Growth of Mouse Femurs During Continual Centrifugation. Nature, vol. 188, 1960, pp. 151–152.
3. Smith, A. J.; and Kelly, C. F.: Influence of Chronic Acceleration Upon Growth and Body Composition. Ann. N. Y. Acad. Sci., vol. 110, 1963, p. 410.
4. Graybiel, A.; and Clark, B.: Symptoms Resulting From Prolonged Immersion in Water: The Problem of Zero G Asthenia. Aerospace Med., vol. 32, 1961, pp. 181–196.
5. McCally, M.; and Lawton, R. W.: The Pathophysiology of Disuse and the Problem of Prolonged Weightlessness: A Review. AMRL–TDR–63–3. Aerospace Medical Research Laboratories, Wright-Patterson Air Force Base, Ohio, 1963.
6. Vallbona, C.; Vogt, F. B.; Cardus, D.; Spencer, W. A.; and Walters, M.: The Effects of Bedrest on Various Parameters of Physiological Function. NASA CR–171 through NASA CR–184, 1965.
7. Berry, C. A.: Status Report on Space Medicine in the United States. Aerospace Med., vol. 40, 1969, pp. 762–769.
8. Berry, C. A.: Summary of Medical Experiences in the Apollo 7 through 11 Manned Spaceflights. Aerospace Med., vol. 41, 1970, pp. 500–519.
9. McCally, M.: Body Fluid Volumes and the Renal Response of Human Subjects to Water Immersion. AMRL–TR–65–115. Aerospace Medical Research Laboratories, Wright-Patterson Air Force Base, Ohio, 1965.
10. White, W. J.: A Space-Based Centrifuge. 1965 Status Report. DAC–P–3436. Douglas Aircraft Company, Santa Monica, California, 1965.
11. Graveline, D. E.: Maintenance of Cardiovascular Adaptability During Prolonged Weightlessness. Aerospace Med., vol. 33, 1962, pp. 297–302.
12. Graveline, D. E.; and Balke, B.: The Physiologic Effects of Hypodynamics Induced by Water Immersion. SAM–60–88. School Aviation Medicine, Brooks Air Force Base, Texas, 1960.
13. Miller, P. B.; Johnson, R. L.; and Lamb, L. E.: Effects of Four Weeks of Absolute Bed Rest on Circulatory Functions in Man. Aerospace Med., vol. 35, 1964, pp. 1194–1200.
14. Taylor, H. L.; Herschel, A.; Brozck, J.; and Keys, A.: Effects of Bed Rest on Cardiovascular Function and Work Performance. J. Appl. Physiol., vol. 2, 1949, pp. 223–237.
15. Birkhead, N. C.; Blizzard, I. I.; Issekutz, B., Jr.; and Rodahl, H.: Effects of Exercise, Standing Negative

Trunk and Positive Skeletal Pressure on Bed-Rest-Induced Orthostasis and Hypercalciuria. AMRL–TR–66–6. Aerospace Medical Research Laboratories, Wright-Patterson Air Force Base, Ohio, 1966.

16. Cardus, D.: Effects of 10 Days Recumbency on the Response to the Bicycle Ergometer Test. Aerospace Med., vol. 37, 1966, pp. 993–999.
17. Vogt, F. B.: Effect of Intermittent Leg Cuff Inflation and Intermittent Exercise on the Tilt Table Response after Ten Days Bed Recumbency. Aerospace Med., vol. 37, 1966, pp. 943–947.
18. Lamb, L. E.; and Stevens, P. M.: Influence of Lower Body Negative Pressure on the Level of Hydration During Bed Rest. Aerospace Med., vol. 36, 1965, pp. 1145–1151.
19. McCally, M.; Piemme, T. E.; and Murray, R. H.: Tilt Table Response of Human Subjects Following Application of Lower Body Negative Pressure. Aerospace Med., vol. 37, 1966, pp. 1247–1249.
20. Lamb, L. E.; Stevens, P. M.: and Johnson, R. L.: Hypokinesia Secondary to Chair Rest from 4 to 10 Days. Aerospace Med., vol. 35, 1965, pp. 755–763.

Effects of Visual Reference on Adaptation to Motion Sickness and Subjective Responses Evoked By Graded Cross-Coupled Angular Accelerations

JAMES T. REASON and ELGIN DIAZ
University of Leicester, England

SUMMARY

Three groups of 10 subjects each were exposed to stepwise increments of cross-coupled angular accelerations in three visual modes: internal visual reference (IVR), external visual reference (EVR), and vision absent (VA). The subjects in the IVR condition required significantly greater amounts of stimulus exposure to neutralize their illusory subjective reactions. They also suffered a greater loss of well-being and a more marked incidence of motion sickness than did subjects in the EVR and VA conditions.

The same 30 subjects were reexposed to the same graded cross-coupled stimulation 1 week later, This time, however, all the subjects were tested under only the IVR condition. All three groups showed some positive transfer of adaptation, but only the IVR-IVR combination required significantly fewer head motions to achieve the same level of adaptation on the second occasion. Taken overall, however, the most efficient and least disturbing route to adaptation at the completion of the second test was via the VA-IVR combination.

The aims of this study were twofold: first, to examine how differences in the nature of the concurrent visual stimulation influence the rate of adaptation to graded cross-coupled angular accelerations; and second, see how adaptation acquired in one visual mode transfers to a different mode when the quality and quantity of vestibular stimulation are approximately the same in both conditions.

At a theoretical level, it was hoped that this investigation would reveal something more about the interaction of visual and vestibular inputs in the production of motion sickness, and also that it would provide further information about the conditions under which adaptation acquired in one situation will confer protection in another. At a more practical level, this work was directed toward the design of an adaptation schedule that was sparing of both time and discomfort and that would give residual protection against motion sickness in a rotating environment.

The theoretical notions that prompted this inquiry owe a great deal to the "conflict" theories of motion sickness advanced by Lansberg (ref. 1), Steele (ref. 2), and Guedry (ref. 3). They were also derived from the now extensive body of research concerned with the adaptive effects and aftereffects associated with displacement of the visual input by various optical devices. As Guedry (ref. 4) pointed out, there are a number of important similarities between adapting to a distorted visual field and adapting to vestibular distortions created by rotation and weightlessness. So compelling are these similarities, in fact, that it is feasible to suppose we are dealing with similar mechanisms of adaptation in both visual and vestibular distortion.

The theory adopted here is concerned with answering the question: What is the essential qualitative nature of the stimulus that produces motion sickness? Since symptoms occur in a wide variety of circumstances, many different types of sensory conflict are likely to be involved in its production. It is suggested that we can isolate six

basic conflict situations, subsumed under two major classes of sensory rearrangement: (1) *visual-inertial rearrangement* (where the term "inertial" includes both the vestibular and nonvestibular proprioceptors), and (2) *canal-otolith rearrangement.*

Under natural conditions of self-propelled locomotion, both pairs of receptors operate synergistically to give us correlated information about spatial orientation. But in the unusual force environments engendered by passive motion, these harmonious relationships can be disrupted to produce three basic conflicts, which we can formulate on purely logical grounds. If A and B represent portions of normally correlated receptor systems (i.e., the visual-inertial or canal-otolith systems), then these three conflict situations can be described as:

Type 1: where A and B simultaneously signal contradictory or uncorrelated information.

Type 2: where A signals in the absence of an *expected* B signal.

Type 3: where B signals in the absence of an *expected* A signal.

Each of these three conflicts in involved in both visual-inertial and canal-otolith rearrangements. Examples of the resulting six conflict situations in which motion sickness is known to occur are shown in table 1.

The theory rests on two basic premises. The first is that situations that provoke motion sickness are characterized by a condition of "sensory rearrangement" in which the information signaled by the vestibular system and other functionally related receptors (i.e., the eyes and the nonvestibular proprioceptors) is at variance with the integrated pattern of sensory inputs *expected on the basis of past experience.* The second premise is that irrespective of what other senses are party to these conflicts, the vestibular system is always implicated, either directly or indirectly, as in the case of visually induced sickness. This also tells us something about the nature of the effective stimulus, namely that it must involve a changing velocity component.

The essential provocative conflict is considered to be that between the current influx from the various spatial receptors and what is stored from previous transactions with the environment. Thus it presumes the existence of a storage unit that

TABLE 1.—*Six Basic Types of Sensory Conflict that Provoke Motion Sickness*

Type	Visual (A)–intertial (B)	Canal (A)–otolith (B)
Type 1 (A and B)	Visual distortion experiments Vehicular transport	Coriolis vestibular reaction
Type 2 (A, not B)	Cinerama Fixed-base car and aircraft simulators	Caloric and electrical stimulation
Type 3 (B, not A)	Swinging in closed cabin Travel without external reference	Rotation about Earth-horizontal axis Counterrotating devices

retains the important informational characteristics of previous inputs from the spatial receptors. Such a "memory unit" would be compatible with Sokolov's "nervous model" (ref. 5), with Groen's "pattern center" (ref. 6), and with Held's "correlation storage" (ref. 7). A theoretical component of this nature is essential to explain both the all-important process of adaptation to an atypical force environment, and its sequel, the phenomenon of "mal de débarquement" or "land sickness" that occurs when the previously typical conditions are reinstated. Within this theoretical framework, adaptation is seen as the gradual updating of the contents of the neural memory store until they are compatible with the prevailing sensory influx. When the incoming sensory information and the contents of the store are matched, the person can be said to be adapted to his immediate environment (refs. 8 and 9).

While visual-inertial rearrangements may exist in isolation, it is clear that canal-otolith rearrangements are likely to be complicated by the presence of incongruous visual information, unless the eyes are closed or covered. The purpose of the present study was to investigate the effects of different types of visual reference upon the type-1 canal-otolith conflict created by the cross-coupled stimulus. In particular, it was concerned with the rate at which healthy subjects adapt to incremental levels of exposure.

Previous research indicates that vision may either enhance or diminish the nauseogenic properties of a particular force environment, depending

on the nature of the visual information. Fixating on an Earth-stable reference tends to reduce susceptibility on swings (ref. 10) and on ships (ref. 11). But when the gaze is restricted to the interior of a compartment that moves with the subject, the incidence of motion sickness is greatly increased.

Three studies in the Pensacola slow rotation room (SRR) have shown that cross-coupled angular accelerations are less disturbing with the eyes covered than when an unrestricted view of the illuminated interior of the device is permitted (refs. 12 to 14). However, Guedry (ref. 15) found that subjects engaged in visually presented problems were markedly less susceptible to "canal sickness" than subjects who executed the same head movements without vision. This difference was attributed to the fact that the vision-present condition involved heightened mental activity, which prevented the subjects from concentrating on their possible lack of well-being, a factor that is known to be important in suppressing motion sickness (ref. 16).

The evidence with regard to the influence of vision on vestibular habituation is complex and often contradictory. It has been reviewed in detail by Guedry (ref. 17). In general, the presence of vision tends to facilitate adaptation to cross-coupled angular accelerations, as judged by the development of "conditioned compensatory reactions" (ref. 17) and the perrotational diminution of the Coriolis oculogyral illusion (ref. 12).

PROCEDURE

The investigation was in two parts, separated by an interval of 1 week. In part I, each of 30 subjects was randomly assigned to one of three conditions of visual reference: (1) internal visual reference (IVR), where the subject's gaze was restricted to the illuminated interior of the rotating device; (2) external visual reference (EVR), where the subject had a full view of the Earth-stable exterior of the device; and (3) vision absent (VA), where the subject was blindfolded. In all three conditions the subjects executed the same prescribed 90° head and trunk flexions to induce qualitatively and quantitatively similar cross-coupled angular accelerations.

In Part II, we examined how the adaptation acquired under these different visual modes transferred to a single mode, that of IVR. Thus the subjects carried out the same head motions as before but under the same internal reference conditions. This was to see whether adaptation to IVR could be accelerated by previous exposure to the EVR and VA conditions. Such a finding would have important implications for the construction of an adaptation schedule designed to give residual protection against motion sickness (ref. 18).

METHOD

The major item of apparatus consisted of the enclosed, draft-proof rotating device depicted diagrammatically in figure 1. The subject sat in a cylindrical compartment 1.73 m high and 1.68 m in diameter. The upper 1.22 m of this compartment was enclosed in transparent acrylic which allowed an unrestricted view of the exterior. This acrylic window could also be screened by heavy curtains running around the inside periphery of the compartment.

When the subject was in the upright position, his head was alined with the axis of rotation. Situated to the front, back, left, and right of him were adjustable head pads. Located at convenient positions near these pads were fixation targets

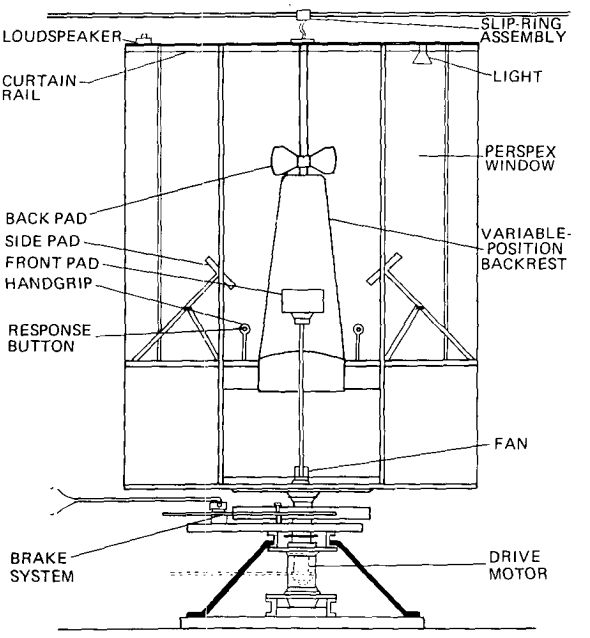


FIGURE 1.—*Diagram of rotating device (curtains omitted for clarity).*

designed to control the direction of gaze during and after the head movements when the eyes were open. They also provided a reference from which to judge aparent visual motion.

The head and body motions were carried out in response to taped instructions delivered through a loudspeaker set into the roof of the compartment. A fan, which circulated air within the compartment, was set into the floor behind the subject, and a number of light-masked ventilation holes were constructed in the roof. The noise of the fan, which was continually in operation during the test runs, effectively masked any slight auditory cues from the bearings and drive system. The interior of the device was illuminated by a 25-watt lamp placed above and to the left of the subject.

Two handgrips were situated on either side of the seat. The subject was instructed to maintain a firm grip on these throughout the test runs. Microswitches (labeled "yes" and "no") were mounted on the handgrips to signal the presence or absence of unusual sensations during and immediately after each prescribed movement.

Techniques for measuring adaptation to incremental levels of cross-coupled angular accelerations, developed in earlier studies using the Pensacola SRR, have been described in detail elsewhere (ref. 19). Essentially the same method was used in the present experiment.

The head movements were grouped into sequences of eight: the four down motions to each pad position, plus the four complementary up movements to the central upright position. In each movement the head passed through 90°, using a combination of trunk and neck flexion, and the taped commands to move occurred at 4-second intervals. At the completion of each individual movement, the subject was required to make a forced-choice detection judgment. One of two alternative responses could be made: either "Yes," meaning that the subject had detected some illusory or unusual sensation due to the rotating environment; or "No," meaning that he was unaware of the effects of the cross-coupled angular stimulus. At the end of each sequence the subjects were asked to make a well-being or malaise estimate on the basis of an 11-point category scale developed in a previous study (ref. 20). The scale ranged from 0 ("I feel fine") to 10 ("I feel awful, just like I'm about to vomit"). These ratings were designed both to monitor the state of the subject during the experimental run and to give an indication of the relative disturbance produced by each visual mode.

Head motions were started at a rotational velocity of 1 rpm in a clockwise direction and continued until the subject had fulfilled an arbitrary adaptation criterion of three consecutive sequences without an affirmative response; that is, 24 discrete movements without any detectable effects due to the cross-coupled stimulus. When this criterion was reached, the velocity was increased by 1 rpm, and the whole procedure was repeated at the next higher angular velocity. This continued until the adaptation criterion was met at the terminal velocity of 6 rpm. When the final perrotational adaptation criterion was met, the device was slowly decelerated to rest. Once stationary, the subject continued to make the same prescribed head motions as before, terminating when the postrotational adaptation criterion had been reached. These postrun movements were designed to evoke postrotationary sensations that are qualitatively similar to the perrotational sensations, but of opposite sign (ref. 19).

Thirty subjects completed both parts I and II, ten subjects in each of the three conditions of visual reference in part I. An additional five subjects were tested but were unable or unwilling to complete part I due to motion sickness.

All the subjects were male graduate students at the University of Leicester. Their ages ranged from 21 to 34 years. Prior to the experiment, each subject filled out a motion sickness questionnaire (ref. 21), and a standardized medical history was taken by the experimenter. The histories indicated that all subjects were in good health and were not under the influence of any medication at the time of the experiment. Only five subjects reported a negative history of motion sickness, and in none was there any indication that this apparent immunity could be attributed to a labyrinthine defect.

RESULTS

Part I

Following the convention adopted in previous studies (ref. 19), two measures of perrotational adaptation were used: (1) the total number of affirmative responses elicited prior to the adaptation

criterion at each step velocity; and (2) the total number of sequences executed prior to the adaptation criterion at each step velocity. Since the number of affirmatives and sequences at each step velocity was likely to be a complex function of the total stimulus exposure up to that point, these cumulative measures of adaptation were considered more meaningful.

Figure 2 shows the mean cumulative totals of affirmative responses plotted against angular velocity on logarithmic coordinates for each of the three visual modes. Each data point represents the mean cumulative total of affirmative responses evoked up to and including that particular velocity. The most notable feature of these results is that the IVR condition evoked about four times as much subjective response as either of the other two visual modes. Analysis of variance showed that these differences were significant at the 1 percent level of confidence.

A similar pattern of results is shown in figure 3,

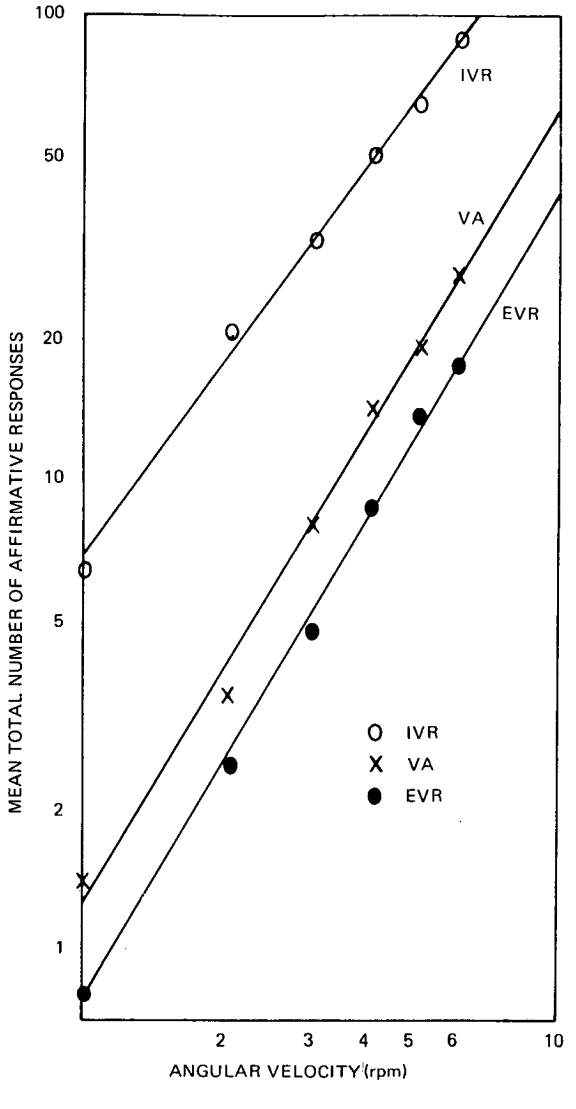


FIGURE 2.—*Mean cumulative totals of affirmative responses elicited up to the adaptation criterion at each step velocity in part I.*

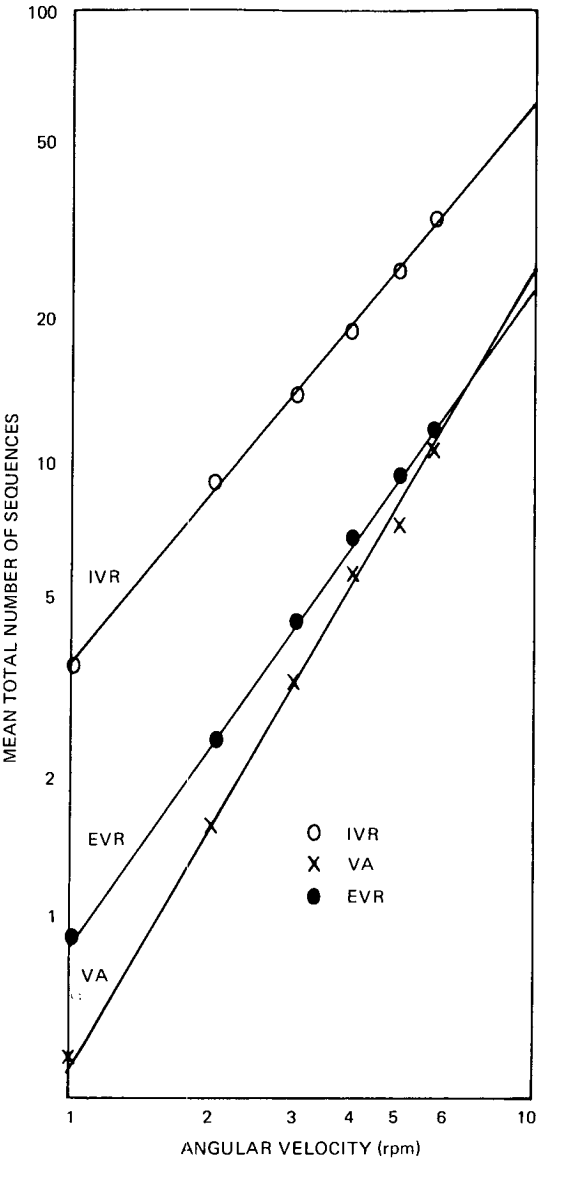


FIGURE 3.—*Mean cumulative totals of sequences executed up to the adaptation criterion at each step velocity in part I.*

parts I and II, analogous to that for the adaptation measures, is shown in table 3. Despite the positive transfer of adaptation, the IVR-IVR condition involved a greater overall decline in well-being than either of the other two combinations.

The motion sickness symptomatology observed in part II is summarized in tables 4a, 4b, and 4c. The most obvious feature was the dramatic reduction in

for Parts I and II Combined

Visual mode combination	Rotational velocity, (rpm)					
	1	2	3	4	5	6
IVR-IVR	0.2	0.4	0.5	1.0	1.0	1.5
EVR-IVR	0	0	0.1	0.2	0.2	0.6
VA-IVR	0	0	0	0.1	0.3	0.8

* Based on scale of 0 to 10; the higher the rating, the greater the loss of well-being.

where mean cumulative totals of sequences executed prior to the adaptation criterion at each velocity are plotted against rpm on logarithmic coordinates. This measure gives an indication of the amount of stimulation required to achieve adaptation, while the mean cumulative totals of affirmatives, shown in figure 2, indicate the amount of subjective response

the greatest producer of sickness. Motion sickness symptomatology, maximum malaise ratings, and motion sickness questionnaire scores for each subject in each visual mode are shown in tables 2a, 2b, and 2c.

Part II

motion sickness reactions in the IVR-IVR group as compared to part I (see tables 2a, 2b, and 2c). The EVR-IVR and VA-IVR groups, on the other hand, showed only a slight increase in symptomatology as compared with part I.

TABLE 4a.—*Summary of Motion Sickness Symptomatology for IVR-IVR Group in Part II*

Subj.	Perrotational MS	Postrotational MS
HA	PI, SW I (2)	NS (1)
DA	NS (0)	SA I (2)
AN	NS (0)	NS (0)
AD	NS (0)	SA I (1)
CO	NS (0)	SA I (2)
FA	HA, SW I (4)	HA, SW I (3)
CR	HA (1)	HA, SW I (2)
CL	SAI (1)	NS (0)
McC	NS (0)	NS (0)
McG	HA (2)	HA (2)

See legend at end of table 2a.

TABLE 4b.—*Summary of Motion Sickness Symptomatology for EVR-IVR Group in Part II*

Subj.	Perrotational MS	Postrotational MS
LE	SA I, P I, SW I (2)	NS (0)
CH	SA I (1)	SA I (1)
BA	NS (0)	P I (0)
PL	SA I (1)	SA I, SW I, vomit (6) T
MO	NS (0)	NS (0)
RA	NS (1)	Sudden vomit (1) T
BR	SA I, SW I, P I (3)	SA II, SW I, P I (5)
SO	SA I (1)	SA I (2)
PL	NS (0)	NS (0)
FO	NS (0)	SW I (1)

See legend at end of table 2a.

TABLE 4c.—*Summary of Motion Sickness Symptomatology for VA-IVR Group in Part II*

Subj.	Perrotational MS	Postrotational MS
OW	NS (0)	NS (0)
TA	NS (0)	NS (0)
WA	NS (0)	SA I (2)
GR	SA I (2)	SA II, SW II (4) T
HA	SA I (1)	SA I (1)
LO	NS (0)	NS (0)
GL	NS (0)	NS (0)
RE	NS (0)	NS (0)
DA	SA II (4)	SA I (2)
BA	SA II, SW II (5) T	

See legend at end of table 2a.

DISCUSSION

Effects of Visual Reference

The principal finding from part I was that the IVR condition evoked considerably more subjective response and required greater stimulus exposure to achieve adaptation than either the EVR or VA condition. It is evident that this result cannot be explained simply by the presence or absence of vision, since the visual modality was involved in the conditions that elicited both the most and the least subjective response, i.e., the IVR and EVR treatments, respectively. Clearly, an explanation of these findings must include some consideration of the *nature* of the visual information present in these two modes. There appear to be a number of alternative, although not mutually exclusive explanations along these lines.

In the first place, we can consider the difficulty of the detection task demanded of the subject and how this was affected by the different conditions of visual reference. It can be argued that the optimal detection conditions were present in IVR. The closed curtain served two purposes. First, it eliminated veridical motion cues from the exterior, thus promoting the sensation frequently reported by subjects that the device was tilting or lurching. They had no cues to the contrary in IVR and, except to the mechanically minded, the external appearance of the device (unlike the Pensacola SRR) did not deny this possibility. Second, the illuminated interior of the curtain provided a sufficiently structured surface to facilitate the perception of the Coriolis oculogyral illusion.

Although the VA mode allowed no information as to the true orientation of the body or the device, it eliminated the Coriolis oculogyral illusion as the basis of an affirmative response. However, this was partly compensated for by a heightened perception of apparent whole-body motion, i.e., the Coriolis sensation.

Finally, there is good reason to believe that the "noisiest" detection conditions prevailed in the EVR condition. Even though the subject maintained a device-stable fixation in this condition, there was enough veridical information coming from the stable surroundings to override his illusory sensations in the competition for his attention.

Although detectability factors probably made

some contribution to the observed differences among the modes, they by themselves fail to account for the corresponding variation in well-being ratings and motion sickness symptomatology. Part of the greater discomfort reported in the IVR condition is likely to be a function of the additional stimulus exposure incurred in the longer process of adaptation. But examination of figures 3 and 4 indicates that well-being declined more rapidly over a given stimulus exposure in the IVR condition that it did in EVR or VA. This finding suggests that it was the quality rather than the quantity of the IVR stimulation that caused the disturbance.

Further support for this notion comes from the fact that there were considerably more spontaneous complaints about the unpleasant nature of the stimulus in IVR than in the other two modes. The alternative explanation, that the IVR subjects were basically more susceptible than the other subjects, founders on the finding that there were no significant differences among the groups in their MSQ scores (although it must be admitted that the average MSQ value for the IVR group was slightly higher than in the other two groups).

Since stimulus exposure was a dependent and not an independent variable in this experiment, it is not possible to state categorically that IVR was intrinsically more nauseogenic than either EVR or VA. But, as we have already seen, there are strong grounds for believing that to be the case, and this belief is further supported by previous studies (refs. 10 and 12 to 14). It seems, therefore, that an explanation of the present findings must extend beyond the nature of the detection task and consider the particular properties of the three visual modes. In other words, we shall proceed on the assumption that the differences in the total affirmative and total sequence measures were not a detection artifact but reflect genuine variation in the intensity of the subjective reactions and the rates of adaptation in the three visual modes.

Perhaps the simplest difference to explain is that between the IVR and EVR conditions. In the latter, the subject received visual information concerning his true orientation from the exterior, and there is ample evidence to show that a veridical visual input will effectively suppress the preception of illusory or inappropriate vestibular sensations (refs. 17 and 22). The almost complete absence of perrotational motion sickness during the rapid ascent through the velocity steps also suggests that the presence of veridical visual information either inhibited the nauseogenic properties of the Coriolis stimulus or superseded it in the bid for central recognition. Certainly the dominance of the visual modality in establishing spatial orientation is likely to play an important part in minimizing the undesirable effects of the Coriolis reaction. In any event, the findings indicate that the provision of external reference is an effective way of reducing both sickness and disorientation in a rotating environment, even when the subject maintains a device-stable fixation point.

The difference between the IVR and the VA conditions, on the other hand, may be accounted for within the framework of the sensory-conflict theory outlined in the introductory section. In both the IVR and VA modes, a canal-otholith conflict of the sort described by Lansberg (ref. 1) and by Guedry (ref. 3) was present both during and immediately after the head motion. But in IVR this was further complicated by an additional visual-inertial conflict. More specifically, we can say that in IVR a type-1 canal-otolith conflict coexisted with a type-3 visual-inertial conflict, i.e., where the inertial receptors signal motion in the absence of expected visual corroboration; whereas the only disturbing characteristic of the VA mode was the type-1 canal-otolith conflict. The presence of the additional conflict in IVR appears to have made a substantial contribution to its disturbing and nauseogenic properties. It is also likely that it was responsible for the greater intensity of the subjective reactions in this mode (see fig. 3).

Finally in this context, an attempt must be made to reconcile the results of the present experiment with the opposite findings obtained in two previous studies (refs. 12 and 15) where the IVR condition promoted a greater degree of adaptation than the VA condition. The first point to consider is that the period of exposure to Coriolis accelerations was much longer in the two earlier studies than in the present investigation. Secondly, adaptation was measured in the Guedry and in the Kennedy et al. studies at discrete intervals, either comparing pretests and posttests or sampling at predetermined points in the perrotational period. In the present experiment a continuous measure of response decre-

ment was used. Thirdly, the nature of the adaptation measures was different in each of the three studies.

In the Guedry study (ref. 15), adaptation was inferred from the decline in nystagmus following a lengthy period of controlled head movement in the Pensacola SRR. It is well known that the nystagmic response, particularly that elicited by Coriolis accelerations, can bear little or no relationship to the strength of the subjective reactions which formed the basis of the adaptation measurements in the present investigation. Another feature of the Guedry study was that the IVR condition was associated with heightened mental activity, whereas the VA condition was not. Since this apparently played an important role in suppressing motion sickness in the IVR group, it may also have influenced rate of adaptation.

The adaptation measure used in the studies by Kennedy et al. (ref. 12) was the extent of the Coriolis oculogyral illusion (OGI) at various points throughout the period of rotation. To achieve adaptation in practically all forms of sensory rearrangement, and certainly in the rotating environment, it is necessary to interact actively with the atypical stimulus conditions (ref. 7). Thus it is possible that subjects in the VA condition showed a relatively small degree of adaptation as measured by the Coriolis OGI because they had been prevented from interacting with the unusual visual properties of the situation. A measure of adaptation that did not rely solely on the visual modality for its expression might have shown a greater amount of neutralization.

Transfer of Adaptation

Previous work has shown that adaptation to cross-coupled angular accelerations is highly stimulus specific and does not generalize readily to qualitatively different stimuli of the same magnitude. Thus Guedry (ref. 17) found that adaptation acquired through one direction of head motion in the SRR did not transfer to head motions executed in a different quadrant.

The findings from part II provide further evidence of the specificity of adaptation and indicate that visual as well as vestibular factors are important in defining the circumstances under which positive transfer will occur. The only occasion when adaptation acquired in part I effected a significant saving in the amount of stimulation required to reach the same level of adaptation in part II was in the IVR-IVR group, where the visual conditions were identical in both cases. Furthermore, the substantial reduction in both symptomatology and loss of well-being in this group during the part-II exposure suggested that considerable residual protection remained when the same conditions were repeated 7 days later.

In both the VA-IVR and EVR-IVR groups, more head motions were needed to reach the terminal adaptation criterion in part II than in part I. In addition, the unfamiliar visual conditions encountered in the part-II exposure provoked a slightly greater decline in well-being and somewhat more symptoms than did the initial session. However, it is important to note that, despite these differences in visual mode, a certain degree of protective adaptation was acquired during the part-I exposure. This is indicated by the fact that the VA-IVR and EVR-IVR groups required less stimulation at a smaller cost in sickness and loss of well-being to reach the terminal criterion in part II than did the IVR group in part I. In other words, their part-I experience counted for something, although not so much as it did for the IVR-IVR group.

The aim of this work was to provide recommendations for the construction of an adaptation schedule that would provide maximum residual protection at the smallest cost in time and discomfort. At the completion of part II, all three experimental groups had acquired the same degree of adaptation under the same conditions of stimulation, namely IVR. The important practical question is: Which of these groups reached this destination with the least expenditure of time and effort and with the least discomfort enroute? The data shown in tables 3 and 4 indicate that, despite the positive transfer, the IVR-IVR combination was still the longest and most troublesome route. In comparison with this combination, both the EVR-IVR and VA-IVR groups achieved significant savings in total stimulus exposure, total subjective response, and motion sickness symptomatology. The most effective combination was the VA-IVR modes where there was a 50-percent saving in sequences and a 43-percent saving in affirmative responses compared to the IVR-IVR combination.

These findings suggest, therefore, that the same degree of protective adaptation can be achieved more quickly with smaller loss of well-being by a combination of the VA and IVR modes than by the IVR condition alone. It still remains to assess whether the protection gained by the VA-IVR combination persists for as long as that acquired by the IVR condition alone; but, on a priori grounds, there is no reason to expect any marked difference.

REFERENCES

1. LANSBERG, M. P.: A Primer of Space Medicine. Elsevier, Amsterdam, 1960.
2. STEELE, J. E.: Motion Sickness and Spatial Perception. A Theoretical Study. ASD TR 61–530. Aeronautical Systems Division, Aerospace Medical Laboratory, Wright-Patterson Air Force Base, Ohio, 1961.
3. GUEDRY, F. E.: Conflicting Sensory Orientation Cues as a Factor in Motion Sickness. Fourth Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–187, 1970, pp. 45–51.
4. GUEDRY, F. E.: Habituation to Complex Vestibular Stimulation in Man: Transfer and Retention of Effects from Twelve Days of Rotation at 10 rpm. Percept. Mot. Skills, vol. 21, monograph suppl. 1–V21, 1965, pp. 459–481.
5. SOKOLOV, E. N.: Neuronal Models and the Orienting Reflex. The Central Nervous System and Behaviour, M. Brazier, ed., J. Macy, 1960.
6. GROEN, J. J.: Problems of the Semicircular Canal from the Mechanico-Physiological Point of View. Acta Oto-Laryngol., suppl. 163, 1960, pp. 59–66.
7. HELD, R.: Exposure History as a Factor in Maintaining Stability of Perception and Coordination. J. Nerv. Ment. Dis., vol. 132, 1961, pp. 26–39.
8. REASON, J. T.: Motion Sickness—Some Theoretical Considerations. Intern. J. Man-Machine Studies, vol. 1, 1969, pp. 21–38.
9. REASON, J. T.: Motion Sickness: A Special Case of Sensory Rearrangement. Advance Sci., vol. 26, 1970, pp. 386–393.
10. MANNING, G. W.; AND STEWART, W. G.: The Effect of Position on the Incidence of Swing Sickness. Rep. Ass. Comm. Av. Med. Res., NRC Canada, 1942.
11. CLAREMONT, C. A.: The Psychology of Seasickness. Psyche, vol. 11, 1931, pp. 86–90.
12. KENNEDY, R. S.; TOLHURST, G. C.; AND GRAYBIEL, A.: The Effects of Visual Deprivation on Adaptation to a Rotating Environment. NSAM–918. Naval School of Aviation Medicine, Pensacola, Fla., 1965.
13. REASON, J. T.; AND GRAYBIEL, A.: Magnitude Estimations of Coriolis Sensations. NAMI–1082, Naval Aerospace Medical Institute, Pensacola, Fla., 1969.
14. GRAYBIEL, A.: Susceptibility to Acute Motion Sickness in Blind Persons. Aerospace Med., vol. 41, 1970, pp. 650–653.
15. GUEDRY, F. E.: Visual Control of Habituation to Complex Vestibular Stimulation in Man. Acta Oto-Laryngol., vol. 58, 1964, pp. 377–389.
16. WENDT, G. R.: Vestibular Functions. Handbook of Experimental Psychology, S. S. Stevens, ed., Wiley, 1951.
17. GUEDRY, F. E.: Psychophysiological Studies of Vestibular Function. Contributions to Sensory Physiology, W. D. Neff, ed., Academic Press, 1965, pp. 63–135.
18. GRAYBIEL, A.: Structural Elements in the Concept of Motion Sickness. Aerospace Med., vol. 40, 1969, pp. 351–367.
19. REASON, J. T.; AND GRAYBIEL, A.: Progressive Adaptation to Coriolis Accelerations Associated with 1-rpm Increments in the Velocity of the Slow Rotation Room. Aerospace Med., vol. 41, 1970, pp. 73–79.
20. REASON, J. T.; AND GRAYBIEL, A.: Changes in Subjective Estimates of Well-Being During the Onset and Remission of Motion Sickness Symptomatology in the Slow Rotation Room. Aerospace Med., vol. 41, 1970, pp. 166–171.
21. REASON, J. T.: An Investigation of Some Factors Contributing to Individual Variations in Motion Sickness Susceptibility. Flying Personnel Research Committee Report No. 1277. Ministry of Defence (Air Force Department), London, 1967.
22. DEARNALEY, E. J.; REASON, J. T.; AND DAVIES, J. D.: The Nature and Duration of After-Sensations following the Cessation of Turning in a Chipmunk Aircraft. Aerospace Med., vol. 33, 1962, pp. 1224–1229.

Perception of the Upright and Susceptibility to Motion Sickness as Functions of Angle of Tilt and Angular Velocity in Off-Vertical Rotation*

EARL F. MILLER II and ASHTON GRAYBIEL
Naval Aerospace Medical Research Laboratory

SUMMARY

Motion sickness susceptibility of four normal subjects was measured in terms of duration of exposure necessary to evoke moderate malaise (M IIA) as a function of velocity (2.5 to 45 rpm) in a chair rotated about a central axis tilted 10° with respect to gravitational upright. The subjects had little or no susceptibility to this type of rotation at 2.5 and 5.0 rpm, but with further increases in rate, the M IIA endpoint was always reached and with ever shorter test durations. Minimal provocative periods for all subjects were found at 15 or 20 rpm. Higher rotational rates dramatically reversed the vestibular stressor effect, and the subjects as a group tended to reach a plateau of relatively low susceptibility at 40 and 45 rpm. At these higher velocities, furthermore, the subjects essentially lost their sensation of being tilted off vertical. In the second half of the study, the effect of tilt angle was varied while the rotation rate was maintained at a constant 17.5 rpm. Two subjects were completely resistant to symptoms of motion sickness when rotated at 2.5° off vertical; with greater off-vertical angles, the susceptibility of all subjects increased sharply at first, then tapered off in a manner reflecting a Fechnerian function. The marked changes in these measured responses were attributed primarily to the macular organs being unnaturally stimulated by off-vertical rotation.

INTRODUCTION

Constant-speed rotation of a subject about his longitudinal axis which has been slightly tilted with respect to gravity produces an unusual and ever-changing pattern of stimulation (refs. 1 and 2). The effect is equivalent to holding the subject stationary in an upright position, with the ability to rotate an acceleration vector around him at an off-vertical angle of incidence equal to the chair's tilt. This mode of stimulation has proven to be highly effective in evoking symptoms that characterize motion sickness. Theoretically, it provides adequate stimulation to the otolith and other gravireceptor organs, but probably not to the semicircular canals. This technique may therefore offer a simple, precise, and highly controllable method of grading motion sickness susceptibility to otolithic stimulation and one that complements those susceptibility tests in which the semicircular canals are the primary or the only otic structures involved (refs. 3 and 4).

Evidence from testing a few highly resistant subjects indicated that a greater provocative effect was derived from increasing the off-vertical angle from 10° to 20° at various rotational rates, but the relative change in effectiveness was not explored (refs. 1 and 2). With the initial method of grading susceptibility a schedule of ever-increasing rotational rates was employed, which often unnecessarily prolonged the test duration and frequently caused a very rapid rise in symptomatology when the adequately stressful rate was finally reached (refs. 1 and 2). As a result, great care had to be exercised to prevent the overshoot of a preselected endpoint, a motion-sickness diagnostic criterion of mild severity (M IIA) (ref. 5). In addition, the original test method exposed the subject to periods of incremental increases in the vestibular stressor level. Low

*This study was supported by contract T–81633, Biomedical Research Office, Manned Spacecraft Center, and by order L–43518, Office of Advanced Research and Technology, NASA.

levels of stressor stimulation were usually compensated initially, and in the process might have served as training toward increasing adaptation, an undesirable factor when determining baseline and relative measurement of susceptibility among subjects.

The purposes of this study were: first, to explore the change in provocative effect of varying the rate of rotation from 2.5 to 45 rpm about a slight (10°) off-vertical axis; and second, by using a velocity within the range of maximum effectiveness, to measure the relationship between motion sickness susceptibility and varying degrees of off-vertical tilt up to 25°.

PROCEDURE

Subjects

Four young Navy enlisted men, ranging in age from 19 to 21 years, who had demonstrated motion sickness susceptibility to off-vertical rotation, volunteered as subjects. Each was found to be healthy by a comprehensive Navy medical examination, given before his acceptance as a research subject, and remained so during the experimental procedure, as reported daily in his preexamination questionnaire (ref. 3). Functional tests of the semicircular canals (ref. 6) and otolith organs (refs. 7 and 8) plus those of postural equilibrium (ref. 9) proved further that these specific systems were functioning well within normal limits.

Methods

A standard Stille rotating chair (model RS–3), mounted on a motor-driven tilt base served as an off-vertical rotation (OVR) chair (fig. 1). The degree of tilt relative to the gravitational upright was registered on a large protractor scale. The subject's head was centered on the axis of rotation and held rigidly against the headrest by adjustable straps across his forehead. Seat belts further secured him to the chair. His eyes were covered with a small padded goggle which prevented vision but did not interfere with the observation by the test conductor of flushing, pallor, and sweating, which are most clearly manifested on the face. The combined weight of the subject and the chair superstructure was statically balanced in a 20° off-vertical position to ensure constant-speed rotation (±0.1 percent) during the OVR test. The chair was then returned to

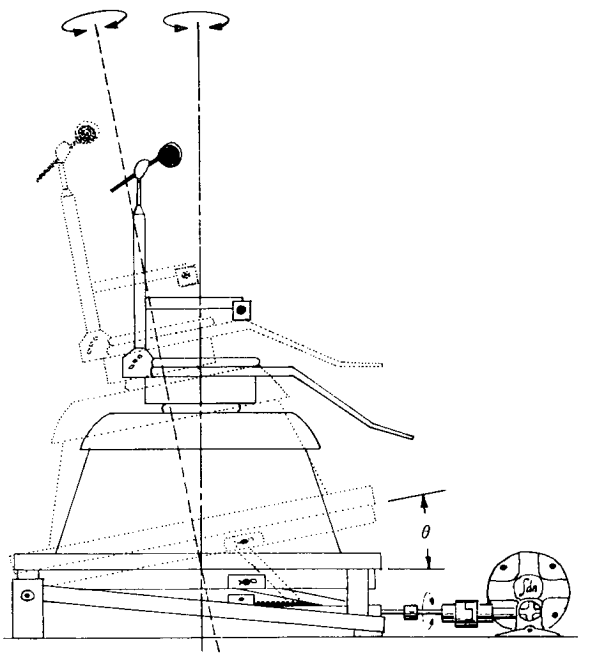


FIGURE 1.—*Diagram of apparatus used in off-vertical rotation test.*

upright and accelerated at 5°/s² until one of several selected terminal velocities (2.5, 5, 10, 15, 20, 25, 30, 40, or 45 rpm) was reached. After no less than 60 seconds' duration the chair was tilted at 5°/s to a tilt position selected from among 2.5, 5, 7.5, 10, 15, 20, and 25 degrees. Off-vertical rotation was continued until moderate malaise (M IIA) was manifested or the time limit of 1 hour had elapsed. If the malaise endpoint was reached, the chair was quickly tilted to the upright, which immediately abolished the stressor stimulus, and decelerated at 5°/s².

Initially, the effect of the chair velocity in an off-vertical position was tested by exposing each subject to a random schedule of the listed test velocities, while maintaining in each case a 10° tilt of the rotational axis from upright. Each subject was tested twice, once in the clockwise and once in the counterclockwise direction of rotation, at each velocity. This procedure was followed by one in which the same subjects were tested once at each of the tilt angles, introduced in random order, while rotating at a constant velocity of 17.5 rpm. That value, based upon an ongoing analysis of results from the first half of this study, was selected

as representing the best estimate of a single rate of rotation which would produce a nearly maximum provocative effect. The scheduled order of presentation of each velocity and tilt angle was randomized not only for each subject, but also among subjects. Although the overt symptoms of M IIA quickly disappeared, at least 24 hours separated the individual trials.

RESULTS

Tolerance of subjects to off-vertical rotation as reflected by the duration required to evoke M IIA is plotted for the four subjects in figure 2 as a function of chair velocity (rpm). It was possible to draw an average subject response curve (solid line) only between 10 and 25 rpm, since the M IIA endpoint was not reached at the other velocities by all subjects. This curve section, however, was extended in an idealized fashion (dotted lines) in both directions in figure 2 to portray the marked general changes in response throughout the entire range of test velocities. SC, the most susceptible subject, reached the endpoint criterion (M IIA) with an average of 43 minutes' exposure at 5 rpm but remained symptomless throughout the 1-hour maximum test period at 2.5 rpm. At 5 rpm subjects TE and BE were unaffected and subject WI reached M IIA just prior to the end of the test. Increasing the velocity from 5 to 10 rpm evoked the endpoint in all subjects, three within 20 minutes. An even greater provocative effect was found at 15 rpm, with all subjects manifesting M IIA within 15 minutes' exposure. The provocative effect, however, could not be enhanced substantially by greater rates of rotation, and in fact at 25 rpm the stressor effect appeared to be lessened. This trend continued while progressing from 30 to 45 rpm. Increasingly longer durations were required to evoke the endpoint, which was not always reached even in the most susceptible subject (SC).

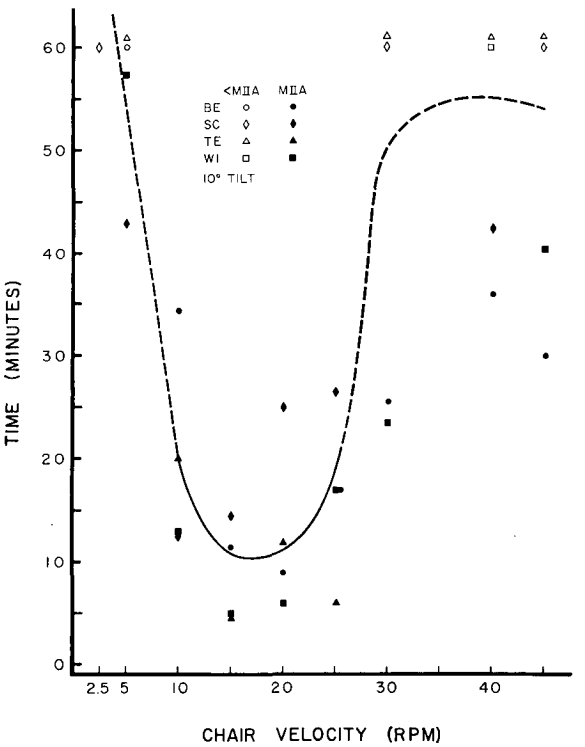


FIGURE 2.—*Subjects' motion sickness susceptibility measured in terms of duration of off-vertical (10°) rotation required to reach the test endpoint (malaise IIA or 60 minutes) as a function of rotational rate (rpm).*

The subject's impressions of bodily movement in space also varied with the rate of rotation. Each subject had the sensation of revolving rather than rotating and in a direction opposite the actual rotation. That is, his head seemed to be moving in a circular path centered on the rotational axis and to be directed always essentially in the same compass direction. At rates below 30 rpm, the subjects reported a smooth revolving bodily movement that generated an inverted cone; the base was traced by the head, and apex usually was located within the area of contact with the chair seat. At a rate of 30 rpm, all subjects felt tilted to some extent, as with the slower rates, but complained that this rate provided substantially "the most difficult ride," with a more pronounced sensation of rocking front to back or side to side as compared to the lower or higher rates. At rates higher than 30 rpm, all subjects began to lose their feeling of being tilted, and at 40 and 45 rpm, they reported "much easier" and "very smooth rides," with the sensation of being at or near upright throughout the period of exposure.

Figure 3 is a plot of the provocative effect, measured in the same terms as figure 2, as a function of the off-vertical angle of tilt at a constant rotation of 17.5 rpm. Two subjects were completely symptom free when rotated about an axis positioned 2.5° from the upright. With greater off-vertical angles, the provocative effect of rotation increased dramatically at first, then leveled off.

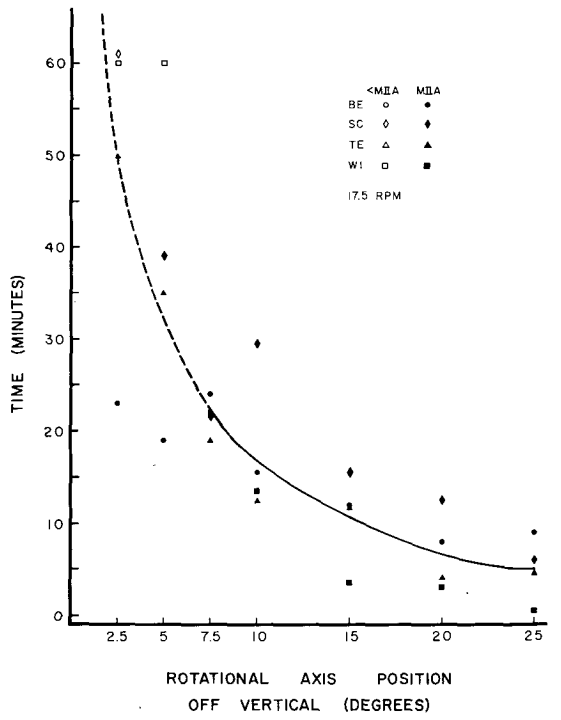


FIGURE 3.—*Subjects' motion sickness susceptibility measured in terms of duration of a constant rotational rate (17.5 rpm) required to reach the test endpoint (malaise IIA or 60 minutes) as a function of off-vertical placement of the rotational axis.*

DISCUSSION

The sweep of the rotating linear acceleration vector (RLAV) stimulated all gravireceptors, but the provocative effect of the RLAV was probably primarily dependent upon its unusual activation of the otolith organs. In a constant off-vertical position the rotational velocity was changed to vary the sweep rate of a constant force in an essentially identical spatial pattern. In the complementary situation a constant velocity was maintained while the angle of incidence of the RLAV with respect to the subject varied with the degree of tilt.

The remarkable decrease and subsequent increase in tolerance that resulted from step increases in off-vertical rotational rate provided another example of the importance of the frequency as well as the intensity and pattern of the stimulus in the production of motion sickness. Wendt (ref. 10) found, for example, that a medium-frequency linear wave motion of 16 to 22 cpm was most effective in making men sick, but that there was a sharp decrease at subsequent higher frequencies. At the low rotational rates used in our study, normal physiological conditions were approximated, and, as expected, most subjects experienced little or no vestibular stress effect. With moderate rates all subjects were found to be susceptible, and their susceptibility over a narrow span of velocities increased precipitously to a critical value. With higher rotational velocities the paradoxical increase in tolerance to off-vertical rotation was found. The slight increase in the provocative effect at 45 rpm from that seen at 40 rpm and the extent to which the more rapid rotational rates failed to eliminate the evocation of motion sickness symptomatology may indicate the possible contribution of nonotolithic proprioceptors that were also stimulated by the RLAV. It is noteworthy, however, that the most susceptible subject (SC), who reached the M IIA endpoint even at 5 rpm, was symptom free at 30 and 45 rpm.

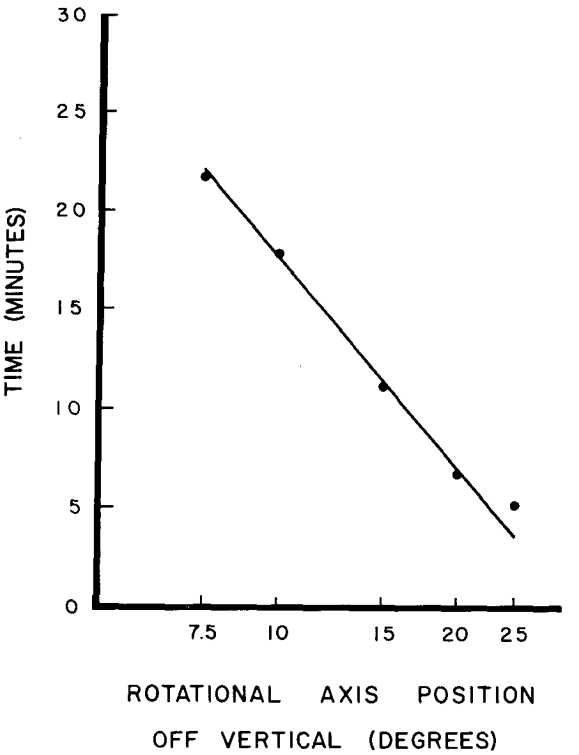


FIGURE 4.—*Linear relationship between duration of exposure required to evoke malaise IIA and logarithmically scaled off-vertical position of axis for constant rotation at 17.5 rpm.*

Figure 4 displays on a logarithmic base the response data of figure 3 within the range of off-vertical angles that was found effective in evoking M IIA in all subjects. The excellent empirical fit of these data with a straight line indicates that the stressor value of a relatively low constant speed (17.5 rpm) varies directly with the axis tilt angle in accordance with Fechner's Law. The nonotolithic receptors, which are also responsive to these experimental conditions, represent secondary stressor influences since they, per se, cannot evoke motion sickness (refs. 2 and 4). The primary genesis of the changes in the provocative effect as a function of off-vertical displacement of the rotational axis (RLAV angle) must therefore be vestibular. More specifically, this effect very likely involves the bizarre activity of cilio-otolith elements, which would tend to respond slavishly to the ever-changing direction of the acceleration vector sweep. If such otolithic activity is the basis for the Fechnerian function found, then it would follow that the change in stressor effect that occurs with simply a shift in off-vertical rotational position (at least within the range tested in this study) would be dependent upon the integrated change in the amount of deformation of the macular sensory hairs, coded in logarithmic terms, in response to the constant dynamic pattern of stimulation.

REFERENCES

1. GRAYBIEL, A.; AND MILLER, E. F. II: Off-Vertical Rotation: A Convenient Precise Means of Exposing the Passive Human Subject to a Rotating Linear Acceleration Vector. Aerospace Med., vol. 41, 1970, pp. 407–410.
2. GRAYBIEL, A.; AND MILLER, E. F. II: The Otolith Organs as a Primary Etiological Factor in Motion Sickness: With a Note on "Off-Vertical" Rotation. Fourth Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–187, 1970, pp. 53–64.
3. MILLER, E. F. II; AND GRAYBIEL, A.: A Provocative Test for Grading Susceptibility to Motion Sickness Yielding a Single Numerical Score. Acta Oto-Laryngol., suppl. 274, 1970, pp. 1–20.
4. MILLER, E. F. II; AND GRAYBIEL, A.: The Semicircular Canals as a Primary Etiological Factor in Motion Sickness. Fourth Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–187, 1970, pp. 69–82.
5. GRAYBIEL, A.; WOOD, C. D.; MILLER, E. F. II; AND CRAMER, D. B.: Diagnostic Criteria for Grading the Severity of Acute Motion Sickness. Aerospace Med., vol. 39, 1968, pp. 453–455.
6. MCLEOD, M. E.; AND MEEK, J. C.: A Threshold Caloric Test: Results in Normal Subjects. NSAM–834, Naval School of Aviation Medicine, Pensacola, Fla., 1962.
7. MILLER, E. F. II: Counterrolling of the Human Eyes Produced by Head Tilt With Respect to Gravity. Acta Oto-Laryngol., vol. 54, 1962, pp. 479–501.
8. MILLER, E. F. II: Ocular Counterrolling. The Vestibular System and Its Diseases, R. J. Wolfson, ed., University of Pennsylvania Press, 1966, pp. 229–241.
9. GRAYBIEL, A.; AND FREGLY, A. R.: A New Quantitative Ataxia Test Battery. Acta Oto-Laryngol., vol. 61, 1966, pp. 292–312.
10. WENDT, G. R.: Experiences With Research on Motion Sickness. Fourth Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–187, 1970, pp. 29–32.

Brain Blood-Flow Changes During Motion Sickness

WALTER H. JOHNSON and JOHN HSUEN
University of Toronto

SUMMARY

Speculation as to the possibility of diminished blood flow in the brain as one of the factors resulting from an increase in skeletal muscle blood volume concomitant with other characteristics of motion sickness led to the present investigation. Thermistors were implanted in the thalamus of dogs and blood-flow changes recorded while they were subjected to sinusoidal movement on a two-pole swing. Results of these initial steps in a proposed long-term project of exploring different areas of the brain are presented.

INTRODUCTION

The well-recognized signs of motion sickness (cold perspiration, yawning with sleepiness, salivation, hyperventilation, skin color changes, nausea, and sometimes vomiting) are undoubtedly indicative of extensive internal alterations of the normal physiology, resulting in a generalized feeling of malaise. Of these, skin pallor is consistently recognized as a prominent symptom, although recent work by McClure (personal communication) indicates that it is more correctly associated with the later stages of nausea. In any case, because of the autonomic activity associated with motion sickness, various investigators have attempted to correlate susceptibility with cardiovascular changes (refs. 1 to 4). Although such studies have failed to establish any significant relationship, skin pallor undoubtedly results from extensive blood-volume redistribution, a conclusion which became evident when attempts were made to quantify the cutaneous effects by venous occlusion plethysmograph techniques (ref. 2). These latter studies also included a comparison between susceptible and nonsusceptible subjects in regard to pulse rate and blood pressure, using various methods including that of direct arterial puncture. It is the object of this presentation to review our preliminary findings and to hypothesize as to possible consequences.

METHODS AND RESULTS

Full details of the venous occlusion plethysmograph procedure have been presented previously (ref. 2), and the significant findings can be summarized as follows:

> Motion-sickness-susceptible subjects demonstrated increased forearm blood flow with the onset of nausea, the degree of which correlated with the severity of the symptoms. In fact, it was often possible by observing the blood-flow increase to predict a signal from the subject that he would soon vomit if the stimulation continued.

Simultaneous measurements showed that the effect involved both forearms and legs. This fact indicates that such volume changes are widespread and may well involve all skeletal masculature.

In general terms, an increase in blood flow through one part of the body must be compensated either by decreased blood flow elsewhere or by an increase in cardiac output; otherwise, reduced arterial blood pressure will result. The human forearm is 85 percent muscle by volume, and skeletal muscle represents 50 percent of the total body mass. If one can assume that the blood flows of the forearm and calf are representative of the total body muscle blood flow, then a slight increase in these

flows must be compensated by a prompt substantial cardiovascular adjustment if normal blood pressure is to be maintained. Since there is little or no apparent change in blood pressure or in the heart rate concomitant with increased flow, in order to maintain this blood pressure it may well be that an increased vasoconstriction in other regions of the body results. That this occurs is evidenced by the marked pallor characteristic of sick subjects. However, even the total cessation of skin blood flow would account for only a small part of the two- to threefold increase that occurs. This led us to speculate as to the possibility of diminished blood flow in the brain as one of several contributing factors. Certainly some physiologic evidence exists for such impairment as evidenced by the symptoms of yawning, sleepiness, and malaise, to name a few.

To investigate this possibility, attempts were made to implant sensors directly into vascular areas of the brain. Dogs were chosen because of their known susceptibility to experimentally produced motion sickness. Appropriate stimulation was induced by the sinusoidal movement of a two-pole simple swing (radius 4 m, arc 80°, and frequency 20 cpm) suspended from the ceiling of the laboratory. The effectiveness of this procedure is evident from the fact that it induced vomiting within 30 minutes in seven of the eight dogs used. A special jacket and harness maintained the dog in the standing position and so assured freedom of head movement, a necessity for appropriate vestibular stimulation.

In order to record blood-flow changes, a thermistor was implanted into the thalamus (a highly vascular area of the brain) with the aid of a stereotaxic device.

In the first dog tested, at the start of swinging the brain temperature fell progressively, starting from the time of onset of excessive salivation. The final minimal level was maintained for 3.5 minutes, and then there was a sudden and definite rise in temperature (decrease in blood flow) immediately prior to and at the onset of vomiting. The swing was then stopped, and the brain temperature gradually returned to its original baseline. The sequence of these changes (excluding return to baseline) is shown in figure 1.

This experiment was repeated on another dog with the thermistor implanted into the thalamus as before and the dog subsequently subjected to swinging. On this occasion, salivation resulted, but no vomiting occurred. The temperature of the thalamus dropped somewhat at the time of salivation. These results are shown in figure 2.

This dog was retested 5 days later, and on this occasion both salivation and vomiting resulted (fig. 3). When the first symptoms indicating the onset of nausea appeared, the temperature of the thalamus dropped as before. After the vomiting, the brain temperature increased somewhat, although the change was not so marked as in the first animal (cf. fig. 1). There is evidence to indicate, however, that the first dog was more susceptible.

The use of a thermistor in these experiments involved measurement of change in resistance of the implanted sensor resulting from alteration in temperature at the location chosen. Another procedure that could be used to measure blood-flow changes consists of the implantation of a thermocouple.

CONCLUSIONS

From the recordings obtained thus far, the brain vascular changes accompanying motion sickness

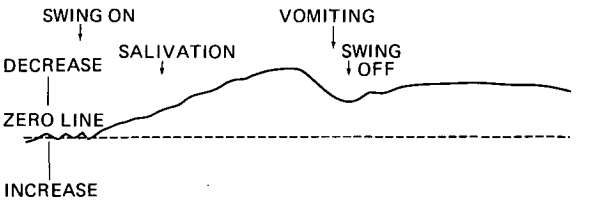


FIGURE 1.—*Temperature recording with thermistor implanted in brain (thalamus) of male dog. Decrease in temperature indicates increase in blood flow.*

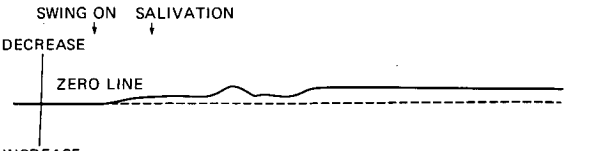


FIGURE 2.—*Recording from thermistor implanted in brain (thalamus) of second male dog.*

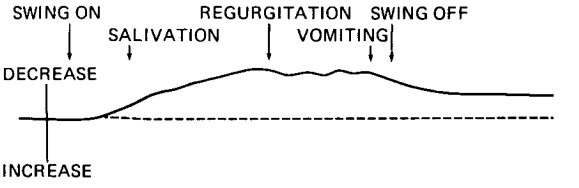


FIGURE 3.—*Repeat recording of second dog.*

appear to consist of an initial increase in flow with the onset of symptoms (salivation), followed by a relative decrease in blood flow at the time of vomiting; following this, there is a further increase in blood flow, which then returns gradually to the pretest level. It is felt that more testing with smaller probes involving other areas of the brain is required to establish whether or not brain blood-flow changes should be added to the list of internal physiologic changes contributing to the syndrome of motion sickness.

REFERENCES

1. MONEY, K. E.: Motion Sickness. Physiol. Rev., vol. 50, 1970, pp. 1–39.
2. SUNAHARA, F. A.; JOHNSON, W. H.; AND TAYLOR, N. B. G.: Vestibular Stimulation and Forearm Blood Flow. Can. J. Physiol. Pharmacol., vol. 42, 1964, pp. 199–207.
3. TAYLOR, W. J. R.; JOHNSON, W. H.; AND SELLERS, E. A.: Cardiovascular Changes with Vestibular Stimulation. Aerospace Med., vol. 3, 1960, pp. 627–638.
4. TYLER, D. B.; AND BARD, P.: Motion Sickness. Physiol. Rev., vol. 29, 1940, pp. 311–369.

Anti-Motion-Sickness Therapy*

CHARLES D. WOOD
Shreveport Medical School
Louisiana State University

SUMMARY

Neither alterations in environmental temperature nor moderate intake of alcohol was found to alter susceptibility to motion sickness in subjects exposed to rotation in the Pensacola slow rotation room.

Scopolamine with *d*-amphetamine was found to be the most effective preparation for the prevention of motion sickness under the experimental conditions of the studies reported here. Promethazine in combination with *d*-amphetamine was in the same range of effectiveness. Drug actions suggest that acetylcholine and norepinephrine may be involved in motion sickness.

INTRODUCTION

In the fourth of this series of symposiums, the possible mechanisms of motion sickness and factors influencing susceptibility to it were reviewed by such authorities as Wendt, Johnson, Steele, and Sjöberg. It is sufficient to state here that a number of factors have been suggested which may alter susceptibility to motion sickness. A partial list of these factors includes temperature, alcohol, drugs, radiation, weightlessness, altitude, fever, psychological factors, and habituation (refs. 1 to 3). Various toxic and pathological conditions, especially those involving the liver, kidneys, or gastrointestinal tract, also may lower the threshold. There is scientific evidence that some nausea-producing conditions will summate with vestibular stimulaton to make an individual more prone to motion sickness (ref. 4). The effectiveness against motion sickness of a large number of substances, as demonstrated by individual case reports in the literature, may be influenced by one or more of the abovementioned factors. For example, several vitamin preparations have produced favorable results in individual cases; however, when these preparations were tested under scientifically controlled conditions on large populations, they were found to be ineffective (refs. 3, 5 and 6). It is possible that some factor may be decreasing the threshold for nausea and that these preparations have produced their favorable effects by an indirect effect rather than a direct effect on motion sickness.

TESTS

Alcohol has long been associated with motion sickness. Overindulgence certainly can produce nausea and is also suspected of diminishing resistance to motion sickness. Smaller doses of alcohol have been reported to increase vestibular sensitivity and to produce nystagmus. Fregly and Graybiel have demonstrated loss of ability to walk on a rail in the acute phase of alcohol intoxication (ref. 7). Since alcohol is a central depressant that appears to affect vestibular activity, it was used to investigate its effect on susceptibility to motion sickness in the slow rotation room (SRR).

Eight subjects were given doses of 86-proof (43-percent alcohol) vodka in orange juice in amounts of 0, 0.2, 0.3, and 0.5 ml/kg body weight. These doses were calculated to produce an approximate blood alcohol level of 0, 40, 60, and 100 mg%. The alcohol was given on an empty stomach, and

*This study was carried out at the Naval Aerospace Medical Research Laboratory, Pensacola, Florida, under the sponsorship of the Office of Advanced Research and Technology, NASA.

the subjects were given a 30-minute rest period to assure maximum absorption before being subjected to rotation. The effectiveness of the alcohol on susceptibility to motion sickness was measured by the number of head movements tolerated. The results are shown in table 1.

This investigation indicates that a moderate intake of alcohol has little if any effect on susceptibility to motion sickness. There was no significant difference produced by any of the doses of alcohol. This one small study indicates that, unless the alcohol itself produces nausea, moderate amounts of alcohol have little effect on the development of motion sickness. Numerous "independent investigators" can attest to the fact that the effects of exposure to motion on the morning after a period of overindulgence will produce an increased sensitivity to motion sickness.

Another factor that has been investigated is environmental temperature. In our project, subjects were exposed to three conditions: 15.5 °C with relative humidity of 50 percent, 23.3 °C with relative humidity of 60 percent, and 35 °C with relative humidity of 90 percent. Each subject was exposed to these conditions for a period of from 10 to 45 minutes while he performed head movements at his baseline speed of rotation. The results indicated no significant difference in sensitivity to motion at any of these temperature (fig. 1). The structures concerned with development of motion sickness are in the body-core-temperature areas. The temperature regulatory mechanisms of the body effectively prevent changes in the internal temperature. Prolonged exposure to high temperatures is required to elevate the body-core temperature. Fever is suggested as a factor that increases susceptibility to motion sickness, although the reports in the literature furnish no support for this proposition. This study confirms a previous report (ref. 8) that environmental temperature is not a factor in susceptibility to motion sickness; however, effects of exposure to temperatures high enough to alter the body-core temperature were not determined.

TABLE 1.—*Effects of Moderate Doses of Alcohol on Susceptibility to Motion Sickness*

Vodka/kg body weight	0	0.2 ml	0.3 ml	0.5 ml
SRR sequences tolerated	14	18	16	16
Best response	3	2	1	2
Approximate blood alcohol	0	40 mg%	60 mg%	100 mg%

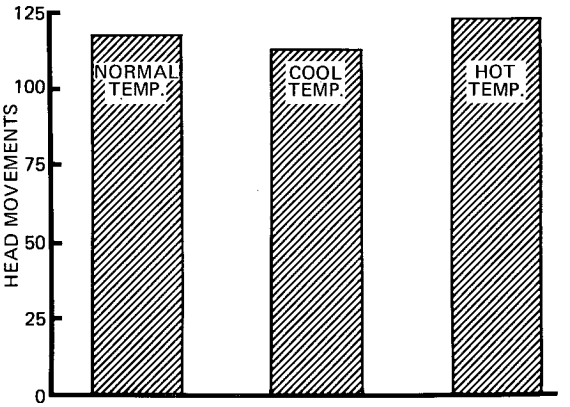


FIGURE 1.—*Effect of environmental temperature on susceptibility to motion sickness.*

PREVENTION AND TREATMENT

The treatment of motion sickness after it has developed is very difficult to assess. Therefore, few definitive studies on the therapy of motion sickness are available. Most of the current treatments are reasonably based on the drugs demonstrated to be effective in preventing the development of motion sickness. The oral route is, of course, ineffective due to the vomiting; however, benefit has been reported from injections or rectal suppositories.

A number of drugs have been tested against motion sickness in the SRR (refs. 9 and 10). The relative effectiveness of these drugs is shown in figure 2. Many drugs are known to produce nausea and, therefore, possibly could increase susceptibility to motion sickness. Morphine and apomorphine apparently act on the chemoreceptor trigger zone by blocking cholinesterase, the enzyme that breaks down acetylcholine. Morphine has been reported by Rubin and Winston (ref. 4) to increase susceptibility to motion sickness produced by rotation in a Bárány chair; the nausea-producing stimuli thus appeared to summate. Dramamine (100 mg) was reported to be effective in preventing the development of this nausea. Physiostigmine is another drug that protects acetylcholine in the central nervous

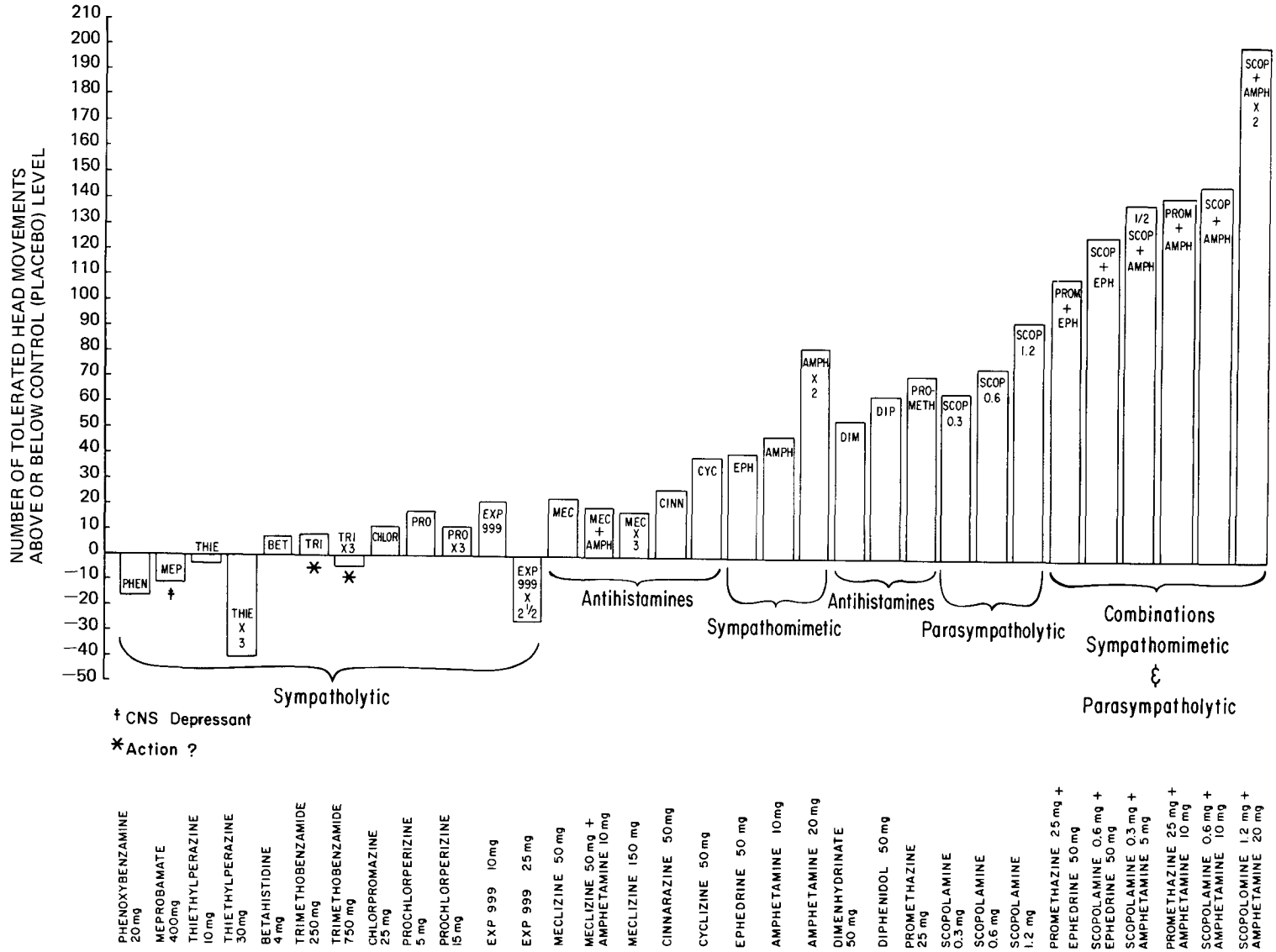


FIGURE 2.—*Relative effectiveness of the drugs tested against motion sickness in the SRR. The drugs are arranged in order of effectiveness of the usually recommended dose, with increased doses and combinations shown adjacent to the recommended dosage. Drug classifications are shown by brackets. Data were gathered from 60 subjects exposed on 500 occasions in a rotating environment using a standardized pattern of head movements. Endpoint for toleration was malaise III.*

system and may produce nausea by allowing it to increase in the vomiting center.

There is considerable evidence that the neurons involved in vestibular reactions are mediated by acetylcholine (refs. 11 to 13). The drugs that block acetylcholine centrally, such as scopolamine, are the most effective drugs for prevention of motion sickness (refs. 3, 5, and 6). Promethazine, the second most effective single drug, has strong central anticholinergic activity and, as a group, the antihistamines are known to have a definite "atropine-like" effect. This central anti-cholinergic activity of the antihistamines is illustrated by the work of Himwich and colleagues with unilateral injections of diisopropylfluorophosphate (DFP) into the cerebral circulation (ref. 14). DFP is a strong blocker of cholinesterase, and the forced circulating produced by the accumulation of acetylcholine in the nervous system was prevented by antihistamines with strong central anticholinergic activity. Antihistamines with weak anticholinergic action failed to block this reaction. They were also ineffective against motion sickness, while those of the stronger group were effective (ref. 6). This evidence indicates that acetylcholine is important in vestibular reactions and motion sickness.

The area of the brain-stem reticular system adjacent to the vestibular nuclei and vomiting center (fig. 3) has been demonstrated to be involved with vestibular impulses (refs. 15 to 19). This area of the reticular system and the vestibular nuclei has been shown to contain neurons that respond only to acetylcholine, intermingled with others that are responsive only to norepinephrine (refs. 20 to 23).

There is fairly general acceptance of the role of acetylcholine and the parasympathetic system in the development of motion sickness. As mentioned above, cells responsive to norepinephrine also appear to be involved (refs. 24 to 28). Tang and Gernandt (ref. 29) have reported strong sympathetic responses resulting from electrical activation of the vestibular nerve. A number of drugs involving central sympathetics and norepinephrine have been tested for effects on motion sickness. The drugs that block sympathetic activity and norepinephrine, such as the phenothiazines and rauwolfia have been reported to increase susceptibility to motion sickness (ref. 6). This was also true of phenoxybenzamine, which is a sympathetic blocking

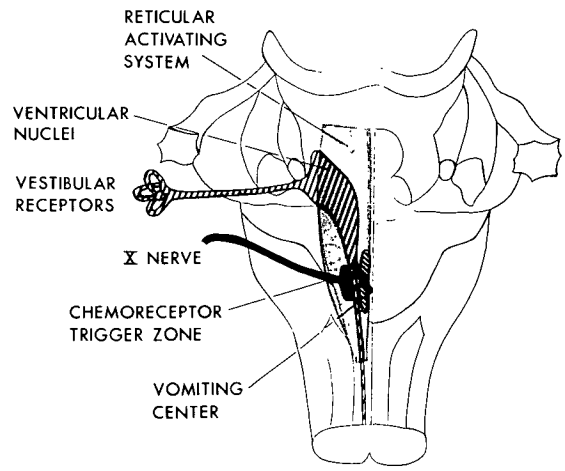


FIGURE 3.—*Spatial relationship of the vomiting center and chemoreceptor trigger zone with the reticular activating system.*

agent with central activity (ref. 10). Drugs that activate the sympathetic system, such as amphetamine, have been reported to protect against the development of motion sickness (ref. 9). In our research, *d*-amphetamine was equal in effectiveness to the midrange of the antihistamines, and ephedrine was somewhat less effective (ref. 10).

The combination of a drug that blocked acetylcholine centrally with a drug that enhanced norepinephrine activity produced the most effective antimotion sickness preparation. This combination was *d*-amphetamine 10 mg with scopolamine 0.6 mg. (The anti-motion-sickness drug used on the Apollo 11 flight was one-half this dosage.) Promethazine 25 mg with *d*-amphetamine 10 mg was only slightly less effective. When ephedrine was substituted for *d*-amphetamine, the effectiveness diminished; it was still well above those of the individual drugs, however. These results suggest that both the acetylcholine and norepinephrine systems are involved in motion sickness.

The SRR provides an excellent laboratory setting for observing the waxing and waning of symptoms as motion sickness develops (ref. 30). The subject may flush, then become pale; his mouth may become dry, or there may be an increase in salivation; stomach awareness may build up and diminish two or three times before motion sickness develops. Both sympathetic and parasympathetic reactions are

seen as motion sickness develops. (They may be byproducts of the central activation of acetylcholine and norepinephrine.)

The fact that the same drugs are effective against Parkinsonism and against motion sickness has often been discussed. The current theory that Parkinsonism results from an imbalance between dopamine and acetylcholine suggests a similar mechanism for motion sickness. Our drug study results suggest that the balance between acetylcholine and norepinephrine may be involved in motion sickness.

CONCLUSIONS

Of the preparations currently available for prevention of motion sickness, the antihistamines appear to be the most satisfactory for mild conditions, such as automobile or commercial air travel. Exposure to more extreme conditions, such as at sea, would indicate scopolamine, promethazine, or the combination of either of these drugs with *d*-amphetamine (refs. 31 and 32). The combination of scopolamine with *d*-amphetamine was consistently the most effective preparation tested in our investigations. Recently, promethazine with amphetamine was tested, and it proved to be in the same range of effectiveness. Because it has fewer disturbing side effects, it may prove to be more useful than the combination containing scopolamine. The hypnotic effect of promethazine may aid sleep if it is given without the amphetamine. Very susceptible individuals may require the more potent preparations even for mild conditions; however, it is well to remember that these drugs only raise the threshold for development of motion sickness and do not prevent it entirely in extreme conditions.

REFERENCES

1. GRAYBIEL, A.: Structural Elements in the Concept of Motion Sickness. Aerospace Med., vol. 40, 1969, pp. 351–367.
2. MONEY, K. E.: Motion Sickness. Physiol. Rev., vol. 50, 1970, pp. 1–39.
3. TYLER, D. B.; AND BARD, P.: Motion Sickness. Physiol. Rev., vol. 29, 1949, p. 311–369.
4. RUBIN, A.; AND WINSTON, J.: The role of the Vestibular Apparatus in the Production of Nausea and Vomiting Following the Administration of Morphine to Man. J. Clin. Invest., vol. 29, 1950, pp. 1261–1266.
5. BRAND, J. J.; AND PERRY, W. L. M.: Drugs Used in Motion Sickness: A Critical Review of the Methods Available for the Study of Drugs of Potential Value in its Treatment and of the Information Which Has Been Derived by These Methods. Pharmacol. Rev., vol. 18, 1966, pp. 895–924.
6. CHINN, H. I.: AND SMITH, P. K.: Motion Sickness. Pharmacol. Rev., vol. 7, 1955, pp. 33–82.
7. FREGLY, A. R.; AND GRAYBIEL, A.: Acute Alcohol Ataxia in Relation to Vestibular Function. Acta Oto-Laryngol., vol. 65, 1968, pp. 468–478.
8. JOHNSON, C.; AND WENDT, G. R.: Studies of Motion Sickness: XVII. The Effects of Temperature, Posture, and Wave Frequency upon Sickness Rate. J. Psychol., vol. 39, 1955, pp. 423–433.
9. WOOD, C. D.; GRAYBIEL, A.; AND MCDONOUGH, R. C.: Human Centrifuge Studies on the Relative Effectiveness of Some Antimotion Sickness Drugs. Aerospace Med., vol. 37, 1966, pp. 187–190.
10. WOOD, C. D.; AND GRAYBIEL, A.: Evaluation of sixteen Antimotion Sickness Drugs Under Controlled Laboratory Conditions. Aerospace Med., vol. 39, 1969, pp. 1341–1344.
11. BRADLEY, P. B.; AND ELKES, J.: The Effects of Some Drugs on the Electrical Activity of the Brain. Brain, vol. 80, 1957, pp. 70–117.
12. SALMOIRAGHI, G. C.; AND STEINER, F. A.: Acetylcholine Sensitivity of Cats' Medullary Neurons. J. Neurophysiol., vol. 26, 1963, pp. 581–597.
13. YAMAMOTO, C.: Pharmacological Studies of Norepinephrine, Acetylcholine and Related Compounds on Neurons in Deiters' Nucleus and Cerebellum. J. Pharmacol. Exptl. Therap., vol. 156, 1966, pp. 39–47.
14. HIMWICH, W. A.; COSTA, E.; CANHAN, R. G.; AND GOLDSTEIN, S. L.: Isolation and Injection of Selected Arterial Areas of the Brain. J. Appl. Physiol., vol. 15, 1960, pp. 303–306.
15. EVIATOR, A.; AND GOODHILL, V.: Comparative Sequential Studies of Effects of Drugs on the Vestibular System of Laboratory Animals. Acta Oto-Laryngol. Suppl. 237, 1968, pp. 1–28.
16. GERNANDT, B. E.; AND THULIN, C. A.; Vestibular Connections of the Brain Stem. Am. J. Physiol., vol. 171, 1932, pp. 121–127.
17. MANNI, E.; AZZENA, G. B.; AND CASEY, H.: Influence of the Labyrinth on Unitary Discharge of the Oculomotor Nucleus and Some Adjacent Formations. Exptl. Neurol., vol. 12, 1965, pp. 9–24.
18. SPIEGEL, E. A.; SZEKELY, E. G.; AND GILDENBERG, P. L.: Vestibular Responses in Midbrain, Thalamus and Basal Ganglia. Arch. Neurol., vol. 12, 1965, pp. 258–269.
19. YULES, R. B.; AND GAULT, F. P.: The Relationship of Nystagmus to Lateral Vestibular Nucleus Stimulation. Exptl. Neurol., vol. 15, 1966, pp. 475–485.
20. TUCKER, G. J.; AND REINHARDT, R. F.: Airsickness and Anxiety. Aerospace Med., vol. 38, 1967, pp. 855–858.
21. WILSON, V. J.; KATO, M.; AND THOMAS, R. C.: Excitation of Lateral Vestibular Neurones. Nature, vol. 206, 1965, pp. 96–97.

22. YULES, R. B.; KREBS, C. Q.; AND GAULT, F. P.: Reticular Formation Control of Vestibular System. Exptl. Neurol., vol. 16, 1966, pp. 349–358.
23. YULES, R. B.: Motion Sickness and the Reticular System. Arch. Otolaryngol., vol. 86, 1967, pp. 355–356.
24. AXELROD, J.: The Effect of Psychotropic Drugs on the Uptake of H^3-Norepinephrine by Tissues. Science, vol. 133, 1961, pp. 383–384.
25. COLLINS, W. E.; AND POE, R. H.: Amphetamine Arousal and Human Vestibular Nystagmus. J. Pharmacol. Exptl. Ther., vol. 138, 1962, pp. 120–125.
26. LOIZOU, L. A.: Rostral Projections of Noradrenaline-Containing Neurones in the Lower Brain Stem. J. Anat., vol. 104, 1969, p. 593.
27. ROTHBALLER, A. B.: The Effects of Catecholamines on the C.N.S. Pharmacol. Rev., vol. 11, 1939, pp. 494–547.
28. VOGT, M.: The Concentration of Sympathin in Different Parts of the Nervous System. J. Physiol., vol. 123, 1954, pp. 451–481.
29. TANG, P. C.; AND GERNANDT, B. E.: Autonomic Responses to Vestibular Stimulation. Exptl. Neurol., vol. 24, 1969, pp. 558–578.
30. GRAYBIEL, A.; WOOD, C. D.; MILLER, E. F. II; AND CRAMER, D. B.: Diagnostic Criteria for Grading the Severity of Acute Motion Sickness. Aerospace Med., vol. 39, 1968, pp. 453–455.
31. DEANE, F. R.; WOOD, C. D.; AND GRAYBIEL, A.: The Effect of Drugs in Altering Susceptibility to Motion Sickness in Aerobatics and the Slow Rotation Room. Aerospace Med., vol. 38, 1967, pp. 842–845.
32. KENNEDY, R. S.; GRAYBIEL, A.; MCDONOUGH, R. C.; AND BECKWITH, F. D.: Symptomatology Under Storm Conditions in the North Atlantic in Control Subjects and in Persons with Bilateral Labyrinthine Defects. Acta Oto-Laryngol., vol. 66, 1968, pp. 533–540.

Artifacts Produced During Electrical Stimulation of the Vestibular Nerve in Cats

PEI CHIN TANG
Naval Aerospace Medical Research Laboratory

SUMMARY

Evoked potentials from various somatic and autonomic outflows have been reported as being elicited by electrical stimulation of the vestibular nerve. Evidence is presented to indicate that evoked potentials in the recurrent laryngeal, the cervical sympathetic, and the phrenic nerve, commonly reported as being elicited by vestibular nerve stimulation, may be due to stimulation of structures other than the vestibular nerve. Experiments were carried out in cats decerebrated by midbrain electrocoagulation, immobilized with Flaxedil, and maintained with artificial ventilation. Electrical stimulation was applied either through bipolar straight electrodes thrust against the wall of the vestibule in contact with the vestibular nerve (the technique commonly used for vestibular nerve stimulation) or through bipolar curved electrodes suspending the nerve above the bone with a new technique of isolating the nerve for stimulation while avoiding stimulation of the bone. Following transection and sealing of the cochlear and the vestibular nerves at the orifice of the internal auditory meatus, stimulation of the wall of the vestibule or any other part of the petrous bone continued to evoke potentials in the recurrent laryngeal and the cervical sympathetic nerves, indicating that the afferent impulses that evoke these potentials must be originating from structures other than the cochlear or the vestibular nerve. Stimulation of the suspended vestibular nerve failed to elicit these responses, indicating that the vestibular input cannot be responsible for these evoked potentials. Stimulation of the suspended vestibular nerve in nonparalyzed cats produced conjugate eye movements, indicating that the isolated vestibular nerve was not damaged during the dissection to free it from the bone. Thus, these findings indicate that stimulation of the petrous bone and not that of the vestibular nerve is responsible for the genesis of evoked potentials in the recurrent laryngeal and the cervical sympathetic nerves. The phrenic response to electrical stimulation applied through bipolar straight electrodes appears to be the result of stimulation of the facial nerve in the facial canal by current spread along the petrous bone, since stimulation of the suspended facial nerve evoked potentials only in the phrenic nerve and not in the recurrent laryngeal nerve. These findings indicate that autonomic components of motion sickness represent the secondary reactions and not the primary responses to vestibular stimulation.

INTRODUCTION

The somatic and autonomic responses to electrical stimulation of the vestibular nerve are very extensive. Vestibular-evoked potentials have been observed in the radial nerve and the ventral roots (ref. 1), in the oculomotor nerve and the reticular formation (ref. 2), in the vagus or the recurrent laryngeal nerve (refs. 3 to 7), in the sympathetic outflow (refs. 5, 7, and 8), in the phrenic nerve (refs. 4, 6, and 7), in the pyramidal tract (ref. 9), and on the cortex (refs. 8, and 10 to 13). Recently it was noted in our laboratory that evoked potentials in the recurrent laryngeal nerve (or the vagus nerve), the cervical sympathetic nerve, and the phrenic nerve, previously reported as being elicited by vestibular nerve stimulation, are most likely the result of stimulation of nonvestibular structures. Preliminary findings are presented in this report.

METHODS

Experiments were performed on cats decerebrated under anesthesia by electrocoagulation of the midbrain tegmentum. They were immobilized with gallamine triethiodide and maintained with artificial

ventilation. The arterial blood pressure and the expired air CO_2 were continuously monitored, and the body temperature was maintained at 39° C during the experiments. With few exceptions, the experimental procedures employed in the present study were essentially the same as those described previously (ref. 7).

The right internal ear was approached ventrally by removing the tympanic bulla, the bony partition of the middle ear, and the promontory. Figure 1 shows the ventral view of the middle and the internal ear of a formalin-perfused cat after the nervous tissues had been stained with osmic acid. The facial nerve is seen lying in the facial canal. The cochlear nerve was sectioned at its exit from the internal auditory meatus, and therefore only the stained nerve stump is visible. The saccule was removed to reveal the main vestibular trunk, which is seen here (fig. 1) supplying the utricle and the lateral ampulla. The anterior ampulla and its nerve, lying beneath the lateral ampulla and its nerve, are hidden from view. The posterior ampulla and its nerve are seen apart from the main vestibular trunk. It would be very difficult, if not impossible, to dissect them out in live preparations with unstained nerves, since the ampulla is hidden beneath the bone and the nerve is firmly embedded in the singular canal. It was therefore no surprise that all attempts by Andersson and Gernandt (ref. 13) failed to expose these structures. On the other hand, they can be destroyed easily in live preparations simply by enlarging the bony opening in the vestibule or by pulling out the ampulla and its nerve from underneath the bone with a fine hook. This was routinely performed in the present study before each experiment unless stated otherwise. In the Andersson-Gernandt approach (ref. 13), only the portion of the vestibule containing the saccule, the utricle, and the anterior and lateral ampullae was exposed. In the present experiments, the main vestibular nerve trunk was exposed all the way to the orifice of the internal auditory meatus, as shown in figure 1, so that after its distal connections to the vestibular organs were severed, there would still be enough length of it left to be suspended in air with a pair of curved stimulating electrodes.

In figure 2, the top diagram shows the ventral view of a cat's head and the location of the shaded area from which the lower sketch was made. The lower sketch was drawn from a fresh preparation and resembles very closely the photograph shown in figure 1, except that it does not show the posterior ampulla and its nerve, since these structures were routinely removed before each experiment. It shows the extent of the exposure and the locations of the

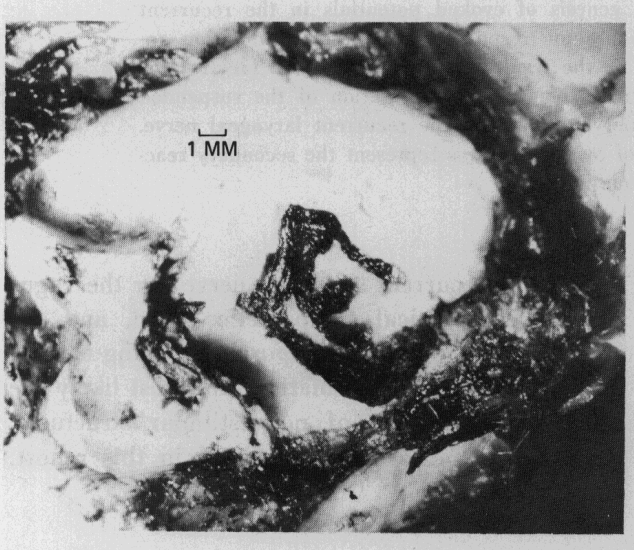


FIGURE 1.—*Ventral view of internal structures of right ear of formalin-perfused cat. Neural structures were exposed by alternate dissecting and staining with 1-percent osmic acid. See figure 2 for identification of structures.*

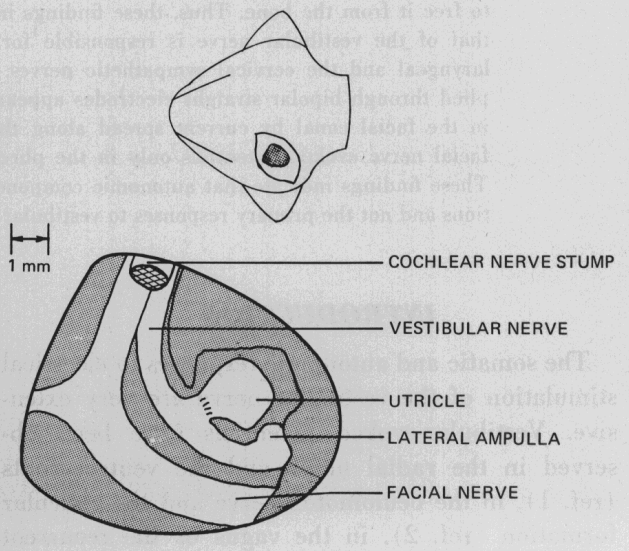


FIGURE 2.—*Shaded area in upper diagram shows location and orientation of lower drawing of internal neural structures of cat's right ear. Posterior ampulla and its nerve, seen lying above and parallel to vestibular nerve trunk in figure 1, are not shown.*

neural structures being stimulated in the present experiments. Unless stated otherwise, the cochlear nerve was routinely sectioned at its exit, as shown in the sketch, and sealed with modeling clay to prevent the outflow of the cerebrospinal fluid and to insulate the nerve from stray currents during electrical stimulation of other parts of the internal ear. This sketch and its variations will be used to illustrate the various experimental procedures.

The stimulating electrodes were made of iridium-platinum wires, 0.13 mm in diameter, insulated with Teflon except at the tips. Two types were used. Curved or L-shaped bipolar electrodes were used to suspend a nerve for stimulation. Straight bipolar electrodes were thrust against the bony wall forming the vestibule and in contact with the vestibular nerve branches, as shown in the upper sketch in figure 3, in a manner similar to that described previously (refs. 3 and 7). Each pair of electrodes was mounted on the carrier of a stereotoxic instrument so that they could be placed at any desired position in the internal or middle ear. Stimulation was made with square-wave pulses of 0.3-ms duration and of different intensities, at frequencies varying from 1 to 20 pps.

The phrenic-nerve efferent potentials were recorded with a pair of silver-silver chloride electrodes from the central cut end of the fourth spinal nerve branch supplying the phrenic nerve on the ipsilateral side of the stimulated ear. The efferent potentials of the contralateral recurrent laryngeal nerve and the ipsilateral cervical sympathetic nerve were similarly recorded from their central cut ends. All the nerves were desheathed and immersed in warm mineral oil during the recording. For recording the eye movements in nonparalyzed animals, silver-silver chloride electrodes were placed one each at the lateral canthi of both eyes and on the ridge of the nose.

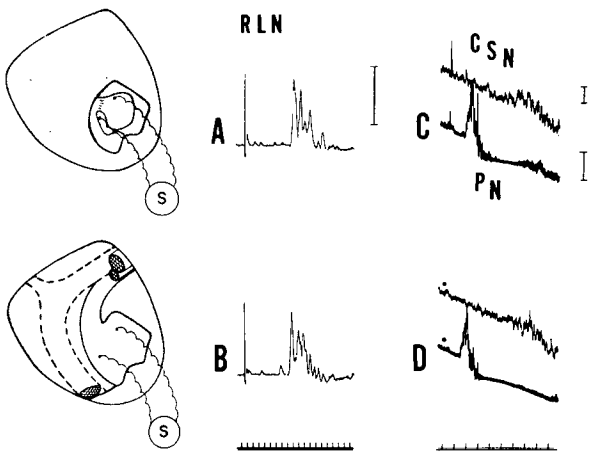


FIGURE 3.—*Upper diagram shows placement of stimulating electrodes before and lower diagram after transection of cochlear and vestibular nerves at internal auditory meatus and of facial nerve outside oval window. RLN, recurrent laryngeal nerve response to single shock stimulation before (A) and after (B) nerve transections. CSN, cervical sympathetic, and PN, phrenic nerve responses to 10-pps stumulation before (C) and after (D) nerve transections. Calibrations: RLN, 500 μV; time, 1 ms; CSN, 50 μV; PN, 1 mV; time, 10 ms.*

RESULTS

Effect of Intravestibular-Nerve Transections

Following transection of the vestibular and the cochlear nerve at the orifice of the internal auditory meatus, the evoked potentials in the recurrent laryngeal, the cervical sympathetic, and the phrenic nerves persisted. The upper diagram in figure 3 illustrates the placement of bipolar stimulating electrodes in the vestibule, against the bone and in contact with the vestibular nerve branches. The cochlear nerve was not sectioned, but the basal portion of the cochlea was removed. The extent of the exposure and the placement of electrodes were essentially the same as described previously (refs. 3 and 7). The lower diagram (fig. 3) shows the same placement of electrodes following transection of the cochlear and the vestibular nerves at the orifice of the internal auditory meatus. Both nerve stumps were sealed with modeling clay to prevent the outflow of cerebrospinal fluid and to insulate them against stray currents from the stimulating electrodes. The facial nerve was transected just outside the oval window but was otherwise left intact in the facial canal. There appears to be no difference between the evoked potentials in the contralateral recurrent laryngeal, the ipsilateral cervical sympathetic, and the ipsilateral phrenic nerves recorded before (A and C in fig. 3) and those recorded after (B and D in fig. 3) transection of the nerves. The latency was about 7 ms for the recurrent laryngeal, 13 ms for the phrenic, and 40 to 60 ms for the cervical sympathetic response.

Transection of the facial nerve at the orifice of the internal auditory meatus in addition to that of the cochlear and vestibular nerves did not appear to affect the evoked potentials in the recurrent laryngeal and the cervical sympathetic nerves. As shown in figure 4A, these potentials persisted after transection of the facial as well as of the cochlear and the vestibular nerves. It soon became apparent that these responses could be elicited by stimulation of any part of the petrous bone. In fact, following removal of the facial nerve, stimulation of the base of the facial canal was effective in evoking potentials in the recurrent laryngeal and the cervical sympathetic nerves. Stimulation of the rim of the remaining portion of the tympanic bulla, which is not part of the petrous bone, was ineffective. Topical application of 2-percent lidocaine to the entire surface of the petrous bone failed to affect these evoked potentials (fig. 4B), indicating that stimulation of nerve fibers or of endings on the surface of the petrous bone is not responsible for these evoked potentials.

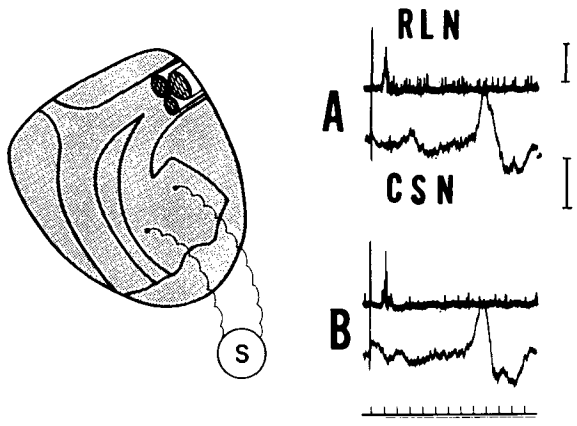


FIGURE 4.—*Diagram illustrates placement of stimulating electrodes following transection and sealing of cochlear, vestibular, and facial nerves at orifice of internal auditory meatus. RLN, recurrent laryngeal, and CSN, cervical sympathetic response to single shock stimulation recorded after nerve transections (A) and after 15-minute lidocaine application to petrous bone surface (B). Calibrations: RLN, 200 μV; CSN, 100 μV; time, 10 ms.*

Effect of Vestibular-Nerve Suspension

The findings in the preceding experiments indicate that stimulation of structures other than the vestibular nerve is responsible for the evoked potentials in the recurrent laryngeal and the cervical sympathetic nerves. However, the possibility is not entirely ruled out that stimulation of the vestibular nerve could also evoke these potentials. In other words, it is still possible, though remotely, that stimulation of either the vestibular nerve or the petrous bone could elicit the same responses. To test this possibility, the vestibular nerve was sectioned at its peripheral end and suspended with a pair of curved electrodes, as illustrated in the diagram in figure 5. The vestibular nerve could be stimulated alone without the stimulation of the petrous bone by suspending the nerve in air during stimulation. Or, the nerve and the bone could be stimulated simultaneously by lowering the curved electrodes so that their undersurfaces touched the bone. Stimulation of the suspended vestibular nerve alone elicited no evoked potential in either the cervical sympathetic or the recurrent laryngeal nerve (fig. 5A). Simultaneous stimulation of the vestibular nerve and the petrous bone resulted in the appearance of evoked potentials in both nerves

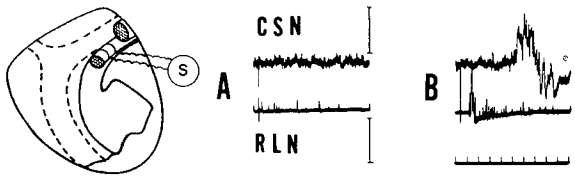


FIGURE 5.—*Diagram illustrates suspension of vestibular nerve with curved stimulating electrodes. CSN, cervical sympathetic, and RLN, recurrent laryngeal response to single shock stimulation of vestibular nerve alone (A) and to simultaneous stimulation of vestibular nerve and petrous bone (B). Calibrations: CSN, 10 μV; RLN, 1 mV; time 10 ms.*

(fig. 5B). It thus appears that stimulation of the vestibular nerve is not responsible for the evoked potentials observed in the recurrent laryngeal and the cervical sympathetic nerves. Petrous-bone stimulation is essential to the genesis of these potentials.

One could, however, still argue in favor of the vestibular origin of these evoked potentials on the assumption that the portion of the vestibular nerve that was in contact with the stimulating electrodes was injured and therefore unresponsive to electric stimulation and that the positive result obtained from simultaneous stimulation of the petrous bone

was due to the stimulation of the uninjured proximal portion of the vestibular nerve through bone conduction of the electric current. To test this possibility, the effects of stimulation of the suspended vestibular nerve, with or without simultaneous bone stimulation, were observed in nonparalyzed decerebrate cats. Simultaneous stimulation of the vestibular nerve and the petrous bone resulted in widespread responses, including contractions of muscles of the face, neck, and upper limbs. Hyperventilation and rise in arterial blood pressure were often observed. These responses did not appear during stimulation of the suspended vestibular nerve without simultaneous stimulation of the petrous bone. The only response that was repeatedly observed with both methods of stimulation was a conjugate deviation of the eyes.

After the right vestibular nerve was sectioned, both eyes of the cat deviated toward the right side, i.e., the side with the lesion, in a manner essentially the same as that following unilateral labyrinthectomy. Stimulation of the central cut end of that nerve suspended with a pair of curved electrodes, without touching the petrous bone, at a frequency of 50 pps, resulted in conjugate deviation of the eyes toward the left side; i.e., the side opposite the lesion and thus opposite the nerve being stimulated (fig. 6). These experiments demonstrated that the vestibular nerve was not injured following its dissection and suspension with bipolar curved electrodes, and the only overt response to its stimulation was a conjugate deviation of the eyes. Other responses appear to be the result of stimulation of nonvestibular structures due to current spread through the petrous bone. These include hyperventilation, rise of arterial blood pressure, and contractions of facial, cervical, and upper-limb muscles.

Origin of Phrenic-Nerve Response

Intravestibular sectioning of the cochlear and the vestibular nerves produced no apparent effect on the evoked potential in the phrenic nerve, but sectioning and sealing of the facial nerve at the orifice of the internal auditory meatus did abolish this phrenic response. The diagram in figure 7A shows the placement of the straight bipolar stimulating

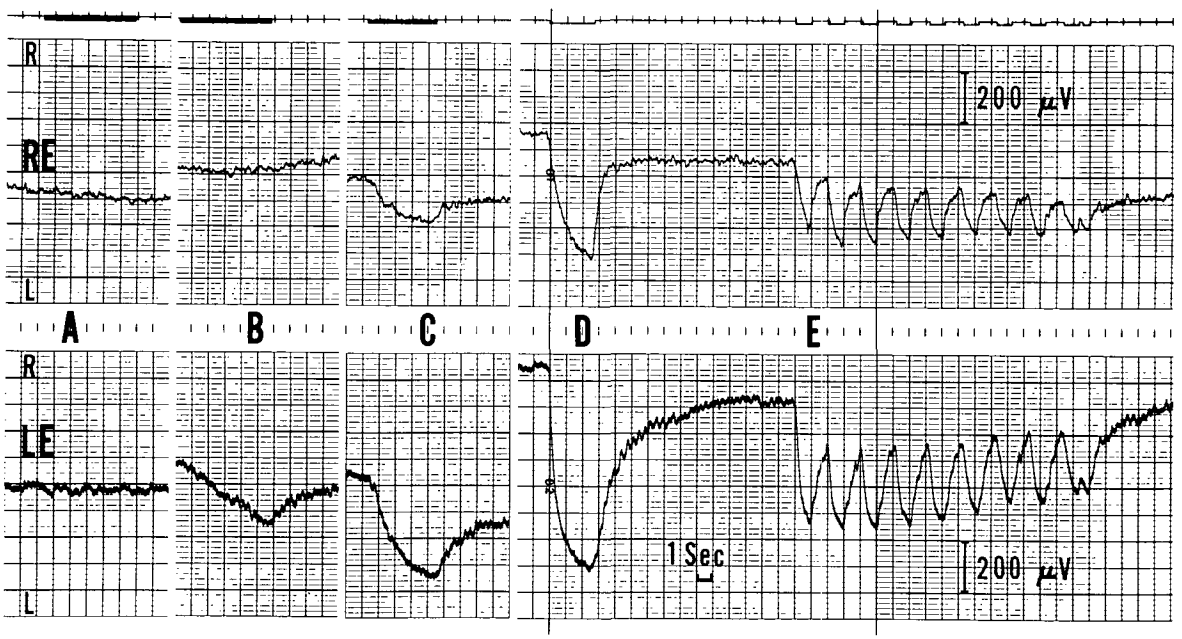


FIGURE 6.—*Electro-oculogram showing conjugate eye deviation in response to electrical stimulation of suspended vestibular nerve alone. R, right side; L, left side; RE, right eye; LE, left eye. Frequency and intensity of stimulation: (A) 50 pps, 10 mA; (B) 50 pps, 22 mA; (C) 100 pps, 10 mA; (D) 200 pps, 10 mA; (E) 200 pps, 10 mA. In E, stimulation was alternately on for 1 s and off for 1 s.*

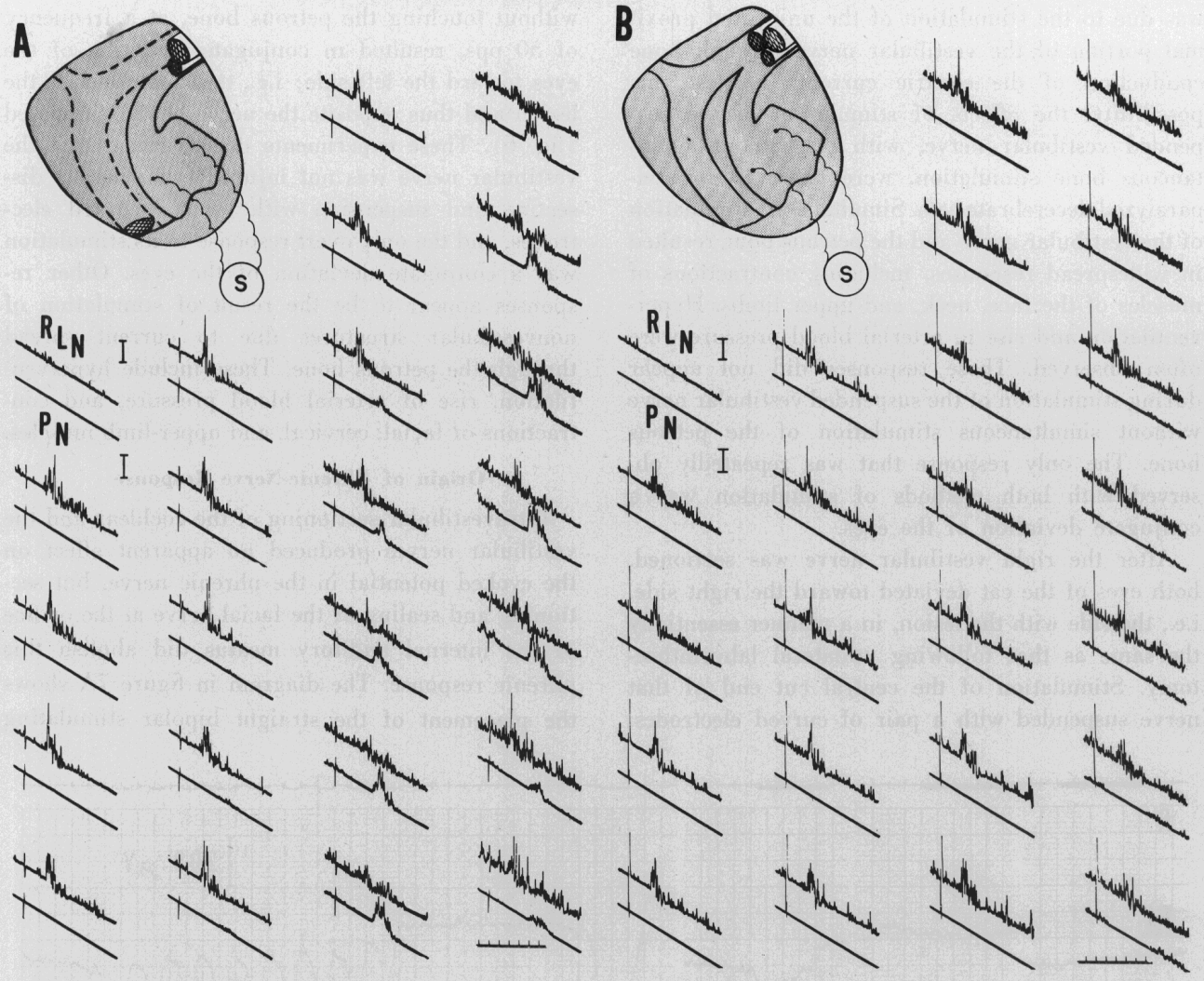


FIGURE 7.—*Diagrams show placement of stimulating electrodes (A) folowing transection and sealing of cochlear and vestibular nerves at orifice of internal auditory meatus and transection of facial nerve outside oval window, and (B) following additional transection and sealing of facial nerve at orifice of internal auditory meatus. RLN, recurrent laryngeal, and PN, phrenic responses to 10-pps stimulation. In both records, stimulation starts from second frame. Calibrations: RLN, 200 μV; PN, 500 μV; time, 5 ms. Note presence of RLN responses in both records but PN responses in A records only.*

electrodes against the bone of the vestibule and the stumps of the cochlear and the vestibular nerves following their transection at the orifice of the internal auditory meatus. These nerve stumps were routinely sealed before the bone stimulation. The facial nerve was transected just outside the oval window but was otherwise left intact in the facial canal. Stimulation of the petrous bone at a frequency of 10 pps evoked responses from both the recurrent laryngeal and the phrenic nerves (fig. 7A). The response of the recurrent laryngeal nerve followed the stimulation faithfully, but there was considerable delay in the appearance of the phrenic response. These observations are in agreement with those reported previously (refs. 3 and 7).

The diagram in figure 7B shows the same stimulating procedure except that an additional transection of the facial nerve was made at the orifice of the internal auditory meatus. All nerve stumps were routinely sealed before stimulation. The phrenic response failed to appear when the petrous bone was again stimulated, but the recurrent laryn-

geal response persisted (fig. 7B). It thus appears that stimulation of the facial nerve is responsible for the genesis of evoked potentials in the phrenic nerve.

An argument in favor of the vestibular origin of the phrenic response could still be made that sealing with modeling clay only occasionally protected the vestibular nerve from being stimulated, and that it was purely coincidental that the phrenic response disappeared following transection and sealing of the facial nerve at the orifice of the internal auditory meatus. To test this hypothesis, the facial nerve was dissected out of the facial canal and suspended in air with a pair of curved stimulating electrodes. Stimulation of the suspended facial nerve at a frequency of 10 pps elicited evoked potentials in the phrenic nerve following the usual latent period, but the recurrent laryngeal response failed to appear (fig. 8A). Then, the stimulating electrodes, still holding the facial nerve, were lowered so that their undersurfaces touched the bony wall of the facial canal. Simultaneous stimulation of the facial nerve and the facial canal produced not only the phrenic but also the recurrent laryngeal response (fig. 8B). It thus appears that the phrenic response results from stimulation of the facial nerve only and not from stimulation of the vestibular nerve by electric current conducted through the petrous bone. On the other hand, it is the petrous-bone stimulation and not the facial-nerve stimulation that is responsible for the genesis of the evoked potential in the recurrent laryngeal nerve.

DISCUSSION AND CONCLUSION

There seems little doubt about the cortical projection of the vestibular afferent, not so much because of the repeated observation of the vestibular-evoked potential on the cortex but mainly because of the care taken to ascertain the vestibular origin of the evoked potentials. Megirian and associates demonstrated that intracranial transection of the eighth nerve abolished the evoked potentials recorded from the contralateral cortex (ref. 8) and from the pyramidal tract (ref. 9). In a study of the cortical projection of the vestibular nerve by Andersson and Gernandt (ref. 13), the individual branches of the vestibular nerve were suspended with hooked stimulating electrodes, thereby limiting the stimulating current to the vestibular nerve.

The same, however, cannot be said about other studies involving electrical stimulation of the vestibular nerve. The allegedly vestibular-evoked potentials recorded from the vagus (or the recurrent laryngeal), the cervical sympathetic, and the phrenic nerves were elicited by stimulations made with a pair of straight electrodes placed in the vestibule in contact with the vestibular branches (refs. 3 and 7). A method of thrusting a monopolar stimulating electrode against the bone of the vestibule was employed in the study of the vestibular-evoked potentials in the somatic motor outflow (ref. 1). Ascertainment of vestibular origin of the evoked potentials with the intracranial transection of the eighth nerve was not attempted in the above studies. In other studies (refs. 2, 4, 6, 8, and 9), reference was made to the method of Andersson and Gernandt (ref. 13), but the actual procedure employed for stimulation was not described.

Evidence presented in this report strongly indicates that the evoked potentials observed in the recurrent laryngeal (or the vagus), the cervical sympathetic, and the phrenic nerves are the results of stimulation of structures other than the vestibular nerve. They can therefore be regarded only as artifacts generated during the presumed stimulation of the vestibular nerve. The phrenic response is probably the result of stimulation of the facial nerve by electric current conducted through the petrous bone. The origins of the recurrent laryngeal and the cervical sympathetic responses are very much in the dark, although it is clearly the petrous-bone stimulation and not the vestibular-nerve stimulation that is responsible for their production.

Involvement of the autonomic nervous system in the genesis of motion sickness has been a subject of much interest and great controversy. Some answers can be found in our present and previous studies. Electric potentials of the vagus nerve, allegedly evoked by vestibular stimulation, have been shown to represent the response of its recurrent laryngeal portion only and not that of its parasympathetic component (ref. 7). The present study revealed further that the allegedly vestibular-evoked sympathetic response is actually elicited by the stimulation of nonvestibular structures. It thus appears that the autonomic nervous system does not play a primary role in the genesis of motion sickness and that the autonomic signs of motion sickness merely

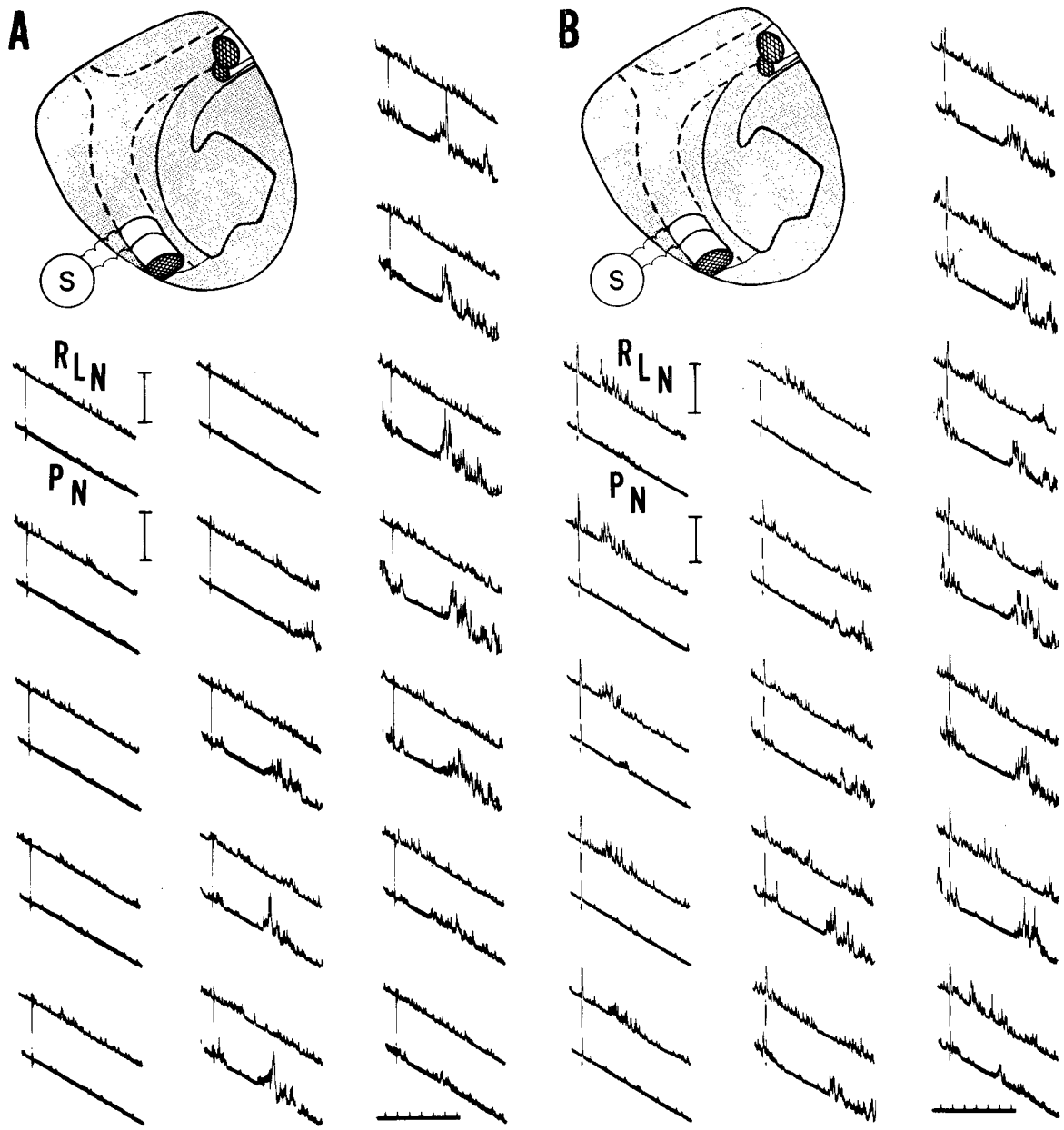


FIGURE 8.—*Diagrams show suspension of facial nerve with curved stimulating electrodes following transection and sealing of cochlear and vestibular nerves at orifice of internal auditory meatus. Electrodes were lifted off the bone in A, thus limiting stimulation to vestibular nerve, and lowered to touch the bone in B, resulting in stimulation of nerve and bone simultaneously. Stimulation at 10 pps starts in first ffames of both records. Calibrations: RLN (recurrent laryngeal response), 500 μV; PN (phrenic response), 500 μV; time, 5 ms. Note presence of PN responses in both records but RLN responses in B records only.*

represent secondary reactions. Our findings thus agree with the concept developed by Graybiel (ref. 14), who regarded these signs as belonging to the V–II manifestations whose site of orgin are normally not vestibular receiving areas.

For years anatomists have maintained that the primary and secondary vestibular neurons send their fibers only to a limited number of destinations, namely the cerebellum, the occular nuclei, and the somatic spinal motor neurons (ref. 15). The vestibular connections to the autonomic nervous system must therefore be very remote. The allegedly vestibular-evoked autonomic responses observed by some physiologists came as a great surprise. The present findings are in complete agreement with the view of the anatomists. The alleged vestibulo-autonomic connections are based on artifacts created by the stimulation of nonvestibular structures.

ACKNOWLEDGMENTS

The scientific advice of Dr. Mark DeSantis and the technical assistance of Mr. Alvin L. Armstrong and Mr. Robert C. Barrett are gratefully acknowledged.

POSTSYMPOSIUM NOTE

Following the suggestions made by Drs. DeSantis and Gernandt during the symposium, intracranial transections of the cranial nerves were performed. The evoked potentials recorded from the recurrent laryngeal nerve and the cervical sympathetic nerve persisted following intracranial transection of the seventh and eighth nerves ipsilateral to the stimulated ear. Intracranial transection of the ipsilateral ninth, tenth, and eleventh nerves abolished the evoked potentials recorded from the ipsilateral sympathetic nerve and the contralateral recurrent laryngeal nerve. These findings appear to prove conclusively the nonvestibular origin of the evoked potentials in the recurrent laryngeal and the cervical sympathetic nerves elicited by stimulation with bipolar electrodes placed against the bony wall of the vestibule in contact with the vestibular nerve branches.

REFERENCES

1. Gernandt, B. E.; Katsuki, Y.; and Livingston, R. B.: Functional Organization of Descending Vestibular Influences. J. Neurophysiol, vol. 20, 1957, pp. 453–469.
2. Gernandt, B. E.: Interactions Between Extraocular Myotatic and Ascending Vestibular Activities. Exptl. Neurol., vol. 20, 1968, pp. 120–134.
3. Akert, K.; and Gernandt, B. E.: Neurophysiological Study of Vestibular and Limbic Influences upon Vagal Outflow. Electroenceph. Clin. Neurophysiol., vol. 14, 1962, pp. 904–914.
4. Gernandt, B. E.: A Comparison Between Autonomic and Somatic Motor Outflow to Vestibular Stimulation. Confin. Neurol., vol. 24, 1964, pp. 140–157.
5. Cobbold, A. F.; Megirian, D.; and Sherrey, J. H.: Vestibular Evoked Activity in Autonomic Motor Outflows. Arch. Ital. Biol., vol. 106, 1968, pp. 113–123.
6. Megirian, D.: Vestibular Control of Laryngeal and Phrenic Motoneurons of Cat. Arch. Ital. Biol., vol. 106, 1968, pp. 333–342.
7. Tang, P. C.; and Gernandt, B. E.: Autonomic Responses to Vestibular Stimulation. Exptl. Neurol., vol. 24, 1969, pp. 558–578.
8. Megirian, D.; and Manning, J. W.: Input-Output Relations of the Vestibular System. Arch. Ital. Biol., vol. 105, 1967, pp. 15–30.
9. Megirian, D.; and Troth, R.: Vestibular and Muscle Nerve Connections to Pyramidal Tract Neurons of Cat. J. Neurophysiol., vol. 27, 1964, pp. 481–492.
10. Walzl, E. M.; and Mountcastle, V.: Projection of Vestibular Nerve to Cerebral Cortex of Cat. Am. J. Physiol., vol. 159, 1949, p. 595.
11. Kempinsky, W. H.: Cortical Projection of Vestibular and Facial Nerve in Cat. J. Neurophysiol., vol. 14, 1951, pp. 203–210.
12. Mickle, W. A.; and Ades, H. W.: Rostral Projection Pathway of the Vestibular System. Am. J. Physiol., vol. 176, 1954, pp. 243–252.
13. Andersson, S.; and Gernandt, B. E.: Cortical Projection of Vestibular Nerve in Cat. Acta Oto-Laryngol., suppl. 116, 1954, pp. 10–18.
14. Graybiel, A.: Structural Elements in the Concept of Motion Sickness. Aerospace Med., vol. 40, 1969, pp. 351–367.
15. Gacek, R. R.: Neuroanatomical Pathways of the Vestibular System. Ann. Otol., vol. 77, 1968, pp. 210–215.

SESSION IV

Chairman: **BRANT CLARK**
San Jose State College

Jacket No. 498-390

The Vestibular System of the Owl

K. E. MONEY and M. J. CORREIA
Defence Research Establishment Toronto

SUMMARY

Five owls were given vestibular examinations, and two of them were sacrificed to provide serial histological sections of the temporal bones. The owls exhibited a curious variability in the postrotatory head nystagmus following abrupt deceleration; sometimes a brisk nystagmus with direction opposite to that appropriate to the stimulus would occur promptly after deceleration. It was found also that owls can exhibit a remarkable head stability during angular movement of the body about any axis passing through the skull. The vestibular apparatus in the owl is larger than in man, and a prominent crista neglecta is present. The tectorial membrane, the cupula, and the otolithic membranes of the utricle, saccule, and lagena are all "attached" to surfaces in addition to the surfaces bearing hair cells. These attachments are very substantial in the utricular otolithic membrane and in the cupula.

INTRODUCTION

The external eye muscles of the owl are "functionless" (ref. 1, p. 642), and the "owl eye cannot be turned in the orbit, even with a pair of pliers" (ref. 1, p. 309). In fact, horizontal or vertical eye movements in the owl appear to be impossible because the back of the eyeball is spool shaped instead of spherical. The owl therefore does not benefit from a vestibulo-ocular reflex, and in order to stabilize the retinal image it must stabilize its whole head. It was felt that a study of the vestibular system of such an animal would be of interest.

PROCEDURES

One snowy owl, *Nyctea scandiaca*, and four great horned owls, *Bubo virginianus*, were observed for head stability (with the wings and claws taped) during manually produced angular movements of the body. Each bird was oscillated through approximately 90° about each of the three orthogonal axes of the skull, starting with the spinal axis vertical. It was also oscillated about the spinal axis with the spine horizontal, as in flight. Frequencies of oscillation ranged from approximately 0.2 to 2.0 Hz, and the tests were done with and without the bird blindfolded. The procedure was repeated on three different days, and in some cases it was recorded on movie film. Small (±5 cm) lateral linear displacements were also imposed on the animal to see whether it could keep the head motionless during such movement. The snowy owl and one great horned owl were sacrificed, and serial sections of their temporal bones were prepared, using the Igarashi procedures (ref. 2) with hematoxylin and eosin staining.

The three remaining great horned owls were subjected to additional rotatory tests. These rotatory tests, about an Earth-vertical axis and about the owl's Z axis (with the horizontal plane of the owl's head in the plane of rotation), were done with the owl blindfolded, at 50, 30, 10, and 3 rpm. After rotation for 2 minutes, the bird was decelerated abruptly (stopping time 5 s) and observed for post-rotatory head movements. In some cases the response was recorded on movie film.

RESULTS AND DISCUSSION

Head Stability and Nystagmus

The owls exhibited a remarkable stability of the head during angular displacement of the body. In two of them (one of which was the snowy owl) this stability was found almost constantly, but in the

other three it was frequently absent, as if the birds chose not to exhibit it. When it was present, the head appeared motionless during angular movement of the body about axes passing through the skull (fig. 1). For amplitudes of approximately 90° this stability was present at frequencies between 0.5 and 2.0 Hz, but it was generally absent at 0.2 Hz.

In one owl, on one occasion when the owl appeared very interested in visually fixing on a nearby object (another owl), the head remained motionless during oscillation at 0.2 Hz, but this was the only instance when such low-frequency oscillation left the head motionless. Head stability was still present, but less marked, with the bird blindfolded. It should be emphasized that the oscillations were achieved manually and that, although the amplitude and frequency were controlled (very roughly), the wave shape was entirely unknown.

Head nystagmus in the owls was markedly variable, both between owls and also in the same owl on the same or different days, and this in spite of careful blindfolding and the use of arousal techniques. After deceleration from 30 or 50 rpm there occurred: no head movement at all; a 30-second period of no head movement (sometimes with the head held displaced in the direction of the expected slow component) followed by a nystagmus with the fast component in the direction of former stimulus rotation, presumably the secondary nystagmus, which was maintained for an additional 30 seconds and which was sometimes followed immediately by two or three beats of nystagmus with fast component in the direction opposite to the direction of the former stimulus rotation (presumably a tertiary nystagmus); a brisk nystagmus with fast component in the same direction as former stimulus rotation, occurring promptly after cessation of rotation, lasting for 30 seconds, and followed by a pendular nystagmus, and finally by a nystagmus with fast component in the direction opposite to former stimulus rotation; a 15-second period of no head movement or of very high-frequency, low-amplitude head movements followed by a primary nystagmus of the type appropriate to the stimulus, sometimes followed by a secondary nystagmus.

After deceleration from 10 rpm, the expected primary nystagmus was observed more frequently than with the stronger stimuli, and it was observed still more frequently and without delays following the 3-rpm stimulus. Even at these lower speeds, however, unusual responses (including no response) were observed frequently. The primary nystagmus

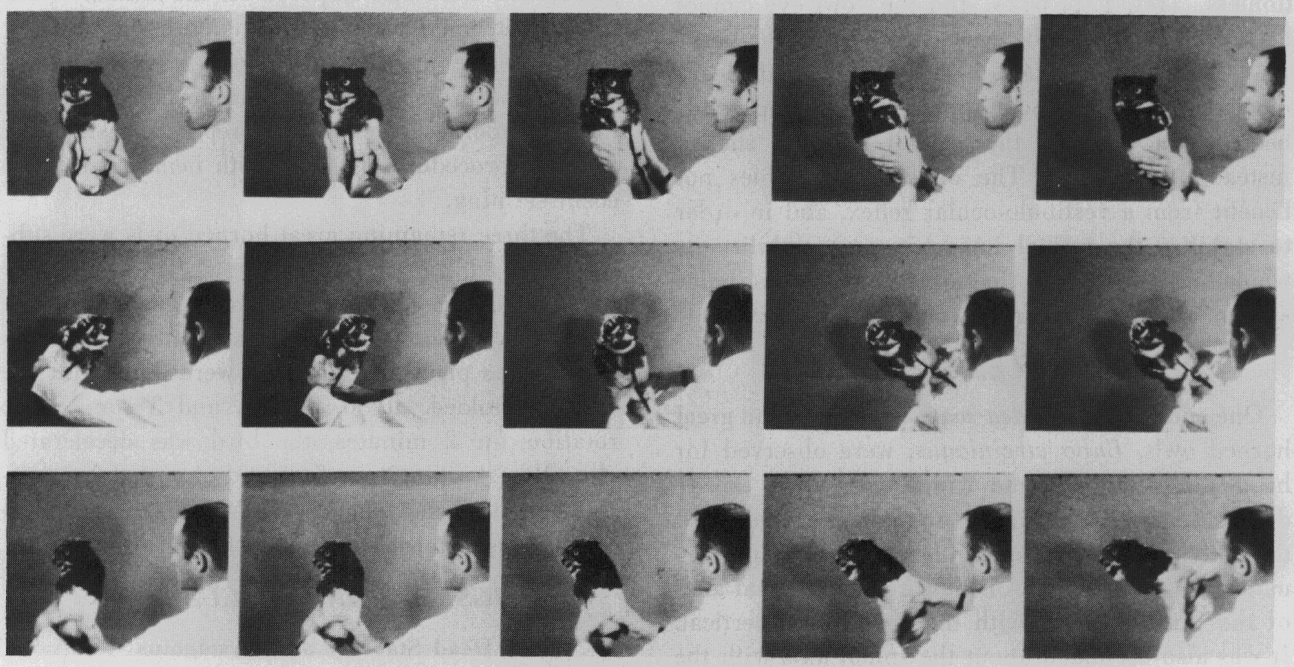


FIGURE 1.—*Head stability of the owl during angular movement of the body. In each of the three lines, adjacent pictures are separated by 5/24 second. In the top line, the rotation of the body is shown by the white feathers on the throat.*

in response to deceleration from 3 rpm often continued for 30 seconds, and it was sometimes followed by a pause and then a secondary nystagmus. The unusual responses to rotatory stimulation in this species, and the extreme variability, can be explained in general terms by postulating an unusually sensitive ear that cannot properly respond to large or unnatural stimuli. Perhaps a more likely explanation is that, compared to the influence on eye muscles, the influence of higher centers on neck muscles is more successful in interfering with vestibular influences, although this is apparently not the case in pigeons, which exhibit predictable and consistent postrotatory head nystagmus. It should also be remembered that head nystagmus itself causes an oscillatory stimulus of the semicircular canals.

Morphology

In spite of the absence of eye movement in the owl, the vestibular apparatus is essentially normal and surprisingly large. The length (circumference) of the horizontal semicircular canal in the owl is 23 mm, of which 3.5 mm is in the vestibule; the length of the posterior canal is also 23 mm, and 4 mm of this is in the crus and vestibule; the superior canal is 32 mm long, of which 5 mm is in the crus and vestibule. The small radii of the membranous ducts are: horizontal, 0.17 mm; posterior, 0.13 mm; anterior, 0.24 mm. As in the pigeon (ref. 3), the membranous duct has a very thick wall and a cross section which tends to be rectangular. The vestibular apparatus in the owl is somewhat larger than in man, but the cochlea in the owl, which is only slightly curved, is only 11 mm long compared with 32 mm in man. The papilla basilaris of the owl, which corresponds to the organ of Corti of mammals, is therefore much shorter than in man, but the papilla in owls is more than 30 hair cells wide (fig. 2), compared with 4 in the organ of Corti in man. The tectorial membrane in the owl is attached not only to the hair cells, but also to a surface without hair cells, as is known to be the case in other birds and in mammals.

As in other birds, the cristae of the posterior and anterior ampullae in owls have eminentae cruciatae, whereas the crista of the horizontal ampulla does not. The eminentiae cruciatae are large stubby fingerlike structures attached to the crista and projecting at right angles to the long axis of the crista.

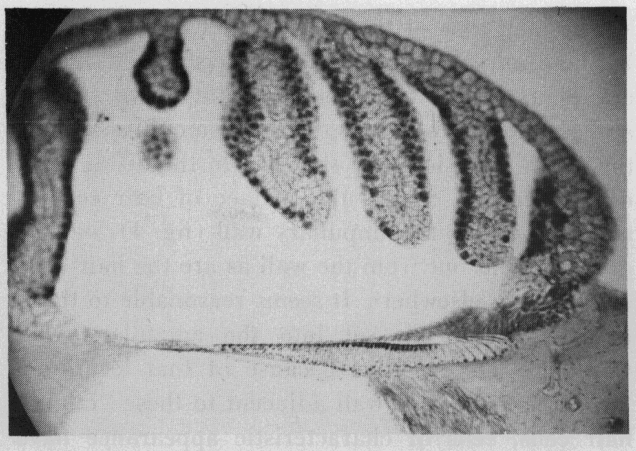


FIGURE 2.—*Papilla basilaris of the snowy owl, left ear. More than 30 hair cells can be seen along the width of the papilla. The auditory nerve can be seen below, and the tectorial membrane (thicker on the right) above. Cut roughly parallel to the horizontal plane of the skull. (×68)*

(The long axis of the crista is the line of the crista across the ampulla and is also the axis about which the cupula is thought to rotate when deflecting.) The eminentiae have, apparently, no nerve supply and no hair cells on the surface (fig. 3).

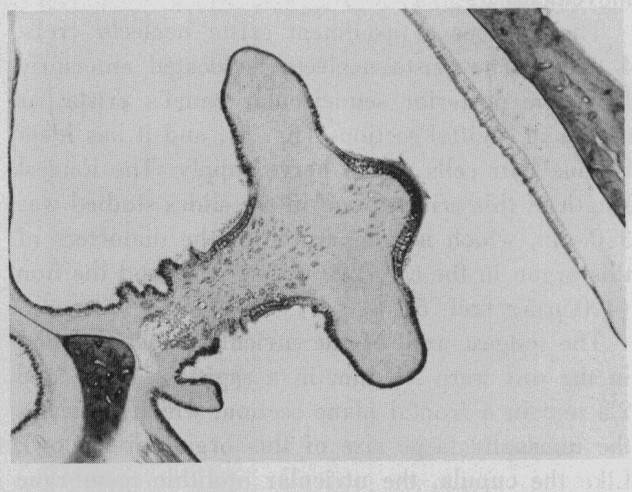


FIGURE 3.—*Crista of the superior canal of the great horned owl, left ear. The plane of the section is roughly parallel to the frontal plane of the skull, and it passes through the base of the crista below the level of the hair cells (lower left), then through the area of hair cells and the eminentiae cruciatae (central), then above the crista to the ampullary wall at the level of the cells of characteristic appearance to which the cupula appears to attach. (×32)*

The long axis of the crista is not a straight line in these ampullae; instead, the crista at its edges curves up, away from its base and nerve supply, and extends onto the sides of the ampulla so that the crista is basically U shaped. On the distal parts of the arms of the U, the surface of hair cells is actually set into the ampullary wall (fig. 4), instead of being held out from the wall as are the hair cells of the crista elsewhere. It seems reasonable to think that the hair cells set into the ampullary wall would be less sensitive because of that location.

On the ampullary wall adjacent to these "inlaid" hair cells, cells of characteristic appearance have on their surfaces pieces of extracellular substances similar to those seen between the hairs of the hair cells (fig. 4), and this suggests that the cupula normally is attached to the ampullary wall beside the crista. These cells of characteristic appearance are also present distally from the most distal hair cells of the crista (the surface of the planum semilunatum), which suggests that the cupula might be attached to the ampullary walls above the crista as well as beside the crista. There is also strong evidence of attachment of the cupula at the base of the crista below the level of the hair cells (figs. 3 and 5). The various cupular "attachments" can be expected to impose severe restrictions on cupular movement.

The owl has a prominent crista neglecta (refs. 4 to 6). The crista neglecta is located anteriorly from the posterior semicircular canal's crista, as shown in sagittal sections (fig. 5), and it has identifiable hair cells and a nerve supply. The longest length of this crista found in the slides studied was 350 μm, which is comparable to the diameters of this organ in the cat (200 to 300 μm) and the lion (400 μm) (ref. 5).

The longest axes of the utricular macula found in the owl were 2.8 mm in a sagittal section and 2.3 mm in a frontal plane section, which indicates the unusually large size of this organ in the owl. Like the cupula, the utricular otolithic membrane has attachments to surfaces in addition to surfaces covered by hair cells (fig. 6). These attachments arise from that layer of the otolithic membrane which is usually referred to as "gelatinous," and their fibers are very substantial and reach areas quite remote from the macula (fig. 7). Since they do not attach to the macula, they are presumably not analogous to similar fibers found in the dogfish (ref. 7), but they are perhaps comparable to less prominent structures present in man (ref. 8) and in the pigeon (plate XVII, fig. 1 in ref. 9). It seems reasonable to think that these attachments

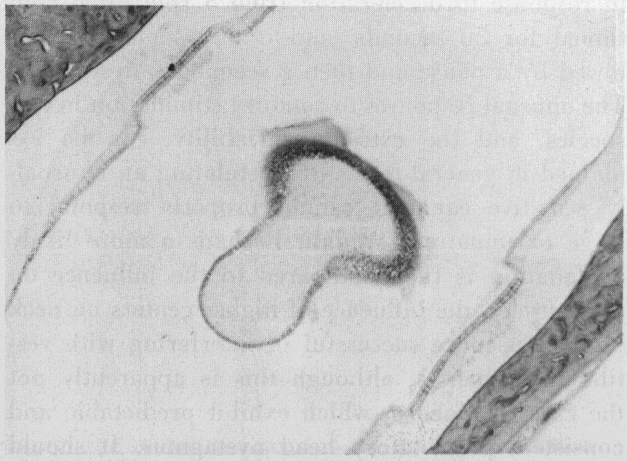


FIGURE 4.—*Crista of superior canal, right ear, in snowy owl, horizontal section. The arms of the U-shaped crista are seen to be set into the ampullary wall, and pieces of extracellular substance (presumably cupula) can be seen on adjacent cells of characteristic appearance. In addition to the arms of the U reaching upward, another prominence extends from the center of the bottom of the U and is included in the section with one eminentia cruciata. (×32)*

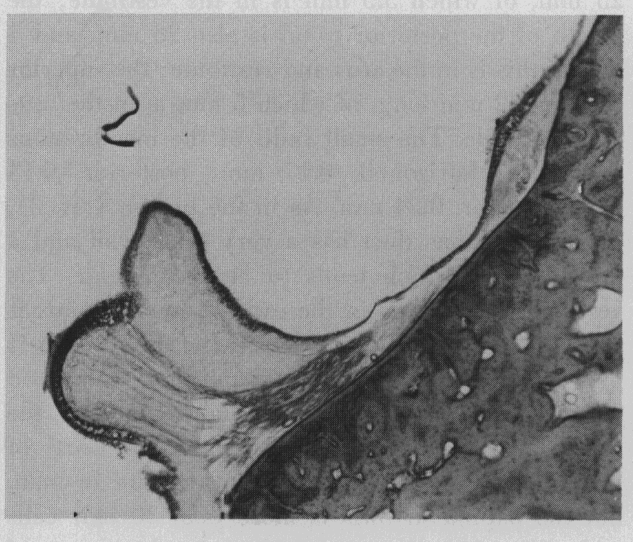


FIGURE 5.—*Crista of posterior canal in right ear of great horned owl, sagittal section. One of the eminentiae cruciatae is present in the section, and also (off to the right) the crista neglecta. (×36)*

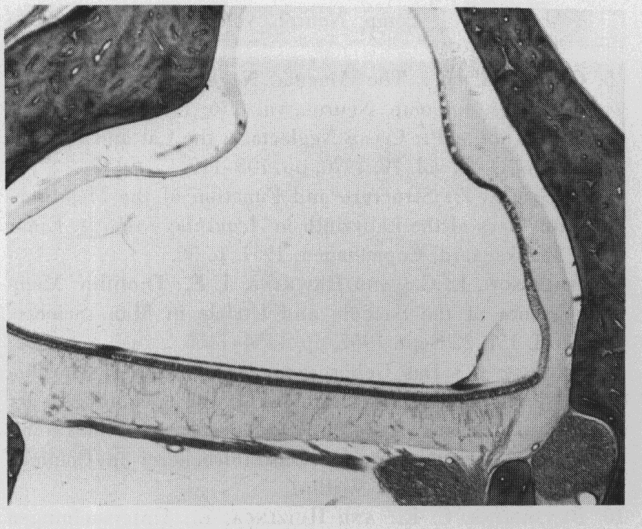


FIGURE 6.—*Utricular otolith organ of right ear of great horned owl, sagittal section, showing endolymph on one side and perilymph on the other. "Holding" or "anchoring" attachments are apparent on the right side of the otolithic membrane. (×25)*

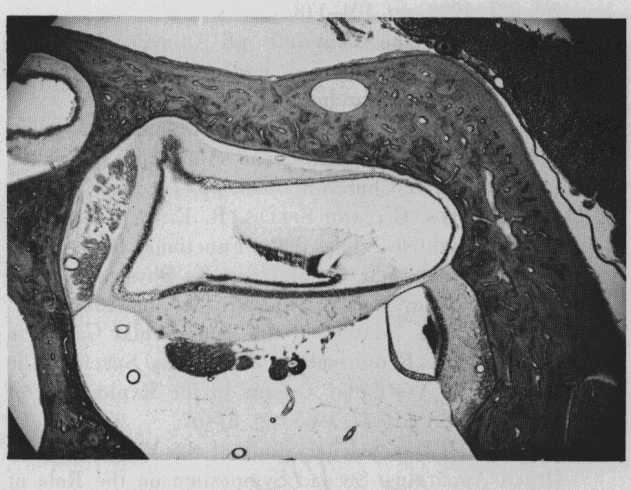


FIGURE 7.—*Utricular and saccular otolith organs in left ear of great horned owl, frontal section. The "holding" attachments on the utricular otolithic membrane are particularly prominent, although they have been displaced from their connection to the membranous wall on the right side. The saccular macula is seen to lie against bone. The relatively large size of the utricular otolith membrane and small size of the saccular one seen here are truly representative. (×19)*

perform the function of holding the otolithic membrane in the appropriate place, so that they could be called "holding" or "anchoring" attachments.

Whereas the utricular macula has endolymph on one side and perilymph on the other side, the saccular macula has endolymph on one side and bone on the other side (fig. 7), as is also the case in mammals. This structural difference between the utricular and saccular otolithic organs suggests that the saccular macula is less subject to displacements in response to linear accelerations than is the utricular macula, and this is probably important functionally. In the owl the saccular otolith organ is much smaller than the utricular one; the longest dimension found in frontal sections was 0.59 mm and in horizontal sections 0.94 mm, and the total depth of the saccule in serial sections confirms these measurements. This surprisingly small size of the saccular macula, in a species without functional eye muscles, is consistent with the idea (refs. 10 and 11) that the saccule of birds has a role in eye movements. The saccular otolithic membrane is thicker than the utricular one, and in several sections a fine, poorly staining fiber can be seen joining the distal surface of the otolithic membrane to the saccular membranous wall (fig. 7). Holding attachments of the gelatinous part of the otolithic membrane can also be found in the saccule, but they are not nearly so numerous or prominent as in the utricular otolithic membrane or in the cupula.

At the distal end of the cochlea is the third otolith organ found in birds, the lagena. As in the saccule, holding attachments (between the gelatinous part of the otolithic membrane and surfaces without hair cells) are present but not prominent. Except for a part of its proximal edge, the macula of the lagena lies against a bony surface (fig. 8) as does the macula of the saccule.

Investigations of the dimensions of vestibular structures in various species have been reported (refs. 12 to 17), and dimensions of the semicircular canals have been related to function. In addition to dimensional differences, however, there are striking differences in the mechanisms of the various vestibular receptors in different species. Some structures, such as holding attachments in the cupula and in the utricular otolith of the owl, might be prominent in one species but difficult to locate (although still important) in other species. It seems reasonable to think that a careful study of structural differences, together with a study of vestibular function, will give insight into the basic physiology of the vestibular system. The present study is a beginning in this direction.

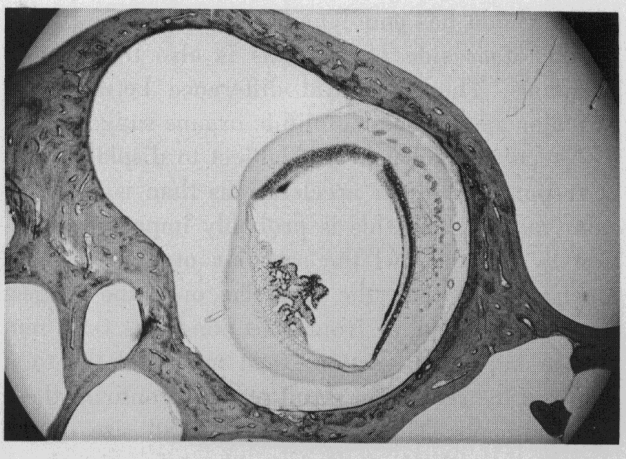


FIGURE 8.—*The lagena in the great horned owl, frontal section. The otolithic membrane, hair cells, and nerve supply are clearly shown, as is a part of the tegmentum vasculosum (which is even more prominent above the papilla basilaris, fig. 2). (×18)*

ACKNOWLEDGMENTS

Technical services were provided by A. D. Nicholas. Histology was by Jerry Laufer. The authors are indebted to J. E. Hawkins and M. L. Wolbarsht for helpful communications concerning this study.

REFERENCES

1. WALLS, G. L.: The Vertebrate Eye and Its Adaptive Radiation. Cranbrook Press, Bloomfield Hills, Mich., 1942.
2. IGARASHI, M.: A Standard Technique for Temporal Bone Preparation. Monograph 13. Naval Aerospace Medical Institute, Pensacola, Fla., 2 March 1966.
3. MONEY, K. E.; BONEN, L.; BEATTY, J. D.; KUEHN, L. A.; SOKOLOFF, M.; AND WEAVER, R. S.: Physical Properties of Fluids and Structures of the Vestibular Apparatus of the Pigeon. Am. J. Physiol., vol. 220, 1971, pp. 140–147.
4. IGARASHI, M.: Redefinition of the Macula Neglecta in Mammals. J. Comp. Neurol., vol. 125, 1965, pp. 287–294.
5. GACEK, R. R.: The Macula Neglecta in the Feline Species. J. Comp. Neurol., vol. 116, 1961, pp. 317–323.
6. MONTANDON, P.: Crista Neglecta in the Cat and Human. Ann. Otol., vol. 79, 1970, pp. 105–112.
7. VILSTRUP, T.: Structure and Function of the Membranous Sacs of the Labyrinth in *Acanthias vulgaris*. Ejnar Munksgaard, Copenhagen, 1951, p. 50.
8. JOHNSSON, L. G.; AND HAWKINS, J. E.: Otolithic Membranes of the Saccule and Utricle in Man. Science, vol. 157, 22 Sept. 1967, pp. 1454–1456.
9. RETZIUS, G.: Das Gehörorgan der Wirbeltier. Morphologisch-histologische Studien. II. Das Gehörorgan der Reptilien, der Vogel und der Säugetier. Stockholm, 1884. Gedruckt in der Centraldruckerei in Commission bei Samson & Wallin.
10. BENJAMINS, C. E.; AND HUIZINGA, E.: Untersuchungen über die Funktion des Vestibularapparates bei der Taube. I Mitteilung. Pflügers Arch. ges. Physiol., vol. 217, 1927, pp. 105–123.
11. BENJAMINS, C. E.; AND HUIZINGA, E.: Untersuchungen über die Funktion des Vestibularapparates bei der Taube. III Mitteilung. Pflügers Arch. ges. Physiol., vol. 221, 1928, pp. 104–118.
12. GRAY, A. A.: The Labyrinth of Animals, Including Mammals, Birds, Reptiles, and Amphibians. Vol. I. London, J & A Churchill, 1907, pp. 1–198.
13. GRAY, A. A.: The Labyrinth of Animals, Including Mammals, Birds, Reptiles, and Amphibians. Vol. II. London, J & A Churchill, 1908, pp. 1–252.
14. MELVILL JONES, G.; AND SPELLS, R. E.: A Theoretical and Comparative Study of the Functional Dependence of the Semicircular Canal Upon Its Physical Dimensions. Proc. Roy. Soc. B., vol. 157, 1963, pp. 403–419.
15. MAYNE, R.: The "Match" of the Semicircular Canals to the Dynamic Requirements of Various Species. The Role of the Vestibular Organs in the Exploration of Space, NASA SP–77, 1965, pp. 57–67.
16. IGARASHI, M.: Dimensional Study of the Vestibular End Organ Apparatus. Second Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–115, 1966, pp. 47–54.
17. TEN KATE, J. H.: The Oculo-Vestibular Reflex of the Growing Pike. A Biophysical Study. Doctoral Thesis, Rijksuniversiteit te Groningen, 1969, pp. 1–167.

SESSION V

Chairman: **JOHN BILLINGHAM**
Ames Research Center, NASA

The Role of Perilymph in the Response of the Semicircular Canals to Angular Acceleration

MAX ANLIKER and WILLIAM VAN BUSKIRK
Stanford University

SUMMARY

A new model for the response of the semicircular canals to angular motion is postulated. This model is based on evidence that the bony canal is not compartmentalized and assumes that the ampulla wall is highly flexible. It is shown that the perilymph induces a cupula displacement far greater than that produced by the endolymph alone. The predicted dynamic behavior of the canals on the basis of this model is found to be consistent with experimental observations.

INTRODUCTION

Steinhausen (refs. 1 to 4) proposed that the semicircular canals respond to angular accelerations in the same manner as would a heavily damped torsion pendulum. According to his model, an angular acceleration of the head causes the bony canals and the membranous structures attached to them to accelerate in a similar manner. The endolymph within the membranous system, however, due to its inertia, lags behind the motion of the head. Because of the small caliber of the membranous canal, this relative motion of the fluid is retarded by a relatively large viscous force. The cupula, which according to his observations fills the entire cross section of the ampulla, exerts a restoring force on the fluid when displaced.

The mathematical formulation of this idea, which in the literature has come to be known as the "torsion pendulum equation," may be written in the form

$$\Theta\ddot{\xi}+\Pi\dot{\xi}+\Delta\xi=-\Theta\alpha \qquad (1)$$

or

$$\ddot{\xi}+\frac{\Pi}{\Theta}\dot{\xi}+\frac{\Delta}{\Theta}\xi=-\alpha \qquad (2)$$

where ξ, $\dot{\xi}$, $\ddot{\xi}$ represent, respectively, the angular displacement, angular velocity, and angular acceleration of the endolymph with respect to the skull and where α is the component of the angular acceleration vector perpendicular to the plane of the canal and is measured with respect to an inertial reference frame. Θ is an inertia term defined by the mass and distribution of the endolymph. The damping coefficient Π denotes the ratio of the torque resulting from viscous forces to the mean angular velocity $\dot{\xi}$ of the endolymph. Δ represents the stiffness of the cupula.

The angular displacement of the endolymph ξ, which is proportional to cupular deflection, is also assumed to be proportional to the sensation of angular velocity as well as to the slow-phase velocity of nystagmus. With this hypothesis it is possible to determine experimentally the constants in the torsion pendulum equation, since we can quantify approximately the sensation of angular velocity and can record the nystagmus induced by an angular acceleration stimulus. The first to do this were van Egmond, Groen, and Jongkees (ref. 5). Using subjective sensation as their variable, they found $\Pi/\Theta=10\ s^{-1}$ and $\Delta/\Theta=1\ s^{-2}$. Niven and Hixson (ref. 6), monitoring nystagmus, found values of the same order of magnitude. Other experimenters have confirmed these results.

Meanwhile, as more accurate measurements of the dimensions of the canals became available, the ratio Π/Θ could also be determined from the anatomical data. Θ, the moment of inertia of the endolymph

ring, can be approximated by the expression

$$\Theta \approx 2\rho\pi^2 r^2 R^3 \qquad (3)$$

where ρ is density of the endolymph, r is the average radius of the canal duct, and R is the average radius of curvature of the membranous canal. If we assume that the endolymph has a parabolic velocity profile as in the case of Hagen-Poiseuille flow in a straight pipe, Π may be approximated by the expression

$$\Pi \approx \frac{\varphi}{2\pi} 16\mu\pi^2 R^3 \qquad (4)$$

where μ is the viscosity coefficient and $\varphi/2\pi$ is that portion of the circumference occupied by the narrow duct. The viscous forces in the utricle are insignificant, since there the radius is much larger and, correspondingly, the flow velocities much smaller. With $\varphi/2\pi = 2/3$,

$$\frac{\Pi}{\Theta} \approx \frac{32\mu\pi^2 R^3}{6\rho\pi^2 r^2 R^3} = \frac{32\mu}{6\rho r^2} \qquad (5)$$

Using the following anatomical data (ref. 7)

$r = 0.016$ cm
$\rho = 1.0$ g/cm^3
$\mu = 0.852$ centipoise

one finds $\Pi/\Theta = 180$ s^{-1}. This value for Π/Θ is an order of magnitude larger than that observed experimentally. To date, no convincing explanation for this discrepancy has been offered.

Alexander Rejtö (ref. 8) proposed that the perilymph might play a more significant role in semicircular canal mechanisms than the endolymph. He referred in his paper to the work of two earlier experimenters, namely Spitzer (ref. 9) and Lorente de Nó (ref. 10), who also held this view. Their assumption was in essence that the walls of the ampulla are sufficiently flexible to be deflected by a flow of perilymph. Because of the larger size of the perilymphatic space, flowing perilymph would experience a lesser viscous force and hence be much more mobile than the endolymph. The basic mechanism is illustrated in figure 1.

The idea of a possible flow of perilymph was vigorously opposed by Dohlman (ref. 11). He argued that his experiments indicated that the membranous ampulla wall was sufficiently rigid to preclude any flow of perilymph. However, his experiments on the ampulla wall were performed after the perilymph was drained. In the absence of the normal perilymphatic pressure the membranous labyrinth could have been excessively pressurized and thus much stiffer than under normal conditions.

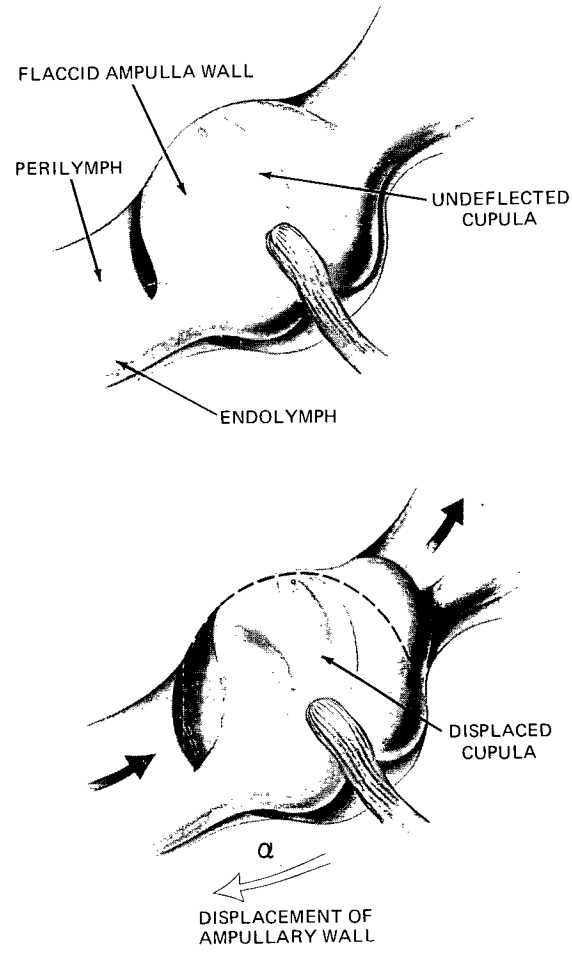


FIGURE 1.—*An illustration of the possible mechanics of cupular deflection. A flow of perilymph displaces the ampulla wall, which in turn produces a cupular deflection. Fluid continuity is maintained by an equal and opposite displacement of the ampulla wall on the other side of the cupula.*

Results from theoretical and experimental wave-transmission studies on canal-duct models (refs. 12 and 13) clearly indicate that the effects of viscosity of the labyrinthine fluids cannot be disregarded in a realistic study of the dynamic properties of the semicircular canals. In view of the small dimensions of the vestibular apparatus, the role played by the viscous forces should be particularly im-

portant in the membranous canal, which has an average cross-sectional radius of only about 0.16 mm in man. Since the cross section of the bony canal is an order of magnitude larger than that of the membranous canal, it seems that the perilymph should indeed be considerably more mobile than the endolymph if the bony canal is not compartmentalized and if the ampulla wall can be considered as a flexible membrane. If this were true, the motion of the perilymph as well as that of the endolymph would contribute to the displacement of the cupula. To assess the possible significance of this observation, we are presenting here an elementary theoretical study of a model simulating the gross features of the canals, including the ampulla, utricle, and cupula.

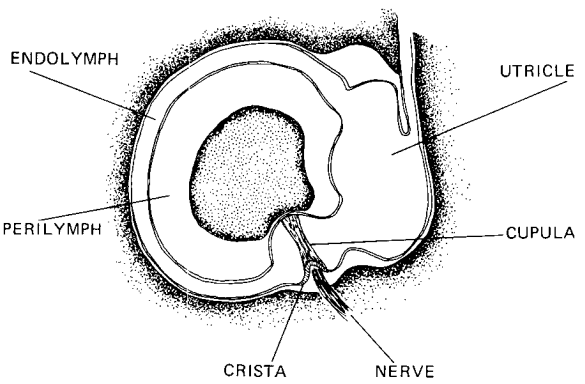


FIGURE 2.—*A cross-sectional view of the horizontal semicircular canal of man (after Igarashi, 1966).*

ANATOMICAL FEATURES

According to a biometric study by Igarashi (ref. 14), we can draw a cross-sectional view of the horizontal semicircular canal as illustrated in figure 2. The membranous canal duct runs along the outer periphery of the bony canal, where it is secured by fibrous banding to the periosteum (ref. 15). As shown, the membranous ampulla takes up the entire cavity formed by the bony ampulla. The two ampullae seem also to be connected with fibrous banding (ref. 15). While the bony canal duct is a confluent channel, there is no evidence of perilymphatic cisterns above the membranous ampulla (ref. 15). This implies that any perilymphatic flow which could occur within the bony canal duct cannot extend past the membranous ampulla. As the central hypothesis for this analysis we assume now that the sides of the membranous ampulla are flaccid or sufficiently flexible to permit the development of a small net flow of perilymph, which theoretically can cause a displacement of the cupula without actually requiring endolymph flow in the membranous canal duct (see fig. 1). To maintain perilymph continuity, there would have to be an equal and opposite volumetric displacement of the ampulla wall into the perilymphatic space on the opposite side of the cupula.

The flow of the perilymph, as well as that of the endolymph, is resisted by viscous forces that are strongly influenced by the geometric features of the canal ducts. A cross section of the duct (after *Gray's Anatomy*) is shown in figure 3. The location of the membranous canal along the outer periphery of the bony canal is again evident, and the relative size of the two canals is clearly illustrated. The shape of their cross sections can be approximated by ellipses which, according to Igarashi (ref. 14), have in man the dimensions of about 1.41 by 1.07 mm in the case of the bony canal and 0.44 by 0.24 mm for the membranous canal. We have therefore as the cross-sectional area of the membranous canal 8.3×10^{-4} cm^2, while that of the bony canal is 1.2×10^{-2} cm^2. If the cross-sectional

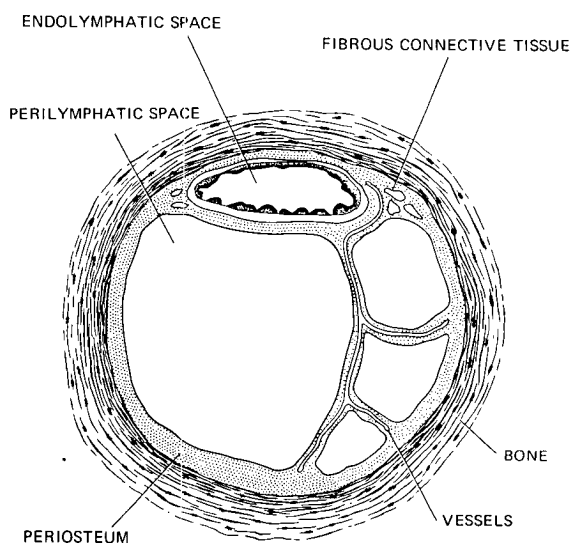


FIGURE 3.—*A cross-sectional view of a semicircular canal duct (after Gray's Anatomy, 1967).*

area of the membranous canal is subtracted from that of the bony canal and some allowance is made for tissue, the remaining perilymphatic cross section is about 1.1×10^{-2} cm^2. Thus, the effective cross-sectional area for perilymphatic flow is more than 13 times that for the endolymph. Also illustrated in figure 3 are vessels that arise from the periosteum and cross the perilymphatic space. If these vessels were taut, the flow of perilymph would be inhibited; but, again according to Igarashi (personal communication), they are loose, thread-like structures that can follow small motions of the perilymph. Unfortunately, much of the information required for a realistic analysis of the canal dynamics is still unknown. For example, we do not have any direct experimental data on the stiffness of the cupula and of the ampulla walls.

A PERILYMPH-ENDOLYMPH-CUPULA MODEL

A realistic model of the dynamic response of the semicircular canals should account for the flexibility of the membranous canal wall and the presence of the perilymph. The system is therefore modeled as follows. The endolymph and perilymph ducts are represented by two toroidal channels running side-by-side as shown in figure 4. The utricle and bony vestibule are simulated as widenings of the respective canals. For this initial study of the entire system, the flexibility of the membranous canal is limited to the region of the ampulla where the membranous wall forms the ampulla wall and blocks the flow of perilymph, as illustrated in figure 1. It is assumed that the ampulla wall offers no resistance to small volumetric displacements of the perilymph. This implies that there is no pressure differential between the endolymph and the perilymph across the ampulla wall on either side of the cupula.

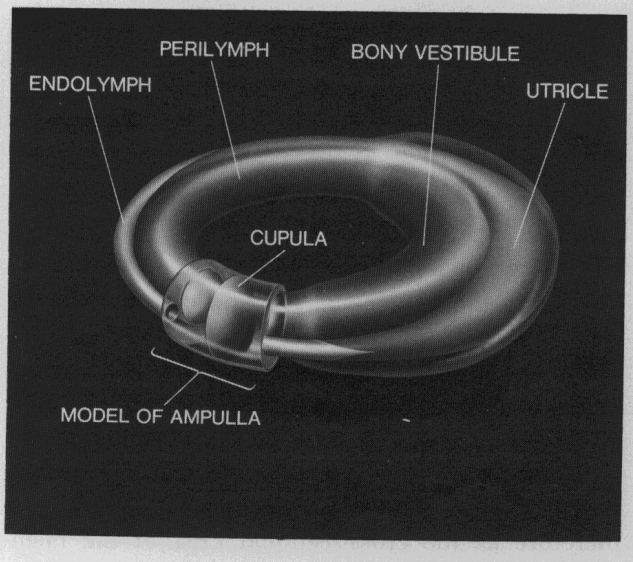


FIGURE 4.—*Theoretical model configuration for the perilymph-endolymph-cupula model.*

The resistance of the cupula to displacements is given by $K_c=\frac{\delta p}{\delta V}$, where δp is the pressure difference across the cupula and δV is the volumetric displacement of the cupula due to both the endolymph and perilymph, i.e.,

$$\delta V=A_eR_e\xi_e+A_pR_p\xi_p \tag{6}$$

where A denotes the cross-sectional area of the duct, and the subscripts refer to endolymph and perilymph. The pressure difference δp can therefore be expressed as

$$\delta p=K_c(A_eR_e\xi_e+A_pR_p\xi_p) \tag{7}$$

and the elastic torques acting on the endolymph and perilymph are, respectively,

$$T_e=A_eR_e\delta p \tag{8}$$

and

$$T_p=A_pR_p\delta p \tag{9}$$

If we disregard the viscous forces in the regions of the bony vestibule and utricle, the equations of motion of the perilymph and endolymph can be written as

$$\Theta_e\ddot{\xi}_e+\Pi_e\dot{\xi}_e+\Delta_{ee}\xi_e+\Delta_{ep}\xi_p=-\Theta_e\alpha \tag{10}$$

and

$$\Theta_p\ddot{\xi}_p+\Pi_p\dot{\xi}_p+\Delta_{pe}\xi_e+\Delta_{pp}\xi_p=-\Theta_p\alpha \tag{11}$$

where ξ_i denotes the mean angular displacement of the particular fluid ($i=e$ refers to endolymph, $i=p$ to perilymph), and where

$$\Theta_i=2\rho\pi A_iR_i^3$$

$$\Pi_i=16\mu_i\pi^2R_i^3\left(\frac{\varphi_i}{2\pi}\right)$$

and

$$\Delta_{ij}=K_cA_iR_iA_jR_j$$

The coefficients in equations 10 and 11 are determined using the following data for the human semicircular canals:

A_e	$=8.3\times10^{-4}$ cm^2	(ref. 14)
A_p	$=1.1\times10^{-2}$ cm^2	(estimate)
R_e	$=0.32$ cm	(ref. 14)
R_p	$=0.30$ cm	(estimate)
ρ_i	$=1.0$ g/cm^3	(ref. 7)
μ_e	$=0.852$ centipoise	(ref. 7)
μ_p	$=0.802$ centipoise	(ref. 7)
$\frac{\varphi_e}{2\pi}$	$=\frac{2}{3}$	(estimate)
$\frac{\varphi_p}{2\pi}$	$=0.6$	(estimate)

With these values, the differential equations of motion are given by

$$\ddot{\xi}_e+170\dot{\xi}_e+(4.1\times10^{-4})$$
$$K_c\xi_e+(5.1\times10^{-3})K_c\xi_p=-\alpha \qquad (12)$$

and

$$\ddot{\xi}_p+11\dot{\xi}_p+(4.7\times10^{-4})$$
$$K_c\xi_e+(5.8\times10^{-3})K_c\xi_p=-\alpha \qquad (13)$$

The stiffness of the cupula K_c is unknown and therefore retained as a parameter. Assuming ξ_e and ξ_p to be of the same order of magnitude, we can drop the third term in equations 12 and 13 and reduce them to the form

$$\ddot{\xi}_e+\frac{\Pi_e}{\Theta_e}\dot{\xi}_e+\frac{\Delta_{ep}}{\Theta_e}\xi_p=-\alpha \qquad (14)$$

and

$$\ddot{\xi}_p+\frac{\Pi_p}{\Theta_p}\dot{\xi}_p+\frac{\Delta_{pp}}{\Theta_p}\xi_p=-\alpha \qquad (15)$$

This system of equations is now solved for a representative function $\alpha(t)$.

A typical head movement of short duration can be approximated by an abrupt onset of a constant angular velocity followed by an equally abrupt cessation. The angular acceleration and deceleration $\alpha(t)$ associated with such a movement is defined as a positive Dirac delta function $\delta(t)$ at the beginning of the motion and a negative one at its termination. Equation 15 stipulates that the perilymph motion has the same character as that of a damped pendulum. If the system is initially at rest and if $\alpha(t)=\delta(t)$, we can write for the Laplace transform of equation 15

$$s^2x_p(s)+\frac{\Pi_p}{\Theta_p}s\,x_p(s)+\frac{\Delta_{pp}}{\Theta_p}x_p(s)=-1 \qquad (16)$$

from which follows

$$x_p(s)=\frac{-1}{s^2+\frac{\Pi_p}{\Theta_p}s+\frac{\Delta_{pp}}{\Theta_p}} \qquad (17)$$

Since physiological evidence suggests that the motion of the cupula is overdamped, it follows that the perilymph motion would also have to be overdamped; therefore,

$$K_{pp}<\frac{\Pi_p^2}{4\Theta_p} \qquad (18)$$

This allows for the approximation

$$x_p(s)\approx\frac{-1}{\left(s+\frac{\Delta_{pp}}{\Pi_p}\right)\left(s+\frac{\Pi_p}{\Theta_p}\right)} \qquad (19)$$

and

$$\xi_p(t)\approx\frac{e^{-\frac{\Pi_p}{\Theta_p}t}-e^{-\frac{\Delta_{pp}}{\Pi_p}t}}{\frac{\Pi_p}{\Theta_p}-\frac{\Delta_{pp}}{\Pi_p}} \qquad (20)$$

By again making use of equation 18, we can further simplify equation 20 and reduce the expression for $\xi_p(t)$ to

$$\xi_p(t)\approx\frac{e^{-\frac{\Delta_{pp}}{\Pi_p}}}{\frac{\Pi_p}{\Theta_p}}\text{for } t>0 \qquad (21)$$

For $\alpha(t)=\delta(t)$ and the system initially at rest, the Laplace transform of equation 14 is

$$s^2x_e(s)+\frac{\Pi_e}{\Theta_e}sx_e(s)+\frac{\Delta_{ep}}{\Theta_e}x_p(s)=-1 \qquad (22)$$

Substituting equation 19 for $x_p(s)$, we find

$$x_e(s)\approx\frac{\frac{\Delta_{ep}}{\Theta_e}}{s\left(s+\frac{\Pi_e}{\Theta_e}\right)\left(s+\frac{\Delta_{pp}}{\Pi_p}\right)\left(s+\frac{\Pi_p}{\Theta_p}\right)}-\frac{1}{s\left(s+\frac{\Pi_e}{\Theta_e}\right)} \qquad (23)$$

and

$$\xi_e(t) \approx \frac{\frac{\Delta_{ep}}{\Theta_e}}{\left(\frac{\Pi_e}{\Theta_e}\right)\left(\frac{\Delta_{pp}}{\Pi_p}\right)\left(\frac{\Pi_p}{\Theta_p}\right)} - \frac{\frac{\Delta_{ep}}{\Theta_e} e^{-\frac{\Pi_e}{\Theta_e}t}}{\left(\frac{\Pi_e}{\Theta_e}\right)\left(\frac{\Pi_e}{\Theta_e} - \frac{\Delta_{pp}}{\Pi_p}\right)\left(\frac{\Pi_e}{\Theta_e} - \frac{\Pi_p}{\Theta_p}\right)} - \frac{\frac{\Delta_{ep}}{\Theta_e} e^{-\frac{\Delta_{pp}}{\Pi_p}t}}{\left(\frac{\Delta_{pp}}{\Pi_p}\right)\left(\frac{\Delta_{pp}}{\Pi_p} - \frac{\Pi_e}{\Delta_e}\right)\left(\frac{\Delta_{pp}}{\Pi_p} - \frac{\Pi_p}{\Theta_p}\right)} - \frac{\frac{\Delta_{ep}}{\Theta_e} e^{-\frac{\Pi_p}{\Theta_p}t}}{\left(\frac{\Pi_p}{\Theta_p}\right)\left(\frac{\Pi_p}{\Theta_p} - \frac{\Pi_e}{\Theta_e}\right)\left(\frac{\Pi_p}{\Theta_p} - \frac{\Delta_{pp}}{\Pi_p}\right)} - \frac{1 - e^{-\frac{\Pi_e}{\Theta_e}t}}{\frac{\Pi_e}{\Theta_e}} \quad (24)$$

Except for very small values of t, the first four terms on the right-hand side of equation 24 are negligible, since $\Delta_{pp}/\Pi_p < \Pi_p/4\Theta_p$, $\Pi_p/\Theta_p \approx \Pi_e/15\Theta_e$, and $\Delta_{ep}/\Theta_e \approx \Delta_{pp}/9\Theta_p$.
Therefore,

$$\xi_e(t) \approx -\frac{1 - e^{-\frac{\Pi_e}{\Theta_e}t}}{\frac{\Pi_e}{\Theta_e}} \quad \text{for } t>0 \quad (25)$$

However, with $\Pi_e/\Theta_e \approx 170$, $e^{-\frac{\Pi_e}{\Theta_e}t}$ also approaches zero rapidly and we can thus conclude that ξ_e almost instantaneously assumes the approximate value

$$\xi_e(t) \approx -\frac{1}{\Pi_e/\Theta_e} \quad \text{for } t>0 \quad (26)$$

From equations 21 and 26 we see that the almost immediate displacements of the endolymph and perilymph are inversely proportional to the respective coefficients Π_e/Θ_e and Π_p/Θ_p. For the anatomical parameters listed, we have therefore $\xi_p > 15\xi_e$ after the onset of head movement, which appears to justify ignoring the third term in equations 12 and 13. If we rotate the head for only a very short duration, the exponential decay of ξ_p is negligible. Finally, when head rotation is terminated, the two fluid systems receive a negative impulse, which essentially returns them to their original positions.

According to equation 6 the volumetric displacement of the cupula is (after inserting anatomical values)

$$\delta V = (2.7\times10^{-4})\xi_e + (3.2\times10^{-2})\xi_p \quad (27)$$

With $\xi_p > 15\xi_e$ for the typical head movement just discussed, the contribution of the perilymph to the volumetric displacement of the cupula is almost 200 times that of the endolymph. Other acceleration patterns produce similar results, which suggest that the cupula displacement is primarily caused by the perilymph and not by the endolymph. If the contribution of the endolymphatic motion to the cupula displacement is ignored, the governing equation for the dynamic response of the perilymph or the corresponding cupula displacement reduces to

$$\ddot{\xi}_p + 11\dot{\xi}_p + 0.0058K_c\xi_p = -\alpha \quad (28)$$

This means that the response of the cupula is still characterized by a torsion pendulum equation but with the significant difference that the bony canal parameters rather than those of the membranous canal define the dynamics of the system.

DISCUSSION

The findings of this study may be summarized as follows:

(1) By assuming that the ampulla wall offers no resistance to small displacements, it was shown that it is primarily the perilymph that causes the deflection of the cupula.

(2) When we allow for perilymph displacement, the equation of motion of the system retains the form of the torsion pendulum equation, with the variable ξ now representing the angular displacement of the perilymph rather than the endolymph.

(3) Based on the dimensions of the perilymphatic space, the theoretically determined coefficient Π/Θ in the torsion pendulum equation is approximately 11 s^{-1}, while the dimensions of the membranous

canal lead to a value of about 170 s^{-1} for the same term.

Experimental determination of Π/Θ through measurements of the slow-phase velocity of nystagmus and through subjective sensations of angular velocity leads to values for Π/Θ which are of the order of 10 s^{-1}. The agreement between the experimentally determined values for Π/Θ and that predicted by our model lends support to the hypothesis that the ampulla wall offers little or no resistance to displacements and thereby allows the perilymph to produce cupular deflection. The validity of this hypothesis, however, can be determined only through experimentation on the ampulla wall itself. The results of this investigation appear to suggest a thorough reexamination of the nature of the ampulla wall.

REFERENCES

1. Steinhausen, W.: Über Sichtbarmachung und Funktionsprüfung der Cupula Terminalis in den Bogengangsampullen des Labyrinthes. Pflügers Arch. ges. Physiol., vol. 217, 1927, pp. 747–755.
2. Steinhausen, W.: Über den Nachweis der Bewegung der Cupula in der Intakten Bogengangsampulle des Labyrinthes bei der Natüralichen Rotatorischen und Calorischen Reizung. Pflügers Arch. ges Physiol., vol. 228, 1931, pp. 322–328.
3. Steinhausen, W.: Über die Beobachtung der Cupula in den Bogengangsampullen des Labyrinths des Lebenden Hechts. Pflügers Arch. ges. Physiol., vol. 232, 1933, pp. 500–512.
4. Steinhausen, W.: Über Modellversuche zur Physiologie des Labyrinthes und über ein Neues Bogengangsmodell. Acta Oto-Laryngol., vol. 27, 1939, pp. 107–122.
5. Van Egmond, A. A. J.; Groen, J. J.; and Jongkees, L. B. W.: The Mechanics of the Semicircular Canal. J. Physiol., vol. 110, 1949, pp. 1–17.
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. Naval School of Aviation Medicine, Pensacola, Fla., 1961.
7. Steer, R. W.: The Influence of Angular and Linear Acceleration and Thermal Stimulation on the Human Semicircular Canal. Sc.D. thesis, Massachusetts Institute of Technology, 1967.
8. Rejtö, A.: Die Rolle der Perilymphe in der Entstehung des Kalorischen Nystagmus. Acta Oto-Laryngol., vol. 27, 1939, pp. 270–280.
9. Spitzer, A.: Über die Funktion des Bogengänge des Ohrlabyrinthes. Mschr. Ohrenheilk, 1925, vol. 59, pp. 1131–1176. Cited by A. Rejtö in Acta Oto-Laryngol., vol. 27, 1939, pp. 270–280.
10. Lorente de Nó, R.: Die Labyrinth Reflexe auf die Augenmuskeln nach Einseitiger Labryinthextirpation. Cited by A. Rejtö in Acta Oto-Laryngol., vol. 27, 1939, pp. 270–280.
11. Dohlman, G.: Die Rolle der Perilymphe bei den Vestibulären Reaktionen. Arch. Ohr-, Nas.-, u. Kehlkheilk., vol. 150, 1941, pp. 25–30.
12. Anliker, M.; and Dorfman, M.: Theoretical Model Studies of Wave Transmission in Semicircular Canal Ducts. Ingenieur-Archiv, August 1970.
13. Anliker, M.; and Van Buskirk, W. C.: Experimental Model Studies of Wave Transmission in Semicircular Canal Ducts. In Proceedings of a Colloquium on Dynamic Response of Biochemical Systems. ASME, New York (in press).
14. Igarashi, M.: Dimensional Study of the Vestibular End Organ Apparatus. Second Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–115, 1966, pp. 47–53.
15. *Gray's Anatomy*. 28th Edition, Lea and Febiger, Philadelphia, 1967.

SESSION VI

Chairman: **CÉSAR FERNÁNDEZ**
University of Chicago

Scanning Electron Microscopy of the Vestibular End Organs

HENRIK H. LINDEMAN, HARLOW W. ADES, and ROGER W. WEST
University of Illinois

SUMMARY

The vestibular end organs, after chemical fixation, were freeze-dried, coated with gold and palladium, and studied in the scanning microscope. Scanning microscopy gives a good three-dimensional view of the sensory areas and allows study of both gross anatomy and microstructures. Gross anatomical features of the structure of the ampullae are demonstrated. The form of the statoconia in different species of animals is shown. New aspects of the structure of the sensory hairs are revealed. The hair bundles in the central areas of the cristae and in the striola of the maculae differ structurally from the hair bundles at the periphery of the sensory regions. Furthermore, some hair bundles consisting of very short stereocilia were observed. The relationship between the cupula and the statoconial membrane to the epithelial surface is discussed.

INTRODUCTION

Recent studies have shown that the vestibular sensory regions are structurally highly differentiated. This is reflected in the organization of the sensory cells, which are morphologically polarized according to a specific pattern (refs. 1 to 5), a feature of great functional significance. Furthermore, there are distinct structural differences between central and peripheral areas of the sensory regions (refs. 5 to 7), which indicate that the different areas are functionally dissimilar. This differentiated organization of the maculae and cristae should be taken into account in both morphological and functional investigations.

The present study is based upon scanning electron microscopy of the vestibular apparatus. The scanning electron microscope in the last few years has been applied to a wide range of biological and nonbiological material. In particular, the instrument has proven to be an important tool in the study of surface structures. Thanks to its large depth of field, it enables us to get a good three-dimensional view of the sensory areas in the inner ear. Moreover, it allows exact localization on the specimen, which, together with its good resolution, makes possible detailed and systematic studies under high-power magnification. Recent investigations indicate that scanning electron microscopy will be a useful complement to other techniques in studies both of the cochlea (refs. 8 to 10) and of the vestibular apparatus (refs. 11 and 12).

MATERIAL AND METHODS

The temporal bones of guinea pig, chinchilla, and cat were used in the present investigation. The animals were decapitated under barbital anesthesia and the bullae removed and opened. Following a wide opening to the vestibule, the inner ear was perfused with a fixative consisting of six parts of 2-percent phosphate-buffered osmic acid and one part of a saturated solution of mercuric chloride (ref. 13). In some cases 1-percent barbital-buffered osmic acid was used as a fixative. The specimens were kept in the fixative at 4° C for 2 to 15 hours. Dissection of the vestibular apparatus was performed in distilled water under the stereomicroscope. The specimens were then prepared for scanning electron microscopy by a technique slightly modified from a description by Marszalek and Small (ref. 14). Each specimen was placed in a drop of double-distilled water on a thin aluminum-foil disk, which was quickly transferred to liquid nitrogen-cooled isopentane at about —150° C. It was next placed on a copper block, precooled in

liquid nitrogen, and the block was transferred to the cold stage of a Pearce tissue drier, where the specimen underwent sublimation under high vacuum (5×10^{-2} torr) at a temperature of —50 to —60° C for 3 to 4 hours. The specimens were then allowed to attain room temperature overnight. The aluminum disks with freeze-dried specimens were mounted on standard scanning electron-microscopy stubs with cement, placed on a rotating table in a vacuum evaporator, and coated with a thin film of gold and palladium. The specimens were studied in a Cambridge Stereoscan scanning electron microscope.

RESULTS AND DISCUSSION

The sensory regions of the vestibular apparatus were studied under primary magnifications varying from 50 to 15 000 times. This range in magnification allowed studies of both the macroscopic anatomy and microstructures. Figure 1 shows a vertical ampulla of a cat with the cupula riding at the top of the crista. According to investigations by Steinhausen (ref. 15), the cupula, under normal conditions, extends from the surface of the sensory epithelium to the roof of the ampulla, and laterally to the plana semilunata. It thus closes the lumen of the ampulla completely to endolymph (ref. 16). However, under the influence of fixatives, the cupula, like the gelatinous substance of the statoconial membrane and the tectorial membrane, shrinks considerably, and thus alters both size and form. It is important to realize that changes seen in the cupula, often assumed to be due to pathological conditions, in most cases are probably the result of fixation and embedding procedures.

The crista is covered by sensory epithelium. Figure 2 shows the lateral crista of a cat, revealing the hair bundles of the sensory cells and myelinated nerve fibers beneath the epithelium, representing the dendrites of the bipolar vestibular ganglion cells. The long axis of the crista extends across the ampulla and is generally described as being perpendicular to the plane of the semicircular duct (refs. 17 and 18). However, the position of the cristae, in the guinea pig particularly the anterior one, shows certain irregularities (ref. 5). In the first place, the crista is not found at right angles to the plane of the semicircular duct (fig. 3), and it also occupies an asymmetric position basally in the ampulla, closer to one lateral wall than to the other.

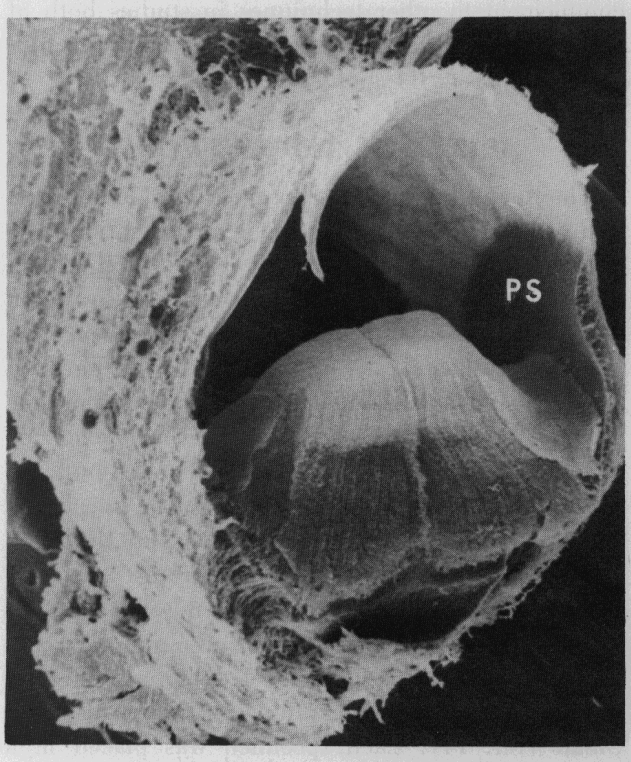


FIGURE 1.—*Scanning micrograph showing ampulla of a vertical semicircular duct in the cat. The cupula is riding at the top of the crista. The planum semilunatum (PS) is located at the lateral wall of the ampulla. (×83)*

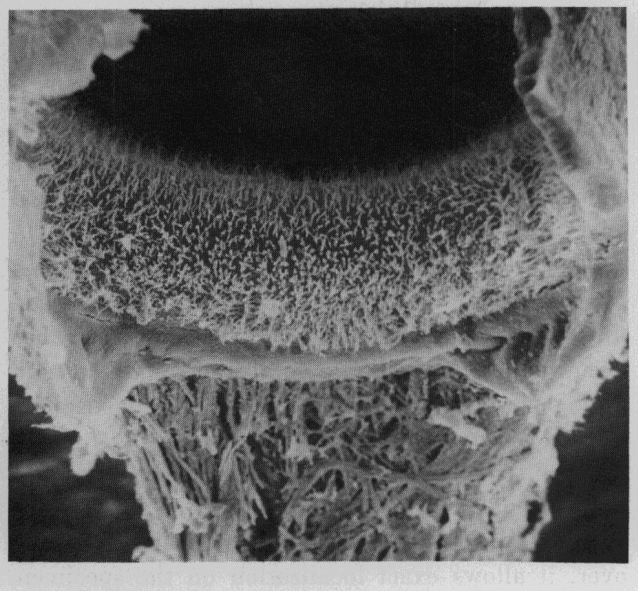


FIGURE 2.—*Photomicrograph showing lateral crista of the cat. Hair bundles protrude from the sensory cells. Beneath the epithelium, myelinated nerve fibers are seen. (×96)*

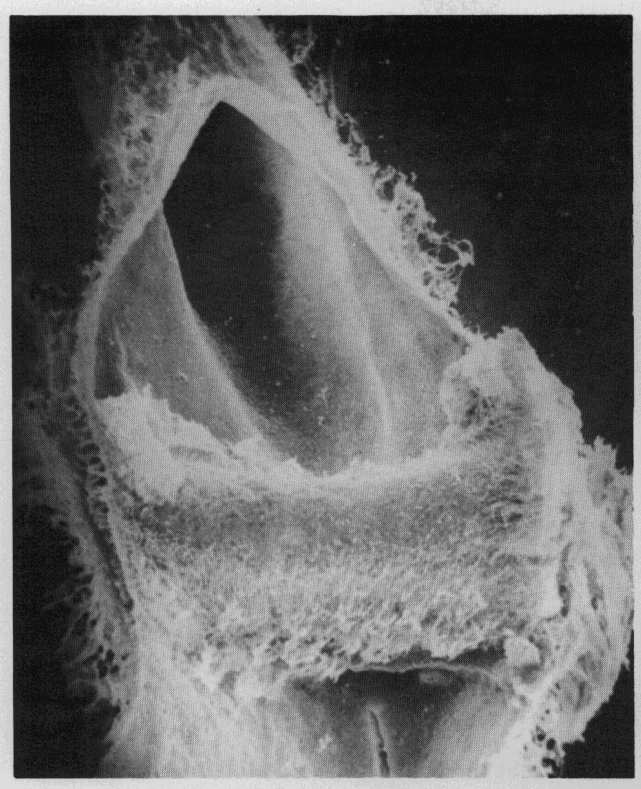


FIGURE 3.—*Scanning micrograph showing the anterior ampulla in the guinea pig. The long axis of the crista is not at right angles to the plane of the semicircular duct. (×85)*

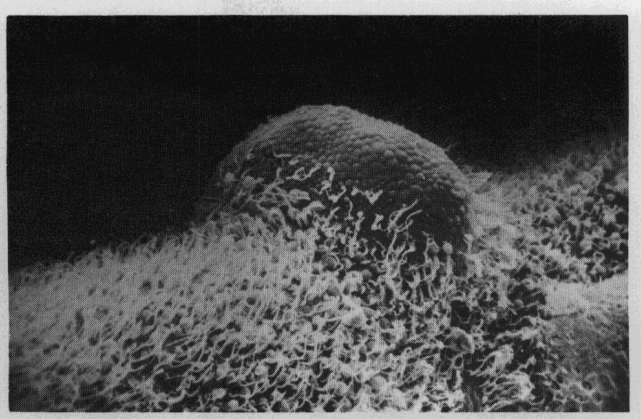


FIGURE 4.—*In the cat the cristae of the vertical semicircular ducts are divided into two parts by a ridge, the septum cruciatum, which contains no sensory cells. (×206)*

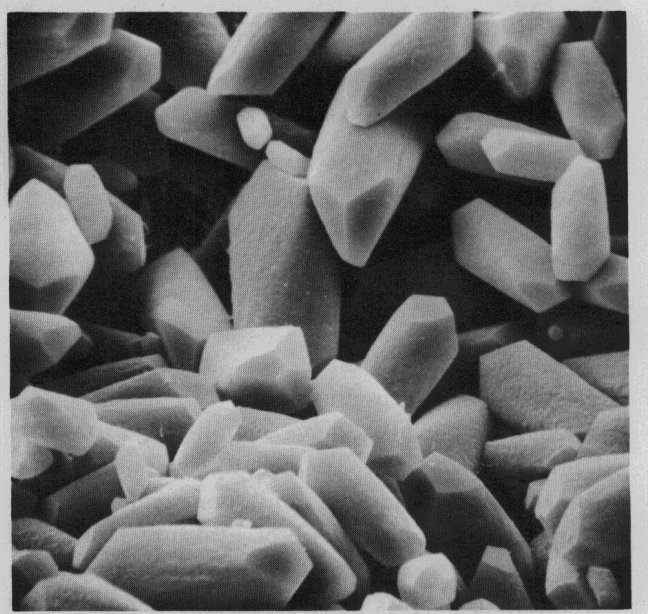


FIGURE 5.—*Scanning micrograph showing statoconia from the macula utriculi in the cat. The crystals have an oblong form. The body is more or less cylindrical and the ends are pointed. Each short side possesses three surfaces. (×1090)*

The anterior and posterior cristae of the cat are incompletely divided into two parts by a transverse ridge, commonly referred to as the septum cruciatum (fig. 4). This ridge is covered by cylindrical epithelial cells and contains no sensory cells.

The plana semilunata are half-moon-shaped areas on the lateral walls of the ampullae (fig. 1) and have attracted considerable interest. They consist of cylindrical cells of complicated structure (ref. 19), indicating a secretory function. Recently Dohlman and collaborators (refs. 20 and 21) showed evidence for a mucopolysaccharide secretion from this area in birds by the use of labeled isotopes.

The macula utriculi and the macula sacculi are covered by statoconial (otolith) membranes of the same form as the sensory epithelium. The statoconial membrane consists of a gelatinous component, directly overlying the hair bundles of the sensory cells, and a mass of crystals—statoconia (figs. 5 to 7). The statoconia lie close together in several layers in the most distal part of the statoconial membrane. Even though the statoconia do not seem to be extensively embedded in the gelatinous substance (which, from a morphological point of view, appears like a cushion for the crystals), they are still relatively firmly held together, and they do not normally detach from the statoconial membrane.

In the scanning microscope the form of the

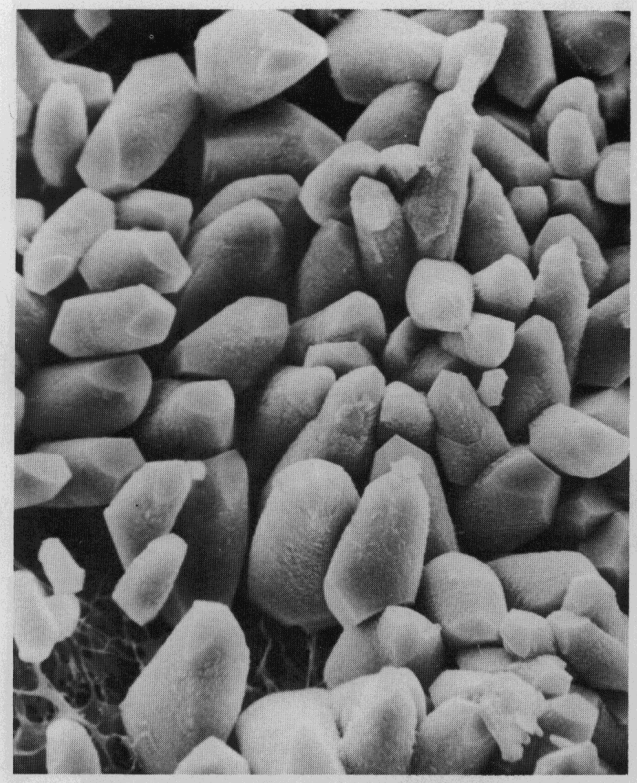


FIGURE 6.—*Statoconia from the macula sacculi of the chinchilla. (×1170)*

crystals is easily seen (figs. 5 to 7). In all the species studied the statoconia have an oblong form with slightly arched sides. Their body is more or less circular in cross section and their ends pointed. In agreement with the observation by Lim and Lane (ref. 12), each short side of the statoconium was found to possess three surfaces which appeared absolutely smooth. The size of the statoconia varies in the guinea pig from about 0.5 μm to about 30 μm, and there are clear regional differences in the size of the crystals as well as in the thickness of the crystal layer (ref. 5). In the striola of the macula utriculi, the crystal layer is thinner than on either side, whereas on the macula sacculi the crystal layer in the striola is thicker than on either side. Moreover, in the striola of both maculae the crystals are all small.

The statoconia have a specific gravity of about 2.71. Recently Veenhof (ref. 22) has shown that each statoconium in mice contains two crystalline substances, calcite and a crystalline organic substance of unidentified chemical structure. He concluded that the formation of statoconia in the mouse takes place from the 13th day of embryonic life until some days after birth. After this period there is no growth of statoconia.

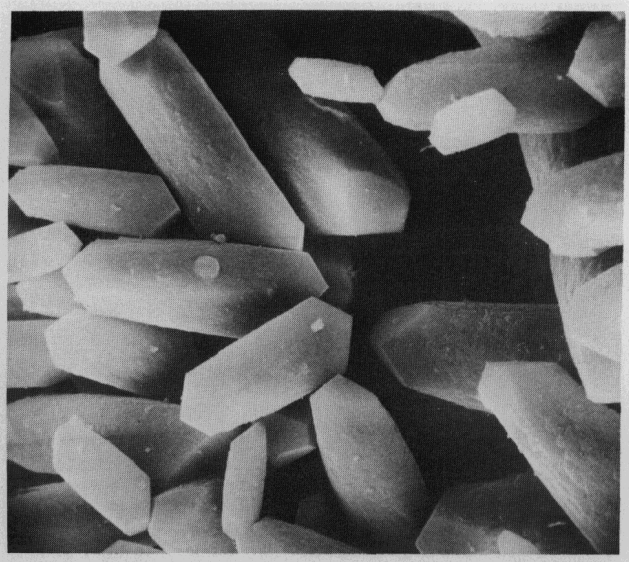


FIGURE 7.—*Statoconia from the macula sacculi of the guinea pig. (×1840)*

Previous studies have indicated that the crystals are vulnerable to fixatives and storage in the cold (refs. 5 and 17), a fact of relevance when discussing whether or not pathological changes are present. In the present study some temporal bones revealed a complete disappearance of the statoconia. In other temporal bones the dissolution was incomplete, leaving only the striola of the macula utriculi without crystals. Furthermore, in some specimens giant crystals were seen, which differed considerably in both size and shape from normal statoconia (figs. 8 and 9). The observations described above were made on fixed specimens; however, similar findings were present in two "normal" guinea pigs, studied under the dissection microscope less than 2 minutes after sacrifice and before fixation. This indicates that dissolution of statoconia and changes in the structure of the crystals may occur intravitally. It may be added that light and electron microscopy of the maculae in the two guinea pigs just mentioned revealed normal sensory epithelia.

Scanning electron microscopy of statoconial membranes from the cat in which the crystals had disappeared allowed studies of the gelatinous substance.

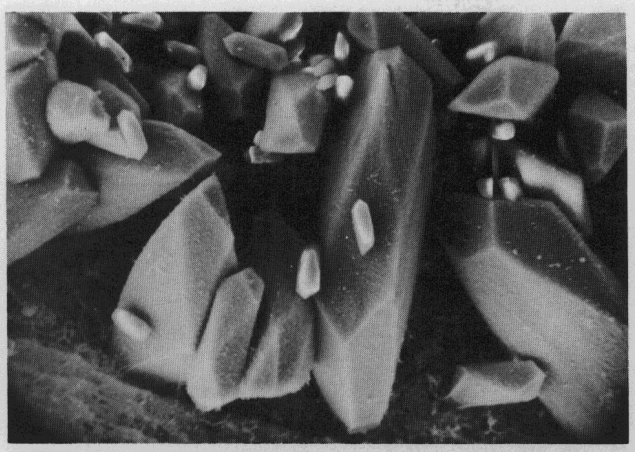


FIGURE 8.—*Giant crystals in the utricular macula of the cat. These crystals differ both in size and form from normal statoconia. (×495)*

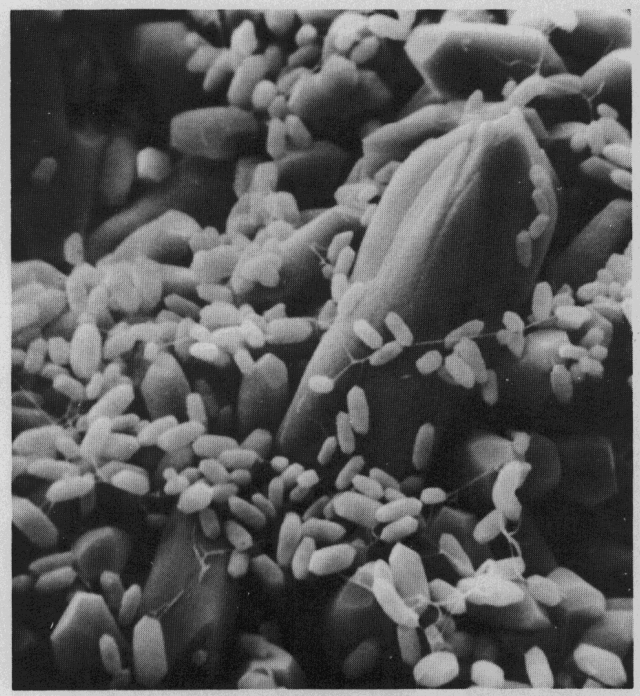


FIGURE 9.—*Giant crystals from the utricular macula of the guinea pig, surrounded by normal appearing crystals. (×1350)*

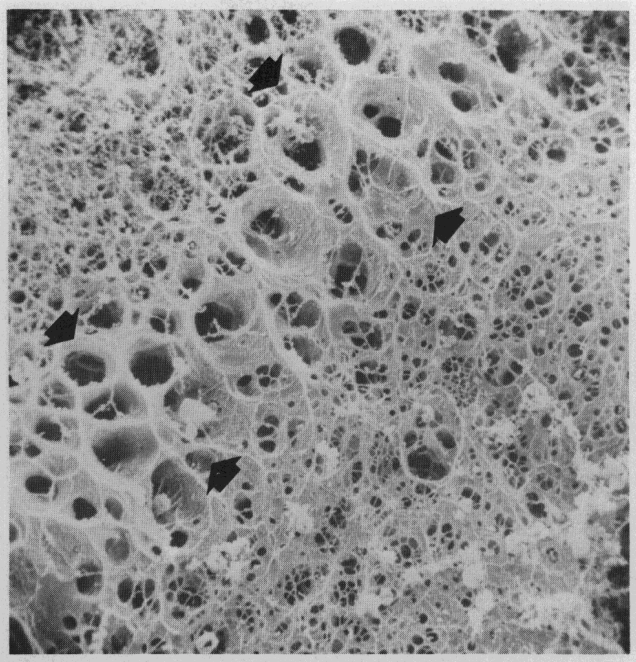


FIGURE 10.—*Scanning micrograph showing the gelatinous component of the statoconial membrane of the saccular macula in the cat. The gelatinous structure shows a significant reinforcement in the striola (between arrows). (×525)*

These studies support previous observations in light microscopy of the guinea pig (ref. 5). The gelatinous substance had a fibrillar appearance and showed a significant reinforcement of this fibrillar structure in the striola (fig. 10) of both maculae. The relationship between the cupula and the epithelial surface has been a matter of dispute (see ref. 5). Kolmer (refs. 23 and 24) was of the opinion that the gelatinous substance extended down to the surface of the sensory epithelium on both cristae and maculae. On the other hand, a subcupular space has often been described as a slit between the epithelial surface and the gelatinous substance. It must be emphasized, however, that apparently all previous studies on this subject have been based on fixed specimens. As already stated, the gelatinous structure is extremely vulnerable to fixation, especially just above the sensory epithelium (ref. 23). The variation of opinions concerning the depth of the subcupular space is obviously due to difference in degree of shrinkage. This conclusion is supported by the present study. Figure 11 shows a definite subcupular space. Figure 12, on the other hand, reveals fine threads of cupular substance extending down between the hair bundles; if there is indeed a subcupular space, it is clearly less than in figure 11. On the maculae, scanning electron microscopy showed evidence of a direct contact between the gelatinous substance and the sensory epithelium,

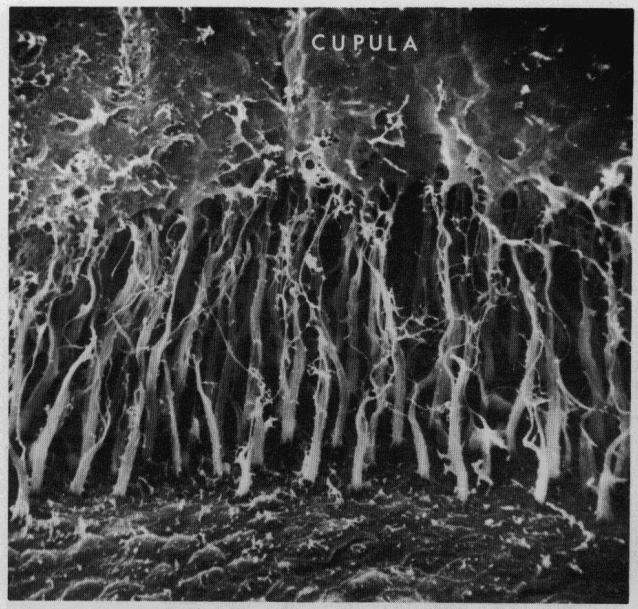


FIGURE 11.—*Scanning micrograph showing the relationship between the epithelial surface, the sensory hairs, and the cupula in the cat. There appears to be a clear-cut subcupular space, even though some fine threads of cupular substance can be seen extending down between the hair bundles. (×1250)*

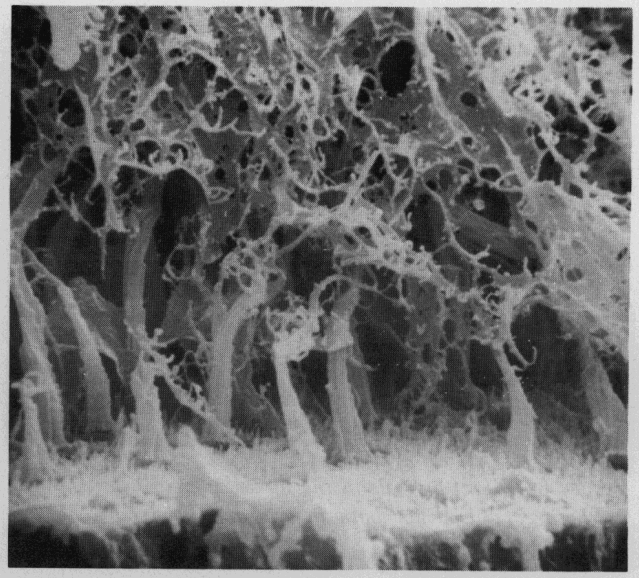


FIGURE 12.—*In this scanning micrograph from an ampulla of another cat, the subcupular space, if indeed there is any, is clearly less than in figure 11. (×2385)*

revealing a meshwork of fine threads, some of which extended to the epithelial surface (fig. 13). This indicates that there is no real subcupular space on the maculae.

Each sensory cell is provided with a bundle of hairs, consisting of one kinocilium and several stereocilia. The stereocilia are anchored in the cuticular plate of the sensory cell in a completely regular pattern. The kinocilium is located at the periphery of the hair bundle and is the longest of the sensory hairs. The stereocilia vary considerably in length, those nearest to the kinocilium being the longest, and the length decreasing gradually with increasing distance from the kinocilium (figs. 14 to 17). The position of the kinocilium on the free surface of the sensory cell is eccentric. The sensory cell is thus morphologically polarized, and the direction of polarization is indicated by the position of the kinocilium in relation to the stereocilia (figs. 14, 16, and 17). This morphological polarization coincides with that of a functional polarization (directional sensitivity) of the sensory cells (refs. 1 and

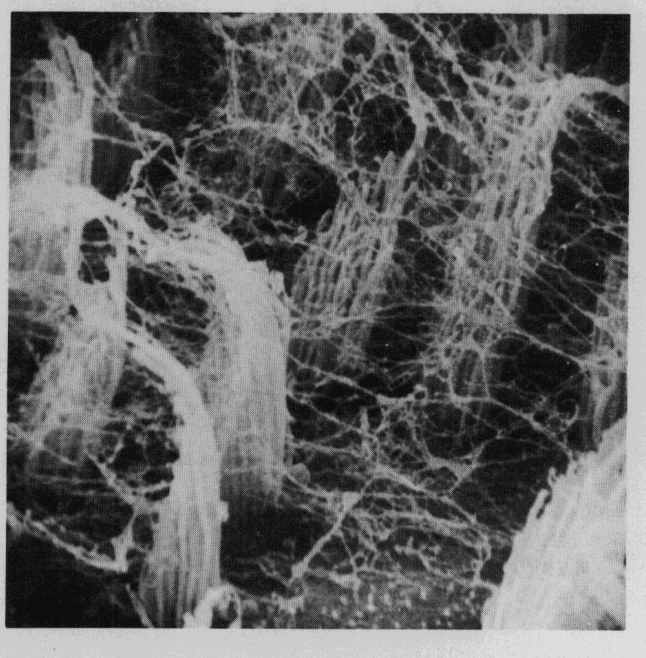


FIGURE 13.—*Scanning micrograph showing the relationship between the gelatinous component of the statoconial membrane of the utricular macula and the epithelial surface on the chinchilla. A meshwork of gelatinous structure is seen between the hair bundles. This extends down to the epithelial surface, indicating that there is no real subcupular space. (×4490)*

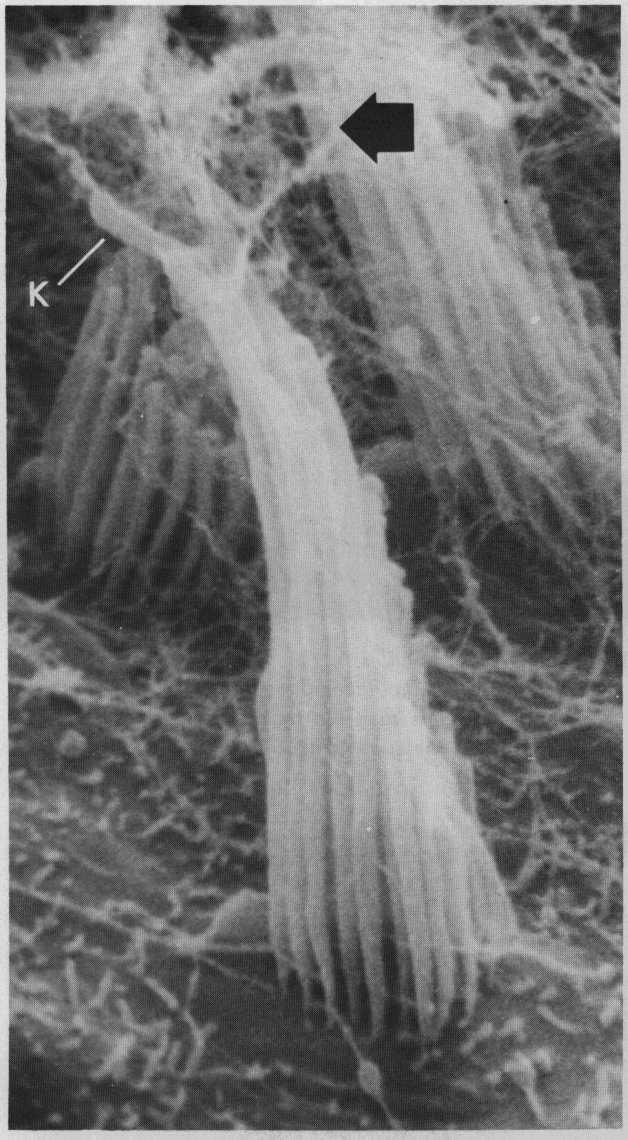


FIGURE 14.—*Scanning micrograph showing a hair bundle on the utricular macula in the chinchilla. The kinocilium (K), which is the longest of the sensory hairs, has an eccentric position at the periphery of the hair bundle. Thus the sensory cell is morphologically polarized. Arrow indicates direction of polarization. The stereocilia show stepwise decreasing length with increasing distance from the kinocilium. (×10 125)*

3). Thus a displacement of the sensory hairs in the direction of the arrow in figure 14 is considered to be accompanied by depolarization of the sensory cell and an increased discharge rate in the afferent nerve fibers (refs. 25 to 27). On the other hand, a displacement of the hairs in the opposite direction is

FIGURE 15.—*Bundles of sensory hairs at the periphery of the macula utriculi in the chinchilla. The stereocilia protrude from the cuticular plate of the sensory cells. The hair bundles have a more or less cylindrical form. Microvilli are seen on the supporting cells. (×6160)*

considered to result in hyperpolarization and a reduction in frequency of impulses in the nerve fibers. Since the functional polarization is reflected in the organization of the hair bundles, a mapping of the morphological polarization will show the functional polarization (directional sensitivity) of the same cells.

Recent studies have shown that the sensory cells of the maculae and cristae are morphologically polarized according to a definite pattern (refs. 1 to 4), a pattern which is basically similar in different mammals, including man (ref. 5). The sensory cells on any single crista are polarized in the same direction. The sensory cells on the lateral crista are polarized toward the utricle; those on the cristae of the two vertical semicircular ducts, away from the utricle.

Each macula is divided into two areas by an arbitrary curved line, with opposite polarization of the sensory cells on either side. This line is situated in the middle of the striola. On the macula sacculi the sensory cells are morphologically polarized

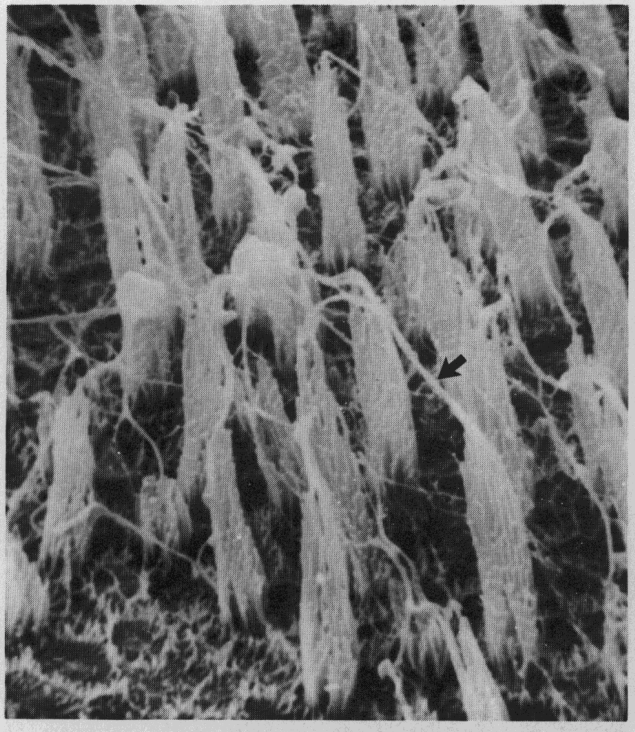


FIGURE 16.—*Scanning micrograph showing hair bundles at the periphery of the utricular macula in the guinea pig. The sensory cells are all polarized in the same direction. Note kinocilium (arrow), which is much longer than the longest stereocilia. (×2640)*

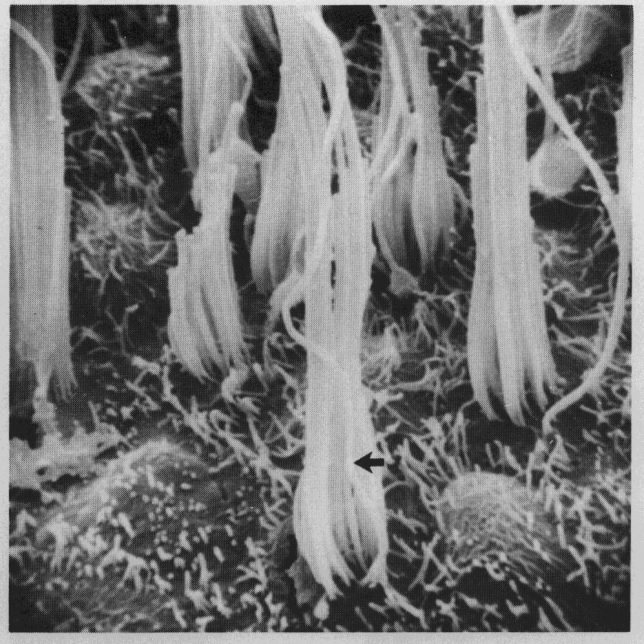


FIGURE 17.—*Hair bundles in the periphery of the crista in the guinea pig. The kinocilium (arrow) appears to be much more flexible than the stereocilia. Numerous microvilli appear on the supporting cells. (×5180)*

away from the line; on the macula utriculi, toward the line. Thus the two maculae differ fundamentally in polarization pattern. It is interesting that they also differ in the general organization of the statoconial membranes. The layer of statoconia in the striola of the macula sacculi is thicker, whereas the layer of statoconia in the striola of the macula utriculi is thinner than on either side of the striola.

The organization of the hair bundles on the maculae and the cristae is the same. However, the hairs on the sensory cells of the cristae are recognized as being generally much longer than the hairs on the maculae (figs. 15 to 19). Furthermore, there are in the guinea pig clear differences in the form as well as in the length of the hairs between the central and the peripheral areas of the sensory regions (ref. 5). Such regional differences in the structure of the hairs can be verified in the scanning microscope (figs. 16 and 20 to 23), revealing new aspects of the structure of the hairs. Similar findings were made in the cat and the chinchilla. The hairs on the

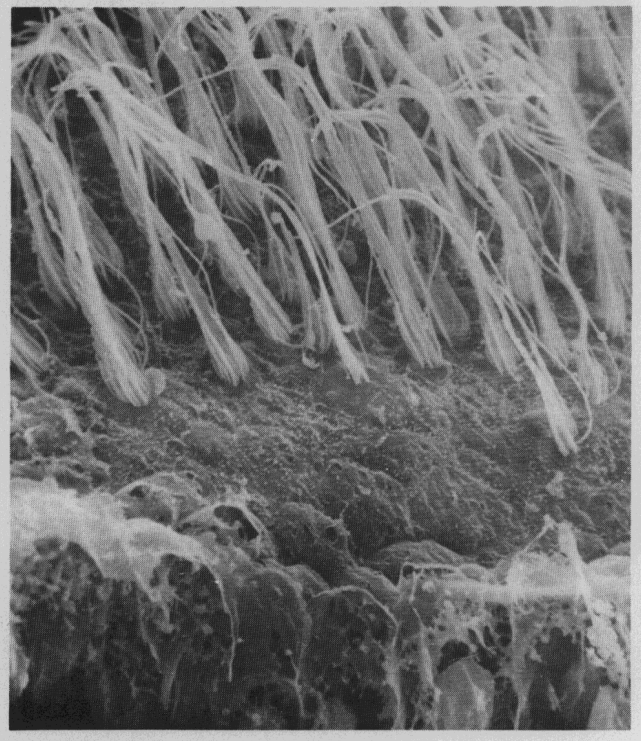


FIGURE 18.—*Sensory hairs at the periphery of the crista of the chinchilla, bordering the planum semilunatum. The hairs are much longer than in the maculae. (×1200)*

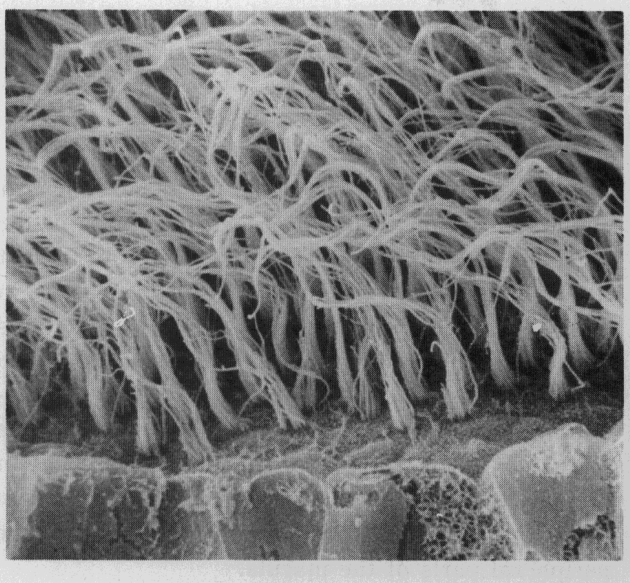


FIGURE 19.—*Sensory hairs bordering the planum semilunatum in the guinea pig. (×1100)*

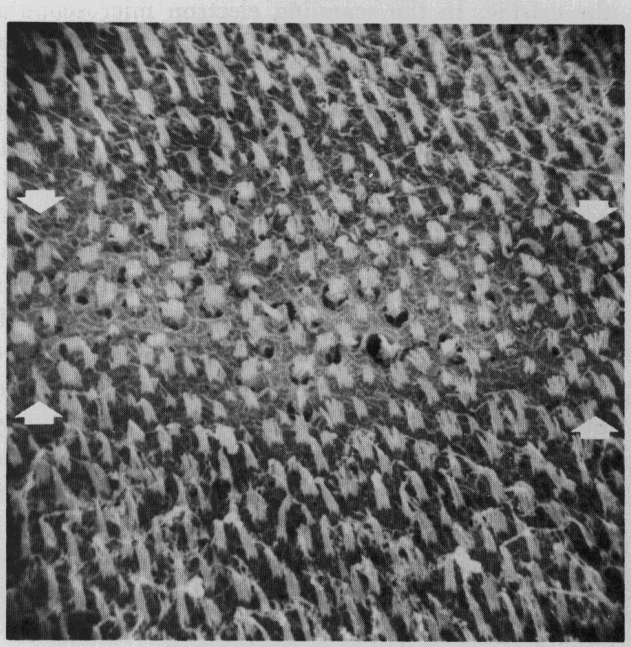


FIGURE 20.—*Scanning micrograph showing hair bundles on the macula utriculi in the guinea pig. The hair bundles have a different appearance in the striola (between arrows) from that on either side of the striola. (×465)*

sensory cells in the periphery of the maculae are aggregated into distinct bundles, which have a rather cylindrical shape close to the epithelial surface, the stereocilia protruding from a more or less circular area of the cuticular plate. The stereocilia are all

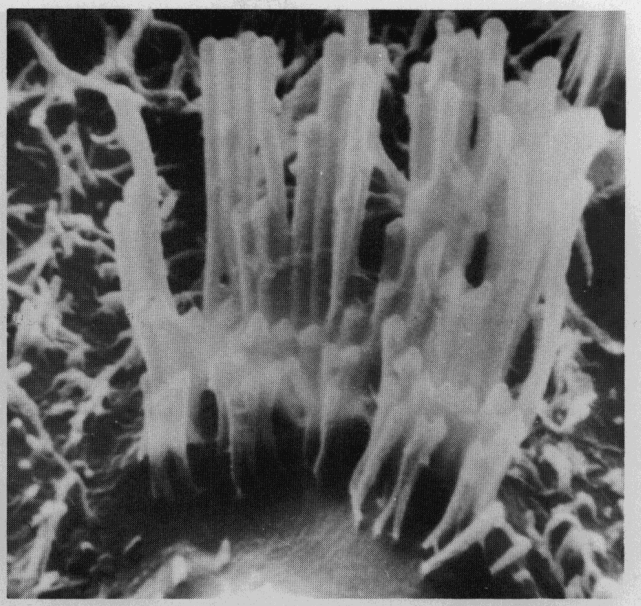


FIGURE 21.—*Hair bundle in the striola of the macula utriculi in the guinea pig. The hairs are arranged mainly in one direction, parallel to the striola. (×8840)*

FIGURE 22.—*Hair bundle from the striola of the macula utriculi in the guinea pig. The stereocilia are club-shaped, the distal portion being much thicker than the proximal. The longest stereocilia are definitely shorter than the longest stereocilia at the periphery. (×9900)*

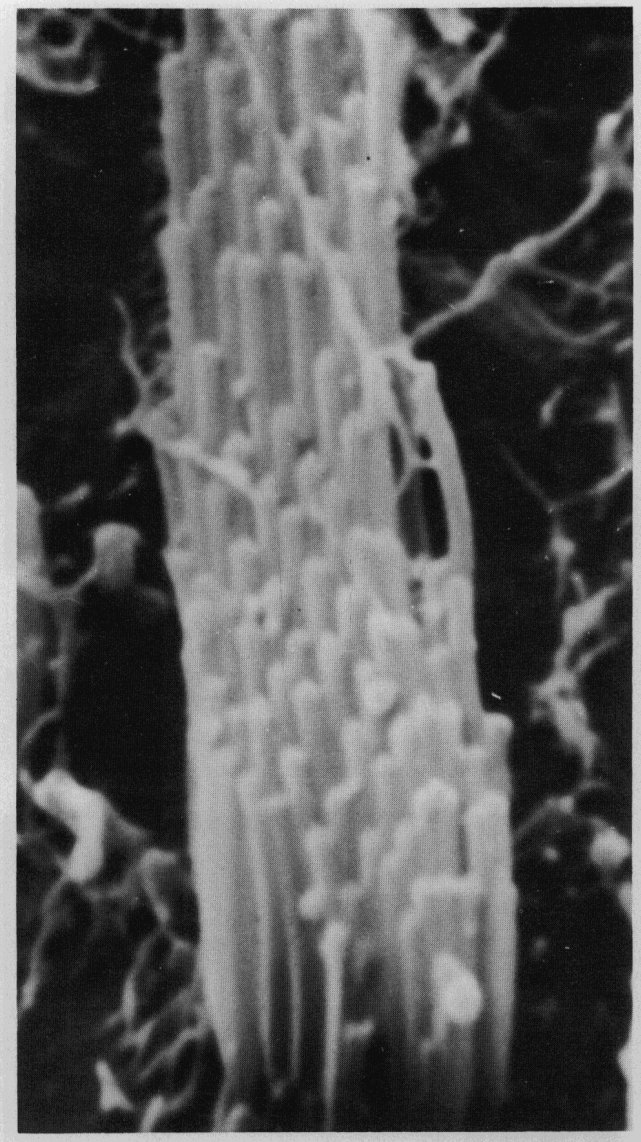


FIGURE 23.—*Hair bundle from the periphery of the same utricular macula as in figure 21. The stereocilia are slender, the distal portion being only slightly thicker than the proximal. (×16 500)*

slender, the diameter of their distal part being only slightly larger than the diameter nearer to the epithelial surface. In the striola (fig. 20) the hair bundles of the sensory cells with a large free-surface area, which in the guinea pig represent type-I cells (ref. 5), appear different. The hairs are arranged mainly in one direction, parallel to the length of the striola, and the hair bundles bear a striking resemblance to the hair bundles of the inner hair cells in the cochlea (figs. 21 and 22). Furthermore, the stereocilia show a distinct club shape, having a much larger diameter in their distal portion than toward the cuticular plate. Moreover, the hair bundles are clearly shorter than in the periphery of the maculae.

In the peripheral parts of the maculae the length of the kinocilium clearly exceeded the length of the longest stereocilia (fig. 16). However, this is not true in the striola; on many of the large cells in this area, no single hair clearly exceeded the others in length. This observation makes it necessary to ask whether some of these cells really lack a kinocilium as do the inner and outer hair cells of the mature organ of Corti. The regional differences in the structure of the hair bundles support the hypothesis that the striola differs functionally from the peripheral parts of the macula.

The present study revealed other aspects of the structure of the hair bundles on the maculae and the cristae. In the scanning electron microscope a small number of the bundles differed from the rest in that the stereocilia were fewer and, above all, much shorter (fig. 24). However, the kinocilia of these cells often appeared to be of normal length. Before concluding that the short bundles represent a normal variety, the objection must be considered that the shortness of the stereocilia may be due to preparation artifacts. It is known that the hair bundles may suffer loss during preparation. In such cases, however, the bundles are torn off at the cuticular plates where the stereocilia are anchored by their thin rootlets (fig. 25), and do not usually break at a distance from the epithelial surface. The short hair bundles thus seem to represent a normal feature, a conclusion which is supported by the observation that the bundles showed the normal gradation in the length of the stereocilia. They were observed both in the periphery and in the striola, where they belonged to the cells with a small free-surface area, the type-II cells (ref. 5).

There are clear regional differences in the form and size of the hairs on the sensory cells of the cristae (ref. 5). The stereocilia in the periphery of the epithelium are long and gracile compared to the short, club-shaped hairs in the central parts. Figures 17 to 19 show hair bundles peripherally on the crista at the border to the planum semilunatum and reveal the kinocilia on one and the same side of the bundles, indicating the direction of polarization.

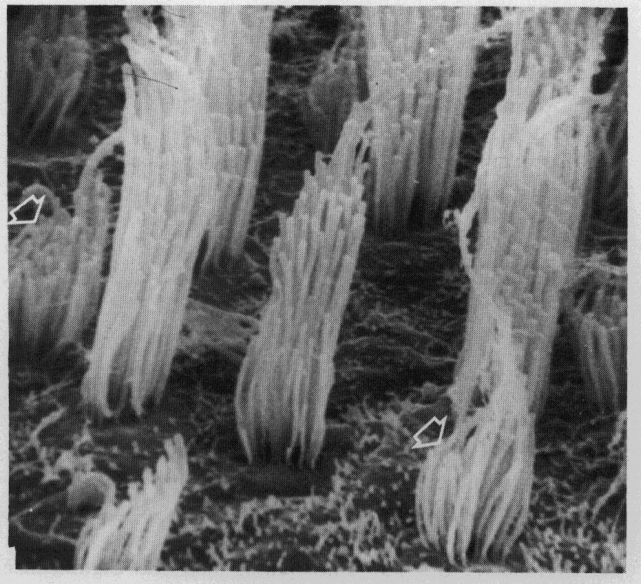


FIGURE 24.—*Scanning micrograph showing hair bundles on the macula utriculi of the chinchilla. Some of the hair bundles consist of stereocilia which are shorter (arrows) than those normally seen; however, the kinocilium of these cells often appeared to be of normal length. (×4650)*

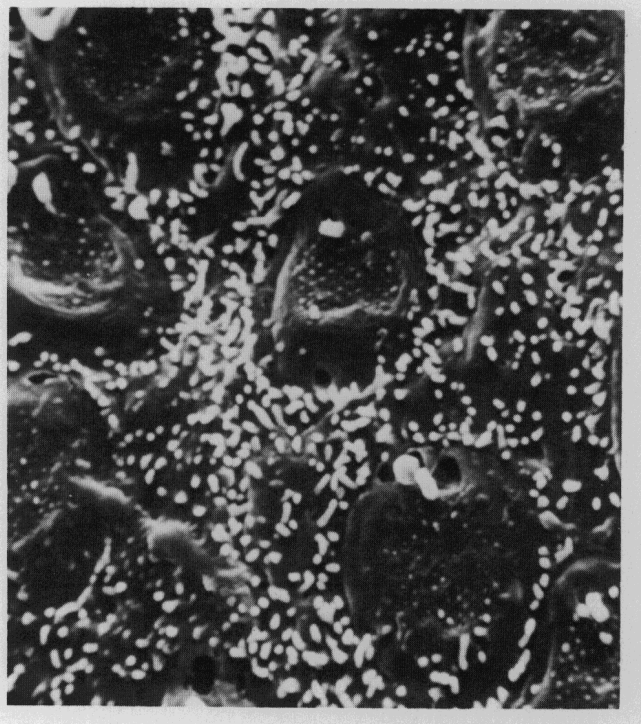


FIGURE 25.—*Scanning micrograph showing the epithelial surface of a crista in the guinea pig. The stereociliar rootlets are regularly arranged in the cuticular plates of the sensory cells. Numerous microvilli are seen on the supporting cells. (×6680)*

REFERENCES

1. LOWENSTEIN, O.; AND WERSÄLL, J.: A Functional Interpretation of the Electronmicroscopic Structure of the Sensory Hairs in the Cristae of the Elasmobranch *Raja Clavata* in Terms of Directional Sensitivity. Nature, vol. 184, 1959, pp. 1807–1808.
2. 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., vol. 34, 1962, pp. 1356–1362.
3. FLOCK, A.: Structure of the Macula Utriculi with Special Reference to Directional Interplay of Sensory Responses as Revealed by Morphological Polarization J. Cell Biol., vol. 22, 1964, pp. 413–431.
4. SPOENDLIN, H. H.: Ultrastructural Studies of the Labyrinth in Squirrel Monkeys. The Role of the Vestibular Organs in the Exploration of Space, NASA SP–77, 1965, pp. 7–22.
5. LINDEMAN, H. H.: Studies on the Morphology of the Sensory Regions of the Vestibular Apparatus. Ergebn. Anat. Entwickl.-Gesch., vol. 42, Heft 1, 1969, pp. 1–113.
6. LORENTE DE NÓ, R.: Études sur l' Anatomie et la Physiologie du Labyrinthe de l'Oreille et du VIIIe Nerf. Deuxième Partie. Quelques Données au Sujet de l'Anatomie des Organes Sensoriels du Labyrinthe. Trav. Lab. Rech. Biol., Univ. Madr., vol. 24, 1926, pp. 53–153.
7. WERNER, C. F.: Die Differenzierung der Maculae im Labyrinth, Insbesonders bei Säugetieren. Z. Anat. Entwickl.-Gesch., vol. 99, 1933, pp. 696–709.
8. LIM, D. J.; AND LANE, W. C.: Cochlear Sensory Epithelium. A Scanning Electron Microscopic Observation. Ann. Otol., vol. 78, 1969, pp. 827–841.
9. ENGSTRÖM, H.: ADES, H. W.; AND BREDBERG, G.: Normal Structure of the Organ of Corti and the Effect of Noise-Induced Cochlear Damage. Sensorineural Hearing Loss, G. E. W. Wolstenholme and J. Knight, eds., A CIBA Foundation Symposium, J. & A. Churchill, London, 1970, pp. 127–156.
10. MAROVITZ, W. F.; THALMANN, R.; AND KAUFMAN ARENBERG, I.: Scanning Electron Microscopy of Freeze-Dried Guinea Pig Organ of Corti. Scanning Electron Microscopy/1970. Proceedings of the Third Annual Scanning Electron Microscope Symposium, O. Johari, ed., IIT Research Institute, Chicago, 1970, pp. 275–280.
11. ENGSTRÖM, H.: The First-Order Vestibular Neuron. Fourth Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–187, 1970, pp. 123–135.
12. LIM, D. J.; AND LANE, W. C.: Vestibular Sensory Epithelia. A Scanning Electron Microscopic Observation. Arch. Otolaryngol., vol. 90, 1969, pp. 283–292.
13. PARDUCZ, B.: Ciliary Movement and Coordination in Ciliates. Intern. Rev. Cytol., vol. 21, 1967, pp. 91–128.
14. MARSZALEK, D. S.; AND SMALL, E. B.: Preparation of Soft Biological Materials for Scanning Electron Mi-

croscopy. Scanning Electron Microscopy/1969. Proceedings of the Second Annual Scanning Electron Microscope Symposium, O. Johari, ed., IIT Research Institute, Chicago, 1969, pp. 231–240.

15. STEINHAUSEN, W.: Über die Funktion der Cupula in den Bogengangsampullen des Labyrinthes. Z. Hals-, Nas.- u. Ohrenheik., vol. 34, 1933, pp. 201–209.
16. TRINCKER, D.: Physiologie des Gleichgewichtsorgans. Hals-Nasen-Ohrenheilkunde, J. Berendes, R. Link, and F. Zöllner, eds., Thieme, Stuttgart, 1965, pp. 311–361.
17. WERNER, C. F.: *Das Labyrinth,* Thieme, Leipzig, 1940.
18. BAST, T. H.; AND ANSON, B. J.: *The Temporal Bone and the Ear*, Charles C. Thomas, 1949.
19. KIMURA, R.; LUNDQUIST, P.-G.; AND WERSÄLL, J.: Secretory Epithelial Linings in the Ampullae of the Guinea Pig Labyrinth. Acta Oto-Laryngol., vol. 57, 1963, pp. 517–530.
20. DOHLMAN, G.; ORMEROD, F. C.; AND MCLAY, K.: The Secretory Epithelium of the Internal Ear. Acta Oto-Laryngol., vol. 50, 1959, pp. 243–249.
21. DOHLMAN, G.: Secretion and Absorption of the Endolymph. Third Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–152, 1968, pp. 101–121.
22. VEENHOF, V. B.: The Development of Statoconia in Mice. N. V. Noord-Hollandsche Uitgevers Mastschappiu, Amsterdam–London, 1969, pp. 1–49.
23. KOLMER, W.: Über das Verhalten der Deckmembranen zum Sinnesepithel der Labyrinthendstellen. Arch. Ohr.-Nas.-u. Kehlk.-Heilk., vol. 116, 1926, pp. 10–26.
24. KOLMER, W.: Gehörogan. *Handbuch der Mikroskopischen Anatomie des Menschen*, vol. III, W. v. Möllendorf, ed., Springer, Berlin, 1927, pp. 250–505.
25. FLOCK, A.; KIMURA, R.; LUNDQUIST, P.-G.; AND WERSÄLL, J.: Morphological Basis of Directional Sensitivity of the Outer Hair Cells of the Organ of Corti. J. Acoust. Soc. Am., vol. 34, 1962, pp. 1351–1355.
26. GÖRNER, P.: Untersuchungen zur Morphologie und Elektrophysiologie des Seitenlinienorgans vom Krallenfrosch (*Xenopus Laevis Daudin*). Z. vergl. Physiol., vol. 47, 1963, pp. 316–338.
27. DIJKGRAAF, S.: The Functioning and the Significance of Lateral-Line Organs. Biol. Rev., vol. 38, 1963, pp. 51–105.

Habituation of Vestibular Responses: An Overview

WILLIAM E. COLLINS
Civil Aeromedical Institute, FAA

SUMMARY

An historical survey of vestibular habituation experiments has been undertaken. Methodological problems are presented briefly, and the influence of arousal on vestibular responses is detailed. Data obtained from animals and from man are treated separately. At least for man, the term "habituation" may be better defined by a dynamic change in the form of vestibular responses than by a simple response reduction.

INTRODUCTION

In vestibular physiology the term "habituation" is frequently used interchangeably with "response reduction" to indicate the effects of repeated stimulation. This phenomenon differs from "fatigue," a temporary state for which brief intervals of rest are usually sufficient for recovery of the response, and from "adaptation," a special type of response decline—one that occurs during a prolonged stimulus. More specifically, "habituation" refers to a relatively long-lasting change; even extended rest periods (48 hours to several weeks) may not be sufficient to restore the response to its original level or form.

The fact that reductions in vestibular responses might occur with repeated stimulation was discussed many years ago. Directional differences in the reactions of experienced dancers were noted by both Ruppert (ref. 1) and Bárány (ref. 2) with reference to nystagmus and by Abels (ref. 3) with reference to dizziness. Responses obtained from such individuals appeared weaker for the practiced direction of turning. Abels (ref. 3) apparently borrowed the term "habituation" from Hitzig to describe the gradually acquired ability to inhibit the symptoms of seasickness. A large volume of literature on habituation has developed since these early reports. Data and views have at one time or another supported the complete spectrum of possible effects of repeated stimulation on vestibular responses (no change, total suppression, response modification).

METHODOLOGICAL PROBLEMS

In spite of considerable literature regarding the influence of repeated stimulation on human vestibular nystagmus, there exists a surprisingly high degree of uncertainty about many of the effects. This is due in part to questions related to arousal factors, in part to the array of nonvestibular stimuli present in various studies, and in part to methods of obtaining and evaluating responses. With regard to methods, nystagmus may be observed directly by the examiner while the subject visually fixates on some object, or, among a variety of other techniques, electronystagmographic tracings or photographic recordings may be obtained (ref. 4).

All of the methods present some problems: Visual fixation obviously interferes with the response (the response is even distorted if the subject wears Frenzel glasses; ref. 5), and quantification, except for duration, has to be limited to a rating at best. Although recording provides the examiner with greater security, testing in total darkness or with eyes closed, in the absence of assigned tasks, probably puts the subject very nearly to sleep. Eye closure presents another problem; it is possible that the eyes may eventually roll up in their sockets (Bell's phenomenon) as the subject relaxes in the test situation, thereby possibly affecting the recording by reducing at least the apparent amplitude of the slow-phase displacement. Further, Fluur and Eriksson (ref. 6) and Fluur and Mendel (ref. 7) have demonstrated that eye closure can produce a

spontaneous vertical nystagmus (most frequently beating upward) or undulating movements of the eyes. Such spontaneous responses do not occur with eyes open in total darkness (ref. 7). Under the open-eyes condition in darkness a large-amplitude nystagmic eye-movement signal is obtained by corneoretinal potential techniques. However, subjects rapidly become unaware of the position of their eyes, and it is possible that they may even close their eyes without realizing it. Further, dark adaptation has been shown to influence the corneoretinal potential (refs. 8 and 9), the method most frequently used to record nystagmus, and the fidelity of tracings obtained by this technique is not always good (ref. 10). All in all, there are problems associated with any of the methods used; therefore, the method must be kept in mind when considering reports of results. Further, in evaluating changes in the nystagmic reaction, the measure used may be of considerable significance. Although duration of nystagmus has been traditionally the most commonly used means of assessing vestibular reactivity, particularly in clinical situations, it is also probably the least indicative of changes. A thorough assessment requires some combination of measures of slow-phase eye displacement, velocity of eye movement, frequency of nystagmic beats, and duration of the response.

THE PROBLEM OF AROUSAL

Animals

Fearing and Mowrer (ref. 11) examined the effects of anesthesia on habituation of rotation-induced head nystagmus in pigeons. They reported no habituation for anesthetized animals and a clear reduction in nystagmus for undrugged birds. From incidental observations made during that study, Mowrer (ref. 12) became probably the first investigator to attempt to manipulate arousal factors and examine their influence on vestibular responses. Working with pigeons, Mowrer (ref. 12) differentiated between "excited" and "unexcited" conditions, depending upon how the birds were prepared for rotation trials, and reported longer durations of nystagmus for the "excited" condition. His interpretation, based also on previous studies, was that excitement influenced the vestibular reaction but that it could not account for all the reduction produced by repeated stimulation. Similarly, Wendt (refs. 13 and 14) noted that immobilized animals might go into sleeplike states ("animal hypnosis") and that various types of stimuli (e.g., auditory or tactual) could restore alertness and reinstate an apparently suppressed nystagmus. Hood and Pfaltz (ref. 15), like Mowrer (ref. 12), reported temporary increases in frequency of nystagmus from rabbits when they appeared excited, and they further noted that injections of adrenalin in rabbits that had shown substantial response declines to repeated rotation produced a partial recovery of nystagmus. An identical result was obtained by Crampton and Schwam (ref. 16), who used intermittent electric shock and auditory signals as excitatory stimuli during repeated rotation of cats.

Drugs have also been used to affect alertness. Cramption (ref. 17) introduced the use of *d*-amphetamine in an effort to maintain arousal levels in the cat; recorded amplitude of nystagmus was increased, but the usual rapid reduction of nystagmus occurred with repeated rotation. Dowd (ref. 18) reported an increase in recorded amplitude, but no change in phase relationships of nystagmus during sinusoidal acceleration of cats treated with *d*-amphetamine; sodium pentobarbital depressed the response. On the other hand, Cenacchi, Fenu, and Gabrielli (ref. 19) reported decreased nystagmus from rabbits with various amounts of *d*-amphetamine.

Wolfe (ref. 20) has noted that the dosage levels of *d*-amphetamine that have been used to maintain animal alertness may have some undesirable side effects; some animals become ataxic and appear to have vestibulo-cerebellar impairment. In addition, the influence of the drug on eye-movement calibrations has been neglected. The importance of the latter was noted by Jongkees and Philipszoon (refs. 21 and 22), who reported enhancement of rabbit nystagmus with hyoscine and also a slight increase in the (corneoretinal potential) eye-calibration signal. Other drugs such as chlorpromazine and cinnarzine, which have a soporific effect, have been shown to reduce rabbit nystagmus (refs. 21 to 23) without altering the eye-calibration signal (refs. 21 and 22).

Man

Although a number of early investigators (e.g.,

refs. 1, 2, and 24) had noted effects on vestibular responses of "nervousness" or "arousal," Wendt (ref. 14) was apparently the first to attempt a definition of "excitement" or "arousal" in terms of conditions basic to the state of the subject, and to relate these to the problem of habituation. He cited two factors in habituation: In one case, repeated rotatory stimulation under conditions that provided opportunities for visual fixation would result in an increasing dominance of the visual stimuli and thereby inhibit nystagmus; in the other case, under conditions of darkness an "inward orientation of attention" would account for nystagmus reduction while an "environment-directed orientation" would restore nystagmus or prevent its decline.

Based primarily on Wendt's notions (ref. 14) regarding mental or attentional states, Collins, Crampton, and Posner (ref. 25) evaluated several instructional procedures aimed at controlling alertness during rotational stimulation of human subjects (tested with eyes open in total darkness) and demonstrated that both quantity and quality of nystagmic output could be manipulated in the same subject by instructions regarding mental activity. If subjects were asked to relax, daydream, and pursue no particular line of thought (reverie), relatively weak nystagmic responses were recorded; vigorous nystagmus was obtained when the same subjects performed continuous mental arithmetic (successive division) or signaled turning sensations in 90° intervals (cf. refs. 26 and 27). Several other studies (refs. 28 to 30) reported that mental tasks or increased alertness would produce a regular nystagmus in place of dysrhythmic responses (see fig. 1), or would prevent apparent adaptation from occurring (refs. 31 and 32).

The results of subsequent research (refs. 33 to 37) pointed to the fact that inward-directed versus outward-directed orientations probably do not account for the influence of the instructions on nystagmic output, since both reverie and mental arithmetic may be classified as inward-directed. (See fig. 2.) What appears to be of importance is that the subject be in a sufficiently aroused or active mental state, whether the arousal arises from attending to his sensations, pursuing a particular line of thought, performing mental calculations, or responding to auditory or other stimuli. It should be noted that not all instructions are effective in this regard. Some tasks, such as counting, may not produce any noticeable effect on nystagmus (ref. 38), while others, such as tasks involving timing behavior, appear to require relatively little attention with repetition (ref. 33).

That assigned tasks or instructions regarding mental activity are important in maintaining vigorous nystagmus or restoring a deteriorated response has been confirmed in a number of studies (e.g., refs. 39 to 45). Instructions have been effective with caloric irrigations (refs. 34, 36, and 46), with drugs (refs. 47 and 48), with alcohol (refs. 49 and 50), with positional alcohol nystagmus (ref. 51), and when used in conjunction with hypnosis (ref. 52). Under some laboratory conditions, mental tasks appeared effective in reducing or preventing motion sickness occasioned by unusual vestibular stimulation (refs. 53 to 55). Further, the absence of alertness or arousal, whatever its neuro-psycho-physiological cause, is probably a major factor in vestibular findings that indicate substantial or total suppression of nystagmic responses in schizophrenic patients (e.g., refs. 56 to 59). Patients who show

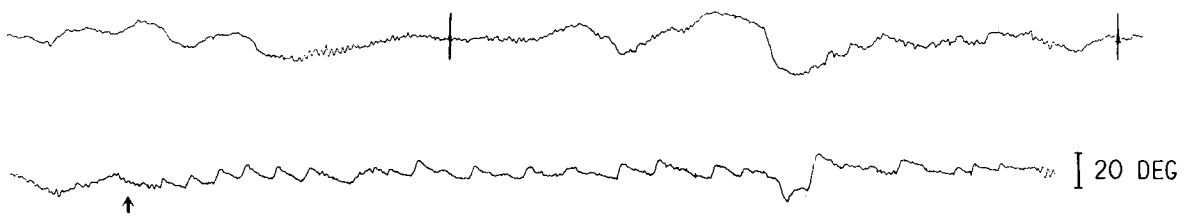


FIGURE 1.—*The effect on vestibular nystagmus of alerting a subject. The vertical bars demarcate the period of angular deceleration, which was 13 s at 41°/s²; the arrow denotes the point at which the subject (eyes open in darkness) was given an alerting task. A 20° eye calibration appears at the end of the tracing. (Reprinted with permission from ref. 35.)*

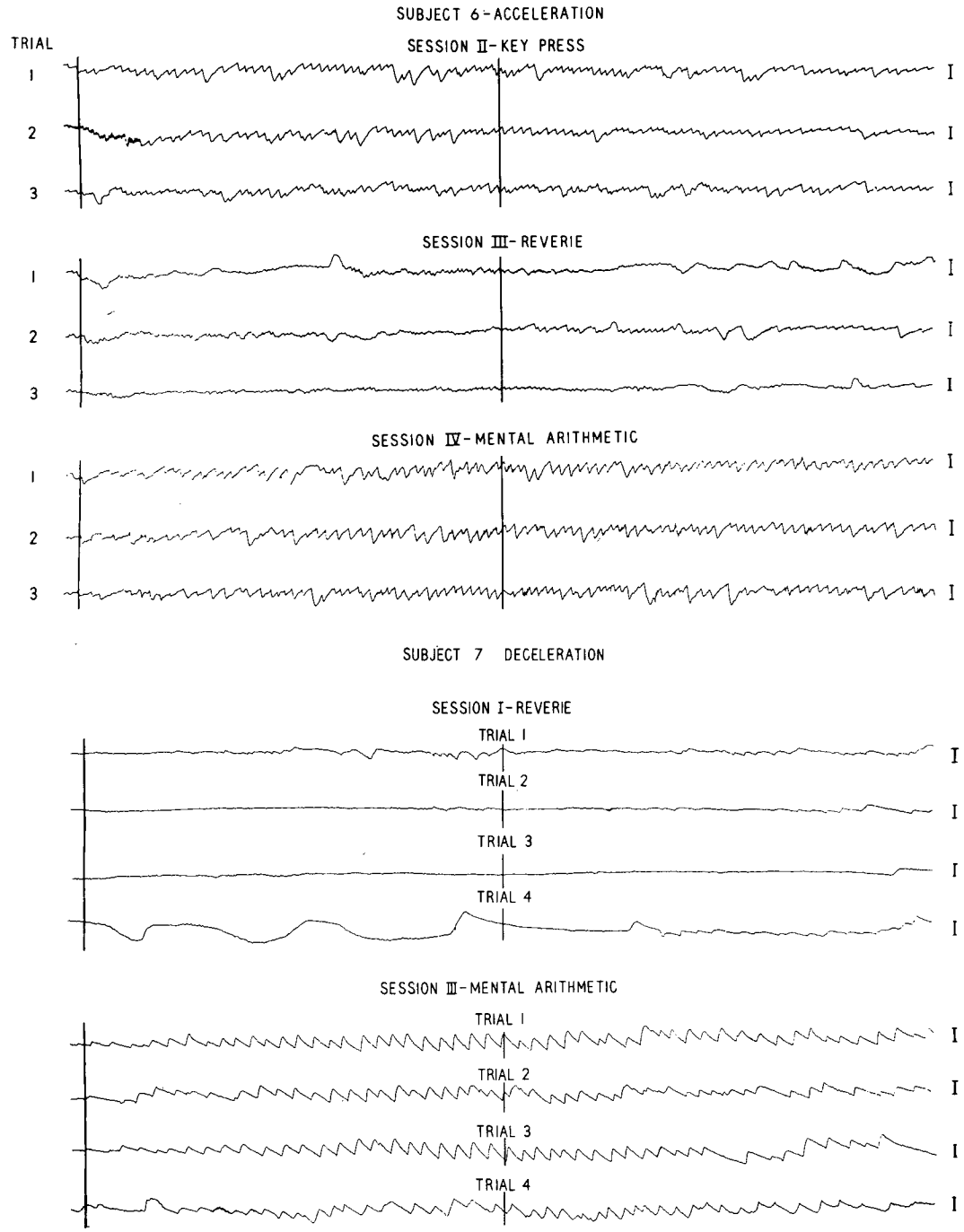


FIGURE 2.—*The influence of instructions on vestibular nystagmus (see text). Vertical bars through the tracings demarcate a 15-s period of angular stimulation at 4.5°/s². Markers at the end of each tracing indicate 20° eye calibrations. Recordings were obtained from subjects with eyes open in total darkness. Sessions were held on separate days; trials were consecutive on a given day. The "reverie" task was given to subject 7 during his first exposure to laboratory vestibular stimulation. (Reprinted with permission from ref. 33.)*

these subnormal vestibular reactions are frequently characterized as apathetic, indifferent, lacking in initiative, and showing a poverty of mental content.

SURVEY OF STUDIES OF ANIMALS

Rotation-Induced Nystagmus

Abels (ref. 3) apparently began experimental evaluation of the habituation of head after-nystagmus in pigeons. Using a cage suspended from cords which were twisted and then allowed to unwind, Abels observed the intensity of the jerks of nystagmus following an abrupt stop of the rotation under conditions either permitting or excluding vision. He noted a marked habituation with repetition under the former condition, and a considerable but lesser response reduction under the latter condition. Abels (ref. 3) reported that the reduction was directionally specific; after repeated rotation in one direction, a reversal of direction would elicit a vigorous response.

The reduction of head after-nystagmus in pigeons tested in the light was confirmed by King (ref. 60) and by Huddleston (ref. 61). Huddleston indicated that the decline was more rapid for squabs than for older pigeons and noted that head after-nystagmus could be completely inhibited by visual stimuli in combination with voluntary movements of the head. Further, although birds rotated in the dark showed a compensatory head movement upon which nystagmus of relatively low amplitude was superimposed, birds rotated in the light showed little compensatory positioning of the head and a relatively higher amplitude of nystagmus.

King (ref. 60) confirmed Abels' finding (ref. 3) of the lesser reduction of nystagmus with vision excluded by testing blind birds, while Fearing (ref. 62), rotating hooded pigeons alternately clockwise and counterclockwise, reported significant reductions in frequency and duration of head after-nystagmus with no apparent effect of one direction of stimulation upon the other (i.e., the responses for each direction appeared to decline independently). In later studies, Fearing (refs. 63 and 64) massed the habituation trials (61 stimulations in a single session) for one group of birds and spaced the habituation trials (10 trials per day for 14 successive days) for other birds; the animals were then subdivided into smaller groups, and each group was retested at a different interval ranging from 2 to 32 weeks. The data led Fearing (ref. 64) to conclude that spacing the habituation trials led to greater retention of habituation than did massing the trials in a single session. Brown (ref. 65) confirmed this effect with ocular nystagmus in cats.

King's study (ref. 60) also introduced a finding that has yet to be experimentally resolved; i.e., in tests following habituation stimuli, the number of nystagmic head movements declined more when the pigeons' heads were fixed (restrained) during the habituation series than when they were free to move in the horizontal plane (vision permitted in all cases). Lumpkin (ref. 66), rotating a single rabbit in the light in an extensive series of tests (142 days), used a head-holder for most of the trials, but tested the animal with its head free on specific occasions. He found considerable differences in the frequency and duration of ocular nystagmus (although both showed habituation) for the two conditions and concluded that head-fixed and head-free habituation were not the same. Earlier, Maxwell, Burke, and Reston (ref. 67) claimed that ocular nystagmus was reduced in the light when rabbits' heads were free more than when they were fixed. However, they simply noted the results of the habituation series for their animals (i.e., there was no pretest-to-posttest comparison of the groups under the same conditions) and, initially, the head-free group had durations which were approximately 30 percent shorter and frequencies of nystagmus which were about 50 percent less than the head-fixed group.

Mowrer's study (ref. 68) of the influence of vision on habituation of head nystagmus in pigeons also contained differences related to head-free stimulus conditions. Mowrer (ref. 68) reported that pigeons tested in darkness showed little or no habituation of the duration of after-nystagmus following a large number of rotations with heads free in illumination, and a clear (directionally specific) decline when the heads were fixed and the room was illuminated during the habituation series. Similarly, when vision was permitted and a striped drum forming a wall around the turntable was rotated with the turntable during the habituation series (thereby preventing optokinetic nystagmus), only a slight bidirectional reduction of head after-nystagmus was obtained during posttests in darkness. (Use of the striped drum also produced very little after-nystagmus dur-

ing light trials, and repeated stimulation resulted in abolition of the response.) However, when the habituation series was in darkness, bidirectional nystagmic reductions were obtained for both the head-fixed and head-free conditions, although, contrary to results reported by King (ref. 60) for tests in which vision was always permitted, response attenuation was greater for the head-free condition. The complete lack of a decline of nystagmus in the dark following repeated head-free stimulation in the light and a directionally specific decline following head-fixed stimulation in the light are difficult to explain, since Mowrer (ref. 68) employed approximately equal rates of acceleration and deceleration during the habituation trials. However, the experiment was a complex one involving head-fixed and head-free conditions; ocular nystagmus with and without head nystagmus; optokinetic nystagmus, enhancing vestibular nystagmus in one direction and opposing it in the other; compensatory head positioning allowed to occur with free heads and prevented with fixed heads; and the possibility that head position varied among the conditions. Mowrer (ref. 69) later examined the effects of interacting vestibular and optokinetic stimulation with pigeons. He indicated that, when vision was permitted during and after rotation, after-nystagmus was inhibited by (1) the tendency to fixate visually on some object, (2) the tendency for an optokinetic response to persist after cessation of rotation, and (3) the tendency for optokinetic head movements during rotation to stimulate the semicircular canals in a way which counteracted the vestibular stimulus occasioned by a deceleration.

King (ref. 60) had also reported that decerebrate pigeons with heads fixed in the light showed less habituation than did normal birds. Halstead (ref. 70) and Halstead, Yacorzynski, and Fearing (ref. 71), using hooded pigeons, found a significantly greater decline in duration of head after-nystagmus in normal birds than in those with cerebellar lesions. Another group of normal birds received a similar series of rotations and demonstrated the same response reduction; half of the birds were then subjected to cerebellar operations. Two weeks later birds with cerebellar lesions showed significant recovery of nystagmus; those that had not been operated on showed no change from the habituated level. Similar findings were obtained from yet another group with a 4-week interval. In both control and decerebrate birds, some habituation was still present after a 3-month interval. Moreover, substantial (control birds) to complete (birds with lesions) "transfer of habituation" was obtained to rotation in the opposite direction. The latter result confirmed the findings of Mowrer (ref. 68), although "transfer" may be an inappropriate term, since the acceleration stimulus used during habituation trials almost surely was of high magnitude and brief duration.

The question of transfer of habituation to the opposite direction of turning as cited by Halstead (ref. 70) and by Abels (ref. 3) involves some confusion regarding stimulation (cf. figs. 3 and 4). Early investigators sometimes overlooked the fact that a rotating subject brought to an abrupt stop actually received vestibular stimulation in both directions (in one direction during acceleration, and in the opposite direction during deceleration). In Abels' situation (ref. 3), the acceleration rate was probably of lower magnitude than that of the deceleration (brake stops); with Halstead (ref. 70), the rates were probably approximately equal. In examining only deceleration responses, and in reversing the direction of turning after a habituation series, one would expect an equally reduced response if the rates of acceleration and deceleration during habituation had been approximately equal (see fig. 4) or, if the rate of the latter were higher, the "unhabituated" direction might be expected to yield a relatively stronger response than that finally evidenced in the "habituated" direction. Data from the two studies support these expectations, and the expectations themselves are based on studies by Griffith (ref. 72) with white rats and by Maxwell et al. (ref. 67) with rabbits, which showed a considerable reduction in frequency and duration of ocular nystagmus following repeated abrupt stops of rotation. When the stopping was then attempted from a faster rate of motion, a more vigorous and longer-lasting nystagmus was elicited. (Even this response, however, was less than that obtained from an identical stimulus prior to the habituation trials.)

Most of the investigations cited above have examined habituation of head after-nystagmus. Before beginning a more thorough look at the habituation of ocular nystagmus, one further study might best be considered here. Fukuda, Hinoki, and Tokita

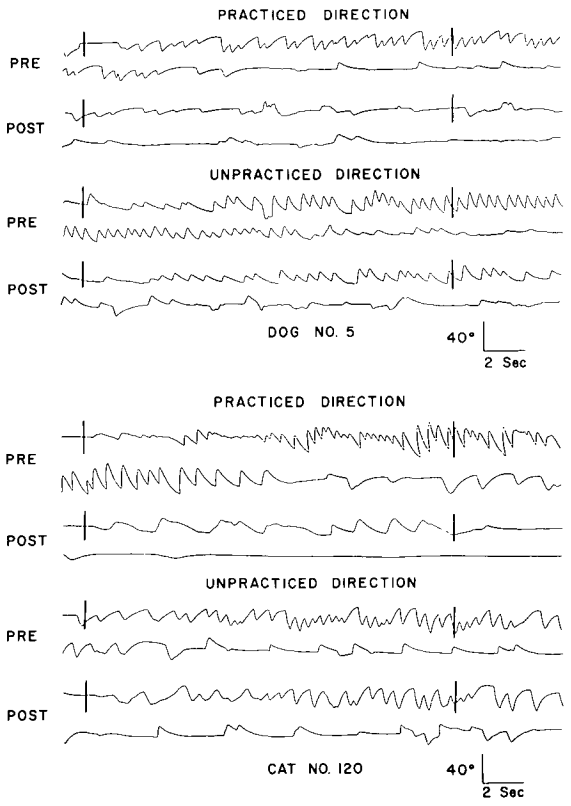


FIGURE 3.—*Directional specificity of habituation of ocular nystagmus in a cat and a dog. A series of 15 clockwise angular accelerations (decelerations were subthreshold) was administered between the pretests and posttests. A directionally specific reduction is evident. The animals were rotated under head and body restraint in total darkness (cf. ref. 84). Vertical bars demarcate the stimulus period; optokinetically derived calibration markers indicate 40° eye excursions. Similar effects are obtained from other animals. (Reprinted with permission from ref. 90.)*

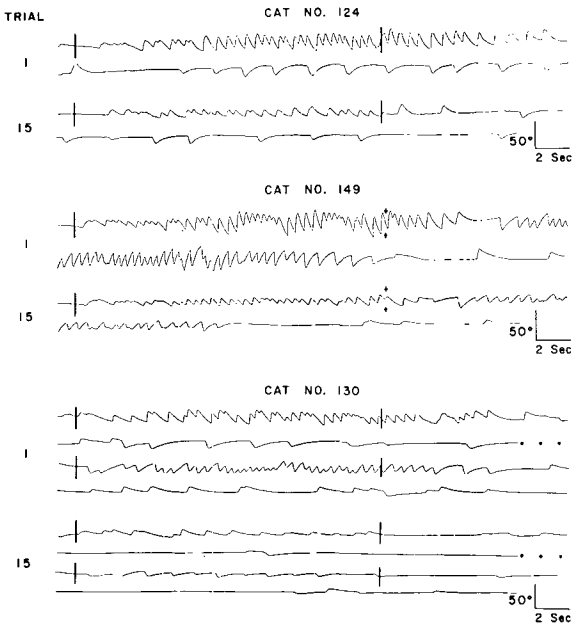


FIGURE 4.—*Equal rates of repeated accelerations and decelerations produce equal reductions in both directions of nystagmus (cat no. 130). Responses need not be permitted to run their course in order for the reductions to occur; cat no. 149 received 15 accelerations which were followed within 0.6 s by decelerations (arrows). Cat no. 124 shows the usual reduction in all phases of the nystagmus response as a result of 15 unidirectional stimulations; strong secondary nystagmus is also evident during the first trial. Vertical bars demarcate the stimulus periods; dots in the lower tracings indicate approximately 80 s of recording not shown (ref. 96).*

(ref. 73) rotated unrestrained, blindfolded leghorns on perches. During accelerations, these birds showed the usual head deviation opposite to the direction of turn, followed by an appropriate head nystagmus. Upon stopping, the head deviation and nystagmus were in the opposite direction. In tests following repeated exposure to rotation in both directions, the birds still showed a head deviation opposite to the direction of acceleration, but now nystagmus was very brief; moreover, this attenuated nystagmus was followed by a deviation of the head in the direction of the turn. The head remained in this position until rotation ended; the head then deviated even further in the direction of the previous turning, a brief nystagmus occurred, and the head returned to the midline. These opposite reactions, conditioned by practice, were accompanied by improved equilibrium during rotation.

As noted earlier, Maxwell et al. (ref. 67) reported reductions of ocular nystagmus from rabbits as a result of repeated rotation with vision permitted. Maxwell (ref. 74), Pilz (refs. 75 and 76), Lumpkin (ref. 66), and Henderson (ref. 77) confirmed this finding. Maxwell and Pilz (ref. 78) compared nystagmic reductions for groups of rabbits (with their heads apparently fixed) rotated in the light and in total darkness. A greater percentage of decline in number of eye movements was obtained for the latter group. However, these findings simply reflected the results of the habituation series, and

optokinetic stimuli were present throughout the light trials. Henderson (ref. 77) noted that rabbits which no longer showed after-nystagmus following repeated stimulation in the light would evidence a vigorous response if they were then blindfolded. Some further tests led him to agree with the conclusions of Mowrer (ref. 69) regarding the influence of optokinetic stimuli.

Maxwell et al. (ref. 67) also reported that, following habituation to rotation, rabbits gave "normal" responses to caloric irrigations. Hood and Pfaltz (ref. 15) made a similar observation with rabbits tested in the dark, and Collins (ref. 79) obtained the same results from cats. However, the animals' heads were fixed at different angles for rotation as opposed to caloric trials in the latter two investigations, and head position was not specified in the Maxwell et al. study (ref. 67). It has been suggested (refs. 80 to 82) that differences in the orientation of specific and nonspecific gravireceptors for the two types of test situations might account for the failure to obtain transfer of habituation from rotational to caloric stimulation.

Hood and Pfaltz (ref. 15) also reported that the response decline obtained by repeated rotation did not show recovery upon retest 2 weeks later. Other tests showed that the interval between stimulations did not affect the course of habituation; i.e., that the number of stimulations performed was critical, regardless of the interval between tests.

More recent investigations have dealt primarily with ocular nystagmus in the cat and have probably effected better overall control of stimulus conditions. Although Prince (ref. 83) reported no change in the duration of after-nystagmus in cats subjected to over 1000 rotation tests, all more recent investigations show clear response reductions following repeated rotatory or caloric stimulation. In all of the studies cited below, the heads of the cats were fixed by special restraining techniques (almost invariably that of Henriksson, Fernández, and Kohut, ref. 84), and electronystagmographic methods were used to record eye movements. Unless otherwise indicated, rotatory stimuli comprised accelerations and decelerations separated by 1 to 3 minutes of constant velocity. Unidirectional rotatory stimulation involved deceleration at stimulus rates below the threshold for eliciting nystagmus.

Crampton and Schwam (ref. 16) and Crampton (ref. 17) reported a marked reduction of ocular nystagmus in cats during the course of a 12-trial series of angular accelerations in darkness. Auditory stimuli or electric shock interspersed throughout a trial produced only partial recovery of habituated nystagmus. The response decline occurred both within a 12-trial series and over the course of similar series administered once a week for 6 weeks. Fairly strong doses of *d*-amphetamine injected into previously untested cats appeared to produce a higher-than-normal amplitude of nystagmus throughout habituation trials, but the response showed the same form of reduction as that of undrugged cats subjected to the same tests (ref. 17). The drug does not appear to affect the time course of nystagmus (ref. 85).

Both Crampton (ref. 17) and Collins (ref. 79) have defined the reduction of nystagmus in the cat as an overall suppression. The greatest decline occurs within the first five stimulations; peak slow-phase eye displacement is sharply reduced, duration of the response becomes attenuated, frequency declines, and secondary nystagmus (a response which follows and is opposite in direction to the primary response; cf. ref. 86) begins earlier and is of lower output with stimulus repetition. The same effects occur with prolonged angular accelerations (36 seconds), and a progressively earlier peaking of the response with a declining output during angular acceleration becomes clearly evident with repeated tests (refs. 85 and 87).

Crampton (ref. 88) also subjected groups of cats treated with *d*-amphetamine to a unidirectional series of six habituation trials in total darkness. An evaluation of pretests and posttests which comprised stimulation in both directions led Crampton to conclude that habituation was directionally specific. Brown and Marshall (ref. 89) confirmed these results with *d*-amphetamine but reported complete recovery of nystagmus after 1 week. They related the latter point to Fearing's work (refs. 63 and 64) on "massed" and "spaced" practice with pigeons and to a study by Brown (ref. 65) in which separate series of unidirectional angular accelerations in darkness were administered to groups of cats. The series for each group were present at different intervals (1 to 14 days). An additional series was given to all cats 2 weeks later, and a final series was presented 1 month after that. The habituation

occurred equally for all groups in spite of the different times between series, but recovery (partial) after rest intervals was clearly affected. Groups with the shortest intervals between series showed the most recovery; those with the longest intervals between series showed no recovery.

Response reductions were also obtained by Collins and Updegraff (ref. 90) from both cats and dogs subjected to unidirectional habituation trials in total darkness. Duration, frequency, and slow-phase measures of nystagmus all showed declines, with duration yielding the least reduction and slow-phase eye movement the most (see fig. 3). Responses to stimulation in the opposite direction were more vigorous, but such that the authors concluded that habituation might be only relatively specific to direction. Following 1 week of rest, only partial response recovery was obtained (ref. 90). Similarly, Winget and Smith (ref. 91) reported a reduction in the duration of ocular after-nystagmus from restrained chickens (eyes closed) repeatedly decelerated from 32 rpm. Winget, Smith, and Kelly (ref 92), however, indicated that such study declines did not occur if animals were exposed to long periods of centrifugation (1.5 to 2.0 g) between habituation stimuli. These authors suggested that centrifugation might alter the functional characteristics of the labyrinth by inducing structural or chemical changes.

Crampton (ref. 93) examined the influence on habituation of optokinetic stimuli (room lights on during accelerations and for 30 seconds thereafter) during unidirectional rotatory stimulation of cats. Whether the optokinetic response enhanced (one group) or opposed (another group) the vestibular nystagmus, the same amount of habituation was evident in pretests and posttests conducted in total darkness as occurred with a group of cats given the same habituation series (eight rotations) in total darkness.

Later, Crampton and Brown (ref. 94) reported that repeated unidirectional rotatory stimulation of the vertical semicircular canals (in darkness) had no effect on responses of the horizontal canals; i.e., there was no transfer of effects from vertical-canal stimulation to the horizontal canals. Although these authors apparently did not record the vertical nystagmus, Collins (ref. 81) demonstrated habituation of all aspects of that response in a series of unidirectional habituation trials in darkness with the animals restrained and placed on their sides. Pretests and posttests showed that the habituation was relatively specific to the direction of nystagmus elicited during practice and, moreover, was relatively specific to the position of the otoliths and other gravireceptors (see fig. 5); the latter pretest-to-posttest comparison was accomplished by placing the animals on the side opposite that maintained during habituation (i.e., changing their positions by 180°), thereby stimulating the same set of canals, but with the otoliths and other gravireceptors in a new position. A similar study by Collins and Updegraff (ref. 95) demonstrated in parrots the same possibility of the specificity of habituation of vertical-canal responses to the position of gravireceptors.

Collins (ref. 96) noted no significant effect on habituation of nystagmus in darkness when physically equivalent accelerations and decelerations followed each other within less than a second, i.e., the deceleration stimulus was applied during the peak of the nystagmic response to acceleration, thereby interrupting that response; nystagmus occasioned by deceleration was permitted to run its course. Habituation was obtained for both directions of response (evaluated in separate unidirectional pretests and posttests) and was of the same magnitude as that obtained both from a group of cats exposed to 2-minute periods of constant velocity between accelerations and decelerations (both directions of response permitted to run their course) in a similar habituation series, and from another group that received unidirectional habituation (subthreshold decelerations). The latter group also confirmed the relative directional specificity of habituation (see fig. 4).

Caloric-Induced Nystagmus

With the exception of Dunlap's (ref. 97) use of unilateral ice-water irrigations with two rabbits, caloric habituation studies of animals have been largely restricted to cats. Dunlap (ref. 97) calorized a single ear of each rabbit in the light three times daily every other day for a total of 39 to 48 trials. No ocular nystagmus was elicited following the 24th trial in one animal and following the 33rd trial in the other. Retests a month later showed recovery; nystagmus was obtained during the first 5 to 6 days of this new series, which was continued

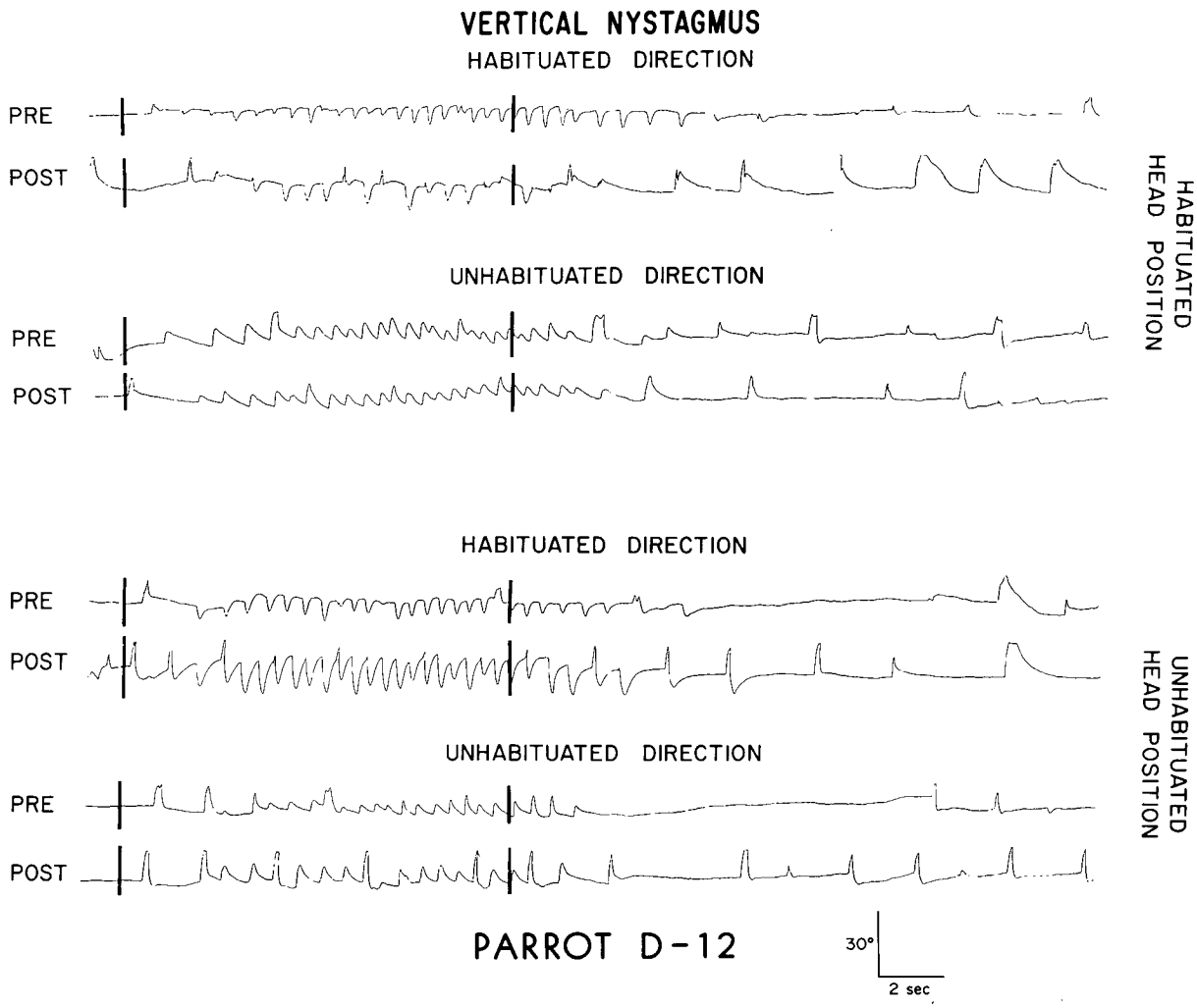


FIGURE 5.—*Specific and nonspecific gravireceptors interact with the semicircular canals in the reduction of vertical nystagmus by repeated rotation of birds (ref. 95) and cats (ref. 81). The ocular tracings above were obtained from a parrot that was exposed to 15 unidirectional accelerations with its head and body fixed in a beak-down position. Pretests and posttests involved both directions of rotation with the beak down (habituated head position) and both directions of rotation with the beak up (unhabituated head position). Nystagmus reduction was relatively specific to both the head position used and the direction of nystagmus elicited during the habituation series. Vertical bars demarcate the acceleration period; a calibration marker appears at the bottom right of the chart. (Reprinted with permission from ref. 95.)*

for 1 to 2 months. The opposite ear was then irrigated and yielded some response, but the reaction was clearly weak; several days of irrigation eventually abolished this nystagmus. The rabbits next were rotated and showed no nystagmic response to stimulation of the horizontal or of the vertical (animals placed on their sides) semicircular canals. When retested 6 months later, the rabbits showed no caloric nystagmus, but both gave vigorous responses to rotation. The heads of the animals may have been in the same plane for both caloric and rotatory stimulation.

Henriksson, Fernández, and Kohut (ref. 84) calorized cats in the light and reported a reduction of nystagmus (particularly maximum eye speed) following several stimulations. In another study

which permitted vision (ref. 98), 10 irrigations, alternately warm to one ear and cool to the other (each driving nystagmus in the same direction), produced more habituation and at a faster rate than that obtained by 10 irrigations of one ear with a single temperature of stimulus; a following irrigation drove nystagmus in the opposite direction and produced vigorous nystagmus; i.e., there was no transfer of habituation to the unpracticed direction of response. In another series of tests, cats were given 10 irrigations of one ear alternately with cool and warm water; another group received a single temperature of stimulus alternately to each ear. Both procedures reduced maximum eye velocity, but the second procedure seemed more effective, in agreement with the related finding noted above. Further tests showed that the same pattern of habituation was obtained whether 10 irrigations were separated by 5-minute intervals or by 24-hour intervals, and that response declines persisted for 2 to 3 weeks.

Transfer of unidirectional habituation was extensively examined. Different groups were given 10 calorizations of a single ear with cool or with warm water, and the same ear was then irrigated with the opposite stimulus temperature (driving nystagmus in the unpracticed direction); normal responses were obtained. Other animals received 10 cool stimuli in the right ear and then, in separate tests, warm water to the same ear and both warm and cool water to the left ear. Nonequivalent stimuli (i.e., stimuli that would drive nystagmus in the unpracticed direction) produced vigorous responses, regardless of the ear tested; equivalent stimuli produced a reduced response. When animals were given 10 irrigations alternating warm and cool water to one ear or using cool water alternately presented to each ear (in both cases nystagmus was driven alternately to the left and the right), a bidirectional decline was obtained, and any following irrigation stimulus (cool or warm applied to either ear) revealed complete transfer of habituation. Fernández and Schmid (refs. 99 and 100) noted that the reduction of nystagmus was most apparent in the frequency of eye movements and in velocity of the slow and fast components, while duration and amplitude of nystagmus might not be affected. Habituation was also obtained from cats with total ablation of the neocortex, with unilateral or bilateral ablation of the temporal lobes, and with hemidecortication.

Proctor and Fernández (ref. 101) examined blindfolded cats with unilateral irrigations and obtained a clear response decline in frequency and amplitude of nystagmus (the latter was not obtained in the studies that permitted vision), and in the velocity of both the slow and fast components. After habituation, blindfolds were removed and an additional test was conducted. Neither direction, nor frequency, nor total number of eye movements was affected (i.e., they were still at the habituated level), but both amplitude and velocity of nystagmus were increased. Pretests and posttests indicated transfer of habituation for an equivalent stimulus presented to the nonhabituated ear; nonequivalent stimuli presented to either ear showed some response reduction (partial transfer). Tests conducted 2 to 12 days later showed only partial response recovery.

Collins (ref. 80) confirmed the response reduction with unilateral caloric irrigations in the dark and noted that the greatest decline occurred within the first five trials. The relative directional specificity of reduction was also confirmed by use of equivalent and nonequivalent unilateral stimuli following habituation. Further, unidirectional rotation tests in total darkness, conducted before and after the habituation series, showed only a small change in nystagmic output. Thus, contrary to Dunlap's findings with rabbits (ref. 97), the response decline following caloric stimulation did not show significant transfer to rotational stimuli. However, the animals' heads were positioned differently for the two stimulus conditions in Collins' study (ref. 80).

In other transfer experiments, Capps and Collins (ref. 102) and Mertens and Collins (ref. 82) employed both bilateral (simultaneous stimulation of one ear with warm water and the other with cool water) and unilateral irrigations in total darkness. A 17-trial series (15 trials plus pretest and posttest) produced significant decrements in slow-phase eye movement and frequency of nystagmic beats (duration was not markedly affected) for either unilateral or bilateral stimulation (see fig. 6). Retention tests were conducted 1 to 4 weeks later and showed partial recovery, which appeared somewhat greater after 4 weeks than after 2 weeks; however, nystagmus appeared to decline more rapidly with several repetitions of the retention-test irrigations. In

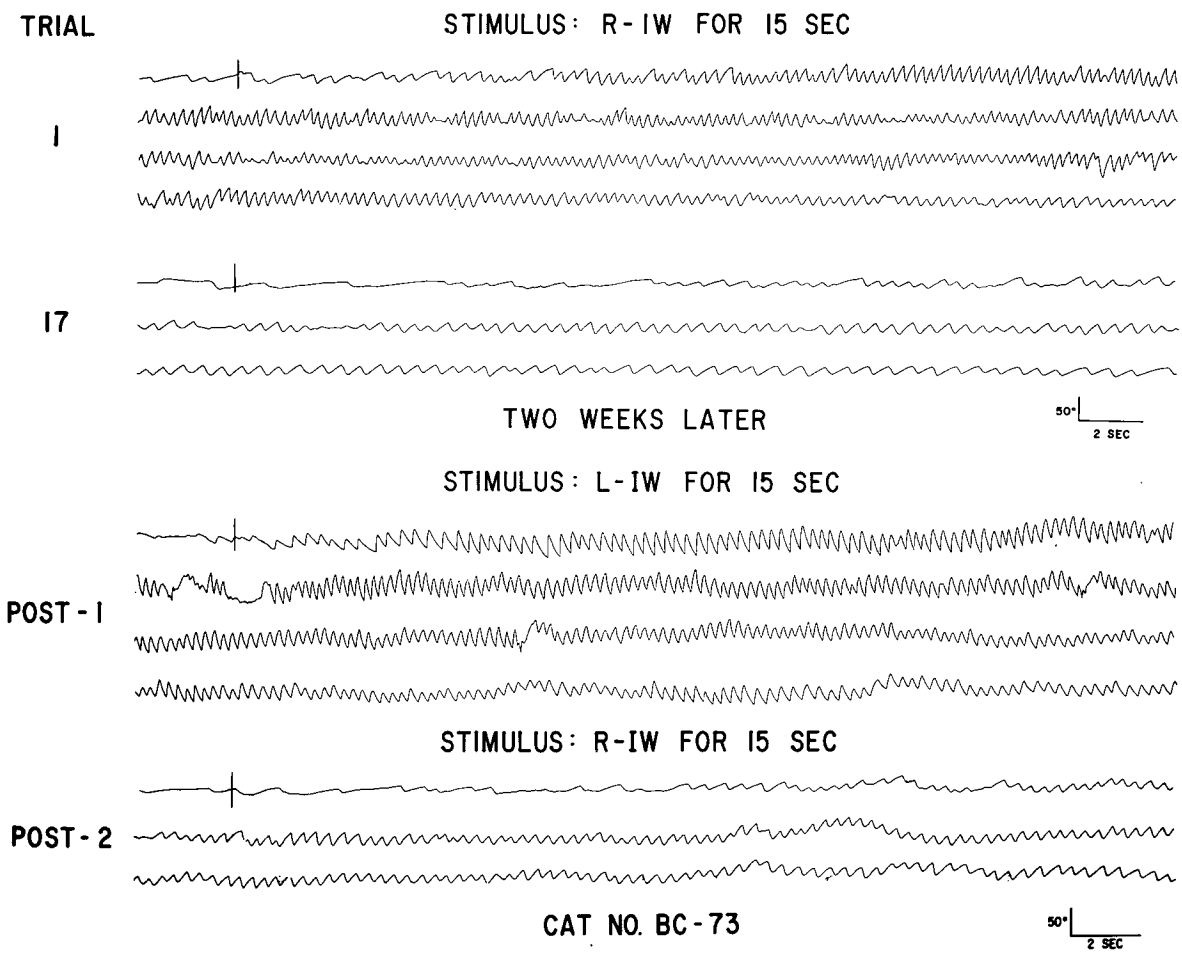


FIGURE 6.—*Caloric nystagmus during the first and last trials of a 17-trial series of unilateral ice-water (IW) irrigations of the right ear. The response is clearly reduced following the caloric habituation series. After 2 weeks, there is some recovery of the responses from the right ear, but the directional specificity of the reduction is still evident when comparisons are made with responses from the left ear. Vertical bars indicate the end of the irrigations; optokinetically obtained calibration markers appear at the right, below each pair of tracings. (Reprinted with permission from ref. 102.)*

examining transfer, Capps and Collins (ref. 102) gave three groups of cats a pretest and a posttest comprising a mild unilateral stimulus (26° C to the right ear); one group received a habituation series of 17 such irrigations, and the other two groups received a bilateral series (26° C to the right and 50° C to the left ear) of 15 habituation trials for a duration of 25 s in one case and 30 s in the other. (Increasing the duration of a bilateral stimulus up to at least 30 s produces a stronger nystagmus; see also ref. 103.) Responses to the posttest unilateral stimulus were reduced to an approximately equivalent level for all three groups. Thus, habituation to bilateral calorizations transferred to a unilateral stimulus of less intensity. Similarly, three new groups received a strong unilateral stimulus (ice water) during pretests and posttests; one group was exposed to unilateral ice-water habituation; the other two were habituated with bilateral irrigations of 15- and 20-s duration, respectively. Although bilateral stimuli produced weaker responses initially than did the unilateral

irrigation, responses to the posttest unilateral stimulus were reduced approximately equally for all groups. Thus, habituation to bilateral calorizations transferred completely to a unilateral stimulus of greater intensity (ref. 102). Further, no effect of the habituation series was evident for optokinetic responses (rotation of a striped drum).

Later, three different intensities of unilateral stimulation were applied to three groups of cats in a 15-trial habituation series (ref. 82). Pretests and posttests indicated that all of the unilateral habituation stimuli produced as great a response reduction to bilateral caloric stimuli as did a series of 15 bilateral irrigations. Thus, unilateral caloric habituation transferred to bilateral caloric stimulation. Additional pretests and posttests involved rotation. Bilateral caloric habituation appeared to have relatively little effect on responses to rotation. Similarly, a group of cats exposed to a series of unidirectional rotations were given pretests and posttests with bilateral calorizations (all driving nystagmus in the same direction); habituation was obtained to the rotatory stimulus, but no significant effects on caloric nystagmus were obtained (ref. 82). These results confirmed previous findings by Collins (refs. 79 and 80) that showed relatively little intermodal transfer of habituation (head position was different for caloric and rotational trials in these studies), but did not agree with Dunlap's results (ref. 97).

A study by Collins (ref. 104) examined the effects of "double irrigations" (simultaneous binaural irrigation with water of equal temperatures) on habituation in the dark. The double irrigation drives the cupulae in opposite directions, thereby cancelling horizontal nystagmus (weak vertical nystagmus may sometimes be produced; cf. ref. 105). Pretests and posttests with unilateral irrigations of the same stimulus temperature indicated that the series of double irrigations produced no nystagmic habituation (see fig. 7).

SURVEY OF STUDIES OF MAN

Rotation-Induced Nystagmus

As noted earlier, Ruppert (ref. 1) and Bárány (ref. 2) reported a unidirectional reduction of nystagmus from dancers who habitually whirled in one direction. In more recent times, Fukuda, Tokita, Hinoki, and Kitahara (ref. 106) have cited shorter-than-normal durations of nystagmus not only for dancers but for athletes as well. Schoolboys involved in a program of vestibular self-stimulation in play activities are reported to show progressively attenuated nystagmus which is interpreted as an improvement of the labyrinthine function by training; athletic ability is concomitantly enhanced (ref. 106).

In laboratory studies, Griffith (ref. 24) rotated subjects (including himself) in the light and reported attenuation of the duration of nystagmus and of the number of eye movements both within a daily series of tests and from day to day; only minor recovery was evident after 2 months of rest. He also noted that, when a rotating subject was stopped and then moved back one quarter turn, a markedly reduced nystagmus duration was obtained. (This probably has implications for "head-free" rotation studies of animals.) Four years later, with no intervening tests, Griffith (ref. 107) conducted another series of rotations on himself in the light and reported some recovery of nystagmus duration and number of eye movements (the response had been abolished after 135 trials in the earlier study), but the nystagmus was not so vigorous as it had been initially, and now only 35 trials were required to abolish the response.

Six subjects in another study (ref. 108) were given a daily series of rotations. After-nystagmus was timed in the light following one revolution of the turning device; when subjects no longer showed any nystagmus, the number of revolutions was increased to two, then to three, and so on. Progressively more trials were required to inhibit after-nystagmus completely as the number of revolutions increased. None of the subjects experienced vertigo at any time, and at the end of the study none showed past-pointing. Griffith (ref. 109) also reported that visual far-fixation after a brake deceleration produced twice the duration and frequency of nystagmus as did near-fixation. Subjects with their gaze directed laterally (in the direction of the fast phase) showed an enhanced duration, but, this too became attenuated with repeated practice. Preventing visual fixation (e.g., with 20-diopter lenses) doubled the duration of after-nystagmus. However, even in the absence of visual fixation, duration of nystagmus declined with repeated elicitation.

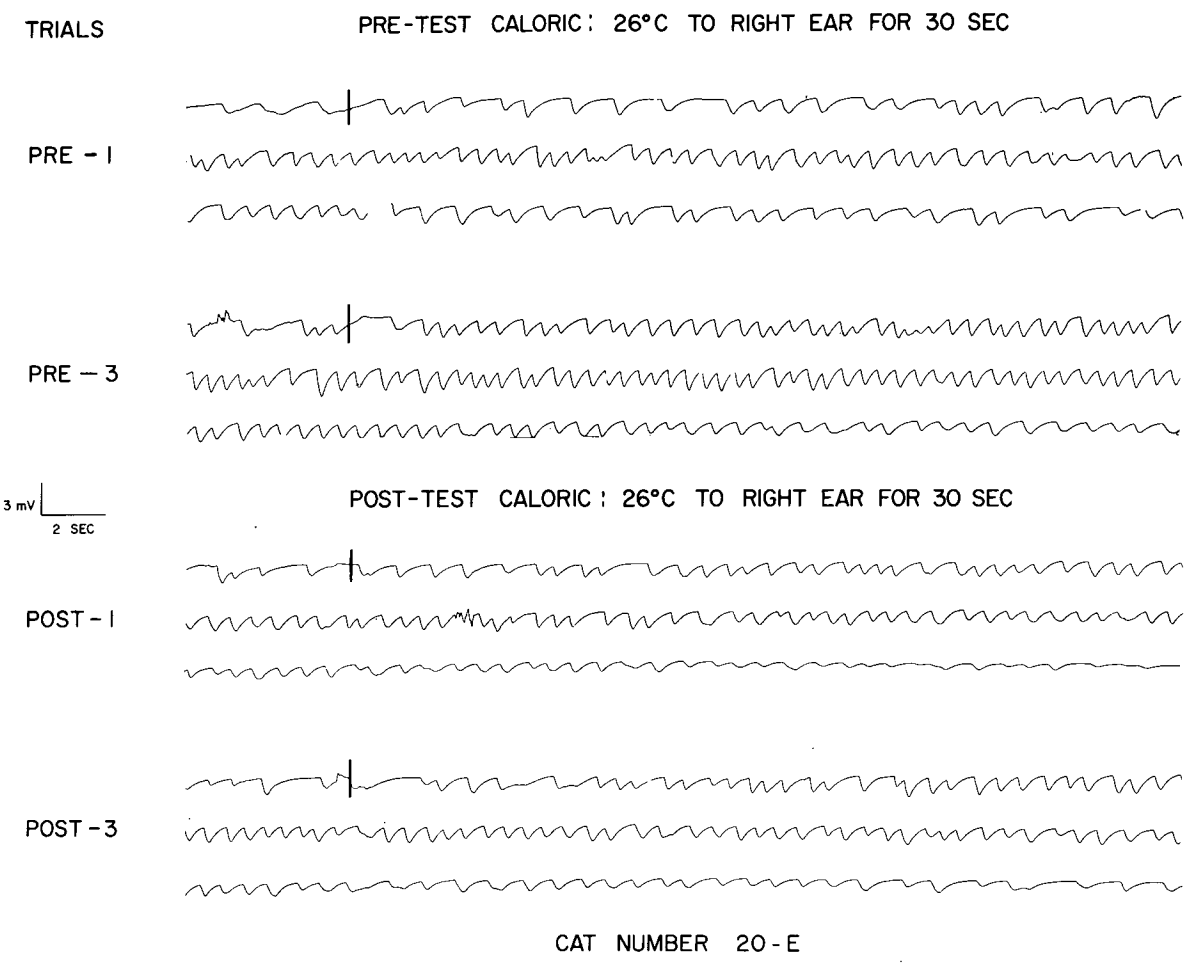


FIGURE 7.—*Repeated peripheral stimulation does not necessarily cause a reduction of vestibular nystagmus. The tracings depicted above are from a cat that received 15 double irrigations (administration of 26° C water to both ears simultaneously) between the unilateral pretests and posttests. Vertical bars denote the end of the irrigation period; all trials were in total darkness. (Reprinted with permission from ref. 104.)*

Holsopple (ref. 110) reported attenuation of after-nystagmus with repeated practice in the light for several rates of deceleration. Subjects were then rotated while wearing 20-diopter lenses and, although they showed an increased nystagmus time, durations were still below initial reactions. One subject was then blindfolded and the duration of his sensation timed; it showed little difference from prehabituation levels. Holsopple (ref. 110) also reported that rotation in the opposite direction yielded vigorous after-nystagmus. He attributed this to the fact that acceleration and deceleration stimuli were not separated by a sufficient period of time. He restated this view in another paper (ref. 111) and particularly emphasized the importance of nystagmus running its course (i.e., that the response must not be interrupted by an opposing stimulus) for habituation to occur. More recently, Brown (ref. 112) obtained significant response reductions for both directions of nystagmus during a series in which accelerations were immediately followed by decelerations in total darkness. Collins (ref. 96) reported a similar finding with cats; however, the form of the habituated response curve appears different in comparing cat and man. Holsopple (ref. 113) also noted that nystagmus time may be seri-

ously affected by position of the head and that "practically every subject exhibits some tendency toward head movement during rotation."

Dorcus (ref. 114) rotated a group of subjects with heads fixed and another with heads free. Duration of after-nystagmus was observed during 50 rotations over a 10-day period. For both groups, duration declined with repetition; responses in the opposite direction, however, were not affected. The head-fixed group clearly showed a greater reduction than did the head-free group. Additional trials were then conducted by fixing the heads of those previously rotated with heads free and freeing the heads of those previously rotated under the head-fixed condition. The response level that had been reached at the end of the habituation series was not changed by changing the head condition. Dorcus (ref. 114) concluded that the two types of habituation were different.

Mowrer (ref. 115) braked subjects to a stop from 12 rpm and observed the duration of after-nystagmus. Twelve trials, in a room with striped walls, alternately required subjects (1) to keep their eyes always open and (2) to have eyes closed during rotation but open when the chair stopped. The latter condition produced greater after-nystagmus than the former, but no decrease with repetition, while the former condition produced some reduction of response duration. Mowrer (ref. 115) then gave another series of 12 rotations to two groups of subjects. Trials were alternately conducted with (1) eyes closed during rotation but open when the chair stopped, and (2) eyes open with the subject fixating on the tip of his finger (one group) or eyes open but a drum over the subject's head (the other group). Both of the latter conditions prevented optokinetic nystagmus and permitted visual still-fixation. There was no difference among the conditions with respect to duration of after-nystagmus. Mowrer (ref. 115) concluded that the reduction of after-nystagmus was due to the post-stimulus persistence tendency of the optokinetic response (i.e., optokinetic after-nystagmus).

Dodge (ref. 116) conducted the first extensive habituation study in which visual stimulation was excluded by subjecting himself to over 600 rotation trials during a 6-day period. Using sudden starts and stops separated by approximately 15 seconds of rotation at 20 rpm, he recorded nystagmus with eyes closed and reported abolition of the response to deceleration. By changing the direction of rotation, he found a 50-percent reduction of nystagmus from prehabituation levels. However, there were complicating factors present; decelerations were introduced while the response to acceleration was still in progress, and alertness was probably uncontrolled. With respect to the latter, Dodge (ref. 116) noted that "the rotation experiment had a soothing soporific character . . . (that) did not carry over into reversed rotation or accidental interference with the customary speed."

Cupulometric data from 320 normal subjects tested with Frenzel-type glasses were obtained by Aschan, Nylen, Stahle, and Wersäll (ref. 117). Duration of nystagmus was directly observed. Using four to six stimulus rates, the authors noted a directional preponderance that depended upon whether subjects were first rotated to the right or to the left for each rate. The durations of nystagmic responses to the first direction of rotation were significantly longer than those obtained to the second direction of rotation for all but the highest stimulus value (52°/s). No such effect was noted for the duration of the after-sensation.

Suzuki and Totsuka (ref. 118) used standard cupulometric procedures and recorded nystagmus from behind closed lids. Subjects were given a series of 10 or more abrupt stops from constant velocities of 5 or 10 rpm. No decline in response duration was reported for the higher stimulus rate, while the lower rate showed considerable trial-to-trial variation; in addition, irregular nystagmus was frequently associated with the shorter durations (probably due to arousal factors). However, Fluur and Mendel (ref. 119) reported a reduction in the duration of recorded nystagmus (closed eyes) after 10 to 12 brake decelerations from 15 rpm. No technique for controlling alertness of the subjects was noted. Additional subjects were braked to a stop from 5 rpm, but no indication of habituation was evident after 10 trails. Unilateral caloric stimuli administered before and after the 15-rpm rotation series showed reduced durations for the direction of nystagmus elicted during the habituation series, and a longer nystagmus time for the opposite direction in most subjects. Several subjects showed a pretest-to-posttest decline in duration to all unilateral caloric stimuli. Similar results were reported by

Pfaltz and Arx (ref. 120), who obtained a reduction in nystagmus during the course of 10 unidirectional rotations in darkness. Posttests elicited both directions of nystagmus and showed a preponderance favoring the unpracticed direction; the difference was still evident 2 weeks later.

Collins (ref. 35) gave 10 subjects each a series of 200 unidirectional accelerations (decelerations were subthreshold) over a 10-day period. Nystagmus was recorded with eyes open in total darkness, and subjects were assigned a variety of tasks to be performed during stimulation. Tests conducted the day before and the day after the habituation series indicated that there was not so much a decline in nystagmus as there were changes in the form of the response. Specifically, for the practiced direction, slow-phase eye displacement declined (30%) throughout the response. However, the number of eye movements increased throughout the stimulus period and for several seconds thereafter (although total frequency showed a slight decline); the response also appeared more regular and perhaps better "tuned" to the stimulus (see fig. 8). These changes were somewhat greater for the practiced direction, but were clearly evident for both directions, and were still present 1 month later with no intervening trials.

Wendt (ref. 121) reported a similar finding,

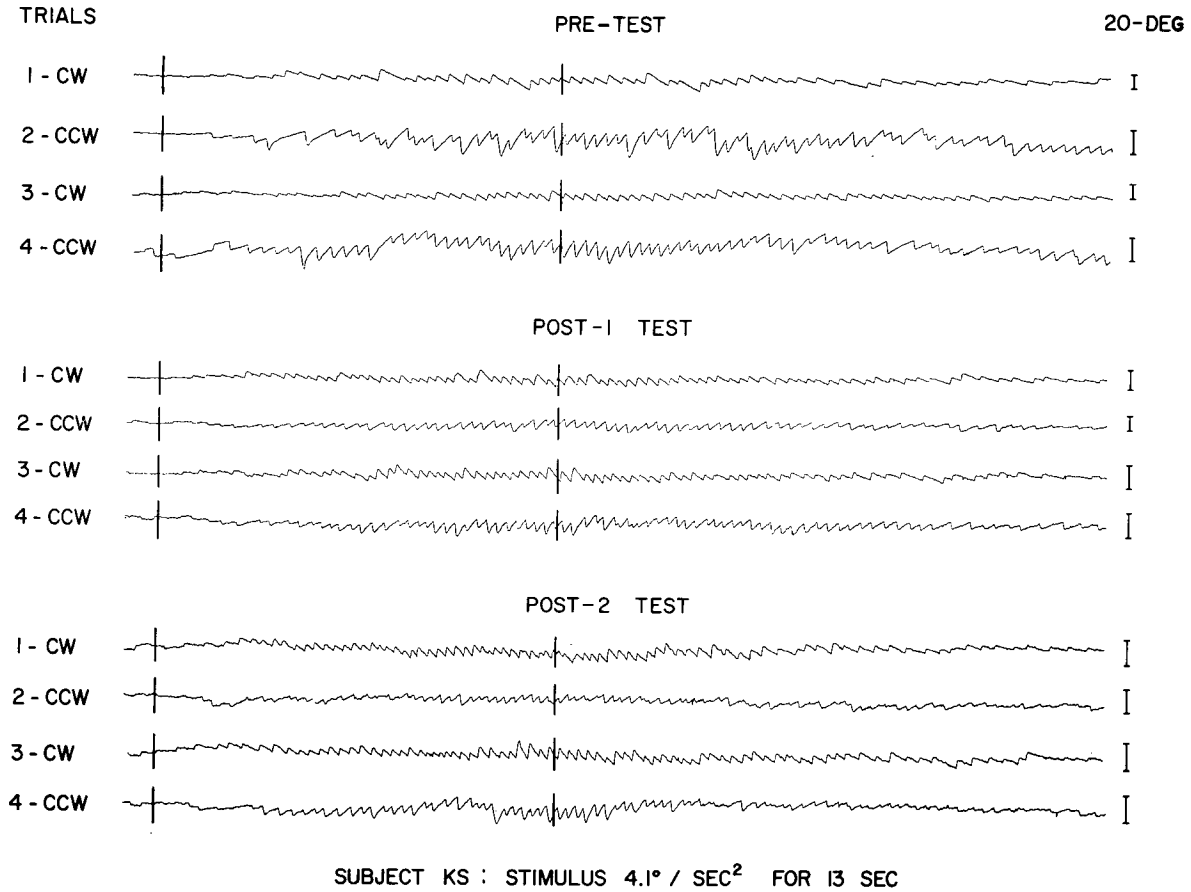


FIGURE 8.—*Tracings of nystagmus obtained from a subject performing mental arithmetic with eyes open in darkness. A 10-day series of 200 clockwise accelerations (all decelerations were subthreshold) occurred between the pretest and post-1 test and produced an increase in the frequency of eye movements. Post-2 tests were conducted 1 month later with no intervening trials. Vertical bars demarcate the period of acceleration; 20° calibration markers appear at the end of each tracing. (Reprinted with permission from ref. 35.)*

using oscillatory stimulation: With repeated testing (subjects' eyes were closed), the amplitude of slow-phase eye movements declined and the number of fast phases increased until, finally, the original form of the nystagmus was lost. Both Collins (refs. 35 and 122) and Wendt (ref. 121) have noted that repeated elicitation of nystagmus from alert subjects in darkness produces, as one major effect, increasingly greater fast-phase activity which modifies the form of the response until, conceivably, the nystagmic aspect may no longer be evident following a very large number of stimulus repetitions. More recently, Torok (ref. 123) and Johnson and Torok (ref. 124) noted an increased frequency of nystagmus following repeated angular stimulation.

Perhaps the most striking examples of the effects of repeated angular accelerations on human nystagmic responses may be found with figure skaters. Skaters have been regarded both anecdotally and as the result of some evaluations (refs. 68 and 125) as having acquired considerable or complete vestibular suppression as a result of their spinning experiences. When tested in total darkness, however, skaters gave vigorous nystagmic responses to a variety of angular stimuli (refs. 122 and 126). When compared with ordinary subjects tested in the laboratory (in total darkness), the skaters showed significantly less slow-phase eye displacement and as high or higher a frequency of nystagmus during the early part of the response than did the ordinary subjects (ref. 122). Tracings also appeared more regular for the skater group. These comparisons between skaters ("practiced" subjects) and non-skaters ("unpracticed" subjects) show considerable similarity to the findings discussed earlier in which pretest-to-posttest comparisons of the same subjects were made after 200 unidirectional rotations (ref. 35). Apparently most figure skaters spin counterclockwise (CCW) on ice (there are notable exceptions); and few, if any, either practice or develop a facility for spinning well in both directions. Since their on-ice spinning rate is too high to permit "visual spotting," they use visual fixation almost invariably during one direction of eye movement (i.e., following deceleration). However, no consistent directional differences appeared for skaters in duration of mystagmus, slow-phase eye displacement, or frequency of eye movements as a result of angular acceleration in total darkness (ref. 122).

These results indicate that vestibular nystagmus (in the absence of visual stimulation) is not readily suppressed in alert subjects as a result of considerable experience with angular accelerations, whether that experience occurs in darkness or with opportunities for visual fixation. The response appears modified, but in a dynamic fashion rather than as the result of an overall suppression.

As noted earlier, Wendt (ref. 14) claimed that nystagmus generated in total darkness would not be abolished in alert subjects. However, he indicated that, with opportunities for visual fixation, the visual stimuli would eventually become predominant and markedly interfere with nystagmus. A number of studies in which subjects were allowed opportunities for visual fixation have reported significant declines (or abolition) of nystagmus (refs. 24, 111, 113, and 114). Perhaps the best example of such habituation can also be found with figure skaters. Both in the laboratory (caloric and rotational stimulation) and following on-ice spins, skaters demonstrate almost total suppression of vestibular nystagmus when they have fixed visual references (refs. 125 and 126). The longest duration of nystagmus recorded (by means of telemetry) from skaters following on-ice decelerations from velocities of 250 to 300 rpm was about 5 s; other skaters gave just a few beats of nystagmus before visual fixation terminated any apparent eye movement (ref. 126). However, removal of the opportunities for visual fixation by means of eye closure would reinstate a vigorous nystagmic response (see fig. 9). More recently, Fukuda et al. (ref. 127), Osterhammel et al. (ref. 128), and Dix and Hood (ref. 129) reported no after-nystagmus from ballet dancers whose eyes were open following their pirouettes, but vigorous nystagmus when their eyes were closed. Previously, Tschiassny (ref. 130) had noted that a postrotatory nystagmus could be obtained from a ballet dancer if "visual spotting" was not permitted.

The findings with figure skaters might lead one to conclude that total or almost total suppression of nystagmus may require repeated vestibular stimulation with opportunities for visual fixation. Such suppression is visual inhibition of the response rather than vestibular inhibition, and the ability to exercise substantial control over vestibular eye movements does not seem to generalize completely

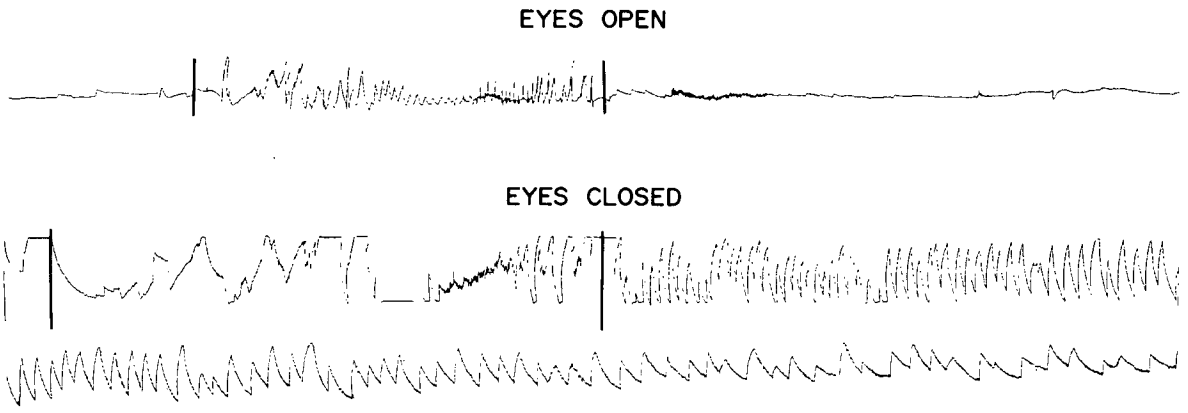


FIGURE 9.—*Eye-movement tracings obtained by means of telemetry during on-ice spins performed by a figure skater. Each strip of tracings represents 33 seconds of recording. Vertical bars demarcate the period of spinning. After-nystagmus is markedly suppressed during visual fixation (cf. ref. 126).*

to situations in which vision is not permitted.* In fact, one study (ref. 131) reported no influence on nystagmic responses in darkness of several types of visual stimulation during repeated rotation (a small, dim target light, an illuminated enclosure, and full room lighting). However, the habituation series for the several tested groups comprised only six trials for a given direction of nystagmus, and the angular accelerations used (24°/s² for 10 s) induced incidents of nausea and vomiting when either the room or the enclosure was lighted.

In evaluating the relative lack of generalization of eye-movement control by figure skaters from the on-ice condition (vision permitted) to the laboratory situation (vision excluded), several factors must be considered: (1) During on-ice spin skaters actively coordinate control of rotation, while in the laboratory they are passively rotated. (2) The stimulus rates during on-ice spins are considerably higher than those generated in the laboratory. (3) Some laboratory findings with ordinary subjects (ref. 132) and with aerobatic pilots (refs. 133 to 135) show directionally specific effects related to visual stimulation.

Aschan (refs. 133 and 134) reported a directionally specific shortening of the duration of nystagmus (cupulometry) in fighter pilots, with the reduction related to the direction in which the pilots most frequently rolled their aircraft during various flight maneuvers. Caporale and Camarda (ref. 135) obtained a similar finding from aerobatic pilots, using accelerations and decelerations of 1°, 3°, and 6°/s² separated by 90 s of constant velocity at 15 rpm. Recordings from behind closed eyes showed a clear directional difference in intensity of nystagmus. Pilots who flew on the left in formations (and therefore kept their heads turned to the right during flight to maintain visual contact with the aircraft ahead of them) showed weak left-beating and normal right-beating nystagmus. Pilots who flew on the right side of formations (with their heads, therefore, turned to the left) evidenced the reverse of this directionally specific difference.

*Generalization of nystagmic modifications from dark to light conditions is also minimal, at least after a short series of habituation stimuli. Marshall and Brown (ref. 44) rotated subjects in an illuminated enclosure before and after a series of 10 habituation trials employing identical stimulus rates. One group received the habituation series with the enclosure illuminated (and visual still-fixation possible); the other group was in total darkness. During all trials, subjects signaled their estimates of turning velocity. Both groups showed a significant reduction in slow-phase eye displacement during the habituation series. In the posttest (enclosure illuminated), the group habituated in total darkness showed no significant change in slow-phase output from the prehabituation level; the group habituated in illumination, of course, had significantly reduced scores. This lack of transfer from dark to light conditions might be accounted for by the fact that, of itself, visual fixation on objects produces a marked decrease in the slow-phase excursion of the eyes, and the reduction obtained after a short series of rotations in the dark may not be sufficient to influence noticeably the greater attenuating effects of visual still-fixation.

Guedry (ref. 132) controlled arousal by tasks and used total darkness during one direction of nystagmus (e.g., CW acceleration), while visual stimulation occurred during the other direction of the response (deceleration from CW rotation). Subjects were given 80 such rotation trials within a 4-hour period. Tests before and after the 80-trial series were conducted entirely in darkness; results indicated a pretest-to-posttest decline in slow-phase nystagmus of about 20 percent for the direction repeatedly elicited in darkness (in good agreement with the results obtained in darkness by Collins, ref. 35) and a pretest-to-posttest decline of about 50 percent for the direction of nystagmus repeatedly inhibited by visual stimulation.

The findings of Aschan (refs. 133 and 134), Caporale and Camarda (ref. 135), and Guedry (ref. 132) do not appear to agree with results obtained from figure skaters, who show equivalent nystagmus regardless of direction when tested in the laboratory, although on ice they make use of visual information only (or at least primarily) during deceleration responses. The lack of agreement may be due not only to active versus passive rotation and to marked differences in stimulus rates, but also to the fact that the skaters do perform on-ice turning maneuvers in both directions, and the rate of these turns (as opposed to rates during their free-style spins) may be equivalent to laboratory stimuli. In addition to these factors, (1) it is possible that visual stimulation may simply speed up the habituation process for the practiced direction of turn and, with many repetitions, transfer of the nystagmic response modification may become complete so that both directions of nystagmus are equivalently modified; and (2) the sensory pattern is different for skaters spinning on ice as compared with laboratory rotations. In the latter case, nystagmus was always elicited with the cupula in its resting position. On ice, skaters build up their acceleration until they feel it has reached its peak and then bring themselves to a stop; the deceleration (followed by visual still-fixation) is thus introduced with the cupula away from its resting position, perhaps at a point of maximum deflection.

All of the above findings have been concerned with what Guedry (ref. 132) has termed "simple vestibular stimulation," i.e., stimulation by angular acceleration about a vertical axis primarily of a single pair of semicircular canals. "Complex vestibular stimulation" may involve simultaneous activation of other pairs of canals and of specific and nonspecific gravireceptors. In this regard, mention should be made of studies involving rotation about a horizontal axis (refs. 53, 54, and 136 to 138) and combinations of angular and centripetal accelerations (refs. 139 and 140). Nystagmus is clearly affected by these conditions. In the first case, nystagmic eye movements did not cease during constant velocities ranging up to several minutes in duration, and a relatively abrupt termination of nystagmus and sensation occurred upon stopping. In the second case, the intensity, plane, and direction of nystagmus changed as centripetal accceleration increased with the lateral canals in the plane of rotation. Bergstedt (refs. 141 and 142) has shown that the intensity of both positional and caloric nystagmus is increased with increasing g-force; this finding was confirmed by Orlov (ref. 143) and by Yuganov (ref. 144), with Yuganov also reporting a decrease in postrotational nystagmus during weightlessness. Thus, it is clear from these and other studies (e.g., refs. 81, 95, 145, and 146) that nystagmus can at least be affected by other motion-sensing or position-sensing systems and, indeed, horizontal nystagmus can even be elicited by periodic linear accelerations (ref. 147).

Complex vestibular stimulation can also be accomplished by means of simultaneous motion about more than one axis (cf. ref. 148) or by active or passive head movements during rotary motion (refs. 149 and 150). Such simultaneous stimulation of more than one pair of semicircular canals, the so-called Coriolis vestibular effect, produces (in addition to sensations) nystagmus with a predominantly vertical component. It should also be noted that there is a lack of congruency between signals from the semicircular canals and those from other sensory systems (e.g., otoliths and proprioceptors) under these unusual stimulus conditions. In examining the question of habituation, most "Coriolis studies" have been conducted with opportunities for visual fixation. Subjects have been permitted to move about freely in a rotating room (ref. 149), or have made restricted head movements during rotation according to set procedures (refs. 55, 151, and 152), or have been exposed to passive movement as well as making active head tilts (ref. 153).

Subjects learn to adapt to the unusual vestibular effects produced by moving about in a lighted rotating room. During long-term studies (64 hours to 12 days) standard head-movement tests with control of arousal were introduced during the course of the prolonged rotational period as well as before and after rotation (i.e., with no rotation). Nystagmus was recorded in total darkness during these standard tests and showed an overall decline during rotation (ref. 149). Following cessation of rotation (in a static situation), subjects demonstrated spontaneous compensatory nystagmus as a result of head movements (refs. 149 and 154). That is, following the adaptation period, while at a standstill, standard head movements produced a nystagmus opposite in direction to that which should have occurred had the same head movement been made during rotation (see fig. 10). Retention and transfer of habituation effects were examined by Guedry (ref. 154), who tested subjects in darkness before and after a 12-day exposure to living in a CCW-rotating room. A clear attenuation of nystagmic responses to head tilts during CCW rotation was obtained in post-habituation tests; little pretest-to-posttest change was obtained for head tilts during CW rotation. Additional posttests were conducted 2 days, 3 weeks, and 3 months later. Nystagmic responses to head movements during CCW rotation showed progressive but incomplete recovery; however, nystagmus obtained as a result of head movements during CW turning fell well below the pretest values in these later tests, to a level approximately equal to the CCW responses, as if to establish a balance at the habituated level.

Studies of Coriolis effects with restricted head movements (in one quadrant of the frontal plane only) have been of shorter durations (4 to 7 hours). Although a large number of head movements were made in the lighted room by the subjects, no spontaneous compensatory nystagmus was obtained following the rotation periods (refs. 151 and 152). However, standard head-movement trials in darkness during rotation were conducted after about 200 head movements in the light and showed a general pretest-to-posttest suppression of nystagmus for the practiced direction of head movement, but there was no effect of the habituation trials on nystagmus generated by head movements in the unpracticed quadrant.

Further research confirmed and expanded these findings. Guedry (ref. 55) had a group of subjects tilt their heads and return them to upright at set intervals with their eyes closed. Other subjects performed the same head movements but were required to solve problems with multiple-choice answers which were projected on a screen during the head movements. All head movements were made in a single quadrant, with most subjects completing 100 tilts and 100 returns. Before and after the 100-cycle series, subjects performed mental tasks in darkness and made head movements in both the practiced and unpracticed directions. A clear pretest-to-posttest reduction in recorded nystagmus (approximately 70%) occurred in the practiced quadrant for subjects who performed the visual problem-solving tasks; the unpracticed quadrant showed only a 15-percent reduction. A similar quadrant-specific reduction was evident in pretest-to-posttest comparisons for the group that had performed the head movements with eyes closed, but the reduction in the practiced quadrant was only about 12 percent; the unpracticed quadrant showed essentially no pretest-to-posttest change.

Caloric-Induced Nystagmus

Although angular acceleration is the adequate stimulus for the semicircular canals, the most frequently used method of eliciting nystagmus in clinics is that of thermally produced endolymph movement in the canals. The recommended procedure is to use water of temperatures 7° C above and below body temperature (ref. 155). Cool water (30° C) drives nystagmus (fast phase) away from the irrigated side; warm water (44° C) drives it toward the irrigated side. Although the stimulus is a gross one, it has the advantage of permitting the testing of each ear separately.

Almost all studies of the effects of repeated caloric irrigations on nystagmic reactions have involved unilateral stimulation. Loch and Haines (ref. 156) timed nystagmus from subjects wearing Frenzel glasses and reported no decline as a result of irrigations performed once daily for 5 to 6 days, although they noted a change in the form of the response (nystagmus became more regular). With subjects' eyes closed, Stahle (ref. 157) found no clear response decline for four irrigations, two of which drove nystagmus to the left and two to the right.

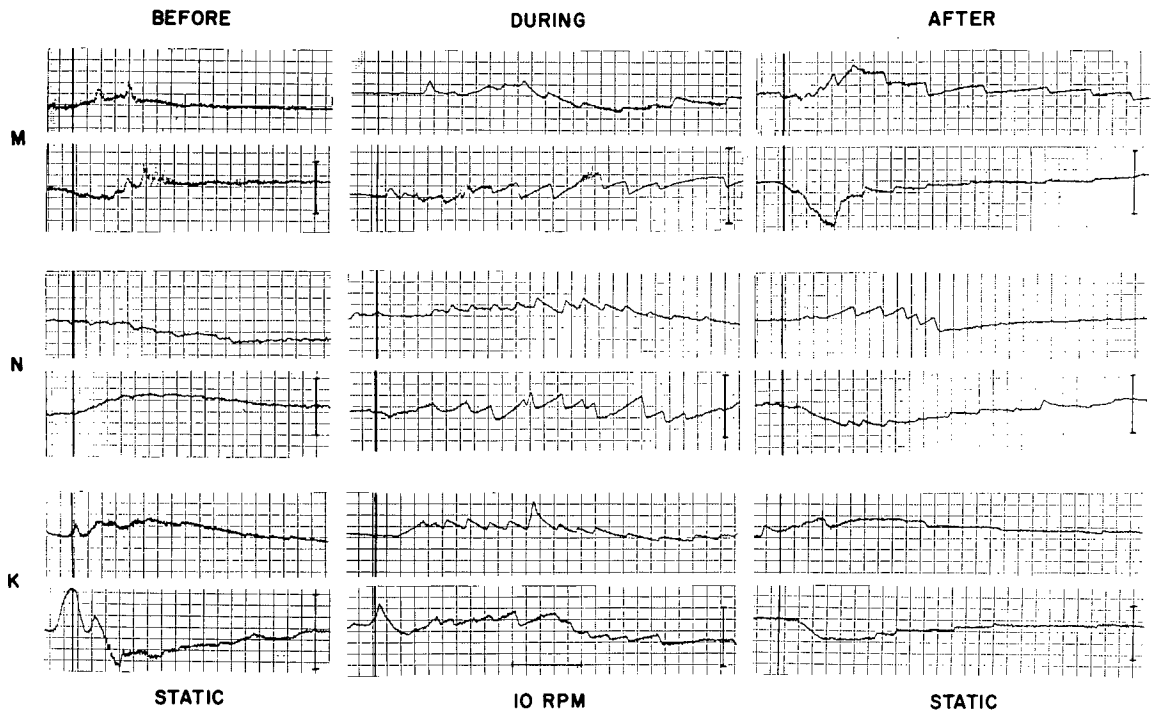


FIGURE 10.—*The development of a conditioned compensatory nystagmus is evident in the tracings obtained in a static situation (i.e., with the rotator stationary) after a 64-hour period of head movements during rotation at 10 rpm (compare the "after" static responses with the "before" static tracings). Note that the compensatory nystagmic reaction is in the direction opposite to that which was produced as a result of head tilts during rotation. The upper tracing of each pair resulted from a 75° head tilt to the left; the lower tracing of each pair represents the response occasioned by returning the head to an upright position. All tracings are of vertical nystagmus for subjects M, N, and K. Vertical bars through the left of each tracing indicate the initiation of head movement; bars through the right of the lower tracing of each pair represent 40° eye calibration markers. (Reprinted with permission from ref. 149.)*

However, he noted that dysrhythmia might occur with repeated stimulation. Lidvall (refs. 29, 38, 158, and 159) performed a series of studies which indicated that a relatively small number of stimuli (4 to 6) would significantly shorten nystagmus duration (recorded from behind closed eyes) and reduce the frequency of nystagmus, while increasing latency and dysrhythmia, whether the intervals between stimuli were short (6 to 8 minutes) or long (1 to 25 days).

Fluur and Mendel (refs. 160 and 161) also recorded eye movements from behind closed lids and obtained significant reductions (about 30%) in response duration (increasing dysrhythmia made quantification of other aspects of nystagmus impractical) following a series of 8 to 12 irrigations.

In their first study, subjects received unilatera habituation trials. In post-habituation tests, th majority of subjects showed a response reductio from pretest levels for unilateral stimuli to eithe ear which drove nystagmus in the habituated direc tion, and a longer nystagmus time for unilatera stimuli which drove nystagmus in the opposit direction. Other subjects showed a pretest-to-post test decline for either cool or warm stimulation i either ear. In the second study (ref. 161), subject were habituated with 8 to 12 unilateral stimulation of one ear; posttests yielded the same results a those obtained from the majority of subjects in th previous study. Next, a new series of unilatera stimuli drove nystagmus in the opposite direction Posttests indicated a decline for that direction o

nystagmus irrespective of the ear irrigated, but responses in the opposite direction (habituated in the first series of stimulations) were now back to the initial, prehabituation levels. The authors concluded that "in a person already habituated by repeated monaural irrigation with hot water it is not possible to superimpose a habituation to cold water in the same ear without influencing the response to the primary habituation." They theorized that habituation influenced the spontaneous activity levels in the two labyrinths and disrupted the balance between them.

However, Pfaltz and Arx (ref. 120) obtained a directional preponderance of nystagmus following a series of unidirectional rotations, and reported no reversal of this difference following a series of unilateral caloric stimuli which drove nystagmus in the opposite direction, although the caloric response declined with repetition (all tests in darkness). They noted that a shift in the ratio between the two directions of nystagmus could be obtained by repeated unidirectional angular accelerations, but not by repeated unilateral caloric irrigations. ("Eine Verschiebung des quantitativen Verhaltnisses zwischen Rechts- und Linksnystagmus kann im Gegensatz zur wiederholten Drehreizung nicht mittels aufeinanderfolgender monauraler thermischer Reize erzielt werden.") A series of binaural irrigations, on the other hand, produced a preponderance of nystagmus favoring the unpracticed direction of eye movement.

Forssman et al. (ref. 162) and Forssman (ref. 163) investigated the effects on caloric habituation of 12 consecutive irrigations in darkness and in illumination (the first and twelfth trials were always in darkness). They reported approximately equivalent reductions in duration and velocity of nystagmus for the two conditions (25% for duration; 55% for velocity). In addition, laterotorsion (i.e., deviation of the head; cf. ref. 164) declined by approximately 40 percent. Dysrthythmia during light trials was considerable (probably due to intermittently effective visual inhibition), but during dark trials was very moderate.

In the above investigations, only Lidvall (ref. 38) reported the use of any alerting technique. After demonstrating a progressive decline in the number of eye movements during four irrigations, Lidvall (ref. 38) noted that an additional trial during which subjects performed mental calculations resulted in an immediate restoration of the response. A study by Collins (ref. 36) examined the influence of an extensive series of unilateral irrigations (10 daily for 4 days) on responses to both unilateral and bilateral irrigations. One group of subjects was tested in total darkness while performing various tasks; the other group actively sought to control nystagmus by fixating on ceiling markers in a lighted room. During pretests and posttests, each subject received six irrigations; two were bilateral (warm and cool water simultaneously presented to the two ears), one driving nystagmus to the left, the other to the right; the remaining four were unilateral irrigations with cool and warm water presented to each ear successively. Results indicated essentially no effect of the habituation series on the duration of nystagmus for either group. For the group habituated in total darkness, no significant decline in slow-phase eye displacement but a significant overall increase in eye-movement frequency appeared (see fig. 11). For the group habituated in the light, no pretest-to-posttest change in frequency of nystagmus occurred, but slow-phase measures declined significantly. For both groups, changes were somewhat greater for the three stimuli provoking responses in the same direction as that of the habituation trials. For the light group no recovery of the slow-phase loss was evident after 1 month of rest; similarly, for the dark group, the increase in frequency of nystagmus was still evident upon retest a month later.

Collins, Schroeder, and Mertens (ref. 165) performed a similar series of 40 irrigations with subjects actively fixating on ceiling markers. One group was given unilateral irrigations, the other bilateral stimulation; nystagmus was driven to the left in both cases. During pretests and posttests, subjects received the series of six cool and warm unilateral and bilateral stimulations noted above. Results indicated that, for both groups of subjects, nystagmus declined unidirectionally. A control group received only the pretest and posttest irrigations and showed no change.

The same study also included four rotation trials prior to any pretest irrigations; the rotations were repeated after all posttest irrigations. No significant

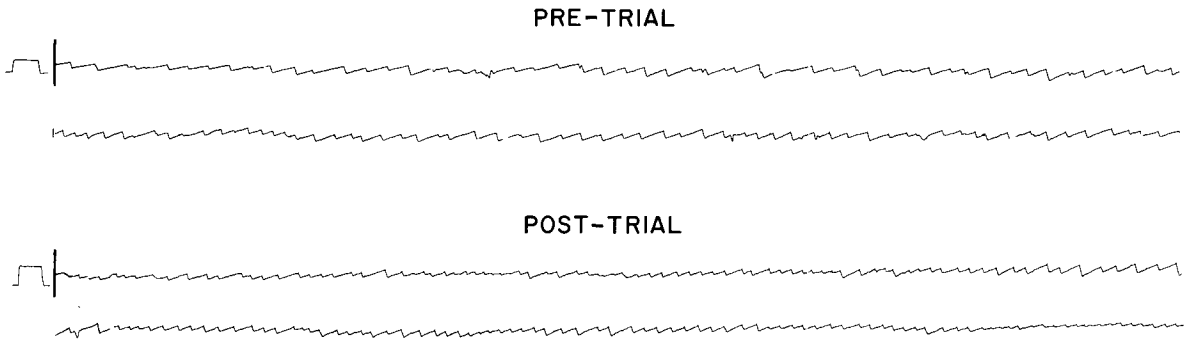


FIGURE 11.—*Caloric nystagmus recorded before and after a habituation series of 40 unilateral irrigations (30°C) to right ear for 30 s each; all trials in darkness with the subject's eyes open). Dysrhythmia and an overall reduction of nystagmus are not necessary consequences of repeated vestibular stimulation; an increase in the number of eye movements, as depicted above, may occur. Vertical bars indicate the end of the irrigation; 20° calibration markers appear at the beginning of each tracing. Each strip of tracings represents 33 seconds of recording (refs. 36 and 165).*

changes were evident in nystagmic output for either direction of rotation-induced nystagmus as a result of the caloric trials.

Rotation-Induced Subjective Reactions

The sensations of motion accompanying semicircular-canal stimulation appear to be more readily affected by repeated exposure to either angular accelerations or calorizations than are nystagmic eye movements. Of considerable interest is the fact that they may follow a time-course different from that of nystagmus.

Griffith (refs. 24 and 108), who reported attenuation of nystagmus following cessation of rotatory stimulation in a lighted room, also noted a decline in the sensation of apparent motion with repeated stimulation. Although Hallpike and Hood (ref. 166) found attenuation of turning sensations that were directionally specific (subjects were rotated with eyes closed), their findings may be more closely related to adaptation effects than to habituation. These investigators used a short test stimulus before and after a prolonged stimulus (e.g., $2°/s^2$ for 75 s) and found that, when the posttest stimulus was in the same direction as the prolonged stimulus, rotary sensations were shorter than the pretest level; when the posttest stimulus was in the direction opposite that of the prolonged stimulus, reactions were of normal (pretest) duration.

A single subject with eyes covered and head approximately erect was given two series of CW rotations by Meda (ref. 167). The first series covered 40 days and comprised 290 trials (stops and starts were sudden and apparently separately by 1 minute of constant velocity at 30 rpm). A fairly systematic decline in the duration of the sensation following deceleration was obtained both during daily trials and from day to day. Tests conducted before and after habituation showed little or no change in (1) the duration of after-sensations with the head anteverted so that the lateral canals were perpendicular to the plane of rotation, (2) the duration of sensations occasioned by head movements either during rotation or immediately after deceleration, and (3) the duration of after-sensations following CCW deceleration. The lack of transfer of habituation from one set of semicircular canals to another set, or between reactions from one set of canals and Coriolis-induced responses, has been confirmed in other studies of nystagmus from man and cat (refs. 81, 94, 151, and 154). However, the failure to obtain a reduction to CCW deceleration is surprising since the subject was apparently exposed to an equivalent stimulus (CW acceleration) during the habituation series, at an equivalent rate (sudden starts and stops), under identical visual conditions (eyes covered). A second series was initiated with the subject's head ventrally flexed. After 100 rotations the after-sensation was reduced by about 50 percent. Tests with head movements

during and following rotation showed no clear effect of the habituation series on the duration of these Coriolis sensations. Additional tests conducted at intervals of 1 to 3 months with the subject's head erect finally showed full recovery of the duration of the after-sensation.

In comparing the technique of cupulometry to the Bárány test procedure, Groen and Jongkees (ref. 168) demonstrated that the sensation cupulogram obtained before and after a set of three CW and three CCW Bárány spins (stops from 30 rpm) showed a marked decline. They felt that the Bárány-type stimulus damaged the vestibular organ and that a maximum velocity of only 10 rpm should be used prior to braking subjects to a stop. However, Brand (ref. 41) obtained response declines for post-rotational turning sensations both within a daily series of brake decelerations from 10 rpm (or less) and from one test day to the next.

Aschan (ref. 133), using cupulometry, reported reduced durations of sensation to rotational stimuli from fighter pilots. Unlike the directionally specific reductions in nystagmus that he found for this group, sensations for both directions of angular stimulation were attenuated. Similarly, Dearnaley, Reason, and Davies (ref. 169) reported shorter durations for experienced pilots than for trainees in the vestibular sensations occasioned by 1-minute-long, 45° banks in an aircraft; within each group there was no difference between left and right turns. The differences between pilots and students were present whether subjects' eyes were closed or visual references were available, although increasing the amount of visual information decreased the sensation for both groups. Preber (ref. 170) had examined pilot trainees before flight training and followed up a group who showed severe airsickness during the training period. These subjects, as a group, had longer durations of sensation during turning tests conducted prior to flight training than did those who did not get airsick; however, after they adapted to the flying situation, tests showed a decline in duration of sensation. Previously, de Wit (ref. 171) had reported progressively greater cupulogram slopes in comparisons of a group of sailors who had never been seasick, a group of students, and a group of sailors who were chronically seasick.

Guedry, Collins, and Sheffey (ref. 30) performed a 50-trial habituation series spaced over 5 days, with trials alternately in total darkness and with a 5-s period of room illumination following deceleration. Pretests and posttests in total darkness showed a decline of approximately 50 percent for total subjective displacement. Partial recovery was evident 9 days later, with no intervening trials.

Benson (ref. 172) gave subjects (closed eyes) impulsive decelerations and, 3 s after they had reached a stop, had them shake their heads for 2 s. The duration of postrotatory sensations was reduced after the headshaking period. With repetition, postrotatory sensations declined both with and without headshaking, but declined independently. There was no difference between nonaerobatic aircrew and ordinary subjects.

Collins' study (ref. 35), employing 200 unidirectional habituation trials in total darkness with subjects performing various attention-demanding tasks, showed a directionally specific reduction of 39 percent in peak subjective velocity and 37 percent in total subjective displacement for the practiced direction of angular acceleration. There was no pretest-to-posttest difference for the unpracticed direction (see fig. 12). One month later, with no intervening trials, total subjective displacement was only 27.5 percent less and peak subjective velocity only 16.5 percent less than the pretest levels for the practiced direction; the unpracticed direction still showed no change from the pretest level. Thus, the subjective reaction declined in a directionally specific fashion, without the subjects attending to the sensation, and in the absence of vision; the response showed partial recovery after 1 month of rest.

Professional figure skaters report some initial dizziness when they resume spinning routines after vacation periods of 1 to 3 months. In skating trim, they appear to experience only very mild and brief dizziness following on-ice spins, but if they close their eyes upon stopping, clear vertiginous sensations occur. Similarly, while they can perform gracefully with open eyes following such spins, closing their eyes produces a loss of balance and an inability to maneuver (ref. 126). In laboratory tests (total darkness) of figure skaters, clear turning experiences were reported to rotation, and there was no difference in the duration of left-turning versus right-turning sensations (ref. 122). When compared with ordinary subjects, however, skaters had significantly shorter durations to stimuli ranging from

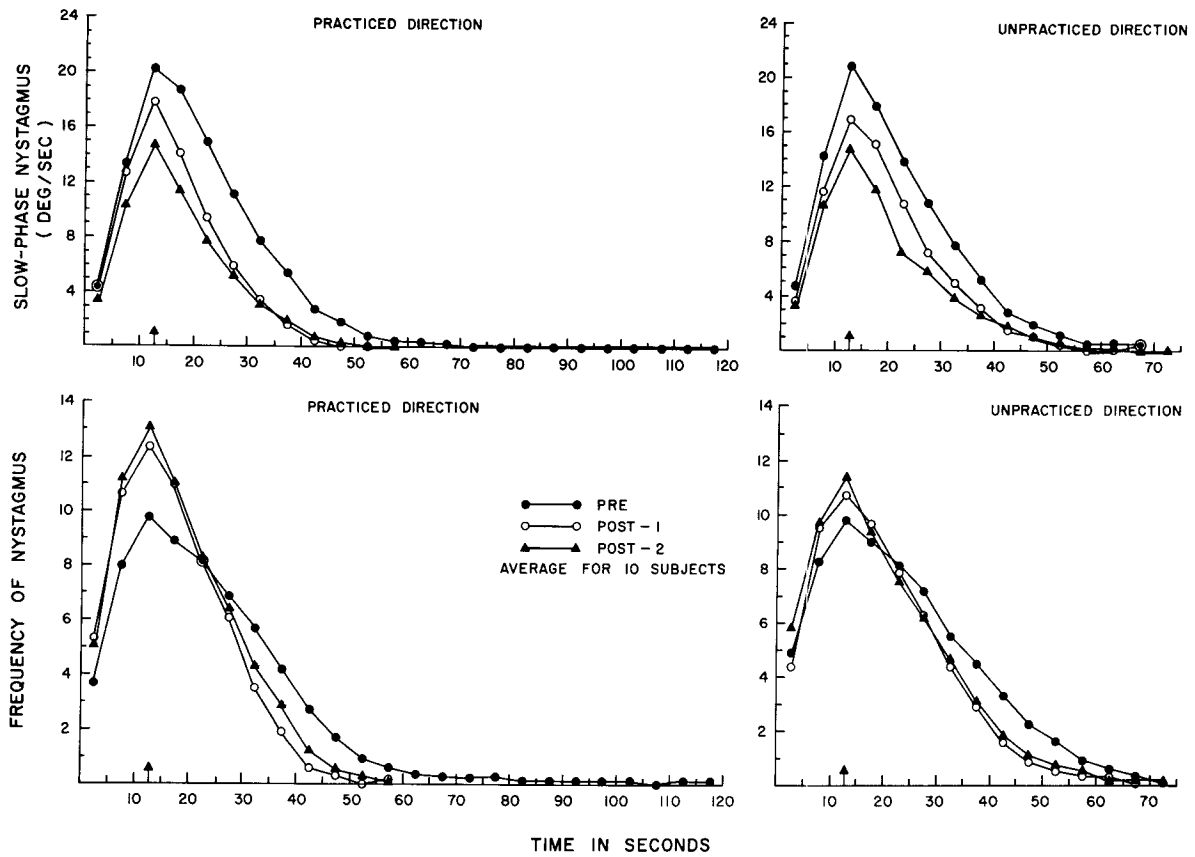


FIGURE 12.—*Subjective velocity estimates made by subjects before (pre) and after (post-1) a series of 200 clockwise accelerations (all decelerations were subthreshold). Post-2 tests were conducted one month later with no intervening trials. The habituation series produced a clear, directionally specific reduction in the intensity of the turning sensation (but not in its duration); a month of rest produced considerable recovery. (Reprinted with permission from ref. 35.)*

$5°/s^2$ for 18 s to $90°/s^2$ for 1 s. It is of considerable interest that the differences between the two groups were proportionately greater for the higher rates of angular acceleration (ref. 122).

The Oculogyral Illusion

During rotational stimulation, an object with a minimal visual surround (a small, dim light source in an otherwise dark room) appears to move in a definite pattern although it and the observer are in fixed positions (ref. 173). This apparent motion, the oculogyral illusion (OGI), has been identified with the sensation of turning rather than with nystagmus (refs. 174 to 176; however, see also ref. 177). Brown and Guedry (ref. 178) examined the duration of the OGI before and after short series of rotations (1) in total darkness, (2) with a target light present, and (3) with 5 s of room illumination following cessation of rotation (target light present). Marked declines in OGI duration occurred for the last two conditions. Previously, Guedry (ref. 179) had reported a successive shortening of the duration of the illusion during a series of trials in which conditions of total darkness and of brief illumination of the test room (shortly after deceleration) were alternated. Directional specificity of this decline in the condition which employed vision was noted in later studies (refs. 180 to 182), and it was observed that a decline of the OGI at one stimulus rate produced a greater response reduction to a stimulus of lesser intensity and a lesser response reduction to a stimulus of greater intensity (ref.

181). A recent study by Brown and Crampton (ref. 131) showed no differential effects on duration of the OGI of rotating subjects in total darkness, with the OGI, with an illuminated enclosure, or in full-room illumination. However, only six habituation trials were conducted for each group in the direction under consideration.

Coriolis Vestibular Sensations

The OGI has also been used in studies of habituation of Coriolis vestibular responses produced by head movements during constant rotation. The array of effects that occur depends to some extent upon the kind of visual information available during the head movements (ref. 183). The sensations produced (as well as the apparent movement of a target light) are combinations of displacement and acceleration (see fig. 13 and ref. 184); e.g., tilting the head to the right during CW rotation produces a sensation of tilting backward and accelerating upward much like a sharp climbing maneuver in an aircraft. Guedry and Montague (ref. 150) reported reduction of the apparent displacement of a target light with repeated head movements. Graybiel et al. (ref. 185) rotated subjects in the light for a 64-hour period during which they made voluntary head movements or were passively tilted in a special chair. Tests during the experiment involved the OGI (the lighted outline of a cube) with the room otherwise dark. Magnitude estimates of the illusion declined for all subjects. In addition, a conditioned compensatory reaction was obtained; when the room was at a standstill at the end of the experiment, subjects made head movements and reported OGI effects that were opposed in direction to those that would have been produced had the same head movement been made during rotation. Similar conditioned compensatory reactions were obtained for nystagmus (ref. 149). Further evidence was obtained for conditioned OGI effects in studies of shorter duration (4 to 8 hours of rotation) with voluntary head movements restricted to a single quadrant in the frontal plane (refs. 151 and 152).

Guedry (ref. 55) exposed two groups of subjects to a 100-cycle series of head movements restricted to a single quadrant; one group performed the movements with eyes closed, and the other group solved problems projected on a screen during the head tilts. Tests were conducted before and after the 100-cycle series (1) during rotation and (2) in a static situation. During rotation, there were clear pretest-to-posttest reductions in OGI for head movements in the practiced quadrant and a lesser reduction for the unpracticed quadrant among subjects in the vision group; the darkness group showed less reduction. In static posttests, the vision group reported OGI effects principally in the practiced quadrant; the darkness group evidenced little or no OGI reactions.

In a study related to the above, but without using the OGI, Guedry (ref. 154) gave pretests and posttests in darkness to subjects who had lived in a lighted, rotating room (10 rpm CCW) for 12 days. The subjects rated the intensity of sensations occasioned by specific head movements during both CW and CCW rotation. Little or no posttest sensations were obtained during CCW rotation; sensations from head tilts during CW rotation were as strong or stronger than the pretest levels. Additional posttests conducted 2 days, 3 weeks, and 3 months later showed recovery, but not to the pretest levels.

Caloric-Induced Subjective Reactions

Sensations generated by caloric irrigations are not uniform among subjects and may even differ from one irrigation to the next for the same subject (ref. 36). Frequently a directional component of the experience cannot be specified (ref. 170). Although bilateral stimulation (simultaneous application of cool water to one ear and warm to the other) frequently produces sensations of apparent angular motion about an Earth-vertical axis, both bilateral and, more often, unilateral calorizations will result in experiences of tilting, floating, dizziness, "arcing" (successive apparent motion through a relatively small angle), combinations of these, or even apparent turning about an Earth-horizontal axis (ref. 36).

Lidvall (refs. 29, 158, 159, and 186) reported a considerable decline in vertigo with as few as four unilateral irrigations and noted that the decline was specific to the direction of cupula deflection in the irrigated ear (ref. 29); i.e., after vertigo habituation of one ear with a cool stimulus, a warm stimulus applied to that ear or any stimulus applied to the opposite ear would produce a strong vertigo response. Forssman et al. (ref. 162) and Forssman

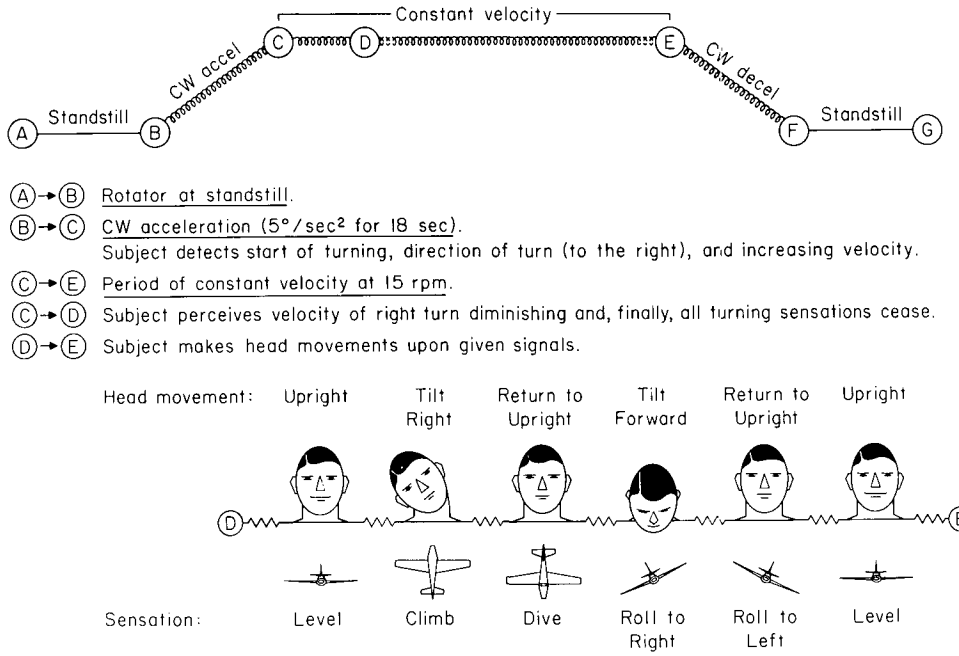


FIGURE 13.—*Subjective reactions occasioned by angular accelerations, angular decelerations, and head movements during rotation (ref. 184). Note that: the sensations are reversed if direction of rotation is reversed; returning the head to upright from a tilt to the right is equivalent to tilting the head to the left; the sensation experienced as a result of deceleration is directionally opposite that resulting from the acceleration and is perceived as a speeding up rather than a slowing down.*

(ref. 163) noted declines in vertigo ratings exceeding 80 percent after 12 irrigations, whether subjects were tested in darkness or in an illuminated room. Hinchcliffe (ref. 187) obtained ratings of caloric vertigo induced by unilateral stimulation and reported marked declines in intensity ratings following five irrigations; a second series administered on the following day showed partial recovery for the first trial and a rapid decline for the remaining tests (subjects' eyes closed).

Collins (ref. 36) kept subjects alert during a 40-trial habituation series (one group was tested in total darkness, another group was engaged in active visual fixation) and tested responses in total darkness before and after the habituation trials. Since the pretests and posttests involved all combinations of irrigations (unilateral and bilateral, cool and warm), the effects of the 40-trial series (cool water to the right ear) on responses in the same and in the opposite "directions" could be evaluated. For both groups of subjects, directionally specific effects were evident. There was an average increase in latency of the sensation and a decline in its duration and rated intensity for all stimuli eliciting responses in the same direction as the habituation trials. Responses in the opposite direction were either less affected (although showing some decline) or, in the case of subjects who received the 40-trial series in darkness, unilateral stimulations produced ratings of greater intensity than the pretest levels. For both groups, a 1-month rest interval resulted in some recovery or complete recovery of the response.

Collins and Mertens (ref. 188) expanded and

partially replicated the above study. Using the same caloric pretest and posttest procedures, they examined two groups who engaged in active visual fixation during a 40-trial habituation series (one group received unilateral stimulation, the other bilateral stimulation) and a control group who received only the pretests and posttests. During the 40 visual-fixation trials, the duration of dizziness and of the apparent movement of the fixation point both showed an irregular but progressive decline during each 10-trial day and over the 4 test days (see fig. 14). The pretest and posttest data (obtained in darkness) showed essentially no change in sensation ratings made by the control group. In agreement with previous work (ref. 36), repeated unilateral stimulation (cool water to the right ear) produced relatively little pretest-to-posttest change in sensation intensity for caloric stimuli that elicited responses in the direction opposite those of the habituation series. Posttest responses to the unilateral and bilateral stimuli that involved cool water in the right ear showed significant declines; warm water to the left ear (a stimulus "equivalent" to that used in the habituation series) did not produce a decline in sensation. This latter result (a lack of transfer) does not agree with the previous finding of Collins (ref. 36), but does agree with Lidvall's report (ref. 29) that unilateral caloric habituation of vertigo is specific to the cupula deflection of the repeatedly stimulated ear. The group who received bilateral habituation demonstrated significant declines in vertigo (49 to 62%) for all three stimuli that produced reactions in the same direction as the habituation series, and a uniform increase (22 to 32%) in sensation ratings for the three nonequivalent stimuli.

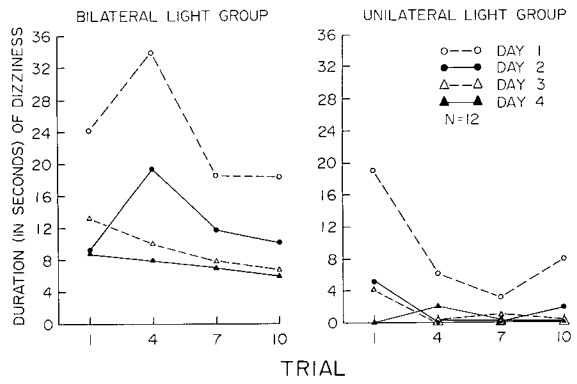


FIGURE 14.—*Mean duration of dizziness calculated from reports made by 12 subjects in each of two test groups during caloric irrigations. Subjects fixated on a ceiling marker throughout each of 10 calorizations per day for four consecutive days. The bilateral group received simultaneous irrigations of the two ears (right 30° C; left 44° C); the unilateral group received 30° C irrigations of the right ear only. The subjective reaction shows considerable decline over the test series (cf. ref. 188).*

Special Effects of Brief Periods of Visual Fixation on Vestibular Responses

Only a few studies have examined the effects of brief periods of visual stimulation on nystagmic and subjective reactions. Brown and Guedry (ref. 178) and Guedry (refs. 180 to 182), as indicated earlier, used brief periods of illumination in studies of the oculogyral illusion. Wendt (ref. 14) noted that, although postrotational nystagmus was inhibited during a period of visual fixation, the response was quickly reestablished during a succeeding period of darkness. Guedry, Collins, and Sheffey (ref. 30) confirmed Wendt's statement (ref. 14), but found that the response was not restored to the level of an uninterrupted reaction and that the sensation of turning showed no recovery.

Collins (ref. 122) introduced 3 s of room illumination 1 s after various rates of deceleration; trials were otherwise in darkness. Subjects actively fixated on wall markers during illumination and attempted to control completely their eye movements. Room illumination (subjects were at a complete standstill) substantially reduced ongoing nystagmus; during the subsequent period of darkness, nystagmus recovered, but never to the level of an uninterrupted response. Of perhaps greater significance, the shortened primary nystagmus was frequently replaced very quickly by a secondary nystagmus, which appeared more vigorous than the secondary reactions obtained (but much later in time) during trials in total darkness (fig. 15). (Markaryan, ref. 189, has also noted strong secondary nystagmic reactions following angular stimulation and a period of visual fixation.) The sensation of turning was suppressed during the visual stimulation and, during the subsequent period of darkness, was reinstated (although considerably reduced) in only a small percentage of cases.

More recently, using "prolonged" angular stim-

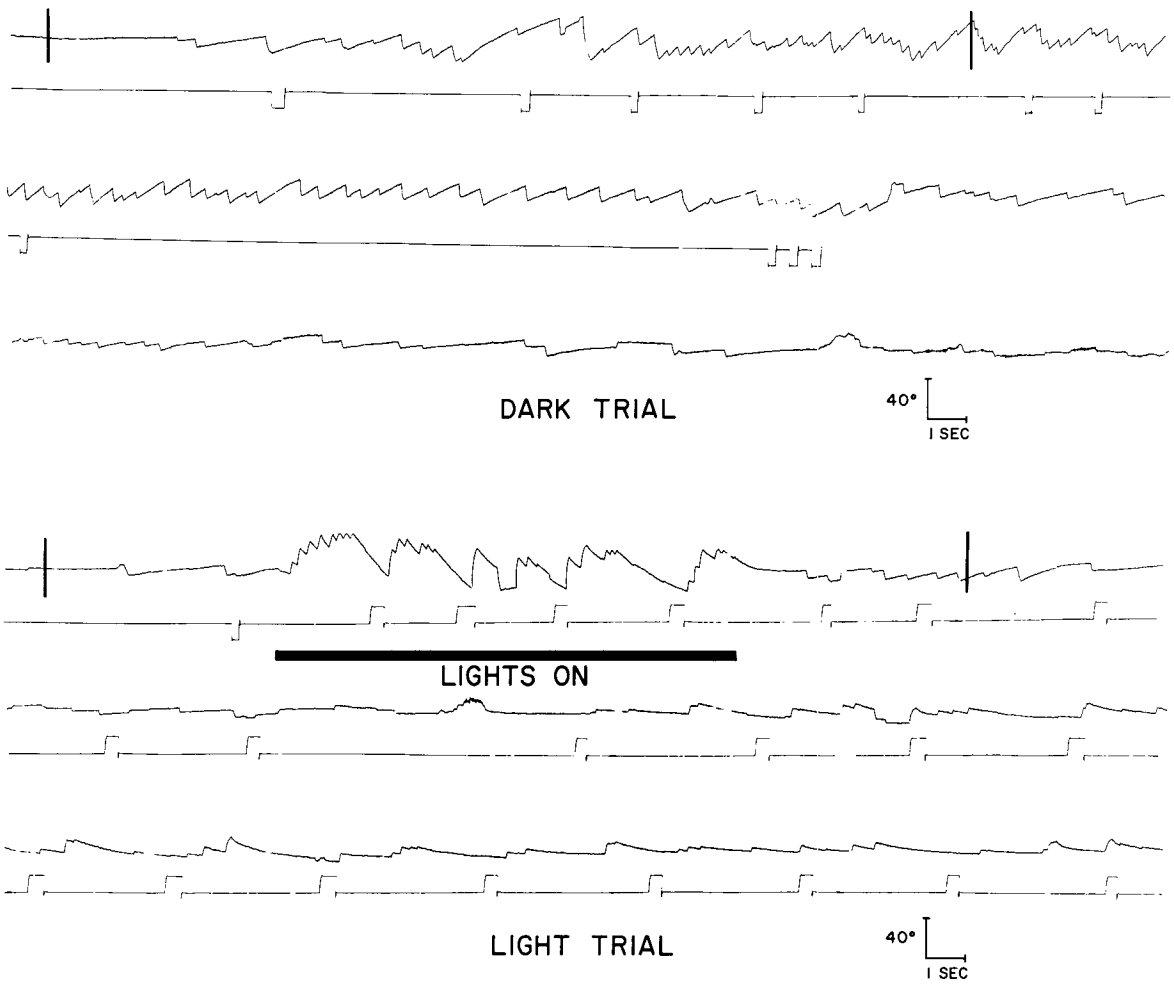


FIGURE 15.—*The influence on nystagmus and subjective turning of a period of room illumination during a clockwise deceleration. Vertical bars through the tracings demarcate the deceleration period. The deflections on the lines just below the nystagmus tracings represent the recorded signals of turning (cf. refs. 26 and 27) experienced by the subject: downward deflections indicate a sensation of turning left; upward deflections indicate a sensation of turning right. Note in total darkness the prolonged primary nystagmus, the lack of secondary nystagmus, and the indication of the usual left-turning sensation from this subject. The period of room illumination during the light trial produces a reversed (optokinetic) nystagmus and a reversal of the indicated direction of turn. Subsequent extinguishing of the lights reverses the direction of primary nystagmus for only a few seconds; the primary response is then replaced by a long-lasting secondary nystagmus. Moreover, this subject did not experience the leftward turning which the canals would have been signaling after the lights were turned off; a long-duration right-turning sensation was reported (subjects called out the direction of turn in addition to manual signals). Other subjects reported no sensation for several seconds after the period of room illumination, and then experienced strong sensations of turning to the right (cf. ref. 190).*

uli (20 s or more), Collins (ref. 190) introduced room illumination for various durations during the deceleration period; the final 6 s of deceleration and the subsequent period at a standstill were always in total darkness. Under these conditions, subjects could see themselves decelerating (although

in darkness the vestibular signals would have produced a sensation of accelerating in the opposite direction; see figs. 13 and 15), and an optokinetic nystagmus was generated opposite to the nystagmus that the semicircular canals would have initiated. Room lights were turned off during the last few seconds of deceleration, and vestibular nystagmus quickly replaced the optokinetic response. However, in comparison with an uninterrupted response, nystagmic output was at a much reduced level, was significantly shorter in duration, and more frequently was succeeded by a vigorous secondary nystagmus. Following the period of room illumination (during the last 6 s of deceleration in darkness), many subjects reported no sensation; some reported a brief sensation of turning in the true direction (in opposition to the vestibular signals). Very shortly after the termination of deceleration, a marked and prolonged "secondary" sensation was reported by almost all subjects.

These data have been interpreted as indications that secondary reactions are of central origin, in agreement with Aschan and Bergstedt (ref. 86). In addition, it has been suggested that they may occur as a result of "prolonged" neural activity in the primary direction (ref. 190); that secondary reactions are processes that oppose primary reactions (perhaps occurring as the consequence of a central imbalance produced by "prolonged" unidirectional activity); and that visual information (under some conditions) is centrally integrated and enhances this already ongoing process, thereby attenuating the primary response (refs. 122 and 190).

A MODIFIED VIEW OF "HABITUATION"

The term "habituation" has been used throughout this review to identify a series of repeated stimulations or to describe the effects of such a series on various responses. Most investigators carefully restrict the meaning of the term to "response reduction" or, considering habituation to represent a valid class of learning, cite Thorpe's definition (ref. 191): "a tendency merely to drop out responses". Although vestibular research findings appear to share many of the general characteristics ascribed to other habituation phenomena (refs. 70, 116, and 192), such definitions fail to describe adequately the dynamic processes of change which are evident as a result of repeated vestibular stimulation under at least some conditions. For example, repeated exposure to Coriolis stimulation in the light reduces nystagmus and subjective reactions in both light and darkness; an overall suppression of the responses appears to occur. However, closer examination indicates that a conditioned compensatory reaction has developed to oppose these reactions (refs. 55 and 149). There is no simple dropping out of responses; instead, there is a gradual inhibition of the original response as the opposed reaction becomes more fully developed. These opposed reactions have appeared most clearly under two nonexclusive conditions: (1) when there is an element of conflict between vestibular and visual signals (and perhaps others of a sensory nature); and (2) where the organism is attempting to adapt to an unusual vestibular environment. In darkness, nystagmus repeatedly elicited by simple vestibular stimulation is not simply reduced in alert subjects; the form of the response is changed, and the change appears to be due to increased activity of the fast phase of the eye movement, which concomitantly reduces the amount of slow-phase eye excursion (refs. 80 and 122). With very brief responses, such as those elicited by mild turns of short arc, increasingly more frequently fast phases may gradually obliterate the original form of nystagmus (ref. 121). "Active modification" would more closely describe the effects of repeated stimulation on human vestibular responses.

The above suggests the possibility of more than one kind of vestibular habituation. In the section on arousal, the notion of psychological or situational habituation was discussed in terms of arousal factors. In addition, repeated vestibular stimulation of animals produces some effects apparently different from those obtained from human subjects. Cats, for example, show a very rapid reduction of all measures of ocular responses (in darkness or in light) similar to what might occur with a nonalert human subject, even though this overall reduction occurs in animals that seem alert, or that have received an analeptic drug, and in which various sensory methods of arousal do not significantly restore nystagmus. Thus, in spite of a rich history of research, there remain considerable gaps in knowledge concerning specific effects of repeated stimulation on vestibular reactions and, moreover, there remain important questions regarding the

extent to which changes in the reaction patterns of animals can be generalized to human response patterns. It is suggested, however, that central processes are activated as a result of repeated vestibular stimulation, and that limiting the term "habituation" to a "simple dropping out of responses" masks dynamic changes and interactions that occur as these responses become modified.

REFERENCES

1. RUPPERT, J.: Untersuchungen über den Drehnystagmus. Zbl. Inn. Med., vol. 27, 1906, pp. 465-478.
2. BÁRÁNY, R.: Weitere Untersuchungen über den vom Vestibularapparat des Ohres Reflektorisch Ausgelösten Rhythmisechen Nystagmus und seine Begleiterscheinungen. Mschr. Ohrenheilk., vol. 41, 1907, pp. 477–526.
3. ABELS, H.: Über Nachempfindung im Gebiete des Kinästhetischen und Statischen Sinnes. Ein Beitrag zur Lehre vom Bewegungschwindel (Drehschwindel). Z. Psychol. Physiol. Sinnesorg., vol. 43, 1906, pp. 268–289 and 374–422.
4. YOUNG, L. R.: Measuring Eye Movements. Am. J. Med. Electron., vol. 2, 1963, pp. 300–307.
5. ASCHAN, G.; BERGSTEDT, M.; AND STAHLE, J.: Nystagmography: Recordings of Nystagmus in Clinical Neuro-otological Examinations. Acta Oto-Laryngol., suppl. 129, 1956, pp. 1–103.
6. FLUUR, E.; AND ERIKSSON, L.: Nystagmographic Recording of Vertical Eye Movements. Acta Oto-Laryngol., vol. 53, 1961, pp. 486–492.
7. FLUUR, E.; AND MENDEL, L.: Recording of Vertical Rotatory Nystagmus. Proc. Oto-Rhino-Laryngol., vol. 25, 1963, pp. 319–323.
8. DOESSCHATE, G. TEN; AND DOESSCHATE, J. TEN: The Influence of the State of Adaptation on the Resting Potential of the Human Eye. Ophthalmologica, vol. 132, 1956, pp. 308–320.
9. DOESSCHATE, G. TEN; AND DOESSCHATE, J. TEN: The Influence of the State of Adaptation on the Resting Potential of the Human Eye (2nd Communication). Ophthalmologica, vol. 134, 1957, pp. 183–193.
10. BYFORD, G. H.: Anomalies of the Human Corneoretinal Potential. FPRC Report 1223 Flying Personnel Research Committee, Ministry of Defence (Air), London, 1964.
11. FEARING, F. S.; AND MOWRER, O. H.: The Effect of General Anesthesia Upon the Experimental Reduction of Vestibular Nystagmus. J. Gen. Psychol., vol. 11, 1934, pp. 133–144.
12. MOWRER, O. H.: Influence of "Excitement" on the Duration of Post Rotational Nystagmus. Arch. Otolaryngol., vol. 19, 1934, pp. 46–54.
13. WENDT, G. R.: An Interpretation of Inhibition of Conditioned Reflexes as Competition Between Reaction Systems. Psychol. Rev., vol. 43, 1936, pp. 258–281.
14. WENDT, G. R.: Vestibular Functions. Handbook of Experimental Psychology, S. S. Stevens, ed., Wiley, 1951, pp. 1191–1223.
15. HOOD, J. D.; AND PFALTZ, C. R.: Observations Upon the Effects of Repeated Stimulation Upon Rotational and Caloric Nystagmus. J. Physiol., London, vol. 124, 1954, pp. 130–144.
16. CRAMPTON, G. H.; AND SCHWAM, W. J.: Effects of Arousal Reaction on Nystagmus Habituation in the Cat. Am. J. Physiol., vol. 200, 1961, pp. 29–33.
17. CRAMPTON, G. H.: Habituation of Ocular Nystagmus of Vestibular Origin. The Oculomotor System, M. B. Bender, ed., Harper & Row, 1964, pp. 332–346.
Ophthalmologica, vol. 134, 1957, pp. 183–193.
18. DOWD, P. J.: Responses of the Habituated Vestibulo-ocular Reflex Arc to Drug Stress. SAM–TDR–64–72, USAF School of Aerospace Medicine, Brooks Air Force Base, Texas, 1964.
19. CENACCHI, V.; FENU, G.; AND GABRIELLI, L.: L'Azoine dell' Amfetamina Sulla Risposta Nistagmica da Stimoli Rotacceleratori. Studio Sperimentale Condotta sul Coniglio. Bull. Soc. Ital. Biol. Sper., vol. 41, 1964, pp. 527–530.
20. WOLFE, J, W.: Evidence for Control of Nystagmic Habituation by Folium-Tuber Vermis and Fastigial Nuclei. Acta Oto-Laryngol., suppl. 231, 1968, pp. 1–48.
21. JONGKEES, L. B. W.; AND PHILIPSZOON, A. J.: Some Nystagmographical Methods For the Investigation of the Effect of Drugs Upon the Labyrinth. Acta Physiol. Pharmacol. Neerl., vol. 9, 1960, pp. 240–275.
22. JONGKEES, L. B. W.; AND PHILIPSZOON, A. J.: Electronystagmography. Acta Oto-Laryngol., suppl. 189, 1964, pp. 1–111.
23. BERGMANN, F.; AND GUTMAN, J.: Effect of Largactil on Various Forms of Nystagmus. Psychopharmacol. Serv. Cent. Bull., vol. 2, 1962, pp. 57–58.
24. GRIFFITH, C. R.: The Organic Effects of Repeated Bodily Rotation. J. Exptl. Psychol., vol. 3, 1920, pp. 15–47.
25. COLLINS, W. E.; CRAMPTON, G. H.; AND POSNER, J. B.: Effects of Mental Activity Upon Vestibular Nystagmus and the Electroencephalogram. Nature, vol. 190, 1961, pp. 194–195.
26. BÉKÉSY, G. VON: Subjective Cupulometry. Arch. Oto-Laryngol., vol. 61, 1955, pp. 16–28.
27. GROEN, J. J.; AND JONGKEES, L. B. W.: The Turning Test with Small Regulable Stimuli. IV. The Cupulogram Obtained by Subjective Angle Estimation. J. Laryngol., vol. 62, 1948, pp. 236–240.
28. MAHONEY, J. L.; HARLAN, W. L.; AND BICKFORD, R. G.: Visual and Other Factors Influencing Caloric Nystagmus in Normal Subjects. Arch. Otolaryngol., vol. 66, 1957, pp. 46–53.
29. LIDVALL, H. F.: Vertigo and Nystagmus Responses to Caloric Stimuli Repeated at Short Intervals. Acta Oto-Laryngol., vol. 53, 1961, pp. 33–44.
30. GUEDRY, F. E.; COLLINS, W. E.; AND SHEFFEY, P. L.: Perceptual and Oculomotor Reactions to Interacting

Visual and Vestibular Stimulation. Percept. Mot. Skills, vol. 12, 1961, pp. 307–324.

31. Guedry, F. E.; and Lauver, L. S.: Vestibular Reactions During Prolonged Constant Angular Acceleration. J. Appl. Physiol., vol. 16, 1961, pp. 215–220.
32. Collins, W. E.; and Guedry, F. E.: Arousal Effects and Nystagmus During Prolonged Constant Angular Acceleration. Acta Oto-Laryngol., vol. 54, 1962, pp. 349–362.
33. Collins, W. E.: Effects of Mental Set Upon Vestibular Nystagmus. J. Exptl. Psychol., vol. 63, 1962, pp. 191–197.
34. Collins, W. E.: Manipulation of Arousal and its Effects upon Human Vestibular Nystagmus Induced by Caloric Irrigation and Angular Accelerations. Aerospace Med., vol. 34, 1963, pp. 124–129.
35. Collins, W. E.: Task-control of Arousal and the Effects of Repeated Unidirectional Angular Acceleration on Human Vestibular Responses. Acta Oto-Laryngol., suppl. 190, 1964, pp. 1–34.
36. Collins, W. E.: Subjective Responses and Nystagmus Following Repeated Unilateral Caloric Stimulation. Ann. Otol., vol. 74, 1965, pp. 1034–1054.
37. Collins, W. E.; and Posner, J. B.: Electroencephalogram Alpha Activity During Mild Vestibular Stimulation. Nature, vol. 199, 1963, pp. 933–934.
38. Lidvall, H. F.: Specific and Nonspecific Traits of Habituation in Nystagmus Responses to Caloric Stimuli. Acta Oto-Laryngol., vol. 55, 1962, pp. 315–325.
39. Barber, H.; and Wright, G.: Release of Nystagmus Suppression in Clinical Electronystagmography. Laryngoscope, vol. 77, 1967, pp. 1016–1027.
40. Benson, A. J.; Goorney, A. B.; and Reason, J. T.: The Effect of Instructions Upon Postrotational Sensations and Nystagmus. Acta Oto-Laryngol., vol. 62, 1966, pp. 442–452.
41. Brand, J. J.: The Effect of Repetition on Measurements of Postrotational Turning Sensation and Nystagmus in Man. Quart. J. Exptl. Physiol., vol. 53, 1968, pp. 312–326.
42. Dowd, P. J.: Factors Affecting Vestibular Nystagmus in Coriolis Stimulation. Acta Oto-Laryngol., vol. 61, 1966, pp. 228–236.
43. Gillingham, K. K.: Mental Arithmetic During Electronystagmographic Testing. Ann. Otol., vol. 78, 1969, pp. 575–586.
44. Marshall, J. E.; and Brown, J. H.: Visual-Arousal Interaction and Specificity of Nystagmic Habituation. USAMRL Report 688, U. S. Army Medical Research Laboratory, Fort Knox, Kentucky, 1966.
45. Torok, N.: The Effects of Arousal Upon Vestibular Nystagmus. Adv. Oto-Rhino-Laryngol., vol. 17, 1970, pp. 76–89.
46. Collins, W. E.; Guedry, F. E.; and Posner, J. B.: Control of Caloric Nystagmus by Manipulating Arousal and Visual Fixation Distance. Ann. Otol., vol. 71, 1962, pp. 187–202.
47. Benson, A. J.; and Brand, J. J.: Some Effects of l-Hyoscine Hydrobromide on Postrotatory Sensation and Nystagmus in Man. Quart. J. Exptl. Physiol., vol. 53, 1968, pp. 296–311.
48. Collins, W. E.; and Poe, R. H.: Amphetamine, Arousal, and Human Vestibular Nystagmus. J. Pharmacol. Exptl. Therap., vol. 138, 1862, pp. 120–125.
49. Schroeder, D. J.: Influence of Alcohol on Vestibular Responses to Angular Acceleration. Aerospace Med. (in press), 1971.
50. Schroeder, D. J.: Some Effects of Alcohol on Nystagmus and "Vertigo" During Caloric and Optokinetic Stimulation. Ann. Otol. (in press), 1971.
51. Harris, C. S.; Guedry, F. E.; and Graybiel, A.: Positional Alcohol Nystagmus in Relation to Labyrinthine Function. NSAM–839. Naval School of Aviation Medicine, Pensacola, Fla., 1962.
52. Aschan, G.; Finer, B. L.; and Habrarth, K. E.: The Influence of Hypnotic Suggestion on Vestibular Nystagmus. Acta Oto-Laryngol., vol. 55, 1962, pp. 97–110.
53. 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. Naval School of Aviation Medicine, Pensacola, Fla., 1964.
54. Correia, M. J.; and Guedry, F. E.: Modification of Vestibular Responses As a Function of Rate of Rotation About an Earth-Horizontal Axis. Acta Oto-Laryngol., vol. 62, 1966, pp. 297–308.
55. Guedry, F. E.: Visual Control of Habituation to Complex Vestibular Stimulation in Man. Acta Oto-Laryngol., vol. 58, 1964, pp. 377–389.
56. Angyal, A.; and Blackman, N.: Vestibular Reactivity in Schizophrenia. Arch. Neurol. Psychiat., vol. 44, 1940, pp. 611–620.
57. Claude, H.; Baruk, H.; and Aubry, M.: Contribution a l'Étude de la Démence Précoce Catatonique: Inexcitabilité Labyrinthique au Cours de la Catatonie. Rev. Neurol., vol. 1, 1927, pp. 976–980.
58. Colbert, E. G.; Koegler, R. R.; and Markham, C. H.: Vestibular Dysfunction in Childhood Schizophrenia. Arch. Gen. Psychiat., vol. 1, 1959, pp. 600–617.
59. Pollack, M.; and Krieger, H. P.: Oculomotor and Postural Patterns in Schizophrenic Children. Arch. Neurol. Psychiat., vol. 79, 1958, pp. 720–726.
60. King, B. G.: The Influence of Repeated Rotations on Decerebrate and on Blinded Squabs. J. Comp. Psychol., vol. 6, 1926, pp. 399–421.
61. Huddleston, O. L.: The Influence of Retinal Stimuli Upon Labyrinthine Head Nystagmus of Pigeons. Am. J. Physiol., vol. 95, 1930, pp. 465–472.
62. Fearing, F. S.: Postrotational Head Nystagmus in Adult Pigeons. J. Comp. Psychol., vol. 6, 1926, pp. 115–131.
63. Fearing, F. S.: The Retention of the Effects of Repeated Elicitation of the Postrotational Nystagmus in Pigeons. I. The Retention of the Effects of "Massed"

Stimulation. J. Comp. Psychol., vol. 30, 1940, pp. 31–40.

64. Fearing, F. S.: The Retention of the Effects of Repeated Elicitation of the Postrotational Nystagmus in Pigeons. II. The Retention of the Effects of "Distributed" Stimulation. J. Comp. Psychol., vol. 31, 1941, pp. 47–56.
65. Brown, J. H.: Acquisition and Retention of Nystagmic Habituation in Cats With Distributed Acceleration Experience. J. Comp. Physiol. Psychol., vol. 60, 1965, pp. 340–343.
66. Lumpkin, R. C.: Postrotation Nystagmus in the Rabbit With Head Fixed and Head Free. J. Comp. Psychol., vol. 7, 1927, pp. 181–185.
67. Maxwell, S. S.: Burke, U. L.; and Reston, C.: The Effect of Repeated Rotation on the Duration of After-Nystagmus in the Rabbit. Am. J. Physiol., vol. 58, 1922, pp. 432–438.
68. Mowrer, O. H.: The Modification of Vestibular Nystagmus by Means of Repeated Elicitation. Comp. Psychol. Monogr., vol. 9, no. 45, 1934, pp. 1–48.
69. Mowrer, O. H.: Some Neglected Factors Which Influence the Duration of Postrotational Nystagmus. Acta Oto-Laryngol., vol. 22, 1935, pp. 1–23.
70. Halstead, W.: The Effects of Cerebellar Lesions Upon Habituation of Postrotational Nystagmus. Comp. Psychol. Monogr., vol. 12, 1935, pp. 1–130.
71. Halstead, W.; Yacorzynski, G.: and Fearing, F.: Further Evidence of Cerebellar Influence in the Habituation of After-Nystagmus in Pigeons. Am. J. Physiol., vol. 120, 1937, pp. 350–355.
72. Griffith, C. R.: The Effect Upon the White Rat of Continued Bodily Rotation. Am. Naturalist, vol. 54, 1920, pp. 524–534.
73. Fukuda, T.; Hinoki, M.; and Tokita, T.: Static and Kinetic Labyrinthine Reflex: Functional Development of Labyrinthine Function With Rotatory Training. Acta Oto-Laryngol., vol. 49, 1958, pp. 467–477.
74. Maxwell, S. S.: The Effect of Habituation on the Rotation Nystagmus as Compared With the After-Nystagmus in the Rabbit. Am. J. Physiol., vol. 68, 1924, pp. 125–126.
75. Pilz, G. F.: On the Relation of the After-Nystagmus to Rotation Nystagmus. I. Direct Effects of Rotations. Am. J. Physiol., vol. 77, 1926, pp. 428–442.
76. Pilz, G. F.: On the Relation of After-Nystagmus to Rotation Nystagmus. II. Modifying Influences. Am. J. Physiol., vol. 77, 1926, pp. 443–458.
77. Henderson, J. W.: Optokinetic and Other Factors Modifying Vestibular Nystagmus. Arch. Ophthal., vol. 37, 1947, pp. 459–471.
78. Maxwell, S. S.; and Pilz, G. F.: On the Relation of Labyrinthine and Retinal Excitations in the Rabbit. Am. J. Physiol., vol. 70, 1924, pp. 118–121.
79. Collins, W. E.: Primary, Secondary, and Caloric Nystagmus of the Cat Following Habituation to Rotation. J. Comp. Physiol. Psychol., vol. 57, 1964, pp. 417–421.
80. Collins, W. E.: Nystagmus Responses of the Cat to Rotation and to Directionally Equivalent and Non-equivalent Stimuli After Unilateral Caloric Habituation. Acta Oto-Laryngol., vol. 58, 1964, pp. 247–258.
81. Collins, W. E.: Influence of Head Position on the Habituation of Vertical Vestibular Nystagmus in the Cat. J. Comp. Physiol. Psychol., vol. 67, 1969, pp. 156–159.
82. Mertens, R. A.; and Collins, W. E.: Unilateral Caloric Habituation of Nystagmus in the Cat: Effects on Rotational and Bilateral Caloric Responses. Acta Oto-Laryngol., vol. 64, 1967, pp. 281–297.
83. Prince, A. L.: Observations on the Physiology of the Otic Labyrinth. The Influence of Prolonged Rotation on the Duration of Postrotatory Nystagmus. Proc. Soc. Exptl. Biol. Med., vol. 17, 1920, pp. 202–203.
84. Henriksson, N. G.; Fernández, C.; and Kohut, R.: The Caloric Test in the Cat. Acta Oto-Laryngol., vol. 53, 1961, pp. 21–32.
85. Collins, W. E.; and Guedry, F. E.: Duration of Angular Acceleration and Ocular Nystagmus from Cat and Man. I. Responses From the Lateral and the Vertical Canals to Two Stimulus Durations. Acta Oto-Laryngol., vol. 64, 1967, pp. 373–387.
86. Aschan, G.; and Bergstedt, M.: The Genesis of Secondary Nystagmus Induced by Vestibular Stimuli. Acta Soc. Med. Upsala, vol. 60, 1955, pp. 113–122.
87. Guedry, F. E.; and Collins, W. E.: Duration of Angular Acceleration and Ocular Nystagmus From Cat and Man. II. Responses From the Lateral Canals to Varied Stimulus Durations. Acta Oto-Laryngol., vol. 65, 1968, pp. 257–269.
88. Crampton, G. H.: Directional Imbalance of Vestibular Nystagmus in Cat Following Repeated Unidirectional Angular Acceleration. Acta Oto-Laryngol., vol. 55, 1962, pp. 41–48.
89. Brown, J. H.; and Marshall, J. E.: Drug Control of Arousal and Nystagmic Habituation in the Cat. Acta Oto-Laryngol., vol. 64, 1967, pp. 345–352.
90. Collins, W. E.; and Updegraff, B. P.: A Comparison of Nystagmus Habituation in the Cat and the Dog. Acta Oto-Laryngol., vol. 62, 1966, pp. 19–26.
91. Winget, C. M.; and Smith, A. H.: Quantitative Measurement of the Labyrinthine Function in the Fowl by Nystagmography. J. Appl. Physiol., vol. 17, 1962, pp. 712–718.
92. Winget, C. M.; Smith, A. H.; and Kelly, C. F.: Effects of Chronic Acceleration on Induced Nystagmus in the Fowl. J. Appl. Physiol., vol. 17, 1962, pp. 709–711.
93. Crampton, G. H.: Effects of Visual Experience on Vestibular Nystagmus Habituation in the Cat. Acta Oto-Laryngol., vol. 55, 1962, pp. 516–526.
94. Crampton, G. H.; and Brown, J. H.: Repeated Vertical Semicircular Canal Stimulation Does Not Habituate Horizontal Nystagmus in Cat. Acta Oto-Laryngol., vol. 58, 1964, pp. 441–448.

95. Collins, W. E.; and Updegraff, B. P.: Possible Interactions Between Gravireceptors and Semicircular Canals in the Habituation of Vertical Nystagmus in Parrots. Acta Oto-Laryngol., vol. 69, 1970, pp. 257–265.

96. Collins, W. E.: Effects on Vestibular Habituation of Interrupting Nystagmic Responses With Opposing Stimuli. J. Comp. Physiol. Psychol., vol. 64, 1967, pp. 308–312.

97. Dunlap, K.: Adaptation of Nystagmus to Repeated Caloric Stimulation in Rabbits. J. Comp. Psychol., vol. 5, 1925, pp. 485–493.

98. Henriksson, N. G.; Kohut, R.; and Fernández, C.: Studies on Habituation of Vestibular Reflexes. I. Effect of Repetitive Caloric Test. Acta Oto-Laryngol., vol. 53, 1961, pp. 333–349.

99. Fernández, C.; and Schmid, R.: Studies on Habituation of Vestibular Reflexes. II. Effect of Caloric Stimulation in Decorticated Cats. Ann. Otol., vol. 71, 1962, pp. 299–321.

100. Fernández, C.; and Schmid, R.: Studies in Habituation of Vestibular Reflexes: III. A Revision. Laryngoscope, vol. 72, 1962, pp. 939–953.

101. Proctor, L. R.: and Fernández, C.: Studies on Habituation of Vestibular Reflexes. IV. Effect of Caloric Stimulation in Blindfolded Cats. Acta Oto-Laryngol., vol. 56, 1963, pp. 500–508.

102. Capps, M. J.: and Collins, W. E.: Effects of Bilateral Caloric Habituation on Vestibular Nystagmus in the Cat. Acta Oto-Laryngol., vol. 59, 1965, pp. 511–530.

103. Collins, W. E.: Some Methodological Considerations in Caloric Tests of Vestibular Function. Third Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–152, 1968, pp. 393–401.

104. Collins, W. E.: Effects of "Double Irrigations" on the Caloric Nystagmus of the Cat. Acta Oto-Laryngol., vol. 59, 1965, pp. 45–54.

105. Riesco-Mac-Clure, J. S.: Caloric Tests: Methods and Interpretation. Ann. Otol., vol. 73, 1964, pp. 829–837.

106. Fukuda, T.; Tokita, T.; Hinoki, M.; and Kitahara, M.: The Physiology of Training the Functional Development of the Labyrinthine Function Through the Daily Repetition of Rotary, Centrifugal, Seesaw and Pendulum-like motions. Acta Oto-Laryngol., vol. 56, 1963, pp. 240–250.

107. Griffith, C. R.: A Note on the Persistence of the "Practice Effect" in Rotation Experiments. J. Comp. Psychol., vol. 4, 1924, pp. 137–149.

108. Griffith, C. R.: The Cumulative Effects of Rotational Increments. Illinois State Acad. Sci. Trans., vol. 13, 1920, pp. 122–134.

109. Griffith, C. R.: An Experimental Study of Dizziness. J. Exptl. Psychol., vol. 3, 1920, pp. 89–125.

110. Holsopple, J. Q.: Factors Affecting the Duration of Postrotation Nystagmus. J. Comp. Psychol., vol. 3, 1923, pp. 283–304.

111. Holsopple, J. Q.: An Explanation for the Unequal Reductions in Postrotation Nystagmus Following Rotation Practice in Only One Direction. J. Comp. Psychol., vol. 4, 1924, pp. 185–193.

112. Brown, J. H.: Interacting Vestibular Stimuli and Nystagmic Habituation. Acta Oto-Laryngol., vol. 62, 1966, pp. 341–350.

113. Holsopple, J. Q.: Some Effects of Duration and Direction of Rotation on Postrotation Nystagmus. J. Comp. Psychol., vol. 3, 1923, pp. 85–100.

114. Dorcus, R. M.: A Comparison of Postrotation Nystagmus Duration For Repeated Stimulation With the Head Fixed and Free. J. Comp. Psychol., vol. 7, 1927, pp. 177–179.

115. Mowrer, O. H.: The Influence of Vision During Bodily Rotation Upon the Duration of Postrotational Vestibular Nystagmus. Acta Oto-Laryngol, vol. 25, 1937, pp. 351–364.

116. Dodge, R.: Habituation to Rotation. J. Exptl. Psychol., vol. 6, 1923, pp. 1–35.

117. Aschan, G.; Nylen, C. O.; Stahle, J.; and Wersäll, R.: The Rotation Test: Cupulometric Data from 320 Normals. Acta Oto-Laryngol., vol. 42, 1952, pp. 451–459.

118. Suzuki, J.; and Totsuka, G.: Postrotatory Nystagmus: Modifications Observed in Experiments With Repeated Rotatory Stimulation. Acta Oto-Laryngol., vol. 51, 1960, pp. 570–578.

119. Fluur, E.; and Mendel, L.: Habituation, Efference and Vestibular Interplay. III. Unidirectional Rotatory Habituation. Acta Oto-Laryngol., vol. 57, 1964, pp. 81–88.

120. Pfaltz, C. R.; and Arx, S. V.: Zur Wirkung Wiederholter Thermischer und Rotatorischer Reize auf das Normale Vestibuläre Endorgan. Acta Oto-Laryngol., vol. 63, 1967, pp. 191–207.

121. Wendt, G. R.: The Nature of Adaptation to Oscillatory Rotation. The Role of the Vestibular Organs in the Exploration of Space, NASA SP–77, 1965, pp. 133–139.

122. Collins, W. E.: Special Effects of Brief Periods of Visual Fixation on Nystagmus and Sensations of Turning. Aerospace Med., vol. 39, 1968, pp. 257–266.

123. Torok, N.: Nystagmus Frequency versus Slow-Phase Velocity in Rotatory and Caloric Nystagmus. Ann. Otol., vol. 78, 1969, pp. 625–639.

124. Johnson, D. D.; and Torok, N.: Habituation of Nystagmus and Sensation of Motion After Rotation. Acta Oto-Laryngol., vol. 69, 1970, pp. 206–215.

125. McCabe, B. F.: Vestibular Suppression in Figure Skaters. Trans. Am. Acad. Ophthal. Otolaryngol., vol. 64, 1960, pp. 264–268.

126. Collins, W. E.: Vestibular Responses From Figure Skaters. Aerospace Med., vol. 37, 1966, pp. 1098–1104.

127. Fukuda, T.; Tokita, T.; Aokia, S.; Watanabe, T.; Hishida, K.; Tashiro, K.; Miyata, H.; and Mazuoka, U.: Study on Nystagmus During Voluntary

Acts: Observation of Rotation in Ballet. J. Oto-Rhino-Laryngol. Soc. Japan (suppl.), vol. 70, 1967, pp. 11–12.

128. Osterhammel, P.; Terkildsen, K.; and Zilstorff, K.: Vestibular Habituation in Ballet Dancers. Acta Oto-Laryngol., vol. 66, 1968, pp. 221–228.

129. Dix, M. R.: and Hood, J. D.: Observations Upon the Nervous Mechanism of Vestibular Habituation. Acta Oto-Laryngol., vol. 44, 1969, pp. 310–318.

130. Tschiassny, K.: Perrotatory Nystagmus in the Ballet Dancer, Pigeon, and Blind Person. Ann. Otol., vol. 66, 1957, pp. 641–648.

131. Brown, J. H.; and Crampton, G. H.: Concomitant Visual Stimulation Does Not Alter Habituation of Nystagmic, Oculogyral or Psychophysical Responses to Angular Acceleration. Acta Oto-Laryngol., vol. 61, 1961, pp. 80–91.

132. Guedry, F. E.: Psychophysiological Studies of Vestibular Function. *Contributions to Sensory Physiology*, W. D. Neff, ed., Academic Press, 1965, pp. 63–135.

133. Aschan, G.: Response to Rotatory Stimuli in Fighter Pilots. Acta Oto-Laryngol., suppl. 116, 1954, pp. 24–31.

134. Aschan, G.: Habituation to Repeated Rotatory Stimuli (Cupulometry) and the Effect of Antinausea Drugs and Alcohol on the Results. Acta Oto-Laryngol., vol. 64, 1967, pp. 95–106.

135. Caporale, R.; and Camarda, V.: La Funzionalita Vestibolare di Alcuni Piloti di Pattuglia Acrobatica. Riv. Med. Aeron., vol. 21, 1958, pp. 12–36.

136. Benson, A. J.; and Bodin, M. A.: Effect of Orientation to the Gravitational Vertical on Nystagmus Following Rotation About a Horizontal Axis. Acta Oto-Laryngol., vol. 61, 1966, pp. 517–526.

137. Benson, A. J.: and Bodin, M. A.: Interaction of Linear and Angular Accelerations on Vestibular Receptors in Man. Aerospace Med., vol. 37, 1966, pp. 144–154.

138. Guedry, F. E.: Orientation of the Rotation Axis Relative to Gravity: Its Influence on Nystagmus and the Sensation of Rotation. Acta Oto-Laryngol., vol. 60, 1965, pp. 30–48.

139. Benson, A. J.; and Whiteside, T. C. D.: The Effect of Linear Acceleration on the Response to Angular Acceleration in Man. J. Physiol., London, vol. 156, 1961, pp. 6P–7P.

140. Lansberg, M. P.; Guedry, F. E.; and Graybiel, A.: Effect of Changing Resultant Linear Acceleration Relative to the Subject on Nystagmus Generated by Angular Acceleration. Aerospace Med., vol. 36, 1965, pp. 456–460.

141. Bergstedt, M.: The Effect of Gravitational Force on the Vestibular Caloric Test. Acta Oto-Laryngol., vol. 53, 1961, pp. 551–562.

142. Bergstedt, M.: Studies of Positional Nystagmus in the Human Centrifuge. Acta Oto-Laryngol., suppl. 165, 1961, pp. 1–144.

143. Orlov, I. V.: Thresholds of Caloric Nystagmus During Rotation at Constant Velocity. Proc. Acad. Sci. USSR, vol. 156, 1964, pp. 972–975.

144. Yuganov, Y. M.: The Problem of Functional Characteristics and Interaction of the Otolithic and Cupular Portions of the Vestibular Apparatus Under Conditions of Altered Gravity. Problems of Space Biology, vol. 4, N. M. Sisakyan, ed., NASA TT F–368, 1966, pp. 48–63.

145. 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., 1964.

146. Owada, K.; and Okubu, K.: The Otolithic Reaction on Nystagmus by Caloric Stimulations. Japan Society of Vestibular Research: Contribution to the Vestibular Physiology and Vestibular Test, Acta Oto-Laryngol., suppl. 179, 1963, pp. 1–6.

147. Niven, J. I.: Hixson, W. C.: and Correia, M. J.: Elicitation of Horizontal Nystagmus by Periodic Linear Acceleration. Acta Oto-Laryngol., vol. 62, 1966, pp. 429–441.

148. Hixson, W. C.; and Niven, J. I.: A Bioinstrumentation Control Center For the Human Disorientation Device. NSAM–848. Naval School of Aviation Medicine, Pensacola, Fla., 1963.

149. Guedry, F. E.; and Graybiel, A.: Compensatory Nystagmus Conditioned During Adaptation to Living in a Rotating Room, J. Appl. Physiol., vol. 17, 1962, pp. 398–404.

150. Guedry, F. E.; and Montague, E. K.: Quantitative Evaluation of the Vestibular Coriolis Reaction. Aerospace Med., vol. 32, 1961, pp. 487–500.

151. Guedry, F. E.; Collins, W. E.; and Graybiel, A.: Vestibular Habituation During Repetitive Complex Stimulation: A Study of Transfer Effects. J. Appl. Physiol., vol. 19, 1964, pp. 1005–1015.

152. Guedry, F. E.; Graybiel, A.; and Collins, W. E.: Reduction of Nystagmus and Disorientation in Human Subjects. Aerospace Med., vol. 33, 1962, pp. 1356–1360.

153. Dowd, P. J.; and Cramer, R. L.: Habituation Transference in Coriolis Acceleration. Aerospace Med., vol. 38, 1967, pp. 1103–1107.

154. Guedry, F. E.: Habituation to Complex Vestibular Stimulation in Man: Transfer and Retention of Effects From 12 Days of Rotation at 10 rpm. Percept. Mot. Skills, Monogr. suppl. 1–V21, 1965, pp. 459–481.

155. Fitzgerald, G.; and Hallpike, C. S.: Studies in Human Vestibular Function. I. Observations on the Directional Preponderance ("Nystagmusbereitschaft") of Caloric Nystagmus Resulting From Cerebral Lesions. Brain, vol. 65, 1942, pp. 115–137.

156. Loch, W. E.; and Haines, H. L.: Habituation to Caloric Vestibular Stimulation. Laryngoscope, vol. 56, 1946, pp. 43–45.

157. STAHLE, J.: Electronystagmography in the Caloric Test. Acta Soc. Med. Upsala., vol. 61, 1957, pp. 307–332.

158. LIDVALL, H. F.: Vertigo and Nystagmus Responses to Caloric Stimuli Repeated at Short and Long Intervals. Acta Oto-Laryngol., vol. 53, 1961, pp. 507–518.

159. LIDVALL, H. F.: Mechanisms of Motion Sickness as Reflected in the Vertigo and Nystagmus Responses to Repeated Caloric Stimuli. Acta Oto-Laryngol., vol. 55, 1962, pp. 527–536.

160. FLUUR, E.; AND MENDEL, L.: Habituation, Efference and Vestibular Interplay. I. Monaural Caloric Habituation. Acta Oto-Laryngol., vol. 55, 1962, pp. 65–80.

161. FLUUR, E.; AND MENDEL, L.: Habituation, Efference and Vestibular Interplay. II. Combined Caloric Habituation. Acta Oto-Laryngol., vol. 55, 1962, pp. 136–144.

162. 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 Oto-Laryngol., vol. 56, 1963, pp. 663–674.

163. FORSSMAN, B.: Studies on Habituation of Vestibular Reflexes. VII. Habituation in Light of Calorically Induced Nystagmus, Laterotorsion and Vertigo in Man. Acta Oto-Laryngol., vol. 57, 1964, pp. 163–176.

164. HENRIKSSON, N. G.; DOLOWITZ, D. A.; AND FORSSMAN, B.: Studies of Cristospinal Reflexes (Laterotorsion): A Method For Objective Recording of Cristospinal Reflexes. Acta Oto-Laryngol., vol. 55, 1962, pp. 33–40.

165. COLLINS, W. E.: SCHROEDER, D. J.; AND MERTENS, R. A.: Relative Specificity of Nystagmus Reduction to Caloric Stimuli in Humans. Preprints of the Annual Meeting of the Aerospace Medical Association, 1970, p. 182.

166. HALLPIKE, C. S.: AND HOOD, J. D.: Fatigue and Adaptation of the Cupular Mechanism of the Human Horizontal Semicircular Canal: An Experimental Investigation. Proc. Roy. Soc. London, vol. 141B, 1953, pp. 542–561.

167. MEDA, E.: Effetti di Repetuti Eccitamenti Rotatori dell Apparato Vestibulare sue Fenmonei Soggettivi da Combiamenta di Perizione del Capo Duranti e Dope la Rotazione. Riv. Med. Aeron., vol. 10, 1947, pp. 316–323.

168. GROEN, J. J.; AND JONGKEES, L. B. W.: The Turning Test With Small Regulable Stimuli. III. The Advantages of Cupulometria Over the Classic Technique of Bárány. J. Laryngol., vol. 62, 1948, pp. 231–235.

169. DEARNALEY, E. J.; REASON, J. T.; AND DAVIES, J. D.: The Nature and Duration of After-Sensations Following in the Cessation of Turning in a Chipmunk Aircraft. Aerospace Med., vol. 33, 1962, pp. 1224–1229.

170. PREBER, L.: Vegetative Reactions in Caloric and Rotatory Tests. Acta Oto-Laryngol., suppl. 144, 158, pp. 1–119.

171. DEWIT, G.: Seasickness (Motion Sickness): A Labyrinthological Study. Acta Oto-Laryngol., suppl. 108, 1953, pp. 1–56.

172. BENSON, A. J.: Modification of Labyrinthine After-Sensations on Shaking the Head. FPRC Report 1210. Flying Personnel Research Committee, Ministry of Defence (Air), London, 1962.

173. GRAYBIEL, A.: AND HUPP, D. I.: The Oculogyral Illusion: A Form of Apparent Motion Which May Be Observed Following Stimulation of the Semicircular Canals. J. Aviat. Med., vol. 17, 1946, pp. 3–27.

174. BYFORD, G. H.: Eye Movements and the Optogyral Illusion. Aerospace Med., vol. 34, 1963, pp. 119–123.

175. VAN DISHOECK, H. A. E.; SPOOR, A.; AND NIJHOFF, P.: The Optogyral Illusion and Its Relation to the Nystagmus of the Eyes. Acta Oto-Laryngol., vol. 44, 1954, pp. 597–607.

176. VOGELSANG, C. J.: The Perception of a Visual Object During Stimulation of the Vestibular System. Acta Oto-Laryngol., vol. 53, 1961, pp. 461–469.

177. CLARK, B.: AND STEWART, J. D.: Comparison of Sensitivity For the Perception of Bodily Rotation and the Oculogyral Illusion. Percep. & Psychophys., vol. 3, 1968, pp. 253–256.

178. BROWN, R. H.: AND GUEDRY, F. E.: Influence of Visual Stimulation on Habituation to Rotation. J. Gen. Psychol., vol. 45, 1951, pp. 151–161.

179. GUEDRY, F. E.: The Effect of Visual Stimulation on the Duration of Postrotational Apparent Motion Effects. J. Gen. Psychol., vol. 43, 1950, pp. 313–322.

180. GUEDRY, F. E.: The Transfer of Habituation to Rotation With Respect to the Directional Aspect of the Vestibular Reaction. NSAM–494. Naval School of Aviation Medicine, Pensacola, Fla., 1953.

181. GUEDRY, F. E.: The Transfer of Habituation to Rotation With Respect to the Magnitude of the Vestibular Stimulus. NSAM–493. Naval School of Aviation Medicine, Pensacola, Fla., 1953.

182. GUEDRY, F. E.: The Retention of Effects of "Massed" and "Distributed" Vestibular Stimulation as Indicated By the Duration of the Oculogyral Illusion. NSAM–492. Naval School of Aviation Medicine, Pensacola, Fla., 1953.

183. COLLINS, W. E.: Coriolis Vestibular Stimulation and the Influence of Different Visual Surrounds. Aerospace Med., vol. 39, 1968, pp. 125–130.

184. COLLINS, W. E.: Effective Approaches to Disorientation Familiarization for Aviation Personnel. FAA Office of Aviation Medicine Report No. 70–17, 1970.

185. GRAYBIEL, A.; GUEDRY, F. E.; JOHNSON, W. H.; AND KENNEDY, R.: Adaptation to Bizarre Stimulation of the Semicircular Canals as Indicated by the Oculogyral Illusion. Aerospace Med., vol. 32, 1961, pp. 321–327.

186. LIDVALL, H. F.: Neurotic Anxiety and Vestibular

Function as Reflected in the Vertigo and Nystagmus Responses to Repeated Caloric Stimuli. Acta Oto-Laryngol., vol. 56, 1963, pp. 445–456.

187. HINCHCLIFFE, R.: Subjective Magnitude of Induced Vertigo as an Index of Caloric Response. J. Laryngol., vol. 81, 1967, pp. 875–885.

188. COLLINS, W. E.; AND MERTENS, R. A.: Influence of Repeated Caloric Vestibular Stimulation on Both Caloric and Rotation-Induced Vertigo. Preprints of the Annual Meeting of the Aerospace Medical Association, 1968, p. 146.

189. MARKARYAN, S. S.: Vestibular Reactions Under the Effect of Various Angular Accelerations. Aviation and Space Medicine, V. V. Parin, ed., NASA TT F–228, 1964, pp. 305–308.

190. COLLINS, W. E.: Modification of Vestibular Nystagmus and "Vertigo" by Means of Visual Stimulation. Trans. Amer. Acad. Ophthal. Oto-Laryngol., vol. 72, 1968, pp. 962–979.

191. THORPE, W. H.: The Concepts of Learning and Their Relation to Those of Instinct. Physiological Mechanisms in Animal Behavior, Cambridge University Press, 1950, pp. 387–408.

192. RATNER, S. C.: Annelids and Learning: A Critical Review. Chemistry of Learning, W. C. Corning and S. C. Ratner, eds., Plenum Press, 1967, pp. 391–406.

Otolithic Influences on Extraocular and Intraocular Muscles*

Bo E. Gernandt
Naval Aerospace Medical Research Laboratory

SUMMARY

A new method for selective stimulation of utricular gravireceptors is described. Utricular stimulation leads to gross activation of the bulbar reticular formation where a strong interaction with evoked spino-bulbo-spinal reflex activity occurs. The utricular neurons encountered by microelectrodes in the lateral vestibular nuclei show four types of elicited activity; two of these display an increased firing rate, and two exhibit pronounced inhibitory effects. Application of a stimulus of long duration and constant intensity to the utricle has shown that rapid adaptation of the peripheral receptors is a prominent feature.

The effects of selective utricular stimulation upon eye movements, as recorded by the corneoretinal potential method, have been studied in experiments on cats and monkeys. Single air-puff stimulation of the gravireceptors of the isolated utricle evoked brisk conjugate eye deviations, demonstrating the existence of strong maculo-ocular connections. The principal aim, investigating the relationship of nystagmus to the otoliths, has been achieved, and it can be firmly stated that prolonged, constant-intensity mechanical stimulation of the utricle can evoke strong primary nystagmus, followed by a secondary nystagmus at the cessation of stimulation.

The action of utricular stimulation on ocular reflexes has been examined further, with particular attention to the effects upon autonomic outflow and intraocular muscle activity. Single air-puff stimulation evoked pupillary reactions in both cats and monkeys: constriction during the fast phase of the brisk conjugate eye movement, and dilatation during the slow phase. In monkeys, during the nystagmus evoked by a prolonged air jet directed at the utricle, the pupils showed initially a strong dilatation with superimposed rapid minor constrictions of a random frequency. After the end of stimulation and at the occurrence of a secondary nystagmus, there was a dilatation during the fast phase and a constriction during the slow phase. As the nystagmus gradually diminished, the pupils concomitantly returned to control size and ceased to fluctuate. Prolonged stimulation also gave rise to an increased stream of efferent impulses in sympathetic fibers to the eye, in synchrony with pupillary dilatation, while the activity of ciliary nerves simultaneously decreased. However, anatomical integrity of the sympathetic connections is not a prerequisite for the presence of these pupillary reactions to vestibular stimulation, since their sectioning did not change the effect.

INTRODUCTION

Because the semicircular canals are more accessible to functional studies, the majority of previous investigations on the labyrinth have been focused on rotation-controlled responses evoked by angular acceleration under terrestrial conditions. Perusal of more recent literature indicates, however, that there has been a slight shift of interest toward otolith organs since it was noticed that gravity-controlled responses may change in character during weightlessness. Some of the present experiments were undertaken to record, analyze, and classify the electrical responses to selective mechanical stimulation of the utricular gravireceptors from single lateral vestibular units.

Other experiments were undertaken in order to

* The animals used in this study were handled in accordance with the "Principles of Laboratory Animal Care" established by the Committee on the Guide for Laboratory Animal Resources, National Academy of Sciences–National Research Council.

An expanded report appears in Exptl. Neurol., vol. 26, 1970, pp. 203–219; vol. 27, 1970, pp. 90–100; vol. 30, 1971, pp. 66–76.

observe by naked eye and record, by electrical means as well as on film for a closer analysis, the presence or absence, direction, and duration of eye movements in response to selective utricular stimulation. These studies help to clarify the functional role of the utricle in controlling conjugate eye movements and evoking nystagmus.

Finally, a study of eye movements evoked by utricular stimulation revealed that pupillary action showed a rhythmic pattern of dilatations and constrictions during and immediately after a period of nystagmus (ref. 1). By putting these latter two phenomena together in a common frame of reference, we can infer that utricular stimulation has two effects, one upon the extraocular muscles and one upon the intraocular muscles.

METHODS

The experiments were performed on cats and squirrel monkeys anesthetized with alpha chloralose (30 to 40 mg/kg, iv). The left utricle was exposed, and air-puff stimuli were directed against this receptor organ according to a technique previously described (ref. 2). The posterior fossa was approached through an occipital craniotomy. After exposure of the floor of the fourth ventricle by cerebellectomy, the lateral vestibular nucleus was penetrated by stereotaxically oriented tungsten microelectrodes for single-unit extracellular analysis of activity elicited by vestibular stimulation. Other procedures, relevant to evaluation of the results, are described later.

FINDINGS

Patterns of Evoked Discharge

While testing the kind of stimulation necessary for the production of a volley of impulses, we varied stimulus strength, duration of individual stimuli, number of stimuli in the conditioning train, and frequency of stimulation. Extracellular recording from the vestibular nuclei, i.e., from second-order neurons, showed that response patterns to air-puff stimulation of the exposed utricle could be separated into four general types (fig. 1). Two of these showed an increased firing rate in response to the stimulus, regardless of the presence or absence of spontaneous firing and without any overt inhibitory action curtailing the stream of evoked impulses (A and B). For some units an extremely faint puff of air was

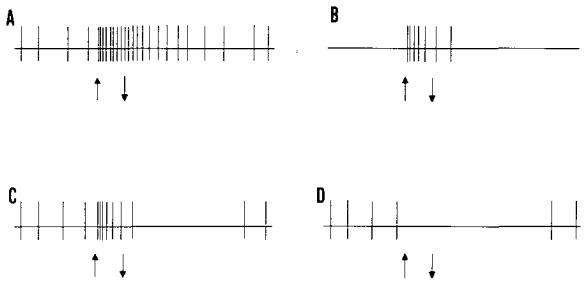


FIGURE 1.—*Types of single-unit discharges (A–D) in response to utricular stimulation, recorded from lateral vestibular nucleus. The arrows mark the period of stimulation.*

sufficient to provoke a response, while in others a stronger stimulus was necessary. Illustrated in figure 2 are threshold measurements of a type-A unit, which was the kind most commonly isolated. An air puff with a force of 0.7 dyne was, for this particular unit, of subthreshold strength (A), but an increase to 1.2 dynes evoked a threshold response (B); thus, a pure onset pattern occurred commonly and was characterized by a single discharge or a fast burst of spikes. In figure 2, C and D, the strength of stimulation was gradually increased to 1.4 and 1.7 dynes, respectively, and a corresponding augmentation of bursts was noticed. It was possible by repetitive utricular stimulation at frequencies of 5 to 10 times per second to facilitate this kind of unit discharge, as manifested by a gradually increasing

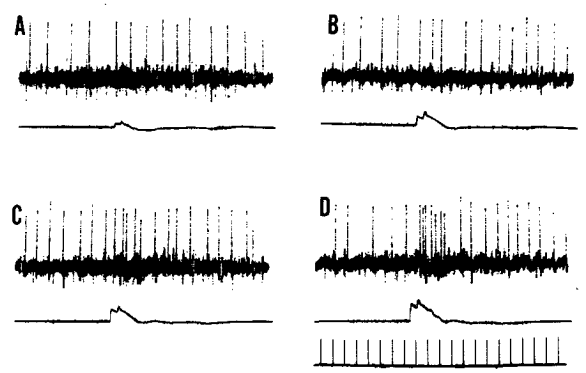


FIGURE 2.—*Single-unit recording from lateral vestibular nucleus. A: subthreshold, 0.7 dyne; B: threshold, 1.2 dynes; C: suprathreshold, 1.4 dynes; D: 1.7 dynes utricular stimulation. Time scale in 10-ms intervals.*

number of impulses in response to each stimulus. Higher frequencies of stimulation, however, caused a considerable reduction in the number of impulses of each burst. Therefore, in studying firing patterns it was necessary to evoke successive stimuli at such a rate as to avoid an undue influence of a preceding stimulus upon the pattern elicited by the next stimulus. A repetition rate of 20 to 30 air puffs per minute insured stable responses.

The other two types of responses exhibited a relatively pronounced inhibitory component. The spontaneous firing of type C of figure 1 showed an initial short burst of impulses immediately followed by a complete inhibition lasting for several hundred milliseconds or sometimes for seconds. By increasing the intensity of the air puff well above threshold value, the initial burst could be eliminated, demonstrating that the stimulus apparently sets in motion a chain of excitatory and inhibitory events; it is the final resultant of these activities that determines the drive upon the neuron. The outcome of this rivalry between two opposing influences, and thus the discharge pattern during an ongoing change in stimulus intensity, is related in some manner to the time derivatives of the stimulus, as well as to its magnitude. The fourth type displayed pure inhibition of varying lengths when threshold stimulation was applied, thus demonstrating that the stimulus may inhibit central neurons in the afferent pathway, perhaps by an inhibitory event preceding the onset of any possible excitation (fig. 1D). During the course of isolating single units within the vestibular nuclei and studying their response patterns elicited by this kind of utricular stimulation, it was always possible to classify them according to these four types of firing patterns. Thus, utricular cells located within the lateral vestibular nucleus, by their varying response patterns to mechanical stimulation of a wide receptive field, offer a running spatial integral of the activity of several first-order neurons that must converge upon them across the synaptic gap.

Adaptation of Utricular Receptors

In order to study adaptation, i.e., the gradual decline in frequency of discharge during application of a stimulus of constant intensity, units showing response patterns of type A or type B to single air-puff stimulation were isolated. When this criterion was met, the stimulation was turned off, and the presence or absence of spontaneous firing was recorded on moving film. After this level was established, an air jet with a constant force of 1 dyne was suddenly directed against the utricle. Figure 3A shows the varying patterns of firing obtained under these circumstances when units belonging to type A were isolated by the microelectrode. The different curves represent the adaptation pattern of individual units, but when many receptors are involved, as in the present case of utricular stimulation, the total amount of adaptation will be proportional to the number of receptors involved. The left-hand side of the curves illustrates the wide differences in spontaneous firing rate among units. The moment stimulation was applied, the frequency of firing showed a sharp rise, followed immediately by decline. Different shapes of the curves

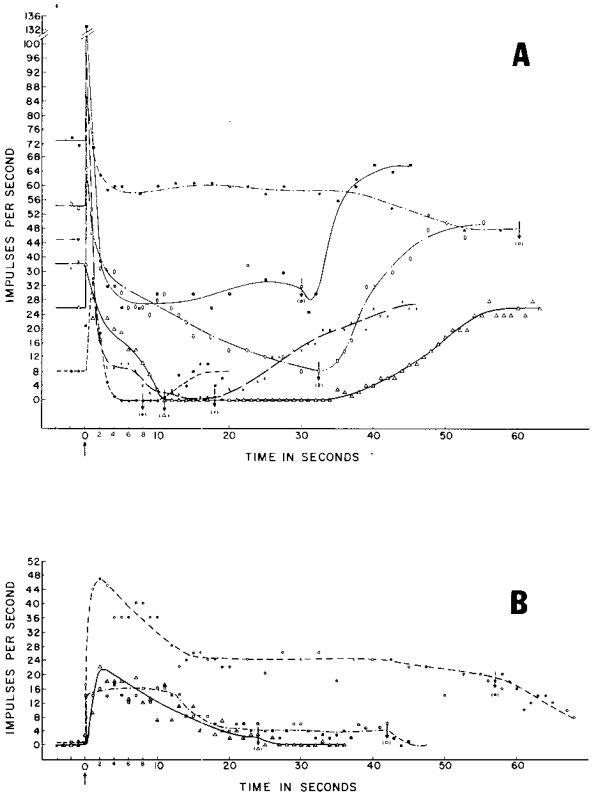


FIGURE 3.—*Adaptation patterns of individual utricular units with (A) or without (B) spontaneous firing and isolated within the lateral vestibular nucleus. Between arrows, a steady-state flow of air with a force of 1 dyne was directed against exposed utricle.*

depicting this decline show wide variations of adaptation of different units; these variances occurred from a complete cessation of firing within 3 or 4 seconds to a gradual drop to the spontaneous level of firing. When the firing of a unit had reached a steady-state level during continuous stimulation, the stimulus was turned off. At this point some of the units immediately started to return to the spontaneous firing level while others remained in the adapted state for several seconds before beginning normal activity.

Figure 3B demonstrates the effect of continuous stimulation on units belonging to type B; i.e., units that did not show any spontaneous activity but responded to air-puff stimulation with an increased firing. These units showed varying degrees of adaptation after the initial response at the onset of stimulation. One unit, represented by the upper curve, displayed only a very slight decline in firing rate in spite of continuous stimulation for almost a minute, while the other two depicted units exhibited a very rapid and almost complete adaptation.

Figure 4 illustrates the effect of continuous stimulation upon units belonging to types C and D. Since they contained strong inhibitory components, they could not be used for studying adaptation, but were included for the sake of completeness.

Utricular Nystagmus

In the next series of experiments (ref. 3) the intact heads of the animals were securely fixed in a rigid holder, and electrodes, placed on the crown at the midline of the skull and either inside the skin at the outer canthus of each eye or medially and laterally of the left eye, were connected to an ac-coupled amplifier (time constant 0.15 Hz/s) feeding a multibeam cathode-ray tube.

Figure 5 demonstrates the effect of continuous utricular stimulation upon eye movements recorded from a squirrel monkey. At the onset of a steady-state flow of air directed against the left utricle and with a pressure of 1 dyne (marked by the downward arrow), there was a sudden upward, vertical (or oblique) deflection of the eye, which was immediately followed by rotatory or horizontal nystagmus with the fast phase to the left (A). During this initial period, the nystagmus at times was wildly irregular, but settled rapidly into the fundamental or primary type. In spite of uninterrupted stimulation, the eye movements became less and less pronounced until they eventually disappeared; only a wider base line showed an increased tonus in the extraocular muscles (B and C). Failure to detect rhythmical potential changes at this stage was probably due to their true absence because of adaptation of the gravireceptors (ref. 2) or to their complete randomization which might be equivalent to continuous functional activity. Following the cessation of stimulation (marked by the upward arrow) there may have been a weak secondary nystagmus in the opposite direction.

Figure 6 further illustrates the kinds of nystagmus that can be obtained by selective utricular stimulation in three different monkeys. All showed

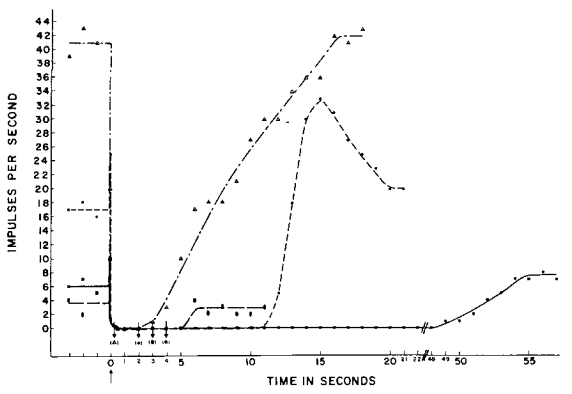


FIGURE 4.—*Adaptation patterns of individual utricular units with a pronounced inhibitory component as first displayed by single-air-puff stimulation. Between arrows, a steady-state flow of air with a force of 1 dyne was directed against exposed utricle.*

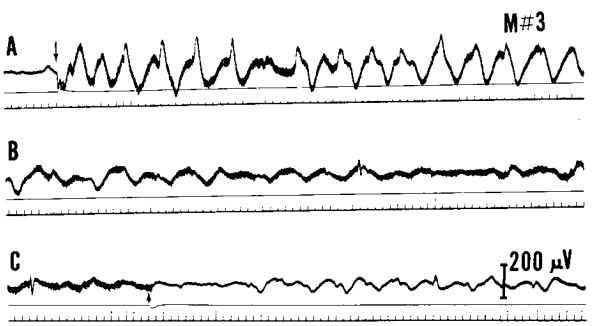


FIGURE 5.—*Continuous record of nystagmus evoked by utricular stimulation in chloralosed monkey. Onset marked by downward arrow and cessation by upward arrow. Time scale intervals are 0.1 s.*

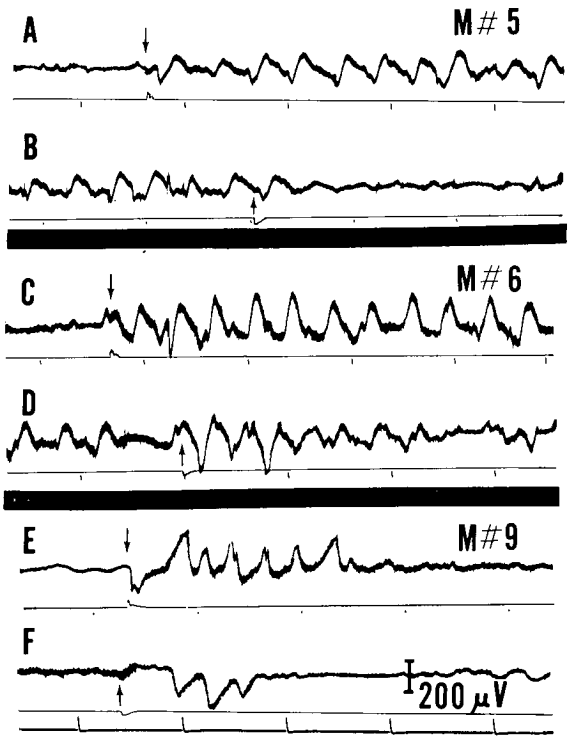


FIGURE 6.—*Nystagmus evoked by selective utricular stimulation in chloralosed monkeys (see text). Onset marked by downward arrow and cessation by upward arrow. Note secondary nystagmus in D and F. Time-scale intervals are 1 s.*

the upward, vertical (or oblique) deflection at the onset of stimulation (A, C, and E). The direction of the nystagmus, however, varied in an unpredictable way; in monkeys Nos. 5 and 6 the fast phase of a horizontal nystagmus was to the right, but in monkey No. 9 it was to the left. Thus, the general rule that each labyrinth endeavors to elicit a nystagmus in its own direction was only partially supported. The nystagmus was always of the associated type; i.e., both eyeballs showed identical movements. At the cessation of stimulation the evoked nystagmus either stopped (B) or showed a reversal in direction (D and F). This secondary nystagmus was of brief duration but sometimes very pronounced. In trying to describe the nystagmus elicited by this form of utricular stimulation, it can be stated that the amplitude of the excursions was of medium size; i.e., between 1 and 2 mm. The nystagmus was quite transitory, but of a high frequency that varied between 120 and 180 beat per minute. It was not possible to establish a reliable rule for the direction of the nystagmus.

In monkey No. 9 it was noted, by visual observation, that the pupils of the eyes constricted in synchrony with the air-puff stimuli. When an air stream of long duration was applied, the pupils showed a rhythmical pattern of constrictions and dilatations after the primary nystagmus had ceased and when the eyes remained in a fixed, deviated position.

Pupil Size Changes during Eye Movements

It is well known that the eye possesses a dual motor innervation, and thus effects induced by vestibular stimulation upon ocular reflexes are always mixed because of impulses being conducted both through somatic and autonomic pathways (ref. 1). However, our understanding of the manner in which somatic sensory and motor neurons in the central nervous system are adjusted to the load that the peripheral vestibular processes supply has been extended and clarified to a much greater degree than has been the case with the autonomic system.

Obviously, vestibular afferents must be linked to the intraocular muscles via one or both sets of autonomic fibers derived from the superior cervical or ciliary ganglia. Since strong responses to vestibular stimulation have been recorded from the cervical trunk of the sympathetic nerve (ref. 4) and because of the proximity of the superior cervical ganglion to the bulla, which has to be exposed in order to reach the vestibule enclosing the labyrinthine receptor structures, the autonomic outflow selected for recording was initially obtained from postganglionic sympathetic fibers. For some years, however, reflex regulation of pupillary size has been attributed more and more to fluctuations in the discharge through the ciliary ganglion, and the functional role of the sympathetic innervation has been progressively minimized. Thus, the dilatation and constriction may not be due to increased or decreased sympathetic firing, but to alteration in parasympathetic activity. This latter concept is supported by the present findings. In spite of being able to record a stream of sympathetic impulses during prolonged blowing directed at the utricle, the pupillary dilatations observed are still present after bilateral severing of the sympathetic fibers to the eyes.

During the nystagmic period the pupils showed a sustained dilatation with superimposed rapid minor dilatations and constrictions of apparently random frequency during the primary nystagmus. At the end of stimulation with the occurrence of a secondary nystagmus, however, these pupil size fluctuations appeared in phase with the nystagmic beats, with the most common pupillary activity being dilatation during the fast phase and constriction during the slow phase. As the secondary nystagmus gradually diminished, the pupils concomitantly constricted to control size and ceased to fluctuate. It was possible to exclude that the changes in pupillary size were due to humoral effects or variations in light intensities affecting the retina during eye movements.

From the experiments described thus far it is not possible to conclude that the efferent limb for the mediation of pupillary dilatation consists of sympathetic fibers; they only offer insight into the kind and magnitude of sympathetic firing evoked in response to utricular stimulation. The rhythmical reflex dilatations of the pupils clearly observed during stimulation may not be due to excitation of the sympathetic system, but to inhibition of the parasympathetic one. In order to determine which of these systems is primarily responsible for these pupillary fluctuations, several approaches were utilized. In acute cats, unilateral destruction of sympathetic fibers to the head by extirpation of the superior cervical ganglion and crushing of any fibers running along the common carotid artery or bilateral severing of the neck sympathetic trunks left the conjugate pupillary responses to vestibular stimulation unaltered. Superior cervical ganglionectomy of chronic preparation in a cat resulted in anisocoria, with the smaller pupil ipsilateral to the destroyed sympathetic trunk, but both dilatation and constriction could still be evoked by air puffs directed at the utricle. Obviously, anatomical integrity of the cervical sympathetic trunk is not a prerequisite for the occurrence of such intraocular responses. The role of the sympathetic nervous supply is merely to provide a general tonic background; the parasympathetic is the autonomic portion chiefly responsible for the effects described. Particularly in the cat, reflex pupillary dilatation appears to be accomplished by pure inhibition of oculomotor tone.

As a corollary to this finding, a recording electrode was placed within the oculomotor complex of cats while the utricle was stimulated with single puffs, and it was noticed that the activity thus obtained showed firing in synchrony with pupillary contraction and cessation of activity coinciding with the period of dilatation. The same firing pattern was obtained by recording from postganglionic, parasympathetic fibers from the ciliary ganglion. Acute or chronic section of the oculomotor nerve intracranially always resulted in a paralytic mydriasis, regardless of whether or not the sympathetic nerve supply to the eye was intact. In these instances no pupil size changes were seen in the denervated eye despite changes that could be elicited in the other eye by vestibular stimulation.

CONCLUSION

It may be concluded that, under the conditions of our experiments, dilatation of the pupils during nystagmus evoked by utricular stimulation is not due to excitation of the sympathetic system, but to inhibition of the parasympathetic system.

A great number of experimental studies have shown that pupillodilatation in cats is due to an inhibition of oculomotor nerve firing and that this reflex response is still obtainable after sectioning of the cervical sympathetic trunk. However, as described by Weinstein and Bender (ref. 5), there may be a difference between cats and primates, pupillodilatation in the monkey being mediated chiefly by activity in the cervical sympathetic trunk, not by parasympathetic inhibition.

Two principal links are available in the chain for connecting the vestibular organ with the paired ventromedial nucleus of Edinger-Westphal, the autonomic part of the third nerve. One is the secondary vestibular neurons ascending from the vestibular nuclei in the medial longitudinal fasciculus to the oculomotor nuclei; the other is more complex and includes multisynaptic connections of the reticular formation, the significance of which was first recognized and justly evaluated by Lorente de Nó (ref. 6).

REFERENCES

1. DeSantis, M.; and Gernandt, B. E.: The Effect of Vestibular Stimulation upon Pupillary Size. Exptl. Neurol., vol. 30, 1971, pp. 66–76.

2. Gernandt, B. E.: Discharges from Utricular Receptors

in the Cat. Exptl. Neurol., vol. 26, 1970, pp. 203–219.
3. GERNANDT, B. E.: Nystagmus Evoked by Utricular Stimulation. Exptl. Neurol., vol. 27, 1970, pp. 90–100.
4. TANG, P. C.; AND GERNANDT, B. E.: Autonomic Responses to Vestibular Stimulation. Exptl. Neurol., vol. 24, 1969, pp. 558–578.
5. WEINSTEIN, E. A.; AND BENDER, M. B.: Pupillodilator Reactions to Sciatic and Diencephalic Stimulation; a Comparative Study in Cat and Monkey. J. Neurophysiol., vol. 4, 1941, pp. 44–50.
6. LORENTE DE NÓ, R.: Vestibulo-ocular Reflex Arc. Arch. Neurol. Psychiat., Chicago, vol. 30, 1933, pp. 245–291.

SESSION VII

Chairman: **LAURENCE R. YOUNG**
Massachusetts Institute of Technology

On Visual-Vestibular Interaction

LAURENCE R. YOUNG
Massachusetts Institute of Technology

SUMMARY

Experimental evidence is presented regarding visual-vestibular interaction, and the results of three studies on the subject are briefly noted. An attempt to put together some of these observations with elementary notions of a visual-vestibular interaction program is shown in the form of a flow-chart representation of a possible model. This is a nonlinear model in which visual and vestibular influences are linearly weighted when they are in relative agreement but switch to the more "believable" one when they are in disagreement. A solution to the human space-orientation problem is depicted by a schema for optimal subjective orientation based on several sensory modalities.

SOME EXPERIMENTAL RESULTS ON VISUAL AND VESTIBULAR STIMULATION

We know that at least two major inputs may result in nystagmus or sensation of rotation; i.e., vestibular stimulation and visual presentation of a moving field. How do these two inputs interact when they are in agreement or when they are in conflict, and how do they relate to the parameters of the stimuli and the dynamics of the sensors? Consider first some experimental data collected by Guedry, Collins, and Sheffey (ref. 1). Human subjects were accelerated to a constant angular velocity about the vertical axis in a darkened room and then abruptly stopped, following the typical program for an acceleration impulse. The expected results show a sudden increase in subjective sensation of rotation (and angular velocity of nystagmus) to a maximum proportional to the prestopping velocity, and then a slow decay back toward zero, as shown in figure 1. The nystagmus and subjective sensation decays follow our adaptation model (ref. 2). Note especially the more rapid decay of the subjective sensation than of the nystagmus. The two arrows mark the beginning and end of the deceleration. It is well known that if the eyes are opened to see an arbitrary single fixation point during this period, nystagmus will stop, but nystagmus and subjective sensation will be restored upon

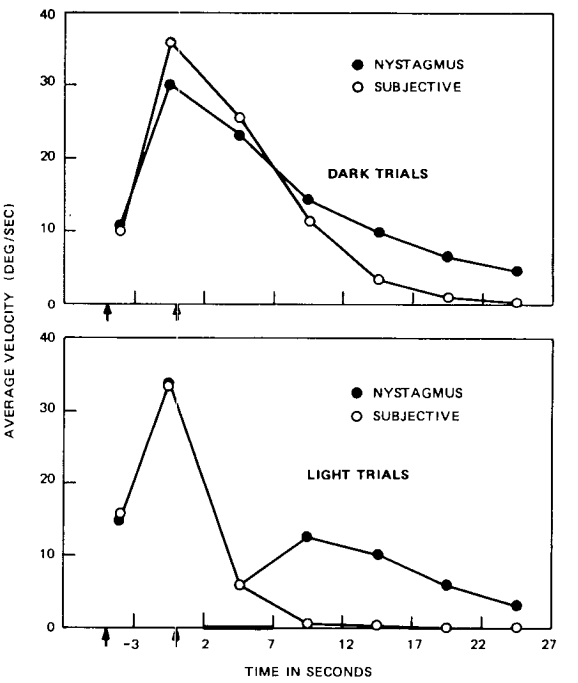


FIGURE 1.—*Effect of room illumination on postrotation nystagmus and subjective sensation (average slow-phase eye velocity and average subjective velocity as a function of time within a trial). After the interval of illumination, the nystagmus shows recovery but the subjective reaction does not. Arrows indicate periods of vestibular stimulation; horizontal bar denotes interval of illumination. (from ref. 1)*

closing of the eyes. However, Guedry et al. turned on the lights in the centrifuge room to show the subject the entire rich visual environment, which was easily associated with the fixed laboratory setting, during the period of 5 s indicated by the solid bar in figure 1b. During the time the lights were turned off again, the nystagmus rose in a period of approximately 1 s to match the normal vestibular decay curve that would have been present had the lights never been turned on. The subjective sensation, on the other hand, never did return following the exposure to the lighted room. The interpretation of these data must be that during the period of exposure to a "compelling visual environment," the visual input dominated the vestibular input and eliminated all nystagmus as well as subjective sensation of rotation. Once this stimulus was gone, however, the vestibular-ocular pathway continued to generate nystagmus, whereas the higher-level subjective sensation system used the remembered information for its set and successfully ignored the continuing vestibular signals.

A related but more complex experimental situation was explored by Melvill Jones (ref. 3). Subjects were accelerated to a constant velocity of 60°/s about the vertical axis. The velocity was maintained for 3 minutes before suddenly decelerating to a stop. The subject's head was tilted so that the stimulation was not in the plane of the horizontal canals but rather was in the yaw and roll axes in the one case and the yaw and pitch axes in another. The rotating platform was surrounded by a fixed screen containing vertical stripes. During the initial acceleration and the first several seconds of the constant velocity, plateau optokinetic and vestibular influences were compatible and resulted in yaw (horizontal) eye movements, which were slightly greater than necessary to achieve retinal stabilization and considerably higher than would be the result of vestibular stimulation. As shown

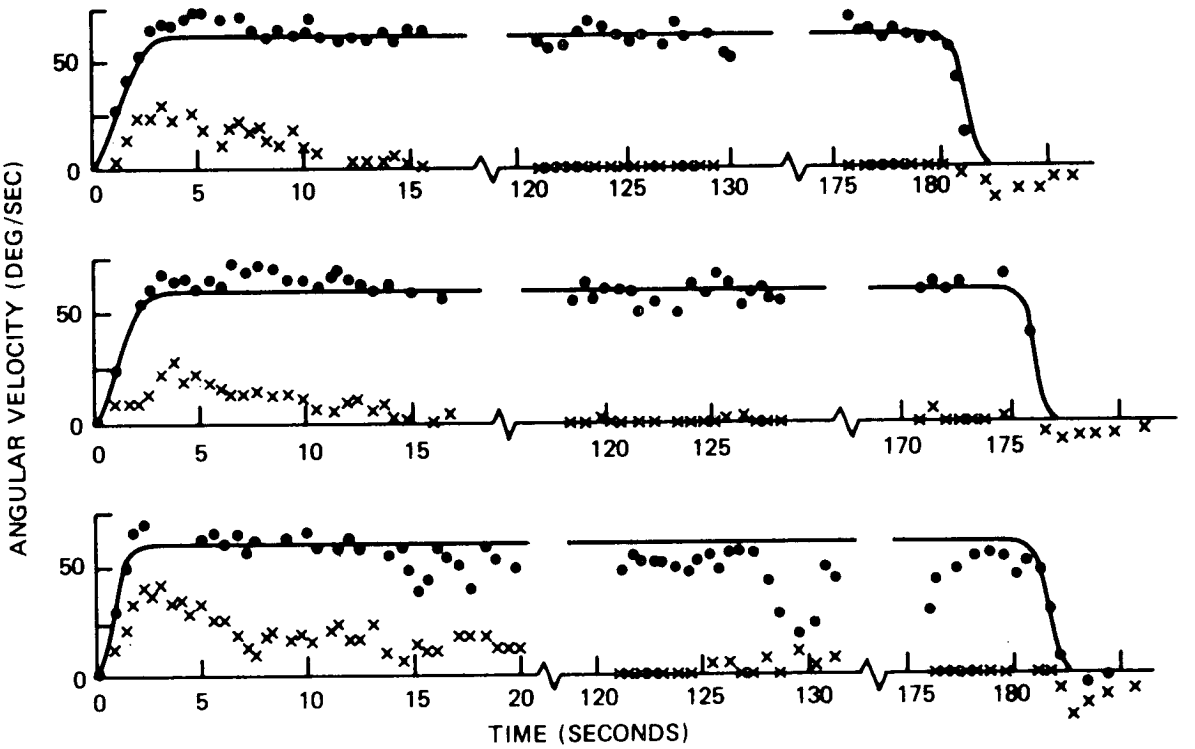


FIGURE 2.—*Compensatory slow-phase eye angular velocities of three subjects on rotation about a vertical axis with head tilted 45° backward. Responses recorded simultaneously in yaw (•) and roll (×) planes of the skull. The continuous line gives the required eye angular velocity in both planes for stabilization of the retinal image. (from ref. 3)*

by the dots in figure 2, the yaw compensatory eye movements remained at the required level of 60°/s throughout the prolonged constant-velocity period, clearly showing that the optokinetic stimulus had taken over from the vestibular stimulus. On the other hand, as shown by the crosses in figure 2, the torsional or roll eye movements, which were never sufficient to be fully compensatory, died out over a period of 10 to 15 s, just as the vestibular stimulation alone would predict, and the optokinetic influence on the roll eye movements was minimal. At the end of the 180-s stimulus, following deceleration the eye movements showed almost no undershoot—again consistent with the optokinetic stimulation. The vestibular stimulation associated with this sudden impulsive stop would be a large undershoot of eye movements down to —40°/s to —50°/s and back to zero. This kind of response was shown at lesser magnitude by the roll eye movements, however. Thus, apparently the yaw eye movements associated principally with stimulation of the horizontal semicircular canals were heavily influenced by the optokinetic stimulus, whereas the torsional eye movements were influenced much less so. When this test was repeated with the subject's head tilted sideways at 45°, so that the stimulus was in the yaw and pitch planes, the results were similar to those for yaw discussed above. The compensatory slow-phase angular velocities for both pitch and roll slightly exceeded those for optokinetic stabilization in the early stages, in which vestibular and visual processes were consistent. In this case, however, the eye-movement velocities for both pitch and yaw (vertical and horizontal nystagmus) decreased slightly from those necessary to maintain fixation following 15 s of constant angular velocity. Although the eye velocity never completely returned to zero, it remained at much lower levels throughout the 3-minute period than had been observed in the yaw eye movements for the yaw-roll stimulation. Thus we see that, for yaw and pitch stimulus, the resulting nystagmus can be a compromise between the eye movements required for optokinetic nystagmus by the visual stimulus and for vestibular nystagmus.

The final bit of experimental evidence to be introduced on visual-vestibular interaction concerns the effect of static visual-field orientation on perception of the vertical. The perception of the apparent vertical (AV), which depends principally on the visual system and the gravireceptors (primarily otoliths), is generally considered to be a result of interpretation of the interaction between visual and vestibular stimuli rather than one or the other. Ever since the tilted-room experiments, there have been efforts to assess the relative influences of visual and vestibular cues. In a recent paper by Udo de Haes (ref. 4) the effect of a tilted visual reference on the perception of the apparent vertical was investigated as a function of the degree of body tilt. He used two visual-field orientations, tilted at angles of ±15° with respect to the true vertical, and recorded the difference between settings of the apparent vertical as a function of body position. This visual-field effect is shown in figure 3. Notice that the visual-field effect, which is the difference between apparent vertical settings for the two visual-field orientations, is less than 5° for the body upright (0°) but rises rapidly to greater than 20° when the subject is lying on his side at 90° and increases further until the subject nearly reaches the inverted position. These data are consistent with the theories of decreased statolith influences on perception and nystagmus in the inverted posi-

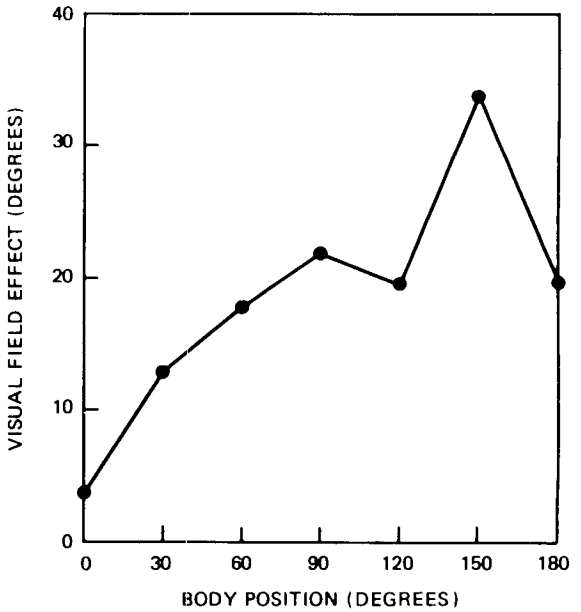


FIGURE 3.—*Interaction between visual system and gravireceptors in perception of apparent vertical. (from ref. 4)*

tion. They also show the changing nature of visual-vestibular interaction in perception of orientation as a function of the compelling nature of the visual, or in this case the vestibular, stimulus. The inverted position, which leads to greater uncertainty in interpretation of statolith outputs, reduces their effect and increases the relative influence of visual stimuli.

FLOW-CHART MODEL OF VISUAL-VESTIBULAR INTERACTION

Figure 4 is a flow-chart representation of a possible model of a visual-vestibular interaction program. It is a nonlinear model in which visual and vestibular influences are linearly weighted when they are in relative agreement, but it switches to the more "believable" one when they are in disagreement. The two inputs are angular velocity stimuli to the vestibular system (ω_v) and to the visual system (ω_e). The vestibular response ($\hat{\omega}_v$) is based on the well-known dynamics of the semicircular canals, including adaptation, and the visual response ($\hat{\omega}_e$) depends upon dynamic response of the visual system, which is approximately equal to one. Notice that the important input on the visual side is the actual angular velocity of the head with respect to the visual field and not the retinal slippage. (Even when the eyes do not move, the effort to maintain fixation may result in a perception of visual-field motion.) These two (visual and vestibular) angular velocities are compared, and if their difference is less than an allowable threshold ϵ, then the single subjective angular velocity or nystagmus velocity ω_s is calculated as a weighted sum of these two, with the weighting coefficients a and b not yet determined. On the other hand, if the two are not compatible, then a hierarchical choice program begins in which normally the visual field is given higher priority. If the visual sensation is "compelling," then it is accepted as "true," and determines the subjective and nystagmus velocity. By "compelling visual stimulus" we refer to either a well-recognized common scene as in the Guedry experiments, or to a highly learned artificial situation as in the case of pilots observing their flight instruments. This can be stated mathematically in terms of the expected variance of this signal exceeding any threshold level α. If it does exceed the threshold level, then one examines the vestibular angular velocity to see if it is compelling. Vestibular angular velocities would be compelling if, for example, one is a well-known rotation stimulus and if the outputs of the statoliths and the semicircular canals are compatible, as discussed earlier. If this is the case (variance of vestibular stimulus less than an acceptable level β), then the vestibular signal is accepted as the subjective angular velocity. If it is not the case, and there is consequently conflict between visual and vestibular stimuli, neither of which is sufficiently compelling, then one predicts the occurrence of disorientation, vertigo, and possible motion sickness. The angular velocity that is sensed may be uncertain; it may switch rapidly back and forth between several different possibilities. This situation, which is disturbing at first, may be the necessary input for habituation, either to a bizarre stimulation situation or to repeated stimulus.

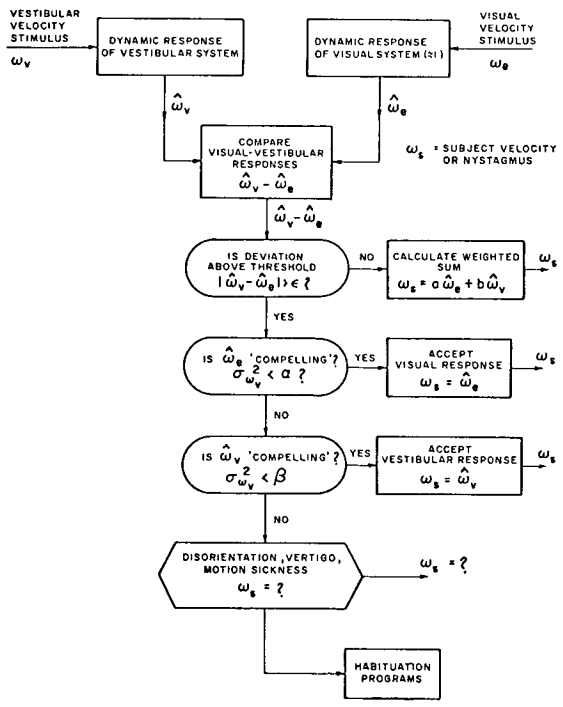


FIGURE 4.—*Flow-chart representation of visual-vestibular interaction model.*

N74-18773

SCHEMA FOR OPTIMAL SUBJECTIVE ORIENTATION BASED ON SEVERAL SENSORY MODALITIES

In this section we consider a possible schema for representing the human dynamic space-orientation problem as an optimal mixing problem. As seen in figure 5, the object of the system is to produce an estimated state (x), which is in some sense an optimal estimate. The state vector may be considered to be a 12-dimensional vector, with elements consisting of the three Euler angles defining head orientation with respect to an inertial reference, their rates of change, three components of linear translation, and their first derivatives. The estimated state is based upon an optimal estimator in which the cost function to be minimized is some function of the state. Whereas a simple weighted sum-of-squares cost function would lead to an analytically tractable problem, it is more likely that an appropriate cost function places greater emphasis on certain state variables in certain regions than others. For example, orientation with respect to the vertical may be of considerably greater interest than heading, when concerned with postural control. The optimum estimator has two types of inputs: (1) those coming from the various sensors and (2) the "expected state." Consider first the sensors that are stimulated by the "true state" or actual motion of the subject. Each of the

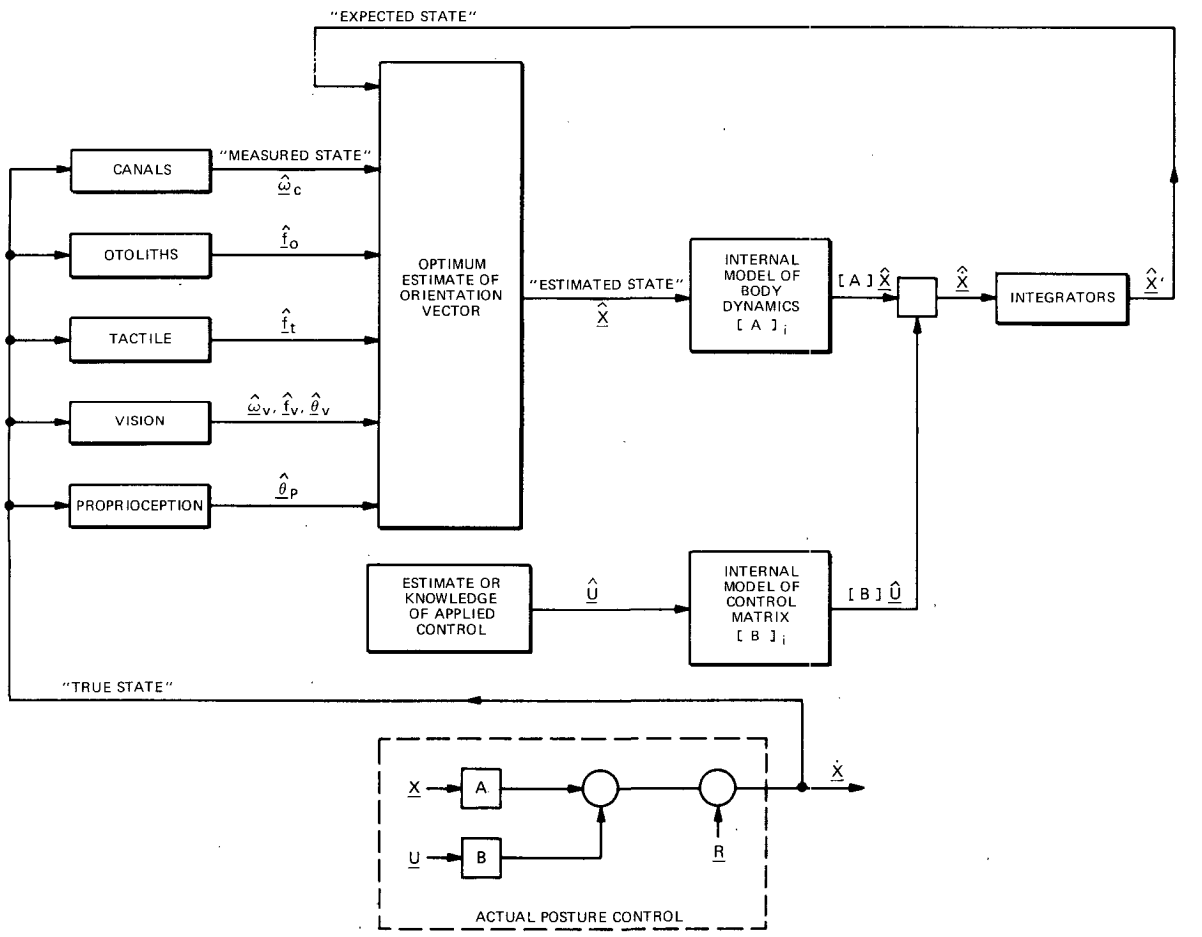


FIGURE 5.—*Human subjective orientation represented as an optimal mixing problem.*

sensors measures one or more of the state variables with characteristic uncertainty and dynamic performance. For example, the semicircular canals measure the angular velocity of the subject with respect to inertial space ($\hat{\omega}_c$), and their dynamic response may be described by the adaptation model. Estimates for noise in this system are difficult to obtain explicitly but may be modeled, for example, by white Gaussian noise with standard deviation equal to the threshold for the oculogyral illusion. Similarly, the otoliths measure specific force; tactile sensation also measures specific force; and the visual channel measures angular velocity, direction of net specific force, and angular orientation.

The outputs of all these sensors may be combined to make an optimum estimate of the true state. This process is analogous to the aerospace optimization problem in which information on altitude obtained from a pressure altimeter, a radar altimeter, and an inertial system must be combined in an optimal way, knowing the noise and dynamic lag associated with each channel, to give the best estimate of current altitude. Although the descriptions of the channel dynamic lags and noises clearly are not completely available, reasonable estimates may be made.

It is equally important in this schema to consider the expected state, which is the major new contribution of this type of model. If one assumes that many of the motions of the body are not arbitrary unexpected motions (like aircraft motion in clear-air turbulence), but rather are the result of deliberate actions on the part of the subject, then the expected head motions may be used to improve the estimate of orientation. Thus, in simple postural control when a subject jumps up, actively moves his head, or begins to run, the vestibular responses that result are nearly always in accordance with the expected head position. One can consider this an extension of the von Holst "efferent copy" notion, in which copies of the motor commands are used to interpret sensory feedback. One can also extend this expected-state notion to the case where the relation between expected head movement and the motor command involves some external machinery with its own dynamics. Examples include the expected angular velocity and lateral acceleration of an automobile when the driver turns the steering wheel or applies the brakes. In this case the reactions of the driver and the passively moved passenger (who was unaware of such actions) differ markedly, whereas the driver and passenger may react identically when the car unexpectedly hits a patch of ice. Consider the trivial example of operating an automatic elevator in which the orientation response when the elevator descends after the passenger has pressed the "up" button is startling. In general, the process can be considered as illustrated in figure 5, in which the subject maintains an internal model of his body dynamics (which enables him to solve the equations transforming body rates to body angles and displacements) and also an internal model of the control matrix $[B]_i$, including any external machinery. His estimate of the control $\hat{u}$ (which really may be muscular force) applied to his estimate of the internal control model $[B]_i$, results in a control vector $[B]\hat{u}$, which is combined with the vector $[A]\hat{x}$ to yield the estimated vector $\hat{x}'$. Any errors in either knowledge of the control matrix or knowledge of the applied control will obviously lead to errors in estimation and produce false illusions regarding orientation.

For limited situations, this schema can be examined to determine the extent to which this optimal mixing notion and the use of the expected state apply to the human space-orientation problem.

REFERENCES

1. Guedry, F. E.; Collins, W. E.; and Sheffey, L.: Perceptual and Oculomotor Reactions to Interacting Visual and Vestibular Stimulation. Percept. Motor Skills, vol. 12, 1961, pp. 307–324.
2. Young, L. R.; and Oman, C.: Model for Vestibular Adaptation to Horizontal Rotation. Aerospace Med., vol. 40, 1969, pp. 1076–1080.
3. Melvill Jones, G.: Interactions Between Optokinetic and Vestibulo-ocular Responses During Head Rotation in Various Planes. Aerospace Med., vol. 37, 1966, pp. 172–177.
4. Udo de Haes, H. A.: Interaction Between Visual and Gravireceptor Stimulation with Respect to the Apparent Vertical. Max Planck Institut für Verhaltensphysiologie, Seeweissen, Germany, 1970.

Subjective and Nystagmus Reactions Considered in Relation to Models of Vestibular Function*

F. E. GUEDRY, JR., R. D. GILSON, and C. W. STOCKWELL
Naval Aerospace Medical Research Laboratory

SUMMARY

Modeling will become increasingly important as more knowledge is accumulated, because it offers advantages in predicting reactions of individuals in a variety of situations, including novel aerospace environments, and in specifying a few parameters which should have considerable clinical significance. However, the need for continuing experimental "crosschecks" of these models has been illustrated by several sets of results which would not have been predicted by any existing models.

INTRODUCTION

In recent years there have been several attempts to develop models of the orientation system that include visual, vestibular, and proprioceptor mechanisms and their functional interactions (refs. 1 to 4). These models are a little disturbing to some of us because they not only predict almost everything we have been trying to understand, but they do it in a foreign language, one written by de Laplace. However, it is not our purpose to criticize these models or their linguistics but rather to point out some of their advantages, while also illustrating the need for a continuing evaluation of their adequacy.

TORSION PENDULUM MODEL

First consider a relatively simple model that most vestibular workers have been using in one way or another for years, a model of the cupula-endolymph system proposed long ago and developed more recently by Van Egmond, Groen, and Jongkees (ref. 5). This model is based on the assumption that the semicircular canal system responds as does a heavily damped torsion pendulum to inertial torques produced by angular accelerations of the head. According to the model, the behavior of the cupula is predictable from a second-order linear differential equation when values for the parameters of the equation have been determined.

Illustrative of some applications of this model are results of a few recent experiments. Several years ago we concluded that measurements of turning sensations associated with angular accelerations are an important aspect of the vestibular evaluation of flight personnel. This conclusion was based on several considerations, including a report by Benson (ref. 6) indicating that directional asymmetries in vestibular turning sensations were present more frequently in pilots whose presenting symptom was vertigo than in other personnel, whereas nystagmus asymmetries did not appear to have the same relevance. Therefore, we attempted to find measures of vestibular turning sensations that would be suitable for application in aviation medicine. We have selected triangular waveforms of angular velocity because such stimuli can be presented quickly and they approximate the type of stimulus received by the canals during natural head movements. We reasoned that commonly experienced stimuli would be more easily and reliably reported than some of the unnatural stimuli commonly used to assess vestibular sensation.

Theoretical cupula deflection during triangular

* This research was sponsored jointly by the U. S. Army Aeromedical Research Laboratory and the Naval Aerospace Medical Research Laboratory and supported in part by the Office of Advanced Research and Technology, National Aeronautics and Space Administration.

waveforms of different durations is illustrated in the upper part of figure 1. The dotted lines represent stimulus velocity (ω), and the curves limiting the hatched area represent cupula displacement (x) as predicted from the torsion pendulum model, equation 1 (shown in fig. 1).

It is generally believed that cupula deflection regulates velocity information; consequently the cupula deflection curve should resemble the subjective angular velocity curve. However, a gain factor K_s, with units, is needed to transform cupula displacement to subjective angular velocity units, equation 2 (fig. 1). This is illustrated in the lower part of figure 1, where K_s was chosen to make peak subjective velocities and peak stimulus velocities correspond fairly closely.

If man receives angular velocity information like this from the semicircular canals and if he is able to integrate this information accurately over time, then angular displacement estimates (ψ_s) should be predicted by equation 3 (fig. 1), which gives the time-integral of the angular velocity curve.

The lower portion of figure 1 shows that short waveforms should yield slight overestimates of angular displacement, whereas long waveforms should yield systematic underestimates. As a matter of fact, this is what happens, as shown by the response measurements taken from subjects who were rotated

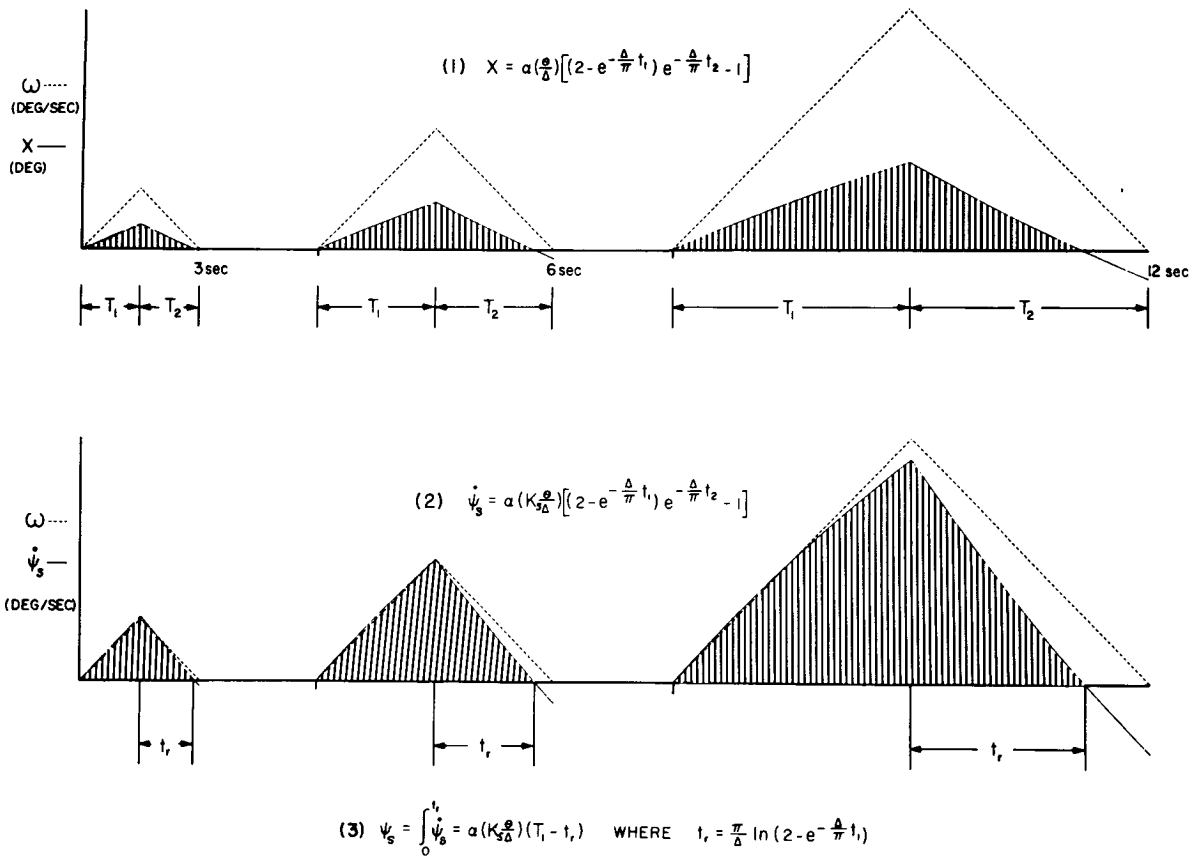


FIGURE 1.—*Theoretical responses to triangular-waveform stimuli of 3, 6, and 12 seconds' duration. The dotted lines represent stimulus angular velocity (ω). In the upper panel, the solid line limiting the hatched area represents cupula displacement (x) from equation 1. In the lower panel, the solid line limiting the hatched area represents subjective angular velocity ($\dot{\psi}_s$) from equation 2. The hatched area in the lower panel represents subjective angular displacement (ψ_s) and is given by equation 3. T_1 is duration of acceleration, T_2 is duration of deceleration, and t_r is the time interval from the end of acceleration to response reversal.*

so that their horizontal canals were stimulated (see fig. 2).

From past research, we estimated $\Pi/\triangle$ to be about 16 s for horizontal-canal responses. Accepting this value for $\Pi/\triangle$ and using $K_s\theta/\triangle = 19.9$ s, equation 3 (fig. 1) gives a good fit to the data shown in figure 2. Similarly, responses from vertical-canal stimuli were predicted. An average value of $\Pi/\triangle$ for the y-axis stimulation of the vertical canals is about 7 s (refs. 7 to 9), and, although a direct estimate of $K_s\theta/\triangle$ for the vertical-canal responses was not available, we estimated its value to be about 53 percent of that for the horizontal-canal responses by analogy from nystagmus data (ref. 10). Thus, $K_s\theta/\triangle$ for the vertical-canal response was estimated to be 0.53×19.9 s, or 10.5 s.

Acceptance of these parametric values for horizontal-canal and vertical-canal responses led to the prediction that subjective displacement estimates during vertical-canal and horizontal-canal stimulation should be about the same for short waveforms, but greater underestimation of vertical-canal stimuli should occur with longer stimulus waveforms. This prediction is illustrated in figure 3; the hatched area is smaller for the vertical-canal (y-axis) reaction than for the horizontal-canal reaction with longer stimulus waveforms. Displacement estimates obtained from a group of subjects are shown in figure 4, where it is apparent that the theoretical curves match the obtained z- and y-axis results very well. Incidentally, these subjects spontaneously reported that they experienced a stronger aftereffect from the vertical-canal (y-axis) stimulation than from horizontal-canal stimulation. According to the model, a stronger after-reaction from the vertical-canal stimulus is to be expected. This difference can be seen in the predicted curves shown in figure 3 and in obtained nystagmus curves (fig. 5).

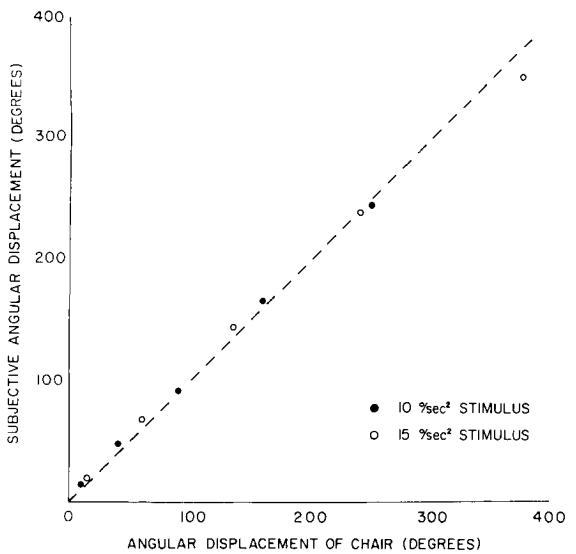


FIGURE 2.—*Mean angular displacement estimates from a group of subjects who were rotated in the plane of their horizontal semicircular canals. (from ref. 11)*

The primary purpose in presenting these results is to illustrate the fact that, when values of two parameters have been determined, it is possible to predict various response characteristics to a range of stimuli. To illustrate this point further, figure 6 shows the responses of three hypothetical subjects with different values for vestibular parameters $\Pi/\triangle$ and $K_s\theta/\triangle$. They should respond differently, as shown for impulse-type stimuli (fig. 6A), sustained accelerations (fig. 6B), and triangular waveforms (fig. 6C). It is apparent that the magnitudes and temporal characteristics of responses should vary considerably among three such individuals to different stimuli. Actually, few if any experiments have been done to test predicted differences in responses in the same subjects with a variety of stimuli. However, differences in responses of these magnitudes are found among apparently normal subjects, as shown in figure 7. The responses of the two subjects in figure 7 resemble the theoretical responses of subjects 2 and 3 in figure 6D.

These considerations illustrate several potential advantages of a mathematical model:

(1) Determination of the values of several parameters for an individual permits prediction of a variety of response characteristics to a range of stimuli.

(2) If the model is appropriate, its parameters should have considerable clinical significance, since they are single index numbers that are descriptive of a range of reactions.

(3) The model aids in choosing and evaluating alternative measures and test procedures. For example, estimation of $\Pi/\triangle$ requires a tedious set of measurements when determined from the decay of nystagmus following an impulse, whereas it can be estimated quickly from the point of reversal of nystagmus (see equation 3, fig. 1).

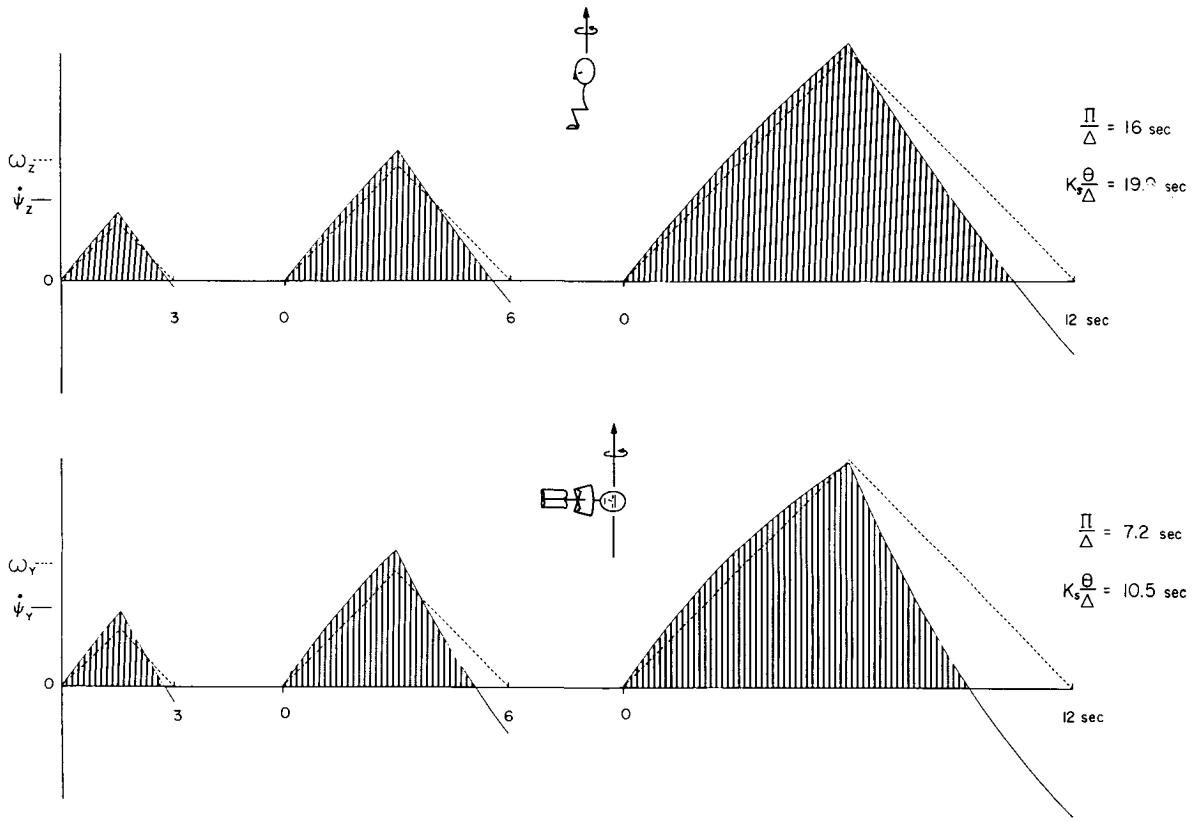


FIGURE 3.—*Theoretical responses to triangular-waveform stimuli of 3, 6, and 12 seconds' duration. Upper panel: horizontal-canal stimulation. Lower panel: vertical-canal stimulation.*

(4) The model can be tested because it makes specific predictions.

The last point brings us to a second topic. Because the simple model makes specific predictions, it is immediately clear where predictions fail. Predictions of this model depart most flagrantly from experimental observations when subjects are exposed to stimuli that are either large in magnitude or long in duration. For some time it has been assumed that these deviations are the consequence of a short-term adaptation process that is not accounted for by the model.

A MORE COMPLEX MODEL: ADAPTATION EFFECTS

Secondary nystagmus and secondary sensation, the decline of primary nystagmus and sensation reactions during prolonged constant angular acceleration, and the differing slopes of nystagmus and sensation cupulograms have been attributed to some kind of adaptation process by a number of investigators. Recently several authors have expressed this idea mathematically. Steer et al. (ref. 1) accepted the basic simple model of the canals as a damped spring system and assumed that adaptation can be represented by a shifting reference level which acts to reduce the difference between cupula-controlled neural inflow and the existing reference level. The mathematical expression of this idea has several advantages. First, if their model is appropriate, we need only to determine two adaptation time constants (one for nystagmus and one for sensation) from which, together with the constants previously described, we can predict individual responses to a much wider range of reactions than before. Second, the model makes explicit various procedures by which these time constants can be

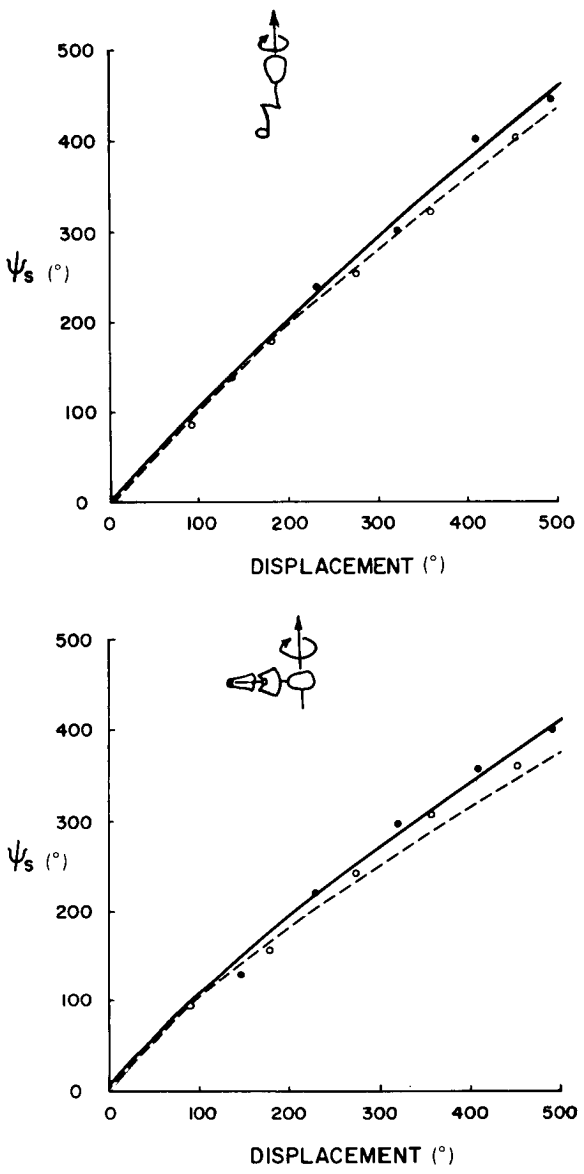


FIGURE 4.—*Mean subjective angular displacement estimates (ψ_S) for a group of subjects who were rotated in the plane of their horizontal semicircular canals (top) and vertical semicircular canals (bottom). Solid lines and solid circles represent predicted and obtained results, respectively, when $\alpha = 15°/s^2$. Dashed lines and open circles indicate predicted and obtained results, respectively, when $\alpha = 10°/s^2$. (from ref. 8)*

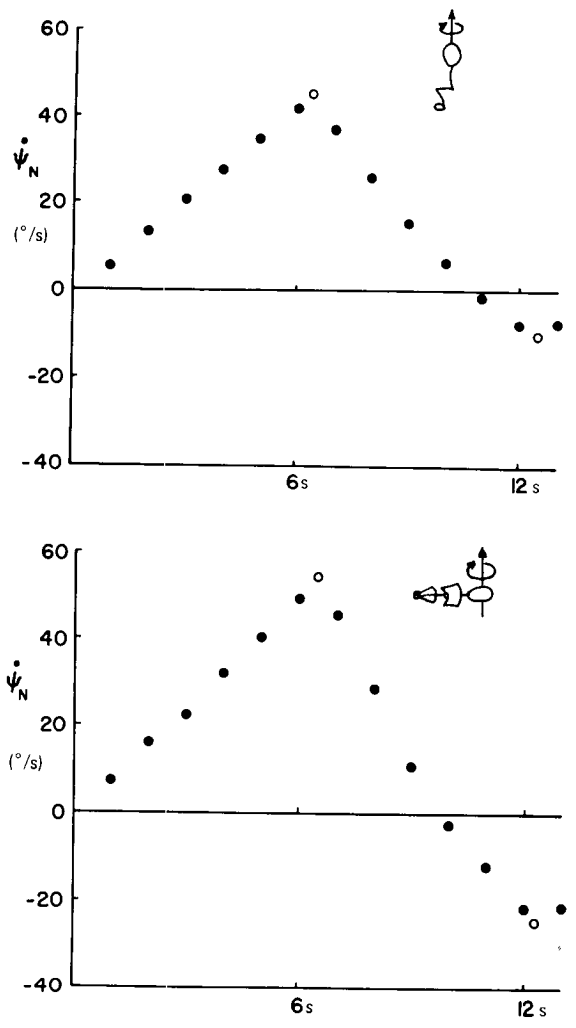


FIGURE 5.—*Nystagmus slow-phase velocity ($\dot{\psi}_n$) during a triangular-waveform stimulus of 12 seconds' duration. Top: horizontal semicircular-canal response. Bottom: vertical semicircular-canal response.*

determined. Third, the model is specific enough to be tested.

It appears that this more complex model accounts for most of the facts which have been considered as evidence of adaptation and which could not be accounted for by the simpler model, although additional checks are necessary. In an experiment currently in progress, we are attempting to find reliable and practical ways to measure adaptation time constants. Specifically, we are measuring the length of time before the response reverses direction (t_r) during triangular waveform stimuli of various wavelengths. (This response measure has been illustrated in fig. 1.) According to the adaptation model, t_r should increase as stimulus wavelength increases

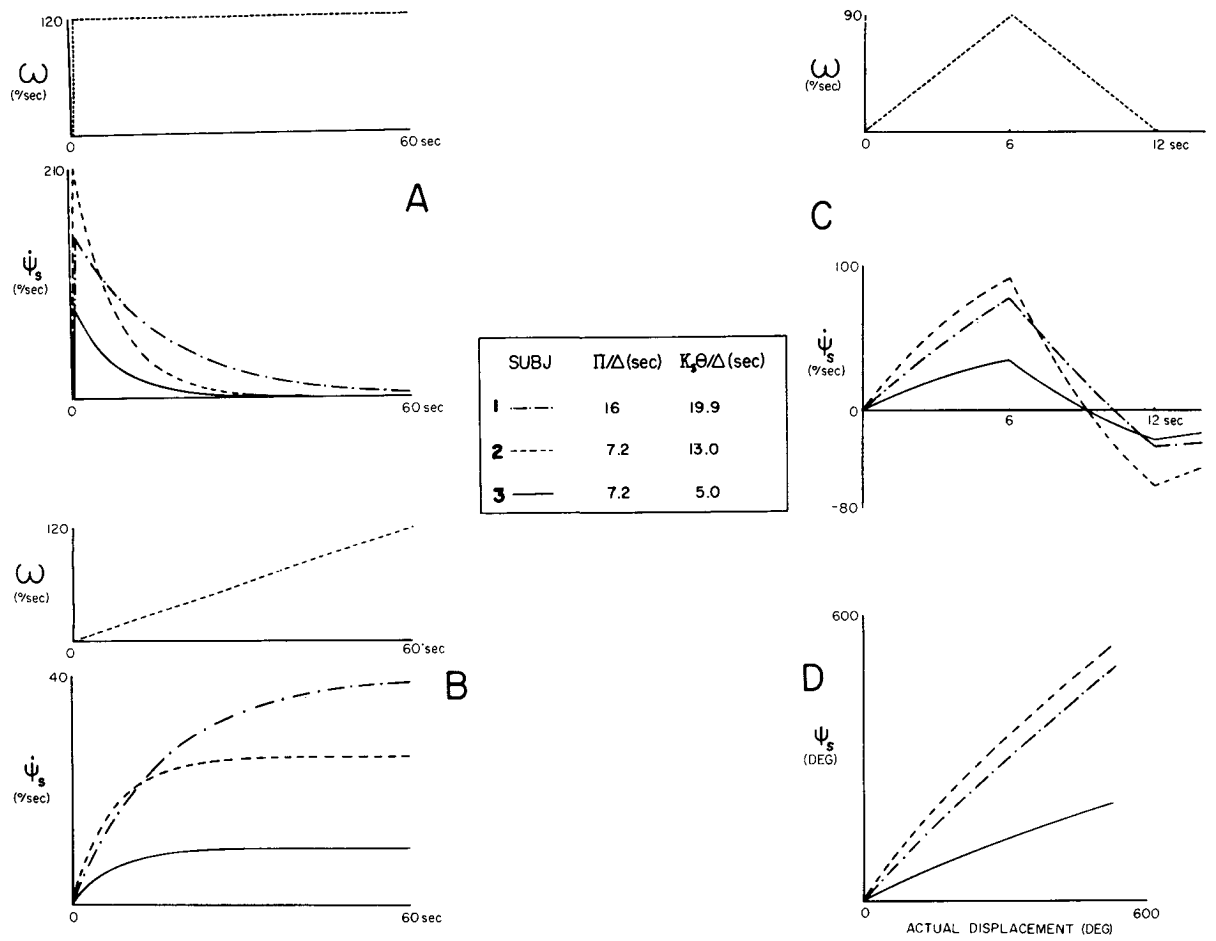


FIGURE 6.—*The theoretical responses of three hypothetical individuals who possess different values of vestibular response parameters,* Π/Δ *and* $K_s\theta/\Delta$. *A: impulse-type stimuli. B: sustained accelerations. C: triangular waveforms.*

up to a certain point and then decrease, as shown in figure 8. The model also predicts that nystagmus t_r should drop off gradually and should be longer than sensation t_r when longer stimulus waveforms are used. Results obtained thus far seem to support the model. However, in some deviant subjects, sensation t_r is neither shorter than nystagmus t_r nor does it drop off sooner. This may mean that we are measuring large individual differences in short-term adaptation, or that the model needs revising, or that we have not hit upon a measure of the adaptation time constant that works for all people.

The pitfalls in assessing a complex process such as adaptation can be illustrated by considering subjective displacement estimates that we obtained from two different groups of subjects exposed to a series of triangular waveforms (fig. 9). Group A grossly underestimated the larger angular displacements. We thought at first that this result might be explained on the basis of short-term adaptation; however, in another group of subjects (group B), displacement estimates were essentially accurate for all stimuli presented. The vestibular stimulus conditions were exactly the same for both groups, and there was no reason to believe that subjects in one group would have significantly different adaptation time constants than those in the other. We now believe that a slight difference in instructions to the

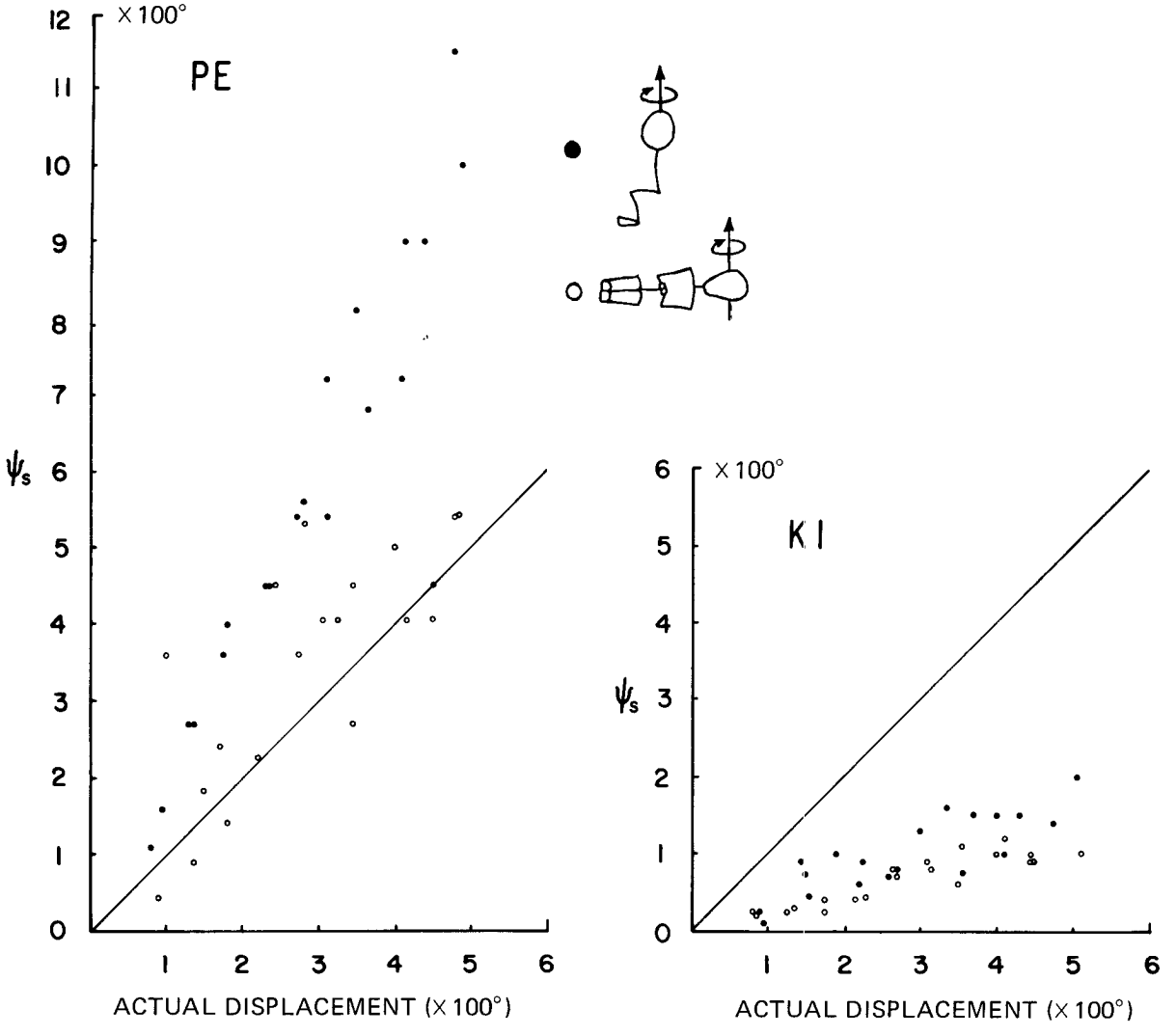


FIGURE 7.—*Mean angular displacement estimates (ψ_s) from subjects PE and KI. (from ref. 8)*

two groups produced this difference in displacement estimates (ref. 11). If so, then when man operates in one mode, his estimates of larger displacements differ from those made when he operates in another mode. Subtleties such as these indicate that we need a firm data base on which to evaluate any model, especially when we are dealing with a complex model that does not have a rational basis but has evolved to explain known stimulus-response relations. With progression from a simple end-organ model, for which many forms of evidence may be adduced, to the more complex adaptation model, for which we have no real guidelines other than the stimulus-response data on hand, we must proceed with caution.

COMPLEX MODELS INVOLVING INTERACTIONS AMONG DIFFERENT SYSTEMS

Still more complex models have been proposed. These models concern interactions among the otolith, the canal, the proprioceptor and the visual systems (refs. 2 and 3). They represent efforts to unify functional relations among these systems, taking into

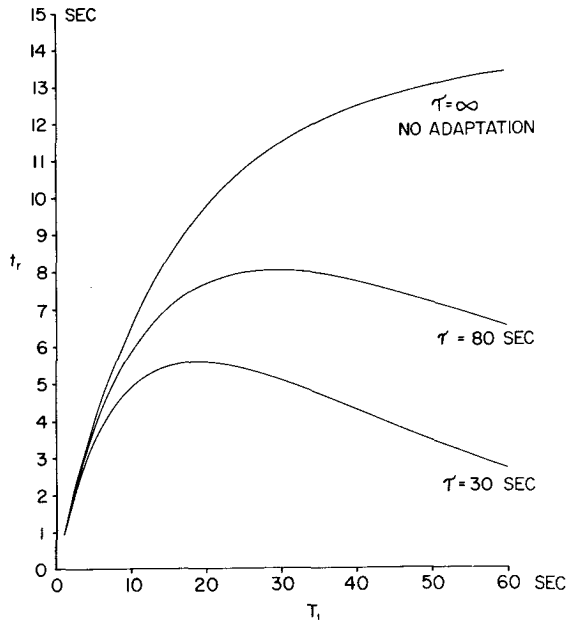


FIGURE 8.—*Relationships between duration of acceleration (T_1) and the interval from end of acceleration to response reversal (t_r) for different values of the adaptation time constant (τ).*

consideration transducer mechanisms of the individual systems involved. Such models are valuable because they produce a systematic plan for experimentation, as well as an overview of how we will eventually have to proceed to understand complex perceptual and behavioral processes.

There is clearly a need for caution, however, in acceptance of these models. Recent experiments on interactions between the visual and vestibular systems can serve as a case in point. In the past few years we have studied the influence of nystagmus on visibility of charts and other objects fixed relative to the head. This work originally stemmed from a request by NASA for an estimate of how long an astronaut's vision would be impaired by nystagmus following rotation about the *y* axis. NASA considered this a potential problem in the event of an abort in the Apollo series of spaceflights. Hixson and Niven (ref. 12) discovered that nystagmus in one direction degraded vision much more than nystagmus in the opposite direction. This finding has been confirmed by subsequent experiments, and it was shown in addition that the tracking of flight

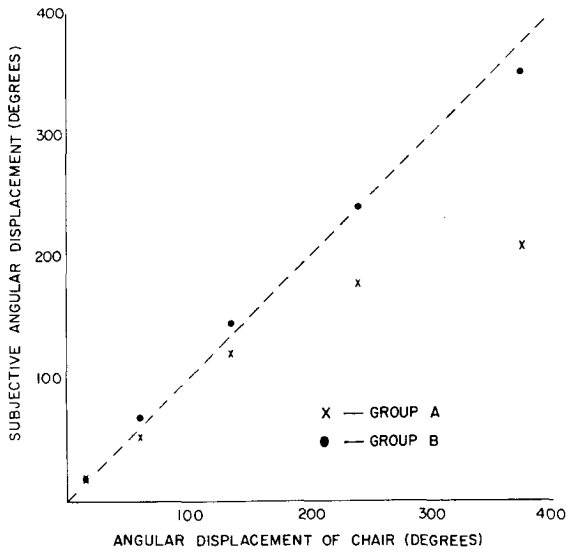


FIGURE 9.—*Mean angular displacement estimates for two groups of subjects who were given different instructions. (from ref. 11)*

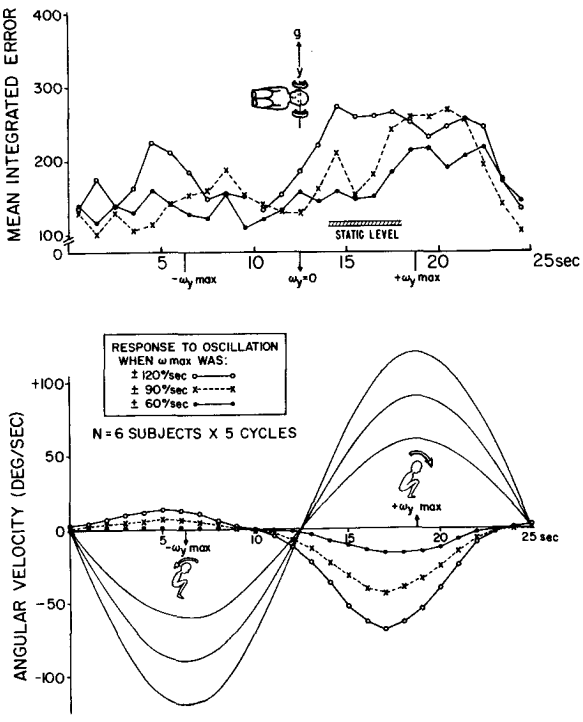


FIGURE 10.—*Tracking error (top) and nystagmus slow-phase velocity (bottom) for a group of subjects who underwent sinusoidal oscillation. (from ref. 13)*

instruments is degraded differentially by nystagmus in these two directions (fig. 10). It appears that there is differential visual suppression of the two nystagmus responses (ref. 13). The point is that these results would not have been predicted in advance by any existing model. If we had attempted to extrapolate from our knowledge of the time constants of the vertical and horizontal canals and from some visual-acuity data obtained during horizontal nystagmus, our predictions would have been nearly correct for backward tumble but in error by a factor of 10 for forward tumble, and this would have led us to false conclusions about performance limits in a potentially important emergency situation.

REFERENCES

1. Steer, R. W.; Young, L. R.; Oman, C. M.; and Malcolm, R.: Progress in Vestibular Modeling. Fourth Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–187, 1970, pp. 353–380.
2. Meiry, J. L.; and Young, L. R.: Biophysical Evaluation of the Human Vestibular System. Status Report on NASA Grant NGR 22–009–156, September 1970.
3. Young, L. R.; Meiry, J. L.; and Li, Y. T.: Control Engineering Approaches to Human Dynamic Space Orientation. Second Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–115, 1966, pp. 217–227.
4. Mayne, R.: The Analogy of the Vestibular Organs to an Inertial Guidance System. Presented at Intern. (World) Cong. Oto-rhino-laryngol., Mexico City, 1969.
5. Van Egmond, A. A. J.; Groen, J. J.; and Jongkees, L. B. W.: The Mechanics of the Semicircular Canals. J. Physiol., vol. 110, 1949, pp. 1–17.
6. Benson, A. J.: Vestibular Asymmetry and Spatial Disorientation in Aircrews. A Symposium on Vertigo as a Problem in Aerospace Medicine, U. S. Naval Air Development Center, Johnsville, Pa., 1967.
7. Melvill Jones, G.; Barry, W.; and Kowalsky, H.: Dynamics of the Semicircular Canals Compared in Yaw, Pitch, and Roll. Aerospace Med., vol. 35, 1964, pp. 984–989.
8. Guedry, F. E.; Stockwell, C. W.; and Gilson, R. D.: Comparison of Subjective Responses to Semicircular Canal Stimulation Produced by Rotation About Two Axes. Acta Oto-Laryngol., vol. 72, 1971, pp. 101–106.
9. Gilson, R. D.; Stockwell, C. W.; and Guedry, F. E.: Nystagmus Responses During Triangular Waveforms of Angular Velocity About the Y and Z Axes. NAMRL–1138. Naval Aerospace Medical Research Laboratory, Pensacola, Fla., 1971.
10. Collins, W. E.; and Guedry, F. E.: Duration of Angular Acceleration and Ocular Nystagmus in Cat and Man. I. Responses from the Lateral and the Vertical Canals to Two Stimulus Durations. Acta Oto-Laryngol. vol. 64, 1967, pp. 373–387.
11. Guedry, F. E.; Stockwell, C. W.; Norman, J. W.; and Owens, G. G.: Use of Triangular Waveforms of Angular Velocity in the Study of Vestibular Function. Acta Oto-Laryngol., vol. 71, 1971, pp. 439–448.
12. Hixson, W. C.; and Niven, J. I.: Directional Differences in Visual Acuity During Vertical Nystagmus. NAMI–1079. Naval Aerospace Medical Institute, Pensacola, Fla., 1969.
13. Benson, A. J.; and Guedry, F. E.: Comparison of Tracking Task Performance and Nystagmus During Sinusoidal Oscillation in Yaw and Pitch. Aerospace Med., vol. 42, 1971, pp. 593–601.

Responses to Rotating Linear Acceleration Vectors Considered in Relation to a Model of the Otolith Organs

ALAN J. BENSON and GRAHAM R. BARNES
Royal Air Force Institute of Aviation Medicine

SUMMARY

The results are presented of experiments in which human subjects were exposed to a linear acceleration vector that rotated in the transverse (xy) plane of the skull, without angular acceleration (counterrotation). As in other experimental situations in which there was rotation of the linear acceleration vector, the lateral eye movements showed a sinusoidal change in slow-phase velocity and an asymmetry or bias in the same direction as vector rotation.

A model is developed that attributes the oculomotor response to otolithic mechanisms. It is suggested that the bias component is the manifestation of torsion of the statoconial plaque relative to the base of the utricular macula and that the sinusoidal component represents the translational oscillation of the statoconia. The model subsumes a hypothetical neural mechanism, based on the differential summation of inputs from complementary halves of the maculae, which allows x- and y-axis accelerations to be resolved. Derivation of equations of motion for the statoconial plaque in torsion and translation, which take into account forces acting in shear and normal to the macula, yield estimates of bias and sinusoidal components that are in qualitative agreement with the diverse experimental findings.

INTRODUCTION

Over the last 5 years it has been clearly demonstrated that a linear acceleration vector that rotates in the transverse *(xy)* plane of the head evokes a horizontal or lateral nystagmus. The experimental techniques that have been employed in these studies are summarized in figure 1. Guedry (ref. 1) was the first to study the effects of rotating human subjects about a horizontal axis. His demonstration of a sustained perrotational nystagmus was confirmed by Benson and Bodin (ref. 2), Correia and Guedry (ref. 3), and Saito et al. (ref. 4). Comparable responses were also observed in the cat (ref. 5) and in the rabbit (ref. 6). More recently, Stockwell et al (ref. 7) (marked as Guedry (1970) in fig. 1) rotated subjects about a longitudinal *(z)* body axis that was inclined to the vertical in order to examine the oculomotor response to a rotating linear acceleration vector with a magnitude in the xy plane of less than 1 g. During rotation about an inclined axis the subject is exposed to a constant acceleration of $g \sin \alpha$ (where α is the angle between the rotation axis and the horizontal) in the z body axis and a component of $g \cos \alpha$ that rotates in the xy plane at an angular velocity ω. A comparable distribution of forces occurs when a subject is rotated about a vertical axis at the end of a centrifuge arm (ref. 8), except that the magnitude of the z-axis acceleration is 1 g and the rotating xy component is the centripetal acceleration.

In all the experimental situations so far mentioned, the subject experienced an angular acceleration and hence stimulation of semicircular canal receptors during the establishment of vector rotation. However, when the angular velocity of rotation of a subject relative to the centrifuge arm is equal and opposite to the speed of rotation of the centrifuge, he experiences no angular acceleration during the initial increase in the speed of rotation of the centrifuge, and hence the responses evoked are not contaminated by prior canal stimulations. This particular condition, termed counterrotation by Graybiel and Johnson (ref. 9), has not been exten-

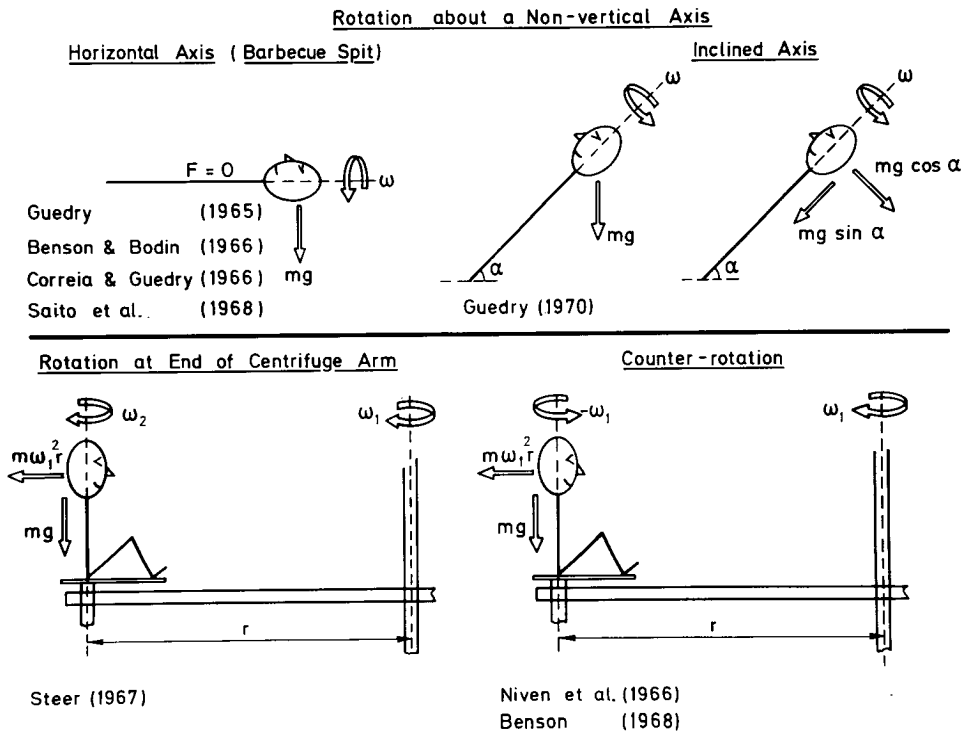


FIGURE 1.—*Types of rotation employed to study in man the lateral eye movements evoked by rotation of the linear acceleration vector in the* xy *plane of the head.*

sively studied. Niven et al. (ref. 10) produced an identical force environment, though by a different technique, and presented qualitative evidence for the evocation of a direction-changing nystagmus in man during vector rotation. A similar nystagmus was observed by Correia and Money (ref. 5) and by Benson et al. (ref. 11) in the cat. Because the nystagmus evoked by rotation about a horizontal axis at speeds of less than 10 rpm was usually unidirectional (refs. 1 to 3), it was considered that a more detailed investigation of the oculomotor response to counterrotation was required, if the results obtained in the different experimental conditions were to be quantitatively compared.

LATERAL EYE MOVEMENTS DURING COUNTERROTATION

Apparatus

A small centrifuge driven by a hydraulic motor (peak torque 340 N-m) was used to produce the rotating force environment (figs. 2 and 3). The seat that carried the subject was mounted on a

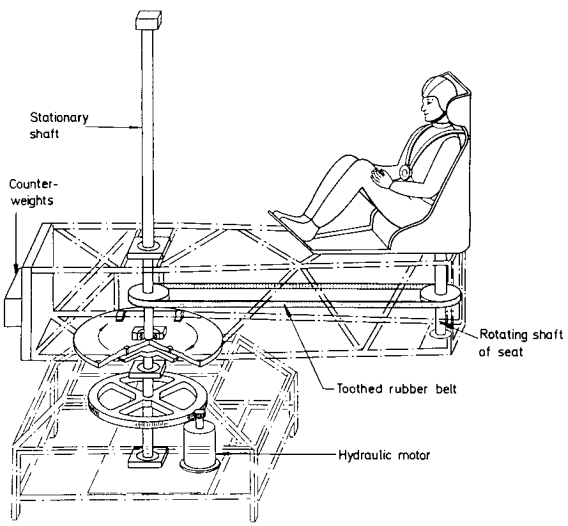


FIGURE 2.—*Isometric view of small centrifuge with counter-rotating seat.*

vertical shaft, 1.5 m from the axis of rotation of the centrifuge. Counterrotation of the seat relative to the centrifuge arm was achieved by means of a

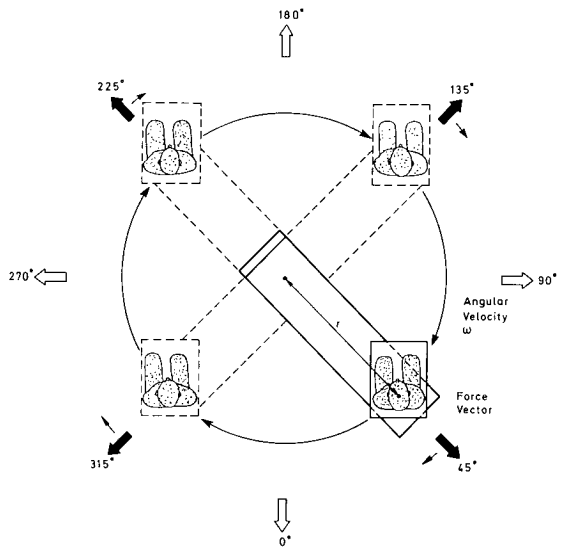


FIGURE 3.—*Plan view illustrating the absence of angular motion of the subject during rotation of the centrifuge arm. Note that the d'Alembert force vector rotates about the subject in the same direction and at the same angular velocity as the centrifuge arm. The filled and open arrows indicate the convention employed for the description of the angular position of the force vector.*

toothed rubber belt, which coupled the shaft carrying the seat to a fixed pulley at the axis of rotation of the centrifuge. Application of maximum torque allowed a speed of 101°/s, equivalent to a centripetal acceleration at the seat axis of 0.44 m/s^2 (0.45 *g*), to be obtained in about 4 s. The speed of rotation was regulated by a pressure-compensated orifice to an accuracy of ±1°/s in the steady state. The subject was held in the seat by a conventional four-strap harness and foot straps; the head was immobilized by a clamp fitted with adjustable side plates and a brow strap.

Lateral eye movements were recorded by the conventional electro-oculographic technique. During rotation the eyes were closed and the head was covered by a small hood. Calibration eye movements were recorded before and after each rotational stimulus.

Results

The lateral eye movement obtained when a subject was counterrotated at 101°/s (centripetal acceleration, 0.45 *g*) is shown in figure 4. Nystagmic eye movements occurred within less than 1 s of the

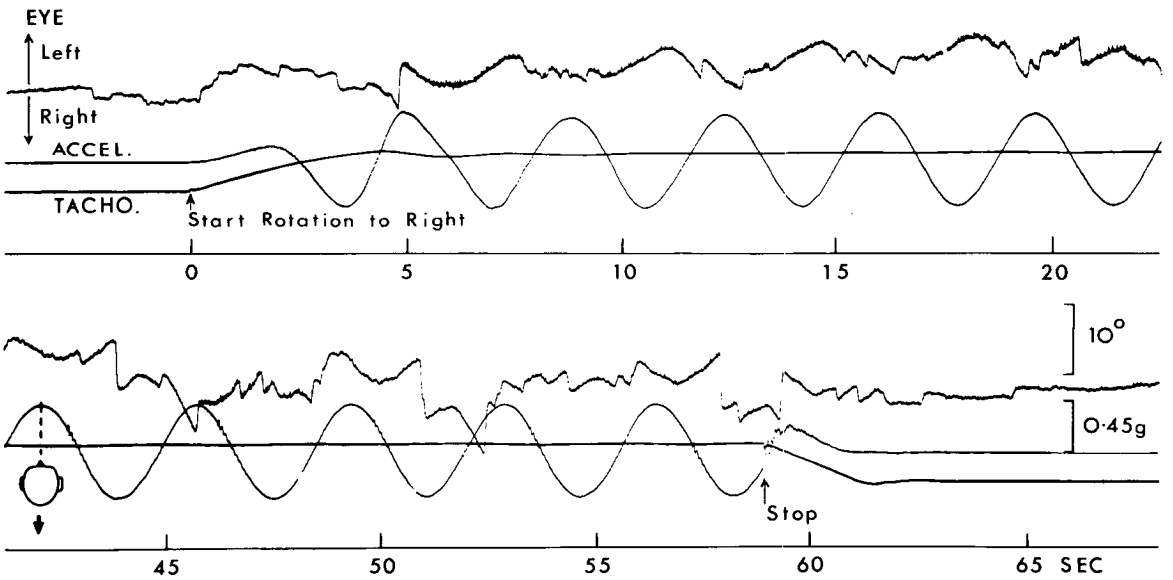


FIGURE 4.—*Typical ultraviolet galvanometer record of the lateral eye movements produced by centrifuge rotation at 101°/s (radial acceleration = 0.45 g) to the right (clockwise when viewed in plan). The traces (from top down) are: lateral eye position, linear acceleration in the anteroposterior (x) axis, and angular velocity of the centrifuge. The datum position of the force vector is indicated by the inset head (lower left) and the eye movement and accelerometer calibrations by the vertical bars (lower right).*

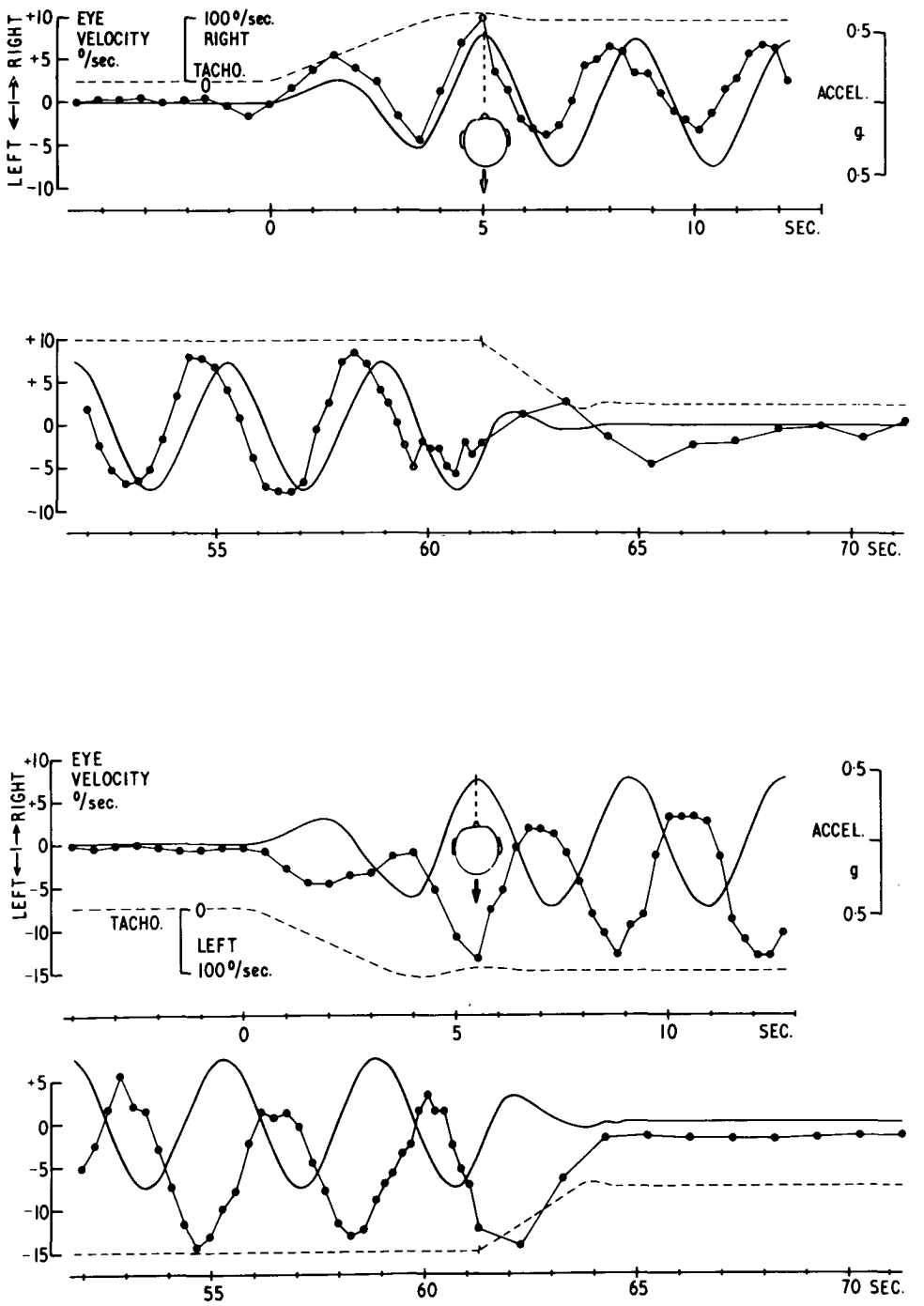


FIGURE 5.—*Angular velocity of the slow-phase component of lateral eye movements at the beginning and end of centrifuge rotation (mean values from eight subjects). The magnitude of the linear acceleration in the x axis is indicated by the continuous line and the speed of rotation by the dashed line. The upper and lower halves of the figure show responses obtained during rotation to the right and to the left, respectively.*

beginning of rotation and increased in amplitude and slow-phase velocity as the speed of rotation increased. These eye movements were caused purely by the changing force environment and not by angular acceleration stimuli to the semicircular canals. Once the speed of centrifuge rotation stabilized, a cyclical direction-changing nystagmus was observed, which was similar in form to that reported by Niven et al. (ref. 10) for a rotating-vector stimulus. This response was maintained over the 60-s period for which rotation was continued, and it decayed rapidly once the centrifuge was brought to rest.

In eight subjects who each experienced counterrotation at 101°/s to the right and to the left, the velocity of the slow-phase eye movements was measured at fixed intervals during the rotational cycle. Mean curves obtained from these data are presented in figure 5. They show clearly the sinusoidal form of the eye-velocity waveform and the lack of a significant poststimulus response. It is also noteworthy that the peak eye velocity, which occurred about 5 s after the beginning of centrifuge rotation, was more or less coincident with the record from the x-axis linear accelerometer. Thus, during rotation of the centrifuge to the right (i.e., in a clockwise direction when viewed in plan) the peak eye velocity to the right occurred when the subject passed through the 0° position (see fig. 3 for nomenclature of vector positions) and was being translated from right to left. Similarly, when the centrifuge rotated in the opposite direction, peak eye velocity to the left occurred when motion of the subject was left to right. From these observations it may be argued that the lateral eye movements produced by the rotating vector are compensatory for translational motion in the transverse (y) axis.

During subsequent revolutions of the centrifuge the peak eye velocity (if regarded as compensatory) was phase advanced upon the peak linear velocity in the y axis. The phase relationship between the stimulus and the response is better shown in figure 6, where eye velocity, averaged over eight revolutions in the steady state, is plotted against vector position. During centrifuge rotation to the right, peak eye velocity to the right occurred about 50° after the force vector passed through the 0° position; with rotation to the left, peak eye velocity to the left was also phase advanced by about 50°. The mean phase advance calculated from the individual records of the eight subjects was 49.5°.

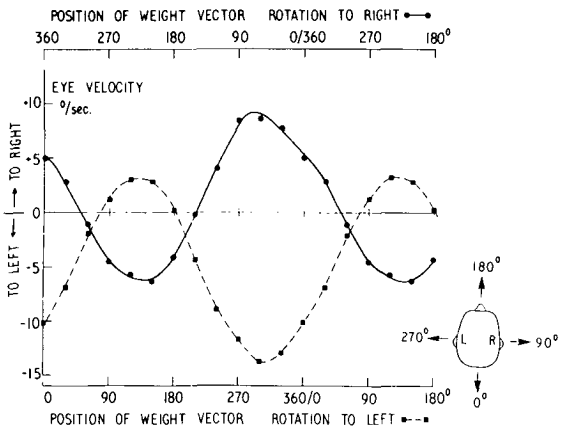


FIGURE 6.—*Relationships between velocity of lateral eye movements and position of the force (or weight) vector during counterrotation at 100°/s (0.45 g). Each point is the mean of 64 observations (8 rev. × 8 subjects). Points for the first half of the rotational cycle are repeated in order to demonstrate the sinusoidal form of the oculomotor response.*

Comparable phase differences were found by Niven et al. (ref. 10) in the lateral eye movements produced by sinusoidal linear oscillation in y body axis. Although these workers expressed the phase error as a lag of eye velocity on acceleration in the y axis, the functional significance of these findings is more apparent if they are considered as a manifestation of phase advance of the eye movement, compensatory to the translational motion in the y axis, upon the linear velocity of the y axis.

The concept of the response being in advance of the stimulus is supported by subjective data. In a subsidiary experiment, subjects were exposed to counterrotation at 77°/s (0.26-g centripetal acceleration) and were asked to signal when they felt they were in the 0° position. Analysis of the results from eight subjects (fig. 7) revealed a mean phase advance of 35.9° in the perception of vector position, a value that was significantly ($p<0.001$) greater than zero. Phase advance in the perception of position during sinusoidal vertical oscillation in

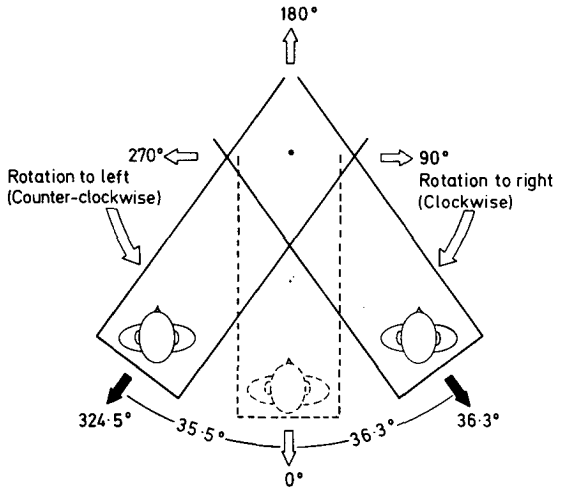


FIGURE 7.—*Mean position of the centrifuge arm when subjects indicated that they were in the 0° position during counterrotation at 77°/s (0.26 g) to the right and to the left (48 observations in each direction from eight subjects).*

the *z* body axis was also reported by Mach (ref. 12) and by Walsh (ref. 13), although the more recent investigation of Meiry (ref. 14) indicated only a progressive increase in phase lag with the frequency of linear horizontal oscillation in the *x* body axis. The significance of these phase measurements as an expression of the dynamics of the otolith organs and their afferent projections awaits interpretation.

The slow-phase velocity of the eye movements evoked by counterrotation were of the characteristic sinusoidal form observed in all experiments in which subjects were exposed to a rotating vector in the *xy* plane. In addition, the plots of slow-phase velocity also exhibit an asymmetry or bias about zero eye velocity. Thus, when vector rotation was to the right, peak eye velocity to the right was greater than to the left; the opposite was true when the centrifuge turned to the left. The mean peak-to-peak magnitude of the sinusoidal component of the response was 18.2°/s when the centrifuge rotated at 101°/s, and the bias component had a mean amplitude of 3.5°/s.

The effect of the rate of rotation of the force vector on the oculomotor response was studied in a separate experiment. Six subjects were exposed to counterrotation at five speeds ranging from 45°/s to 100°/s, which gave centripetal accelerations of 0.09 to 0.45 *g*. Eye velocity was measured over six cycles in the steady state at each rotational speed, and from these measurements mean eye-velocity curves were prepared (fig. 8) and the magnitude of the sinusoidal and bias components calculated. Unfortunately, the construction of the centrifuge did not allow the length of the radius arm to be changed; it was therefore not possible to study separately the effect of the magnitude of the *xy* vector and its rate of rotation.

Figure 8 demonstrates the preservation of the approximately sinusoidal form of the eye-velocity curve at all the speeds of vector rotation employed. The amplitude of both the sinusoidal and bias components was positively correlated with the speed of centrifuge rotation and hence the magnitude of the rotating vector (fig. 9). Statistical analysis showed that the correlation between sinusoidal component and acceleration was significantly better than with angular velocity. The higher variance of the bias component prevented a comparable differentiation to be made for this measure.

The curves suggest that phase angle (peak eye velocity referred to 0° and 180° vector positions) increased as the angular velocity and magnitude of the vector decreased, but this trend was not substantiated by statistical analysis. The mean phase advance was 61.4°.

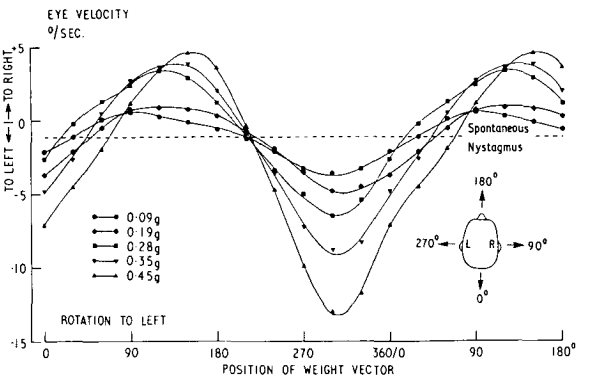


FIGURE 8.—*Relationships between angular velocity of lateral eye movements and position of the force (or weight) vector during counterrotation to the left at speeds of 44.5°/s to 100.3°/s (centripetal accelerations 0.09 g to 0.45 g). Each point is the mean of 36 observations (6 rev. × 6 subjects). The mean velocity of the spontaneous nystagmus of the six subjects when at rest is indicated by the horizontal dashed line.*

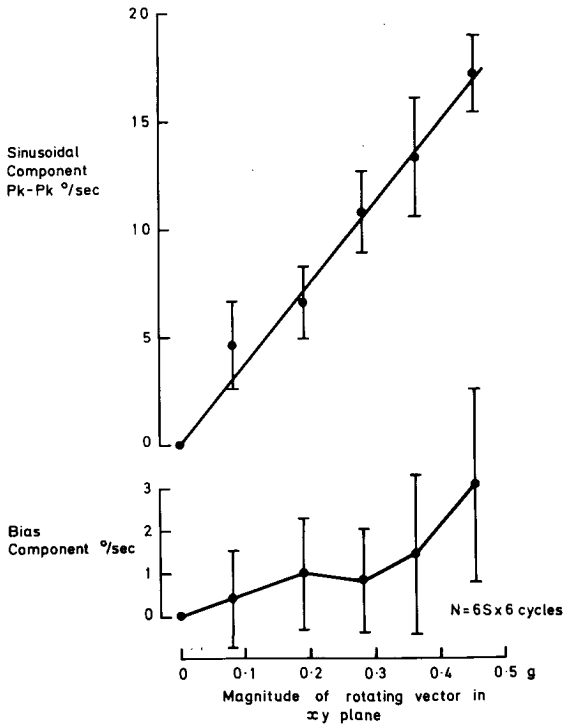


FIGURE 9.—*Relationships between the amplitudes of the sinusoidal and bias components of the oculomotor response to the magnitude of the rotating linear acceleration vector. Each point is the mean of 36 observations (as in fig. 8); the vertical bars show ±1 S.D.*

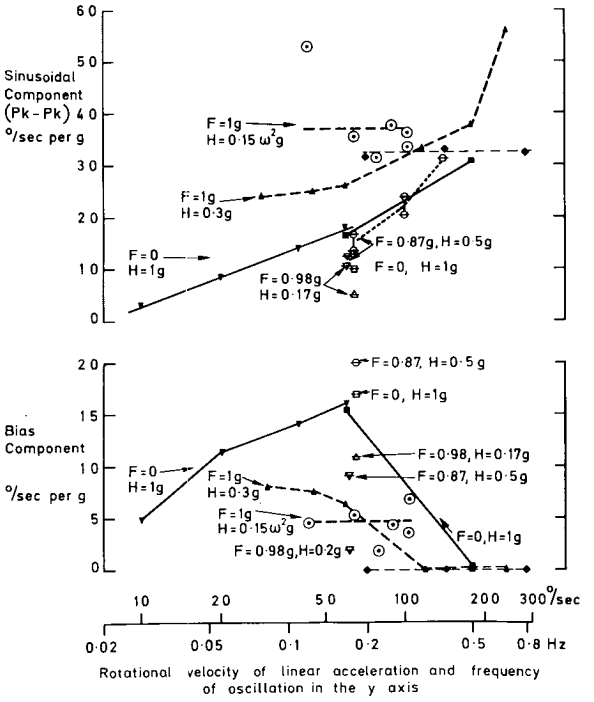


FIGURE 10.—*Comparison of normalized sinusoidal and bias components of lateral eye movements evoked by rotating or oscillating linear acceleration vectors. The solid lines connect mean values from experiments in which the magnitude of the vector was 1 g; the dashed lines indicate resultant accelerations in excess of 1 g.*

▼ — *Benson and Bodin: horizontal z-axis rotation; eight subjects (ref. 2).*

■ — *Correia and Guedry: horizontal z-axis rotation; seven subjects (ref. 3).*

⏢ ⊟ ⊖ — *Stockwell, Turnipseed, and Guedry: rotation about z axis inclined to gravitational vertical; eight subjects (ref. 7).*

▲ — *Steer: rotation about z axis at end of centrifuge arm; radial acceleration (in xy plane) 0.3 g, six subjects (ref. 8).*

◆ — *Niven, Hixson, and Correia: linear oscillation on horizontal track; peak acceleration in y axis ±0.58 g, four subjects (ref. 10).*

⊙ — *Counterrotation; from figure 9.*

▽ — *Benson (unpublished); six subjects.*

COMPARISON OF RESPONSE TO COUNTERROTATION WITH OTHER EXPERIMENTAL FINDINGS

The eye movements recorded during counterrotation exhibited the cyclical change in eye velocity and bias in the direction of vector rotation observed in other experiments in which subjects were exposed to a rotating vector in the *xy* plane (refs. 1 to 4, 7, 8, 10, and 15). However, there were quantitative differences in the magnitudes of the two components of the response that were related to the manner in which vector rotation was achieved. These differences are illustrated in figure 10, where the amplitudes of the sinusoidal and bias components per unit acceleration stimulus (i.e., normalized for 1 *g*) are plotted against the angular velocity of the vector. The use of normalized data facilitates comparison between the different experimental conditions, but there is little experimental evidence, other than that presented in figure 9, to show that the amplitudes of either the sinusoidal or bias components are a linear function of the intensity of the *xy* force vector. The experimental studies, summarized in figure 10, were carried out in different laboratories on different groups of subjects; nevertheless, there is a strong suggestion that rota-

tion of the subject with respect to a centripetal acceleration (as employed by Steer, ref. 8, and in the present experiments) produced a larger sinusoidal component and a lower bias component than when subjects were rotated about a horizontal or inclined axis (refs. 1 to 3 and 7).

It has been proposed that the eye movements produced by a rotating vector might be attributable, at least in part, to the atypical stimulation of ampullary receptors (refs. 2, 8, and 11), though the studies carried out in experimental animals by Janeke (ref. 6) and by Correia and Money (ref. 5) tend to discredit this hypothesis. The experimental evidence favors the view that these eye movements are a manifestation of the adequate stimulation of macular receptors by the rotating force vector (ref. 16).

It is commonly accepted (ref. 17) that it is the specific force in shear acting parallel to the plane of the macula that determines the deflection of the statoconial plaque and the rate of firing of the sensory cells of the macula. If this is so, then comparable responses should be obtained for a given magnitude of rotating vector, irrespective of the magnitude of the component normal to the plane of the macula. But both the sinusoidal and bias components of the response are altered by the intensity of the specific force acting in the longitudinal (z) body axis.

The sinusoidal change in eye velocity associated with a rotating xy vector can readily be attributed to the resolution of the y-axis component of the force vector by otolithic neural mechanisms (ref. 18). The bias component defies such a simple mechanistic explanation. Benson et al. (ref. 11) have postulated that a signal related to the rate and direction of rotation of the vector is derived from the neural integration of impulses from sequentially activated macular cells and is responsible for the bias component. Yet it is difficult to reconcile this hypothesis with the presence of an inverse relationship between the relative magnitudes of bias and sinusoidal components in different experimental conditions. For if the magnitude of the sinusoidal component reflects the intensity of the fluctuating discharge of the macular receptors, then the size of the bias component should alter in a comparable manner, irrespective of the way in which vector rotation was achieved.

In an attempt to explain the nature of the oculomotor responses obtained in the different experimental conditions, the mechanics of a simplified otolith organ exposed to accelerations acting in shear and normal to the macula have been examined.

A THEORETICAL MODEL OF THE OTOLITH ORGAN AND THE LATERAL EYE MOVEMENTS PRODUCED BY A ROTATING LINEAR ACCELERATION VECTOR

In order to attempt an analysis of the behavior of the otolith under dynamic excitation, it is necessary to consider a simplified model of the organ, based on established anatomical features (ref. 19). As shown in figure 11, the statoconia are considered as a concentrated mass supported by a viscoelastic stem that also separates the statoconial plaque from the sensory epithelium of the macula. It is assumed that the elastic restraint of the statoconial mass is provided by the stereocilia of the sensory cells and that the bending of the cilia is proportional to the bending movement of the gelatinous stem.

Equation of Motion For the Statoconial Mass

During exposure to linear accelerations of magnitudes H in shear and F perpendicular to the macula, the equation of motion of the statoconial plaque of effective mass m may be represented as

$$(m\ell D^2+\lambda\ell D+k\ell)\, r=m\ell H+mrF$$

where

r=deflection of the statoconial mass relative to the surface of the macula
λ=damping coefficient of the viscoelastic stem
k=elastic coefficient of the viscoelastic stem
ℓ=length of the stem

and D is the differential operator.

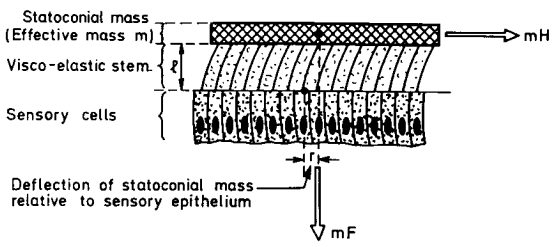


FIGURE 11.—*Diagram of otolith organ with statoconial mass deflected by shear force* mH *and normal force* mF.

Dividing through by $m\ell$ and simplifying gives

$$[D^2+2c\omega_o D+(\omega_o{}^2-\omega_f{}^2)]r=H \qquad (1)$$

where

$$\omega_o{}^2=k/m\ell$$
$$c=\lambda/2\omega_o m\ell$$
$$\omega_f{}^2=F/\ell$$

Response to Step Input of Acceleration in Shear

The steady-state solution of equation 1 is

$$r=\frac{H}{(\omega_o{}^2-F/\ell)} \qquad (2)$$

Thus an acceleration (F) perpendicular to the otolith contributes to its deflection as well as acceleration in shear (H). However, if the shear acceleration is zero, the effect of the perpendicular acceleration is nullified.

Response to a Sinusoidal Input of Acceleration in Shear

The steady-state solution of equation 1 to a sinusoidal input ($H \sin \omega t$) is

$$r=\frac{H \sin(\omega t-\psi)}{\sqrt{[\omega^2-(\omega_o{}^2-\omega_f{}^2)]^2+4c^2\omega^2\omega_o{}^2}} \qquad (3a)$$

where

$$\psi=\tan^{-1}\frac{2c\omega\omega_o}{[\omega^2-(\omega_o{}^2-\omega_f{}^2)]} \qquad (3b)$$

The angle ψ is the phase lag between the input acceleration and the output deflection.

Response to a Constantly Rotating Acceleration in Shear

In order to determine the response of the model to a constantly rotating acceleration in shear, it is necessary to consider its behavior in two dimensions rather than in one as before. The mode of deflection is that shown in figure 12, where the input acceleration is represented by the vector $\underline{H}$. The position of the sensory epithelium of the macula is defined by the fixed set of axes x, y, and the vector $\underline{H}$ is rotated at a constant rate ω with respect to these axes, making an angle ωt with the x axis at time t. It is assumed that the deflection vector $\underline{r}$ also rotates with respect to the fixed set of axes, and consequently equation 1 can be written in vector notation as:

$$[D^2+2c\omega_o D+(\omega_o{}^2-\omega_f{}^2)]r=H \qquad (4)$$

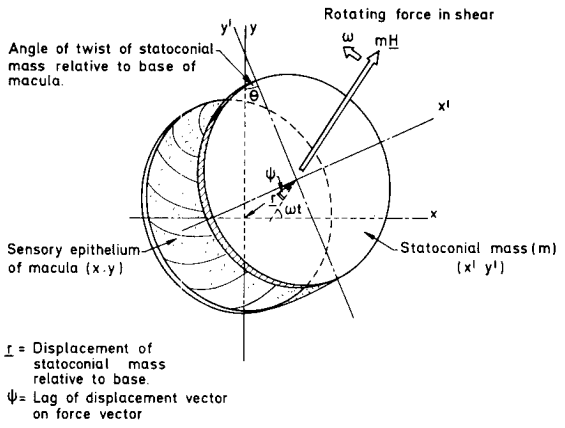


FIGURE 12.—*Diagram of an idealized macula showing translational and torsional displacement of the statoconial mass by a shear force* mH *rotating in the plane of the macula at angular velocity* ω.

Now the acceleration vector $\underline{H}$ may be resolved into orthogonal components along the x and y axes so that

$$H=H \cos \omega t\underline{i}+H \sin \omega t\underline{j} \qquad (5)$$

where $\underline{i}$, $\underline{j}$ are unit vectors in the x and y directions, respectively. Similarly, $\underline{r}$ may be resolved into orthogonal components,

$$\underline{r}=x\underline{i}+y\underline{j} \qquad (6)$$

Substituting equations 5 and 6 in 4 gives two equations for the x and y directions:

$$\begin{aligned}[D^2+2c\omega_o D+(\omega_o{}^2-\omega_f{}^2)]x&=H \cos \omega t\\ [D^2+2c\omega_o D+(\omega_o{}^2-\omega_f{}^2)]y&=H \sin \omega t\end{aligned} \qquad (7)$$

The simplest solution for equation 7 is obtained when H and ω are constant. Then

$$\begin{aligned}x&=\frac{H}{A}\cos(\omega t-\psi)\\ y&=\frac{H}{A}\sin(\omega t-\psi)\end{aligned} \qquad (8)$$

where ψ is given by equation 3b and

$$A=\sqrt{[\omega_o{}^2-(\omega_o{}^2-\omega_f{}^2)]+4c^2\omega^2\omega_o{}^2} \qquad (9)$$

These equations give a constant deflection r given by

$$r=\sqrt{x^2+y^2}=\frac{H}{A} \qquad (10)$$

It can be seen from equation 7 that the deflection vector $\underline{r}$ makes an angle $(\omega t - \psi)$ with the x axis at time t. From this it may be concluded that the deflection vector rotates at the same constant rate ω as the input vector $\underline{H}$, but that it is always lagging behind $\underline{H}$ by the constant angle ψ. As a consequence, the input vector does not pass through the center of the base of the macula, but to one side of it. The offset shear force produces a torque given by the equation

$$T = mHr \sin \psi$$

This torque is resisted by the torsional stiffness of the gelatinous stem, which will thus be twisted. If the stem is assumed to be linearly elastic in torsion, the angle of twist (θ) of the mass with respect to the macula is given by the equation

$$\theta = \frac{Hr \sin \psi}{k_t} \tag{11}$$

where k_t is the torsional stiffness of the stem.

The component $\sin \psi$ is found from equation 3b:

$$\sin \psi = \frac{2c\omega\omega_o}{A} \tag{12}$$

Substituting equations 10 and 12 in 11 and putting $p = \omega/\omega_o$ and $p_f = \omega_f/\omega_o$ gives

$$r = \frac{H}{\omega_o^2 \sqrt{[p^2 - (1 - p_f^2)]^2 + 4c^2p^2}} \tag{13}$$

and

$$\theta = \frac{k_\theta H^2 p}{[p^2 - (1 - p_f^2)]^2 + 4c^2p^2} \tag{14}$$

where $k_\theta = 2c/\omega_o^2 k_t$.

Thus the model predicts that the application of a constantly rotating acceleration in shear would produce not only the expected deflection of the statoconial mass in shear, but also a twist of the statoconial mass with respect to the sensory epithelium of the macula.

Neural Transduction of Statoconial Plaque Motion

Experimental studies of the activity of primary afferent neurons of macular receptors (ref. 20) and of cells in the vestibular nuclei, which relay such macular afferents (refs. 21 and 22), have indicated that alteration in the magnitude of the shear acceleration (H) is signaled by a change in the discharge frequency of the sensory cells. Two components of the neuronal response may be recognized (refs. 20 and 23): the one, a static response in which the discharge frequency is proportional to the sustained shear acceleration; and the other, a dynamic response in which the sensory cell signals the rate of change of the shear acceleration. Now it is known from both anatomical (ref. 24) and electrophysiological studies (refs. 20 to 22 and 25) that the sensory cells of the macula, like those of the ampulla, are optimally stimulated by a bending movement of the stereocilia towards the kinocilium. However, the cells of the macula, unlike those of the ampulla, do not exhibit a uniform directional sensitivity or *morphological polarization*. As shown in figure 13, the directional sensitivities of sensory cells in each half of the macula have an approximately radial distribution and are of opposite polarity on either side of the striola.

But how are the neuronal signals from these multitudinous receptors, with differing directional sensitivities, organized within the central nervous system to produce responses related to the direction and intensity of the specific force? This topic is too broad to be adequately discussed within the framework of this paper. However, it is relevant to describe a possible mechanism for the processing

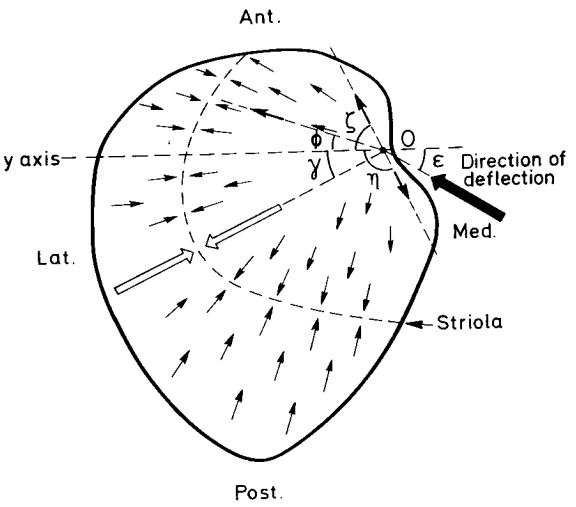


FIGURE 13.—*Plan view of a left utricular macula, illustrating the directional sensitivity of the sensory cells, based on Lindeman (ref. 19). The open arrows represent the directional sensitivity of the integrated neural activity of all the receptors in each half of the macula.*

of signals from the utricular maculae and to attempt to explain the genesis of lateral eye movements by otolith mechanisms.

The assumption central to our argument is that the sensory cells of the macula are not topographically represented within the central nervous system, but that neuronal signals from each half of the macula, as divided by the striola, are handled collectively. The justification for this hypothesis is twofold: First, each afferent neuron is innervated by a number of sensory cells, but this aborizing network does not cross the striola (ref. 19). Second, the organization of macular afferents is likened to ampullary afferents where the physiological response reflects the differential total input from complementary pairs of canals (refs. 26 and 27).

Consider a deflection of the statoconial mass in a direction that makes an angle ϵ with the y axis, as shown in figure 13. The sensory cells, which are morphologically polarized in this direction, will show a maximum change in activity (e) above their resting activity (f). The activity of a cell having a directional sensitivity that makes an angle ϕ to the y axis will be $e \cos(\phi - \epsilon) + f$, for the same displacement of the statoconial plaque (ref. 21). The total signal E from one half of the macula is the sum of the signals from all the receptors with directional sensitivities ranging from ζ to η (fig. 13).

Thus

$$E = \int_{\eta}^{\zeta} [e \cos(\phi - \epsilon) + f] d\phi$$
$$= e[\sin(\zeta - \epsilon) + \sin(\eta + \epsilon)] + F$$

where f is the resting discharge of all cells in each half macula.

Simplifying,

$$E = e \cos(\epsilon + \gamma) + F$$

where $\gamma = (\zeta - \eta)/2$ and $e = e2 \sin[(\zeta + \eta)/2]$. Thus the directional sensitivity exhibited by the integrated activity of a half macula can be represented by an idealized receptor (indicated by the open arrow in fig. 13) with a directional sensitivity at an angle γ to the y axis.

Consider now the sustained deflection of the statoconial mass under the influence of linear accelerations H_x and H_y acting in the sagittal and transverse axes of the skull, respectively. Then the total neural signal (E) from the anteromedial (am) and posterolateral (pl) halves of the right (R) and left (L) utricular maculae may be represented as

$$E_{L(am)} = k(-H_y \cos\gamma - H_x \sin\gamma) + F, \; E_{R(am)}$$
$$= k(+H_y \cos\gamma - H_x \sin\gamma) + F$$
$$E_{L(pl)} = k(H_y \cos\gamma + H_x \sin\gamma) + F, E_{R(pl)}$$
$$= (-H_y \cos\gamma + H_x \sin\gamma) + F$$

where k is a constant of proportionality.

Now it is suggested that the signals from the maculae are processed within the central nervous system by a mechanism similar to that which applies for canal afferents, namely the differential summation of complementary inputs.

Thus, if the left posterolateral and right anteromedial maculae are considered as a complementary pair with the left anteromedial and right posterolateral maculae, then the resulting neural signal is

$$E_x = E_{L(pl)} - E_{L(am)} - E_{R(am)} + E_{R(pl)}$$
$$= 4kH_x \sin\gamma$$

and hence carries information only about the y-axis acceleration. Similarly, if the anteromedial and posterolateral halves of each macula are considered as complementary pairs, then the total signal is

$$E_y = E_{L(pl)} - E_{L(am)} + E_{R(am)} - E_{R(pl)}$$
$$= 4kH_y \cos\gamma$$

which is influenced only by the x-axis acceleration. Since the angle γ is on the order of 45° in man and the other mammals examined by Lindeman (ref. 19), the activity of the two neural centers, represented by E_x and E_y, should have comparable sensitivity for displacement of the statoconial plaque in the x and y axes, respectively (fig. 14).

Under the influence of a force vector ($m\underline{H}$) which rotates in the xy plane, displacement in the x and y axes is described by the periodic function $kH \sin \omega t$, and the statoconial plaque is twisted through an angle θ, which is proportional to the torque developed by the offset shear force. Translational motion of the statoconial plaque in the x and y axes will, according to the postulated mechanism, be resolved by the neural centers (represented by E_x and E_y), which accordingly should exhibit a sinusodial modulation in their activity. On the other hand, torsional displacement of the

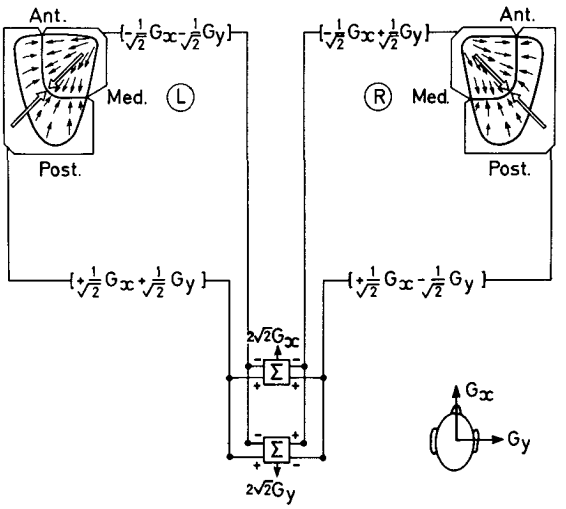


FIGURE 14.—*Diagram of a possible mechanism by which neural signals related specifically to* x- *and* y-*axis accelerations are extracted within the CNS by the differential summation of the activity of complementary half maculae. For the purpose of this diagram the directional sensitivity of each half macula was assumed to make an angle* (γ) *of 45° to the* y *axis. The convention employed for the nomenclature of the specific forces due to positive accelerations in the* x *and* y *axes is shown at lower right.*

statoconial plaque about the center of the macula will stimulate preferentially those receptors that lie toward the periphery of the macula and have a directional sensitivity tangential to the torsional axis of the macula.

Now, if such tangentially oriented receptors were equally distributed in each half of the macula, there would be no alteration in the integrated neural signal when torsional movement of the statoconial plaque occurred. However, this condition is probably not fulfilled, since the sensory cells on the anterior border of the macula are more tangentially oriented than those on the medial border. Hence torsion of the statoconial plaque in a clockwise direction (when viewed in plan) should cause an increase in the integrated activity of the left posterolateral macula, a decrease in the integrated activity of the left anteromedial macula, and the converse alteration in activity of the two halves of the contralateral macula. This would produce a signal (E_y) in the neural center, equivalent to a linear acceleration $+H_y$, but no change in the neural center represented by E_x. Similarly, torsion of the statoconial plaque in a counterclockwise direction would influence E_y in the same manner as a $-H_y$ stimulus. When the linear acceleration vector rotates at a constant rate, it is postulated that the torsional movement of the statoconial plaque is also relatively constant and that the intensity of the neural signal will not be influenced directly by the dynamic response of the sensory cells.

However, the neural transduction of the sinusoidal displacement of the statoconial plaque will be dependent upon an adaptive or rate-sensitive term (ref. 23). Hence,

$$E_y = k_B\theta + k_S r \sin \omega t \sqrt{1+p^2a^2}$$

where a is the sensitivity to the rate of change of deflection of the statoconial plaque, k_B and k_s are constants of proportionality, and r and θ are given by equations 13 and 14.

The final stage in the development of the model is to propose that the neural signal E_y determines the angular velocity of the lateral (horizontal) eye movements evoked by the rotating vector. Here again the model is based on the known organization of ampullary afferents where, for example, the differential signal from the two complementary lateral canals determines the angular velocity of compensatory lateral eye movements (ref. 28). However, for the input from the otolithic maculae, it is necessary to introduce a high-pass filter, similar in principle to that proposed by Mayne (ref. 23), which allows the alternating otolithic signals to be routed to eye-velocity control mechanisms, similar if not identical to those utilized by canal afferents. The low-frequency or steady-state activity determines the compensatory deviation of the eye that occurs in the presence of a linear acceleration vector with constant magnitude and direction. The demonstration of convergence (ref. 29) of canal and otolithic afferents in the vestibular nuclei in animals exposed to a rotating linear acceleration vector (ref. 11) lends support to this hypothesis.

Behavior of the Model Under Specific Conditions

The response of the model to a rotating linear acceleration in shear (H) and a constant normal acceleration (F) was calculated with values of H and F appropriate to those experimental conditions in which eye-velocity measurements have been made in man (fig. 1). These were:

(a) Rotation about an Earth-horizontal axis, where $H=1\ g$, $F=0$.
(b) Rotation in a centrifuge, where $H=0.3\ g$, $F=1\ g$.
(c) Counterrotation, where $H=0.15\omega^2\ g$, $F=1$ g.
(d) Rotation about an inclined axis, where $\alpha=80°$, $H=0.17\ g$, $F=0.98\ g$; or $\alpha=60°$, $H=0.3\ g$, $F=0.87\ g$.

The Bias Component

Since the experimental data were presented normalized for shear acceleration (H), the bias component (B) in normalized form is given by a modified form of equation 14:

$$\frac{B}{H}=\frac{k_B H_p}{[p^2-(1-p_f^2)]^2+4c^2p^2} \qquad (15)$$

where B is the slow-phase eye velocity of the bias component, which is proportional to the angle of twist (θ) of the statoconial plaque.

Equation 15 contains two unknown parameters, c and p_f^2. From the experimentally determined curves relating bias component to the rotational frequency of the H vector, it is apparent that in the $F=0$ condition a peak response occurred at 0.2 Hz or 1.2 rad/s. When equation 15 was plotted against p, this function also exhibited a peak ($\hat{p}$), the position of which may be shown to be a function of the damping coefficient (c). The value of c must be such as to satisfy the equation

$$\hat{p}=\frac{1.2}{\omega_o}$$

The natural frequency of the system can be determined by considering the static deflections of the statoconial mass under the influence of a 1-g acceleration in shear. This will be given by equation 1 (with $F=0$ and $H=1\ g$). However, since the statoconial mass is embedded in a viscous medium of approximately half its density, the effective deflection of the mass will be halved. Therefore,

$$\omega_o^2=g/2r_o$$

where r_o is the deflection under 1-g acceleration in shear. Now if r_o is on the order of 20 μm (ref. 30),

$$\omega_o\approx 500 \text{ rad/s}$$

and

$$\hat{p}=2.4\times10^{-3}$$

The value of the damping coefficient to give this value is

$$c=200$$

This is a very high value and represents a highly overdamped system, but it is not unrealistic.

For condition b, which represents rotation at the end of a centrifuge arm, the normal acceleration (F) is 1 g. But, as in condition a, the effective value of the acceleration is approximately halved. Thus p_f^2 will be given by

$$p_f^2=\frac{g}{2\omega_o^2\ell}$$

By putting $p_f^2=0.5$ in equation 15 and plotting against p, the relative magnitudes of $\hat{p}$ for conditions a and b were obtained. This condition gives a value for ℓ of 40 μm, which is known to be of the correct order (ref. 31).

After the values of parameters c and p_f^2 were determined, the magnitude of the normalized bias component was computed over the frequency range (0.02 to 0.8 Hz) used in the experimental studies. It may be seen from figure 15 that the form of the calculated bias component is in general agreement with the experimental results. In particular, the model accounts for the smaller magnitude of the bias component in condition b than in condition a, and for the frequency shift of the peak response. The model also predicts a falling bias component with increasing frequency, but at a lower rate than that found experimentally.

For counterrotation (condition c), H was frequency dependent. When the appropriate function was substituted for H in equation 15, the model yielded a bias component that increased progressively with frequency and was equal to magnitude to the bias component for condition a at approximately 0.25 Hz and for condition b at approximately 0.18 Hz. The experimental results were not of the predicted form, perhaps because of the high variance of the normalized bias component at low values of H. However, it is of interest that equality with conditions a and b was reached at approximately 0.28 Hz and 0.19 Hz, respectively.

Calculation of the normalized bias for rotation about an inclined axis (condition d) indicated that the response should increase with the angle of tilt from the vertical. With the exception of one point ($H=1\ g$, $F=0$ of Stockwell et al., ref. 7) the

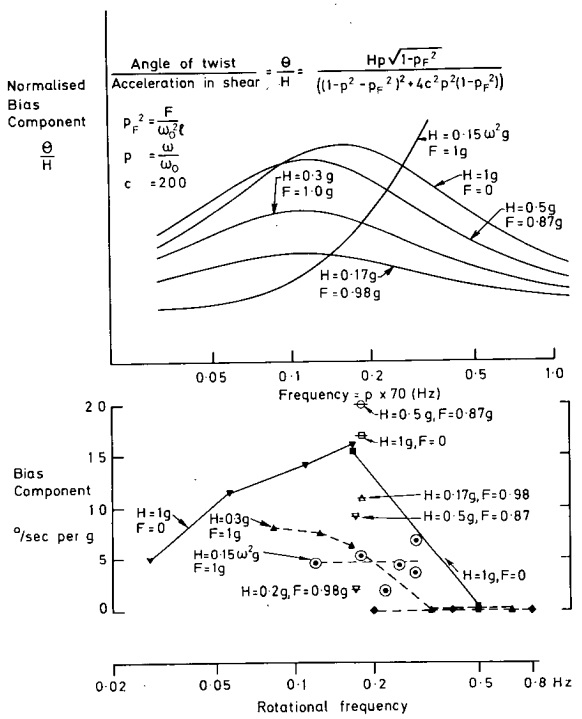


FIGURE 15.—*Comparison of experimentally determined bias component with theoretical model. The lower half of the figure is the same as in figure 10.*

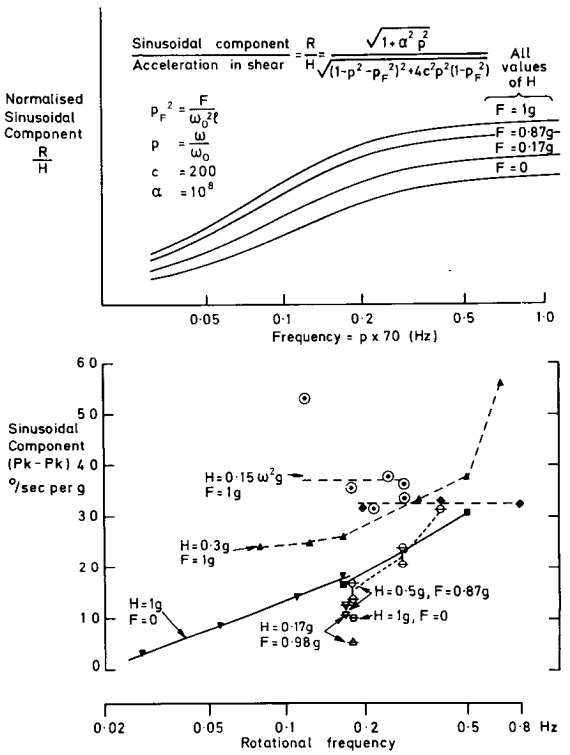


FIGURE 16.—*Comparison of experimentally determined sinusoidal component with theoretical model. The lower half of the figure is the same as in figure 10.*

experimental results are in ordinal agreement with the theoretical prediction, but quantitative differences exist between the replicate measures made in different laboratories.

The Sinusoidal Component

The peak amplitude of the normalized sinusoidal component is given by a modified form of equation 13:

$$\frac{R}{H}=\frac{ks\sqrt{1+p^2a^2}}{\sqrt{[p^2-(1-p_f^2)]^2+4c^2p^2}}$$

where R is the peak amplitude of slow-phase velocity of the sinusoidal component.

The constant a is not known; the value $a=10^8$ was used in order to produce theoretical curves of approximately the same shape as those obtained experimentally. However, the feature of the model lies not in the position of the inflections in the plot of sinusoidal component against rotational frequency (fig. 16), but rather in the prediction that the size of the normalized sinusoidal component is in part dependent upon the magnitude of the normal acceleration (F). As with the bias component, the agreement between the model and experimental results is good for conditions a and b. For counterrotation (condition c) the experimentally determined values tend to be high, especially at the low rotational speeds, but maintain an ordinal relationship to the $F=0$ condition and so are in accord with the theory. Similarly, ordinal agreement is present between calculated and experimental values for inclined-axis rotation, with the exception of the 10° tilt condition ($H=0.17\ g$), where the oculomotor response is of low velocity and the variance of the normalized measure is proportionally high.

CONCLUSION

The model that has been developed attempts to describe the dynamic behavior of the utricular maculae under the influence of a fixed acceleration

acting normal to the surface of the macula and a linear acceleration in shear that rotates at a constant speed. The novel features of the model are that it accounts for the bias component of the oculomotor response by a twisting of the statoconial mass with respect to the base of the macula, and that it presents a parsimonious mechanism for the central integration of neural signals from macular receptors.

The equations derived to describe the magnitude of bias and sinusoidal components give plots which, though by no means exact, are in general agreement with the results of experiments carried out in man. However, agreement between model predictions and experimental results does not necessarily imply that the physiological mechanism is the same as the theoretical model.

REFERENCES

1. GUEDRY, F. E.: Orientation of the Rotation Axis Relative to Gravity: Its Influence on Nystagmus and the Sensation of Rotation. Acta Oto-Laryngol., vol. 60, 1965, pp. 30–49.
2. BENSON, A. J.; AND BODIN, M. A.: Interaction of Linear and Angular Accelerations on Vestibular Receptors in Man. Aerospace Med., vol. 37, 1966, pp. 144–154.
3. CORREIA, M. J.; AND GUEDRY, F. E.: Modification of Vestibular Responses as a Function of Rate of Rotation About an Earth-Horizontal Axis. Acta Oto-Laryngol., vol. 62, 1966, pp. 297–308.
4. SAITO, I.; WADA, H.; YAGURA, S.; AND KATO, N.: On the Influences of Gravitational Acceleration to Nystagmus. Aerospace Med., vol. 6, 1968, pp. 6–12.
5. CORREIA, M. J.; AND MONEY, K. E.: The Effect of Blockage of All Six Semicircular Canal Ducts on Nystagmus Produced by Dynamic Linear Acceleration in the Cat. Acta Oto-Laryngol., vol. 69, 1970, pp. 7–17.
6. JANEKE, J. B.: On Nystagmus and Otoliths. A Vestibular Study of Responses as Provoked by a Cephalo-caudal Horizontal Axial Rotation. Doctoral thesis, University of Amsterdam, 1968.
7. STOCKWELL, C. W.; TURNIPSEED, G. T.; AND GUEDRY, F. E. JR.: Nystagmus Responses During Rotation About a Tilted Axis. NAMRL–1129. Naval Aerospace Medical Research Laboratory, Pensacola, Fla., 1971.
8. STEER, R. W. JR.: The Influence of Angular and Linear Acceleration and Thermal Stimulation on the Human Semicircular Canal. Sc.D. thesis, Massachusetts Institute of Technology, 1967.
9. 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 Counterrotating Room. Ann. Otol., vol. 72, 1963, pp. 357–373.
10. NIVEN, J. I.; HIXSON, W. C.; AND CORREIA, M. J.: Elicitation of Horizontal Nystagmus by Periodic Linear Acceleration. Acta Oto-Laryngol., vol. 62, 1966, pp. 429–441.
11. BENSON, A. J.; GUEDRY, F. E.; AND MELVILL JONES, G.: Response of Semicircular Canal Dependent Units in Vestibular Nuclei to Rotation of a Linear Acceleration Vector without Angular Acceleration. J. Physiol., vol. 210, 1970, pp. 475–494.
12. MACH. E.: *Grundlinien der Lehre von den Bewegungsempfindungen.* Englemann, Leipzig, 1875.
13. WALSH, E. G.: Sensations Aroused by Rhythmically Repeated Linear Motion-Phase Relationships. J. Physiol., vol. 155, 1960, pp. 53P–54P.
14. MEIRY, J. L.: The Vestibular System and Human Dynamic Space Orientation. NASA CR–628, 1966.
15. BENSON, A. J.: Lateral Eye Movements Produced by a Rotating Linear Acceleration Vector. J. Physiol., vol. 197, 1968, pp. 85P–86P.
16. GUEDRY, F. E.: Effects of Concomitant Stimulation of the Semicircular Canals and Otoliths by "Barbecue Spit" Rotation, Rotation about a Tilted Axis and Other Forms of Stimulation. Excerpta Med. Intern. Cong. Ser. 206, Amsterdam, 1970, pp. 440–448.
17. JONGKEES, L. B. W.: On the Otoliths: Their Function and the Way to Test Them. Third Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–152, 1968, pp. 307–330.
18. YOUNG, L. R.: Effects of Linear Acceleration on Vestibular Nystagmus. Third Symposium on the Role of the Vestibular Organs in Space Explorations, NASA SP–152, 1968, pp. 383–391.
19. LINDEMAN, H. H.: Studies on the Morphology of the Sensory Regions of the Vestibular Apparatus. Ergbn. Anat. EntwGesch., vol. 42, 1969, pp. 1–113.
20. LOWENSTEIN, O.; AND ROBERTS, T. D. M.: The Equilibrium Function of the Otolith Organs of the Thornback Ray *Raja Clavata.* J. Physiol., vol. 110, 1950, pp. 392–415.
21. MILSUM, J. H.; AND MELVILL JONES, G.: Trigonometric Resolution of Neural Responses from the Vestibular Otolith Organ. Digest 7th Intern. Conf. Med. Biol. Eng., Stockholm, 1967, p. 203.
22. MELVILL JONES, G.; AND MILSUM, J. H.: Neural Responses of the Vestibular System to Translational Accelerations. 1970, in press.
23. MAYNE, R.: Physics of the Vestibular Organs. Handbook of Sensory Physiology, vol. VI, Heidelberg, Springer-Verlag, 1970, in press.
24. SPOENDLIN, H. H.: Ultrastructural Studies of the Labyrinth in Squirrel Monkeys. The Role of the Vestibular

Organs in the Exploration of Space, NASA SP–77, 1965, pp. 7–22.

25. BEERENS, A. J. J.: Stimulus Coding in the Utricular Nerve of the Cat. Doctoral thesis, University of Amsterdam, 1969.
26. LOWENSTEIN, O.: Peripheral Mechanisms of Equilibrium. Brit. Med. Bull., vol. 12, 1956, pp. 114–118.
27. SHIMAZU, H.; AND PRECHT, W.: Inhibition of Central Vestibular Neurons From the Contralateral Labyrinth and its Mediating Pathway. J. Neurophysiol., vol. 29, 1966, pp. 467–492.
28. OUTERBRIDGE, J. S.: Experimental and Theoretical Investigation of Vestibularly Driven Head and Eye Movements. Thesis. McGill University, 1969.
29. DUENSING, F.; AND SCHAEFER, K. P.: Über die Konvergenz Verschiedener Labyrinthärer Afferenzen auf Einzelne des Vestibulariskerngebietes. Arch. Psychiat. Nervenkr., vol. 199, 1959, pp. 345–371.
30. DE VRIES, H.: The Mechanics of the Labyrinth Otoliths. Acta Oto-Laryngol., vol. 38, 1950, pp. 262–273.
31. IGARASHI, M.: Dimensional Study of the Vestibular End Organ Apparatus. Second Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–115, 1966, pp. 47–54.

Systems Analysis of the Vestibulo-Ocular System*

ROBERTO M. SCHMID
Istituto di Elettrotecnica ed Elettronica, Laboratorio di Controlli Automatici, Politecnico di Milano, Italy

SUMMARY

The vestibulo-ocular system is examined from the standpoint of system theory. The evolution of a mathematical model of the vestibulo-ocular system in an attempt to match more and more experimental data is followed step by step. The final model explains many characteristics of the eye movement in vestibularly induced nystagmus. The analysis of the dynamic behavior of the model at the different stages of its development is illustrated in time domain, mainly in a qualitative way.

INTRODUCTION

Most of the problems related to clinical investigations of biological systems may be stated as identification problems. Sometimes it is a matter of simple parametric identification, since the anatomy and the physiology of the system under consideration are perfectly known. In other cases one has to deal with more complex identification problems, involving both the structure of the system and the values of its parameters.

Black-box identification of dynamic systems consists in discovering the dynamics of a system from the recording of its response to a proper stimulus. Mainly for linear systems, there are very powerful techniques for solving this problem (ref. 1).

In the specific case of the vestibular system, this approach is clinically impossible, since the output of the vestibular system cannot be measured directly. Therefore, one has to look for an indirect measurement of the vestibular output. Since the vestibular system interacts with the oculomotor system, and the output of the latter, that is, eye movement, can be easily measured, the first idea was to use eye movement in vestibularly induced nystagmus for an indirect measurement of the vestibular output.

It is a common practice in the investigation of the semicircular canals to assume that nystagmus slow-phase velocity is proportional to cupula deflection. Since the relationship between head angular rotation and cupula deflection has been well established on the basis of many theoretical and experimental studies (refs. 2 to 5), slow-phase eye velocity is used to identify canal parameters (refs. 6 to 8). However, the assumption that the dynamics of the neural processing may be described by a simple integrator leads to an oversimplified model which cannot explain many important phenomena, first of all adaptation.

Young and Oman (ref. 9) and Malcolm (ref. 10) have proposed two more sophisticated models that can predict adaptation. The entire pattern of the nystagmus beats, their variable amplitude and frequency, remain unexplained by these models. An attempt in this direction has been made by Sugie and Melvill Jones (ref. 11) and by Outerbridge (ref. 12). On the other hand, their models cannot explain adaptation.

At this point it is worthwhile to seek a deeper insight into the mechanism that produces eye movement in vestibular nystagmus. This paper presents the evolution of a mathematical model in an attempt to match more and more experimental data. Restricting the investigation to eye movement produced by rotational stimuli of the lateral semicircular canals, an attempt is made first to explain

* Work supported by C.N.R., Rome, Italy.

some characteristics of the nystagmus patterns, mainly in sinusoidal rotational tests. The model is then modified to explain also adaptation and secondary nystagmus in postrotational tests. Finally, a last modification is introduced to justify a nonzero final value of the cumulative eye position and the possible existence of a third phase in postrotational nystagmus.

For simplicity, some minor mathematical details are purposely avoided, while the analysis of the dynamic behavior of the model at the different stages of its development is illustrated in time domain, mainly in a qualitative way.

FIRST-APPROXIMATION MODEL

The starting point of the present research has been a very interesting information flow diagram proposed by Melvill Jones and Milsum in 1965 (ref. 13) to interpret the main neurological pathways of the visual stabilization system. In their diagram the central nervous system (CNS) receives information from four main feedback channels: visual, vestibular, and two proprioceptive (eye in skull, skull on body). Using this information, the CNS produces, by means of the extraocular muscles, rotation of eyeballs in their orbits such that the fovea remains stationary on a moving target.

Melvill Jones and Milsum's information flow diagram becomes greatly simplified if it is referred to the operating conditions usually realized in rotational tests for clinical investigations of the lateral semicircular canals. Assuming a subject sitting on a chair rotating about a vertical axis in total darkness with body and skull fixed relative to the chair, refer to the information flow diagram in figure 1. The visual and the neck proprioceptive channels are not considered. The reference signal θ_R for visual fixation is set equal to zero, since it is also assumed that the subject will not maintain a fixed gaze in a particular direction. The angles defined in the inset "uniocular" sketch in figure 1 represent the rotations in the horizontal plane of the skull and body ($\theta_s=\theta_b$), eye (θ_e), and eye in skull ($\theta_{er}=\theta_e-\theta_s$).

The dynamics of the pair of lateral semicircular canals may be described by a linear block in spite of the unidirectional sensitivity of the vestibular sensory hair cells (ref. 14). Actually, the two canals

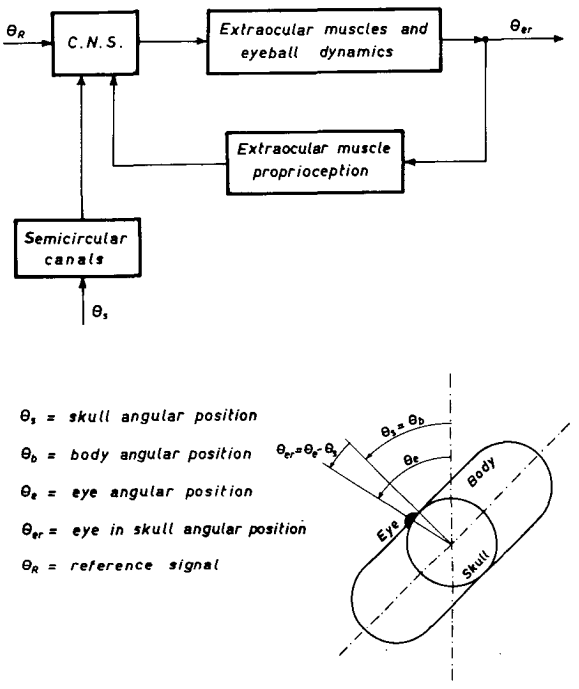


FIGURE 1.—*Simplified information flow diagram for the vestibulo-ocular system.*

act as a push-pull system, thus canceling the effect of the nonlinear response of the single canals (ref. 15). Assuming the same static and dynamic behavior for all the elements in the two parallel pathways of the push-pull system,* and owing to the consequent linearity of the overall system, the output θ_v of the complex of the two canals and their nerve processes can be related to skull angular position θ_s by the following transfer function:

$$\frac{\theta_v(s)}{\theta_s(s)}=\frac{-k_1 s^2}{(1+sT_1)\ (1+sT_2)} \qquad (1)$$

where T_1 and T_2 are the short and long time constants, respectively, of the canals. They have been assumed to be the same for both the canals. Their relationship to canal physical parameters has been well established (ref. 16).

* This assumption turns out to be fairly true for normal subjects. It would be interesting to deduce eventual differences in the static and dynamic characteristics of the two canals from the analysis of the nonlinearity in the overall response.

The negative sign has been introduced into the transfer function of equation 1 to point out that head and eye movement should be in opposite directions to obtain compensation.

Attempting to construct a model for the remaining part of the system, one has first to answer the following question: What is the role (if any) of the extraocular muscle receptors in vestibularly driven eye movement? Stretch reflex in human extraocular muscles has been thoroughly discussed by Robinson in his excellent review of the oculomotor control system (ref. 17). Reporting the divergent opinions of different investigators, he concluded, "At the moment the situation can only be summarized by saying that, although stretch afferent signals exist, no one knows with any certainty where in the brain stem they go and what use the central nervous system makes of them."

From an automatic-control-system point of view, with reference to the diagram in figure 1, the problem can be stated as follows: Do the extraocular muscle receptors play an important role so that the vestibulo-ocular system behaves as a closed-loop control system even in the absence of the visual information, or is their role so unessential that the vestibulo-ocular system should be considered an open system when the visual feedback is excluded?

By accepting the first hypothesis and following a tempting suggestion by Milsum that separate position and velocity proprioceptive units are concerned with eye position and velocity control, respectively (ref. 18), the model in figure 2 can be imagined.

Two interacting feedback control loops govern smooth and saccadic eye movement, respectively. The reference signal of the first loop, shown in the lower part of figure 2, is the vestibular output θ_v, which is compared with the output of the velocity proprioceptive units considered as feedback transducers of eye angular velocity. The difference signal θ_d is then integrated and added to the output of a block representing the mechanism for saccadic movements to obtain the force applied to the extraocular muscles. Integration is needed because the output of the semicircular canals is nearly proportional to skull angular velocity over the frequency range of natural head movements (refs. 13 and 19), while compensation should be obtained for head position. The reference signal θ_R for the saccadic control loop, shown in the upper part of figure 2, is set equal to zero for the above-mentioned reasons. A measure of relative angular position of the eye is fed back to the block controlling the saccadic movements by the position proprioceptive units.

Examining the model illustrated in figure 2, one

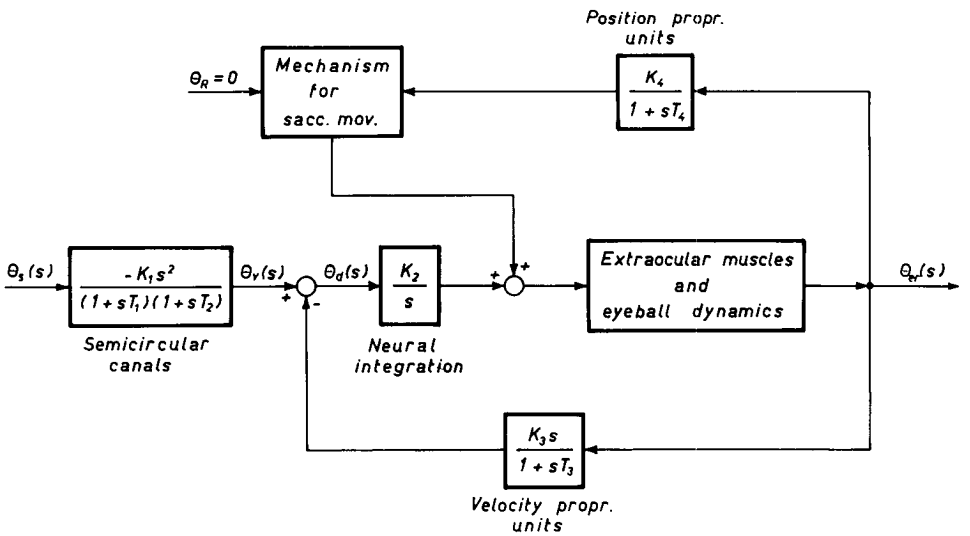


FIGURE 2.—*Model of the vestibulo-ocular system with separate position and velocity proprioceptive units.*

is led to the conclusion that the nystagmus fast phase should disappear when the extraocular muscle receptors are made ineffective. There is a good deal of experimental evidence against this fact. McCauch and Adler (ref. 20), showed long ago that the fast phase is not triggered by a peripheral mechanism such as proprioception, and their results have been later confirmed by other investigators (ref. 21). The simple way to overcome this point is to imagine a central model of the oculomotor system receiving information from the vestibular system and generating a signal θ'_{er}, proportional to eye relative angular position as shown in figure 3. It is tempting to speculate that the information given by the position proprioceptive units is used to adjust the parameters of this central model.

For purposes of simulation and to facilitate the analysis of the model's dynamic behavior, it is perfectly equivalent to assume that the signal θ'_{er} is directly obtained by multiplying the output θ'_{er} by a constant K_5. This is what will be done later on.

The feedback through the velocity proprioceptive units may be thought of as an inner control loop included within the complete system, which uses the visual feedback for the main feedback control loop. Actually, control engineers often introduce this kind of feedback compensation to improve the dynamic characteristics of their control systems. Since there is a great deal of physiological evidence that the nystagmus slow phase is also influenced very little by the extraocular muscle stretch reflex (ref. 21), the model in figure 3 can be simplified by dropping this feedback pathway.

The model proposed for the mechanism for saccadic movements is very close to that suggested by Young to explain saccades in his sampled-data model for eye-tracking movements (refs. 22 and 23). Actually, there is no consistent reason for thinking of different mechanisms for saccadic jumps in eye tracking and in vestibular nystagmus.

As shown in figure 4, which illustrates the complete first-approximation model, a signal proportional to relative angular position of the eye is compared with a reference signal $\theta_R=0$. When the error signal overcomes a threshold value of α, it goes into a sampler, which operates at sampling interval T. The synchronization of the sampler is assumed to be set to coincide with the beginning of

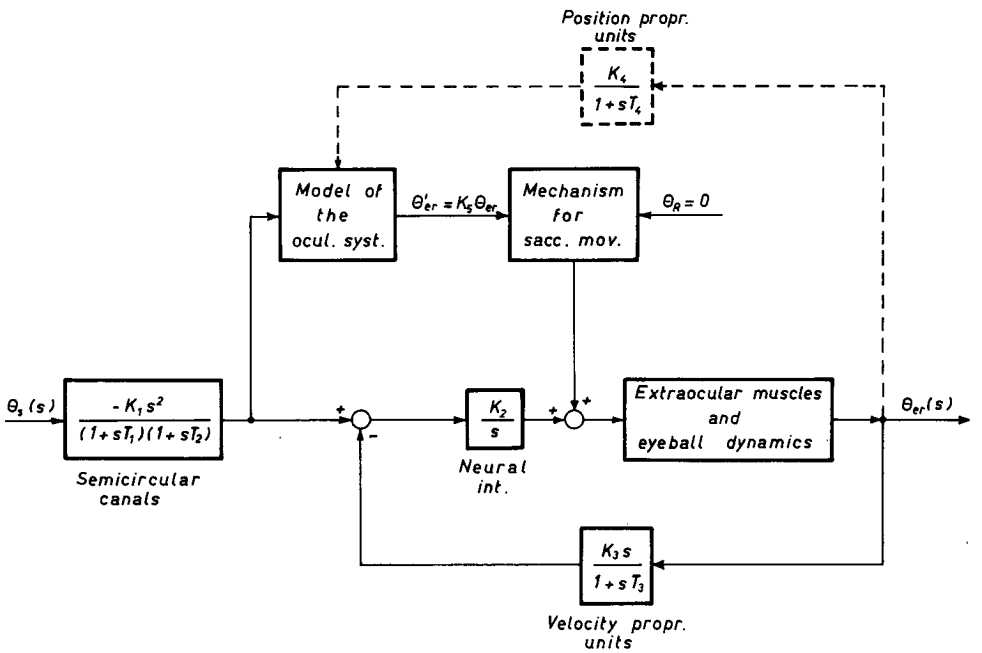


FIGURE 3.—*Model of the vestibulo-ocular system in which a signal θ'_{er} proportional to eye relative angular position is generated by a central model of the oculomotor system.*

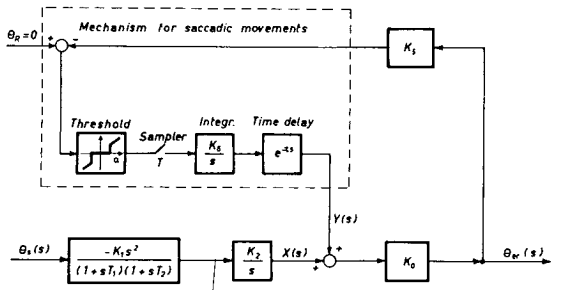


FIGURE 4.—*First-approximation model.*

eye motion. The impulses generated by the sampler are delayed by τ seconds and integrated to give the steps that produce the fast component of the nystagmus. Good results in fitting experimental data have been obtained by taking $K_5=1$ (this value has been fixed arbitrarily*), $\alpha=6°$ to $7°$, $T=200$ to 220 ms, and $\tau=160$ to 180 ms. These values of T and τ are in good agreement with the mean values indicated by Young et al. for the average refractory period for saccadic movements and for the reaction time, respectively (refs. 22 and 23).

To describe the dynamics of the extraocular muscles and eyeballs, the transfer function proposed by Westheimer (ref. 24) or that suggested by Robinson (ref. 17) can be used. Even if these transfer functions are quite different, both indicate that the dynamics of the extraocular muscles and eyeballs is extremely fast when compared with the long time constant of the semicircular canals. Thus, another simplification has been introduced, neglecting the dynamics of this block, which has been characterized only by a constant gain K_0.**

TIME-DOMAIN ANALYSIS OF THE FIRST-APPROXIMATION MODEL

A detailed mathematical analysis of the model in figure 4 has been presented elsewhere (ref. 15). The aim of this section is to describe the dynamic behavior of the first-approximation model in time domain, mainly in a qualitative way.

Assume that a sinusoidal rotational test has to be simulated. The input $\theta_s(t)$ will be a sinusoidal function. At steady state, the output $\theta_v(t)$ of the pair of canals and the output $x(t)$ of the integrator in the primary pathway will also be sinusoidal.

Let the instant at which $x(t)$ goes through zero be considered, and assume that at the same instant the output $y(t)$ of the mechanism for saccadic movements is also equal to zero. At the end of this analysis it will be clear that $y(t)$ is practically equal to zero at the end of each period of $x(t)$. Finally, for the sake of simplicity, let the instant considered coincide with a sampling instant, and let also K_0, K_5, and k_6 be equal to 1.

Since $K_0=1$, as long as $x(t)$ is less than the threshold value of α, it follows that

$$\theta_{er}(t)\equiv x(t)$$

(first slow component in curve A of fig. 5).

When the threshold is reached or exceeded at a

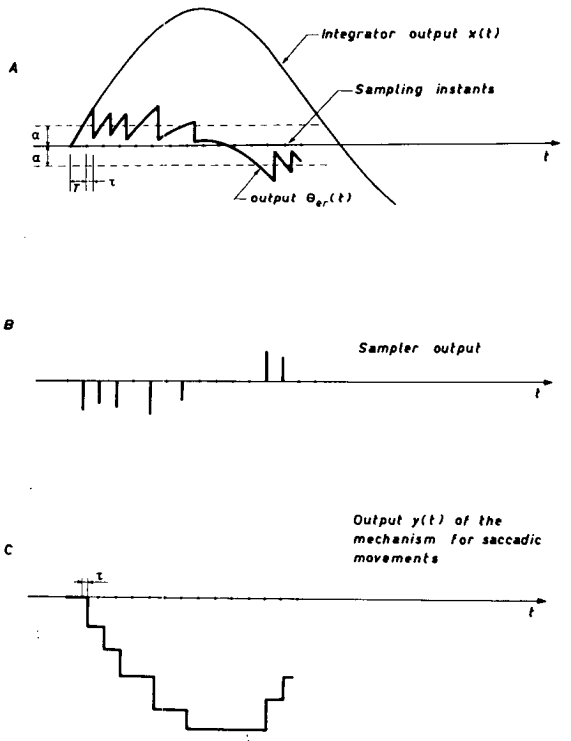


FIGURE 5.—*Qualitative response of the first-approximation model to a sinusoidal input.*

* It should be noted that, from the analysis of the nystagmus records, one can get an idea of the approximate value of the product $K_5\alpha$. No suggestion can be derived for the value of each one of the two parameters K_5 and α.

** This simplification implies that the fast components in the simulated nystagmus follow a vertical line instead of decaying exponentially.

sampling instant KT, the sampler generates an impulse (curve B), which is converted into a step with amplitude equal to $\theta_{er}(KT)$, since $K_5=K_6=1$. τ seconds later, $y(t)$ assumes instantaneously the step value (curve C), and the first fast component is produced. The output θ_{er} does not return exactly to zero; because of the time delay τ, there is a "reset error" equal to $\theta_{er}(KT+\tau)-\theta_{er}(KT)$.

After the end of the first fast component,

$$\theta_{er}(t)=x(t)-\theta_{er}(KT)$$

until the threshold is reached again at a sampling instant (second slow component in curve A), and so on.

If K_0 has to be set at other than 1, it is still possible to fix arbitrarily $K_5=1$ in order to maintain the value of α unchanged, but K_6 must be taken as equal to $1/K_0$.

Even from this rough description of the model behavior, one can easily understand that the model in figure 4 can predict the following charactertistics of eye movement in vestibularly induced nystagmus.

(a) The amplitude of the nystagmus beats is not constant.

(b) The duration of the slow components is not constant.*

(c) The eyes do not always return to the resting position at the end of the fast component, and the reset error is not constant.

There is another characteristic of the vestibular nystagmus produced by head sinusoidal rotation

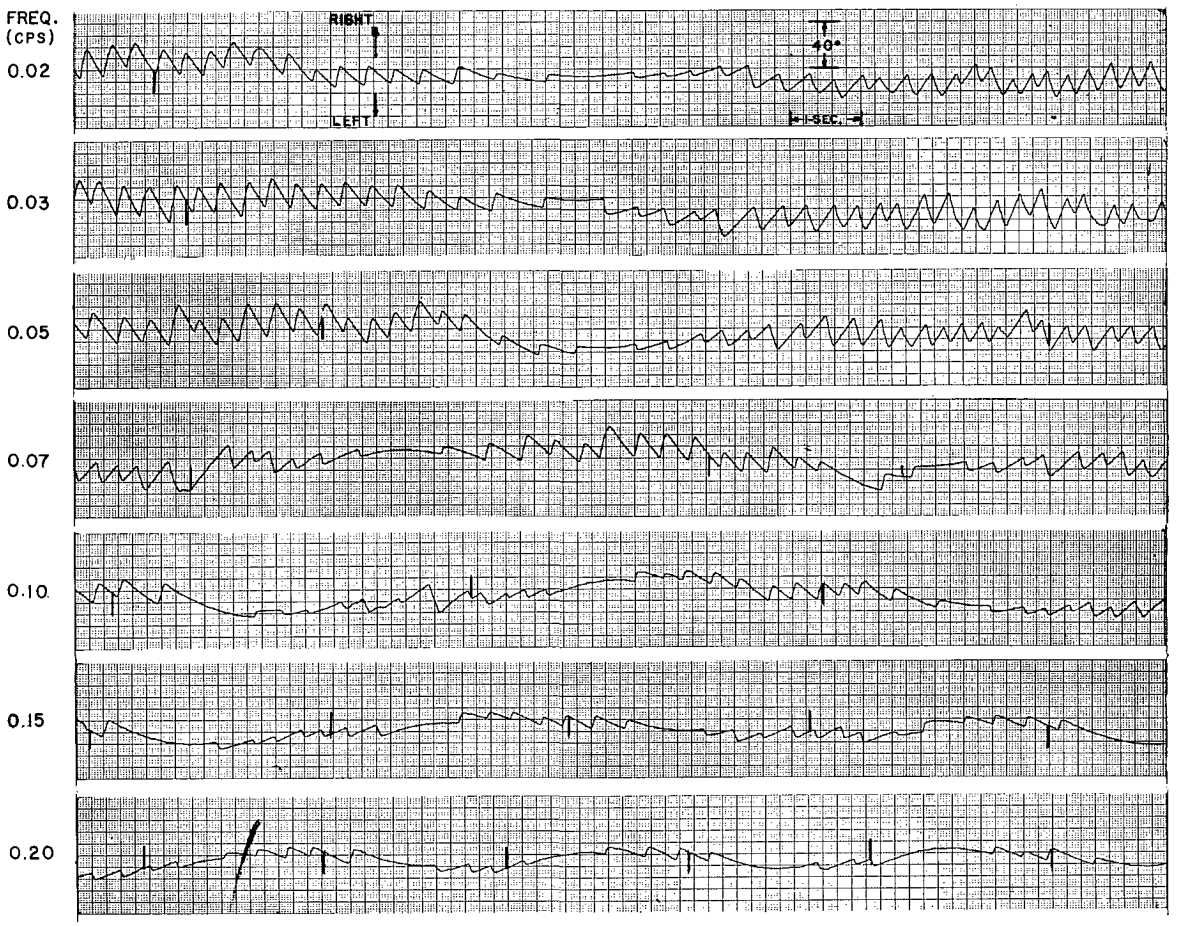


FIGURE 6.—*Recordings of horizontal eye nystagmus arising from rotational stimulation of horizontal semicircular canals with sinusoidal angular accelerations of variable frequency and fixed magnitude. (from ref. 25)*

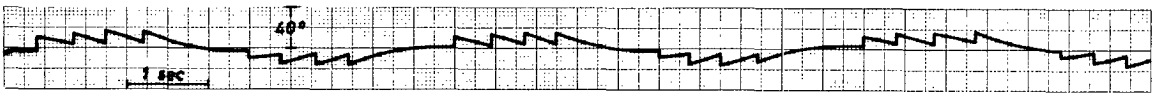


FIGURE 7.—*Result of the simulation of the sinusoidal rotational test which gave the last tracing in figure 6. Model input:* $\theta_s(t) = 25.5 \sin 0.4\pi t$. *Model parameters:* T_1, *neglected;* $T_2 = 16$ *s;* $K_0 = K_5 = K_6 = 1$; $K_1 \cdot K_2 = 14$; $a = 6°$; $T = 200$ *ms;* $\tau = 180$ *ms.*

that can be explained by this model. Figure 6 shows some dc recordings of horizontal eye nystagmus obtained by Niven et al. (ref. 25) during sinusoidal rotational tests of variable frequency and fixed peak angular acceleration. Examining these records, one can easily note that, as the frequency increases, the nystagmus beats seem to modulate a sinusoidal wave. In other words, the reset error varies sinusoidally, and its amplitude increases with the frequency.

Such behavior can be easily explained on the basis of the model in figure 4, if the reset error is expressed in terms of cumulative eye position θ_{ec}. By definition, the cumulative eye position is obtained by eliminating the fast phase of nystagmus and fitting together the smooth portions (ref. 26). Thus, the cumulative eye position is given by the model output when the saccadic pathway is maintained open; that is,

$$\theta_{ec}(t) = K_0 x(t)$$

If the input θ_s (t) is sinusoidal, $x(t)$ and then $\theta_{ec}(t)$ are also sinusoidal at steady state. Therefore, it can be written as

$$\theta_{ec}(t) = A \sin \omega t \qquad (2)$$

Carrying on simple calculations (ref. 15), it is possible to prove that the reset error at the end of the ith fast component is given by

$$e(K_iT+\tau) = \theta_{ec}(K_iT+\tau) - \theta_{ec}(K_iT) \qquad (3)$$

Then, from equation 2,

$$e(K_iT+\tau) = A \sin \omega(K_iT+\tau) - A \sin \omega K_iT \qquad (4)$$

where K_i is an integer depending on the number of sampling intervals which have preceded the ith fast component.

The discrete function (equation 4) can be considered as obtained by sampling the continuous time function

$$e(t) = B \cos \omega \left(t + \frac{\tau}{2}\right) \qquad (5)$$

at the instants $K_iT+\tau$. The peak amplitude B is given by

$$B = 2A \sin \frac{\omega \tau}{2} \qquad (6)$$

From equation 5 it follows that the reset error predicted by the model is a discrete sinusoidal function. From equation 6 it follows that the ratio between the peak amplitude B of the reset error and the peak amplitude A of the cumulative eye position increases with the frequency.

Finally, the model in figure 4 predicts for sinusoidal head rotations of fixed frequency a number of nystagmus beats in a period of the stimulus increasing with the amplitude of the stimulus. However, there is an upper limit on such a number given by

$$N(f) = \frac{1}{fT}$$

where f is the frequency of the stimulus and T is the sampling interval. This kind of saturation has been noted by others (ref. 27).

Figure 7 shows the result of the simulation of the sinusoidal rotational test which gave the last tracing in figure 6 ($f = 0.2$ Hz). The values assumed for the model parameters are indicated in the same figure.

Unfortunately, the model in figure 4 fails in predicting the system response to postrotational stimuli. If a velocity step input is considered, the signals $x(t)$ and $y(t)$ will follow the pattern indicated in curves B and C, respectively, of figure 8. At steady state, the output $\theta_{er}(t)$, which depends on $x(t)$ and $y(t)$, is zero, but the initial resting conditions are never reached again. This statement

* The model predicts that the duration of the slow components is always an integer multiple of the sampling period.

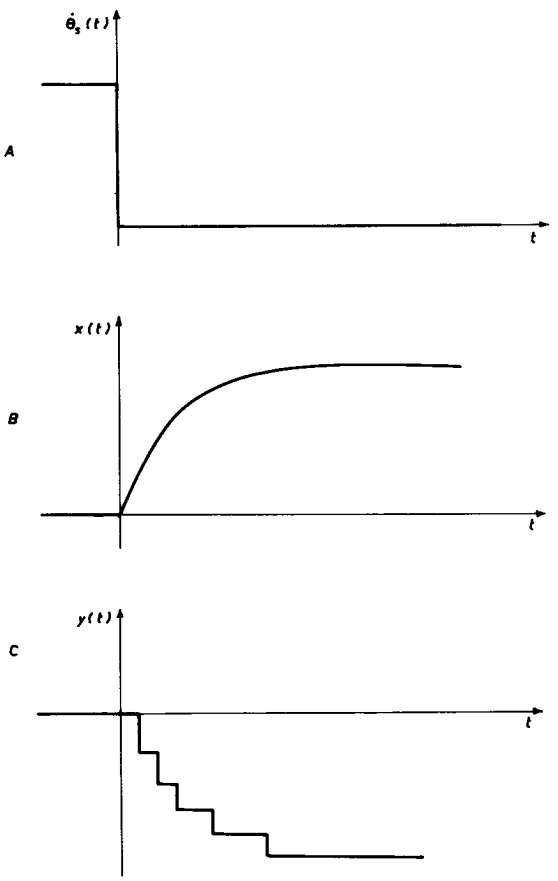


FIGURE 8.—*Qualitative response of the first-approximation model to a velocity step input.*

obviously cannot be accepted. Moreover, the signal $x(t)+y(t)$ will always maintain the same sign, so no secondary nystagmus can be expected. Finally, the cumulative eye position will have the same pattern as $x(t)$ (curve B), while the experimental data indicate for $\theta_{ec}(t)$ a kind of adaptation. That is, $\theta_{ec}(t)$ increases exponentially with a time constant approximately equal to the canal long time constant, reaches a maximum, and then decreases very slowly.

SECOND-APPROXIMATION MODEL

In order to make the model able to predict also the system response to postrotational stimuli, the diagram in figure 4 must be slightly modified. Namely, the perfect integrator in the primary pathway has to be transformed into an "integrator with leak," as shown in figure 9, which gives the block diagram of the second-approximation model.

The transfer function of the integrator with leak is given by

$$\frac{X^*(s)}{\theta_v(s)} = \frac{\mu_1}{1+sT_a}$$

where $\mu_1=1/\beta$, $T_a=1/K_2\beta$, and β is the leak factor.

Assuming the time constant $T_a \gg T_2$ ($T_a \approx 100$ s), the frequency response of the continuous part of the model will not change very much. In particular, the analysis of the model response to a sinusoidal

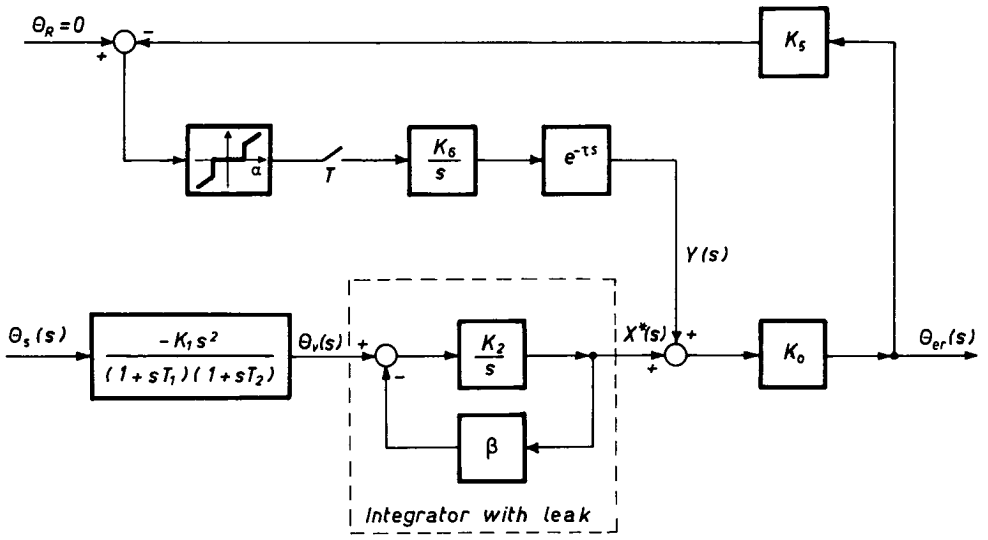


FIGURE 9.—*Second-approximation model.*

input carried out in the preceding section will give the same results if it is referred to the diagram in figure 9 instead of that in figure 4.

Taking the transfer function between nystagmus slow-phase velocity and head angular acceleration results in

$$\frac{\dot{\theta}_{ec}(s)}{\ddot{\theta}_s(s)} = \frac{K(s)}{(1+sT_1)(1+sT_2)(1+sT_a)} \qquad (7)$$

where $K=-K_1\mu_1K_0$. This transfer function is of the same kind as that proposed by Young and Oman (ref. 9) to explain adaptation. The values indicated by those authors for the time constant T_a and the gain K are 125 s and 840 s, respectively. These values can be obtained by fixing $K_1=K_0=1$ and letting $\beta=1.2\times10^{-3}$ and $K_2=6.7$.

Figure 10 shows in a qualitative way the model response to a velocity step input. At steady state, the signals $x^*(t)$ (curve B) and $y(t)$ (curve C) return to zero, as does the output $\theta_{er}(t)$. Thus the initial resting conditions are restored. The signal $x^*(t)+y(t)$ changes its sign when the output of the integrator with leak begins to decrease, and a secondary nystagmus appears after a latency period due to the fact that the maximum of $x^*(t)$ is very flat ($T_a \gg T_2$) (curve D). During the latency period it is

$$K_0K_5 \mid x^*(t)+y(t) \mid <\alpha$$

and thus there are no nystagmus beats.

It should be noted that during the nystagmus latency period the output $\theta_{er}(t)$ is neither always zero nor constant. In other words, the model in figure 9 does not predict any discontinuity in slow-phase eye velocity from primary to secondary nystagmus. The latency period has been explained by Young and Oman (ref. 9) by introducing a 3.8°/s threshold for eye velocity in a direct path from canals to eyes (ref. 9). As pointed out by Malcolm (ref. 10), this would imply a discontinuity in slow-phase eye velocity, which does not appear in the records he obtained. Moreover, if such a velocity threshold existed, the transitions from right to left nystagmus in response to a head sinusoidal rotation would be very different from those shown in the tracings of figure 6.

The cumulative model output, proportional to $x^*(t)$, shows the expected pattern for the cumulative eye position. Neglecting the canal short time

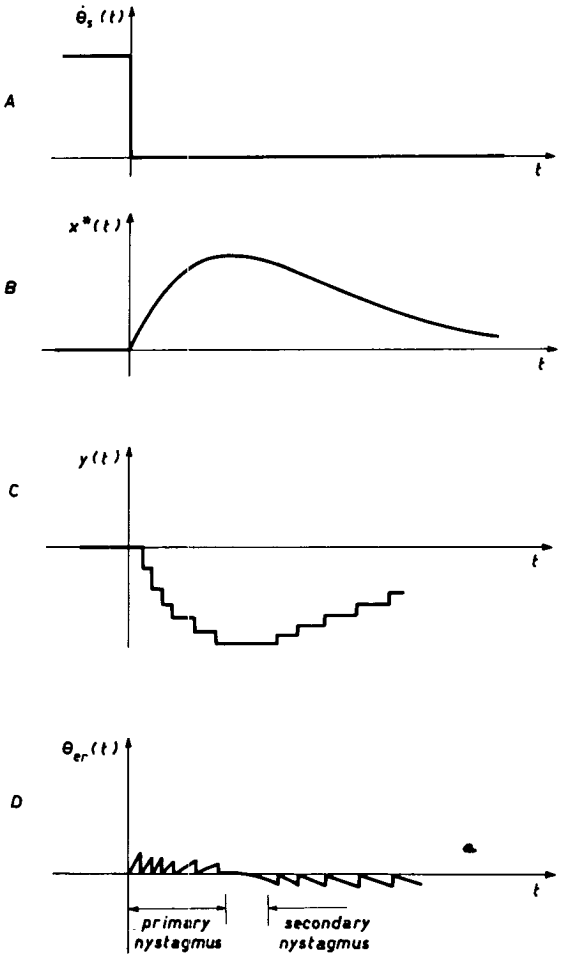


FIGURE 10.—*Qualitative response of the second approximation model to a velocity step input.*

constant T_1, and taking the inverse Laplace transform of equation 7, the slow-phase eye velocity in response to a step change in head angular velocity (head acceleration impulse) is given by

$$\theta_{ec}(t)=\frac{KV}{T_a-T_2}\left(\frac{1}{T_2}e^{-t/T_2}-\frac{1}{T_a}e^{-t/T_a}\right)$$

where V is the amplitude of the head-velocity step.

Thus, the cumulative eye position is given by

$$\theta_{ec}(t)=\frac{KV}{T_a-T_2}\left(e^{-t/T_a}-e^{-t/T_2}\right) \qquad (8)$$

The cumulative eye position reaches its maximum value (and the slow-phase eye velocity goes through zero) for

$$t=t_{\max}=\frac{T_a T_2}{T_a-T_2} \ln \frac{T_a}{T_2} \tag{9}$$

From equations 8 and 9,

$$\theta_{ec}(t_{\max})=\frac{RV}{T_a-T_2}\left(\frac{T_a}{T_2}\right)^{\frac{T_2}{T_a-T_2}}\left(1-\frac{T_2}{T_a}\right) \tag{10}$$

The model in figure 9 can be improved further by making it able to explain some additional findings reported in the clinical literature on vestibular nystagmus.

First of all, it is a common experience in postrotational tests that the secondary nystagmus is elicited only if a sufficiently large step change in angular velocity is produced.

Secondly, several clinicians (refs. 28 and 29), who are accustomed to considering the total angular displacement in postrotational nystagmus as an index of the vestibulo-ocular system behavior, report that there is frequently a remarkable difference (something like 20 to 30 percent) between total eye displacement in primary and secondary nystagmus. This means that the cumulative eye position in response to a velocity step input should follow a curve like that shown in figure 11, with a nonzero final value of f_v. In terms of slow-phase eye velocity, this also means that area I in figure 11 should be greater than area II.

Finally, a short tertiary nystagmus in the same direction as the primary nystagmus has been sometimes observed (ref. 30).

At the moment, there is no model that can predict all these findings. In particular, the model in figure 9 predicts that a secondary nystagmus will always follow a primary nystagmus. The final value $\theta_{ec}(\infty)$ computed from equation 8 is zero. Finally, no tertiary nystagmus can be predicted, since the signal $x^*(t)+y(t)$ cannot change its sign twice.

It will be shown in the next section that a little modification of the model in figure 9 will make it capable of also predicting these interesting aspects of human-subject responses to postrotational stimuli.

THIRD-APPROXIMATION MODEL

The third-approximation model is shown in figure 12. It has been obtained from the model in figure 9 simply by transforming the perfect integrator in the saccadic pathway into an integrator with leak.

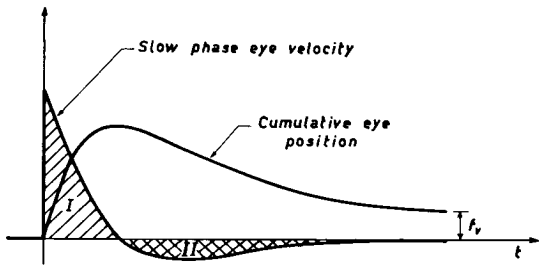


FIGURE 11.—*Cumulative eye position and slow-phase eye velocity for a step change in head angular velocity.*

The input-output transfer function $G(s)$ of this new integrator with leak is given by

$$G(s)=\frac{\mu_2}{1+sT_s}$$

where $\mu_2=1/\gamma$, $T_s=1/K_6\gamma$, and γ is the leak factor.

It is assumed that the time constant T_s of the integrator with leak in the saccadic pathway is greater than the adaptation time constant T_a defined in the preceding section.

The unit impulse response $g(t)$ of the integrator with leak is obtained by taking the inverse Laplace transform of $G(s)$; that is,

$$g(t)=K_6 e^{-K_6\gamma t}$$

Due to the large value of T_s, the first part of the response does not differ too much from a step function of amplitude K_6.

Consider the response of the model in figure 12 to a step velocity input (fig. 13). During the primary nystagmus the leak of the saccadic integrator does not greatly influence the model response; a small increase in the maximum value of the cumulative output may be produced by this leak. The time at which the maximum is reached will still be given by equation 9.

To simplify the analysis of the remaining part of the model response, let us assume that $x^*(t)$ and $-y^*(t)$ have the same value for $t=t_{\max}$,‡ and let this value be denoted by $\theta^*_{ec}(t_{\max})$. Also let K_0 be equal to 1.

Then,

‡ In the model of figure 12, the maximum value that $x^*(t_{\max})+y^*(t_{\max})$ can assume depends on the values of K_0,K_5, and α. For $K_0=K_5=1$, this value cannot exceed α.

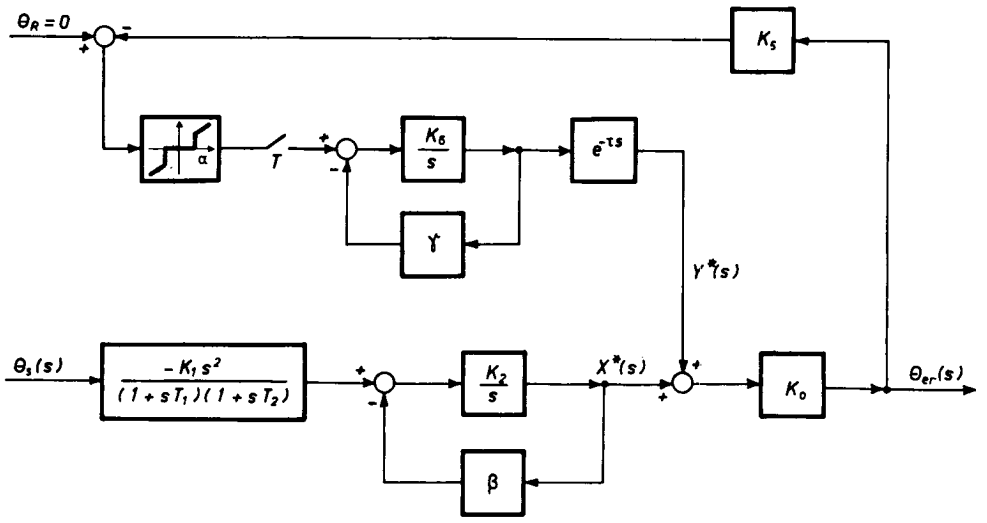


FIGURE 12.—*Third-approximation model.*

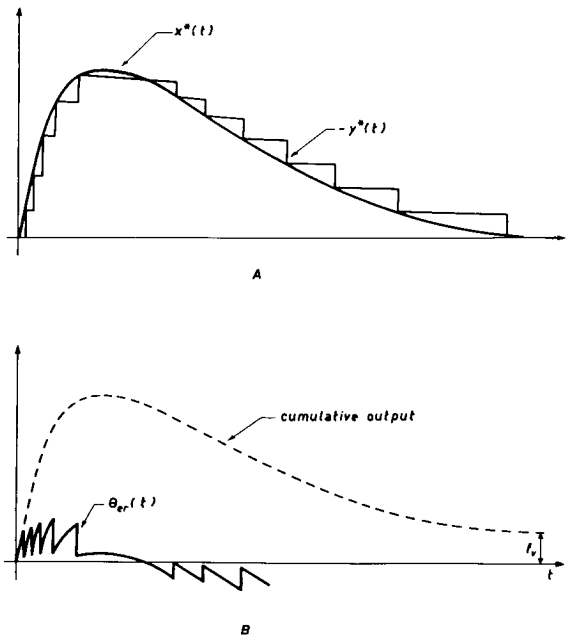


FIGURE 13.—*Qualitative response of the third-approximation model to a velocity step input.*

$$\theta_{er}(t) = \theta^*_{ec}(t_{\max}) \left[e^{-\frac{t-t_{\max}}{T_s}} - e^{-\frac{t-t_{\max}}{T_a}} \right] \quad (11)$$

for $t \geq t_{\max}$ and $\theta_{er}(t) < \frac{\alpha}{K_5}$.

The maximum of the function in equation 11 for $t > t_{\max}$ is given by

$$\max \theta_{er}(t) = \theta^*_{ec}(t_{\max}) \left(\frac{T_s}{T_a}\right)^{-\frac{Ta}{Ts-Ta}} \left(1 - \frac{T_a}{T_s}\right) \quad (12)$$

Substituting $\delta = \frac{T_a}{T_s} (0 < \delta < 1)$ in equation 12,

$$\max \theta_{er}(t) = h(\delta)\, \theta^*_{ec}(t_{\max}) \quad (13)$$

where

$$h(\delta) = (1-\delta)\, \delta^{\frac{\delta}{1-\delta}}$$

For a given value of δ, it follows from equation 13 that, for $t > t_{\max}$, $\max \theta_{er}(t)$ is proportional to $\theta^*_{ec}(t_{\max})$, which in turn is proportional to the amplitude of the step velocity input, for given values of T_2 and T_a.

Thus, for small step inputs, it can result that

$$\max \theta_{er}(t) < \frac{\alpha}{K_5}$$

and no secondary nystagmus will appear.

Larger inputs will give

$$\max \theta_{er}(t) > \frac{\alpha}{K_5} \quad (14)$$

and a secondary nystagmus will appear in the model output as shown in figure 13, where for

generality it has been assumed that $x^*(t_{max}) \neq -y^*(t_{max})$.

Assume that equation 14 is satisfied and a secondary nystagmus exists. Then the cumulative output will follow the dashed curve shown in figure 13B. A nonzero final value f_v is produced by the leak of the integrator in the saccadic pathway in the period of time which follows the end of the primary nystagmus. The whole leak can be expressed as the sum of the leaks during the latency period and during each slow component of the secondary nystagmus. Due to the short duration of such intervals with respect to the time constant T_s, it can be reasonably assumed that $y^*(t)$ decays stepwise linearly, as shown in figure 13A. The slope p_i of the ith linear portion of $y^*(t)$ is given by

$$p_i = -\frac{y^*(t_i^+)}{T_s}$$

where $y^*(t_i^+)$ denotes the values of $y^*(t)$ at the beginning t_i^+ of the ith portion. Thus p_i increases during the primary nystagmus and decreases during the secondary nystagmus.

Figure 14 illustrates in a qualitative way how the third-approximation model can predict a tertiary nystagmus. Actually, if T_s is greater than T_a, but not by much, and the amplitude of the step velocity input is large enough, it can happen that the signal $x^*(t)+y^*(t)$ changes its sign twice. The last change is due to the fact that $x^*(t)$ decays exponentially, while $y^*(t)$ decays stepwise linearly.

CONCLUSION

The evolution of a mathematical model of the vestibulo-ocular system in an attempt to match more and more experimental data has been presented, mainly in a qualitative way. The final model seems to explain same peculiarities of the nystagmus patterns and to predict adaptation, secondary nystagmus, and some other interesting aspects of human subjects' responses to postrotational stimuli.

Much more work is needed to confirm experimentally and theoretically the validity of the proposed model. However, even at this stage of its development, the model can suggest some interesting experiments and guide in the interpretation of their results. A large program is being executed to determine quantitatively the model parameters and to establish more precisely to what extent the model can predict average responses of normal subjects to angular rotation in a horizontal plane.

If the validity of the model is confirmed, it will be interesting to establish on a quantitative basis what the errors are that occur when one derives the values of the canal long time constant and adaptation time constant from the curves of the slow-phase eye velocity or cumulative eye position in postrotational nystagmus, using fitting procedures, without taking into account the effect of the leak of the saccadic integrator.

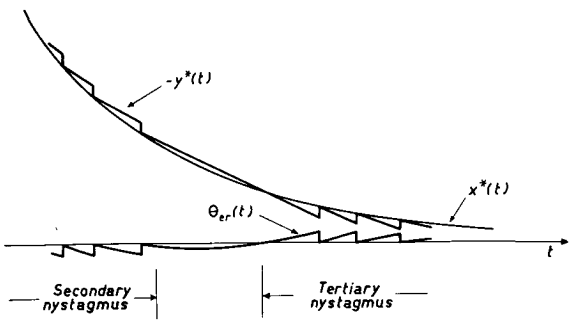


FIGURE 14.—*Possible origin of a tertiary nystagmus.*

ACKNOWLEDGMENTS

The author wishes to thank Dr. M. Stefanelli and Dr. E. Anzaldi of the Istituto di Elettronica of the University of Pavia (Italy), and Prof. E. Mira of the Clinica Otorinolaringoiatrica of the University of Pavia (Italy), for helpful discussions and suggestions.

REFERENCES

1. EYKHOFF, P.; VAN DER GRINTEN, P. M. E. M.; KWAKERNAAK, H.; AND VELTMAN, B. P. TH.: Systems Modelling and Identification. Third IFAC Congress, London, 1966.
2. STEINHAUSEN, W.: Über den Nachweis der Bewegung der Cupula in der Intekten Bogengangsampulle des Labyrinthes bei der Natürlichen Rotatorischen und Calorischen Reizung. Pflügers Arch. ges. Physiol., vol. 228, 1931, pp. 322–328.
3. VAN EGMOND, A. A. J.; GROEN, J. J.; AND JONGKEES, L. B. W.: The Mechanics of the Semicircular Canal. J. Physiol., vol. 110, 1949, pp. 1–17.
4. GROEN, J. J.: The Semicircular Canal Systems of the Organs of Equilibrium. I. Phys. Med. Biol., vol. 1, 1956, pp. 103–117; II. Phys. Med. Biol., vol. 1, 1956–57, pp. 225–242.
5. STEER, R. W., JR.: The Influence of Angular and Linear

Acceleration and Thermal Stimulation on the Human Semicircular Canal. Sc. D. thesis, Massachusetts Institute of Technology, 1967.

6. MELVILL JONES, G.; BARRY, W.; AND KOWALSKY, N.: Dynamics of the Semicircular Canals Compared in Yaw, Pitch and Roll. Aerospace Med., vol. 35, 1964, pp. 984–989.
7. GUEDRY, F. E.: Orientation of the Rotation-Axis Relative to Gravity: Its Influence on Nystagmus and the Sensation of Rotation. Acta Otolaryngol., vol. 60, 1965, pp. 30–49.
8. BENSON, A. J.; AND BODIN, M. A.: Effect of Orientation to the Gravitational on Nystagmus Following Rotation About a Horizontal Axis. Acta Otolaryngol., vol. 61, 1966, pp. 517–526.
9. YOUNG, L. R.; AND OMAN, C. M.: A Model for Vestibular Adaptation to Horizontal Rotation. Fourth Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–187, 1970, pp. 363–368.
10. MALCOLM, R.: A Quantitative Study of Vestibular Adaptation in Humans. Fourth Symposium on the Role of the Vestibular Organs in Space Exploration, NASA SP–187, 1970, pp. 369–380.
11. SUGIE, N.; AND MELVILL JONES, G.: A Mathematical Model of the Vestibulo-ocular System. Digest of the Sixth International Conference on Medical Electronics and Biological Engineering, Tokyo, 1965.
12. OUTERBRIDGE, J. S.: Experimental and Theoretical Investigation of Vestibularly Driven Head and Eye Movement. Sc. D. thesis, McGill University, July 1969.
13. MELVILL JONES, G.; AND MILSUM, J. H.: Spatial and Dynamic Aspects of Visual Fixation. IEEE Trans. Bio-Med. Eng. BME–12, 1965, pp. 54–62.
14. EWALD, J. R.: *Physiologische Untersuchungen über das Endorgan des Nervus Octavus.* Wiesbaden, Bergmann, 1892. 318 pp.
15. SCHMID, R.: AND STEFANELLI, M.: A Mathematical Model for the Vestibulo-ocular Reflex. Tech. Rep. LCA. 70–13. Istituto di Elettrotecnica ed Elettronica, Politecnico di Milano, Italy, 1970.
16. MAYNE, R.: The Match of the Semicircular Canals to the Dynamic Requirements of Various Species. The Role of the Vestibular Organs in the Exploration of Space, NASA SP–77, 1965, pp. 57–67.
17. ROBINSON, D. A.: The Oculomotor Control System: A Review. Proc. IEEE, vol. 56, 1968, pp. 1032–1049.
18. MILSUM, J. H.: *Biological Control Systems Analysis.* McGraw-Hill, 1966, p. 338.
19. MAYNE, R.: The Dynamic Characteristics of the Semicircular Canals. J. Comp. Physiol. Psychol., vol. 43, 1950, pp. 309–319.
20. MCCAUCH, G. P.; AND ADLER, F. H.: Extraocular Reflexes. Am. J. Physiol., vol. 100, 1932, pp. 78–88.
21. MATYUSHKIN, D. P.; AND AVER'YANOVA, I. A.: Coordination of Slow and Fast Ocular Nystagmus. Fiziol. Zh. SSSR, vol. 55, 1969, pp. 544–551.
22. YOUNG, L. R.: A Sampled Data Model for Eye Tracking Movements. Sc. D. thesis, Massachusetts Institute of Technology, 1962.
23. YOUNG, L. R.; FORSTER, J. D.; AND VAN HOUTTE, N.: A Revised Stochastic Sampled Data Model for Eye Tracking Movements. Fourth Annual NASA–University Conference on Manual Control, NASA SP–192, 1969, pp. 489–508.
24. WESTHEIMER, G.: Mechanism of Saccadic Eye Movements. Arch. Ophthal., vol. 52, 1954, pp. 710–724.
25. NIVEN, J. I.; HIXSON, W. C.; AND CORREIA, M. J.: An Experimental Approach to the Dynamics of the Vestibular Mechanisms. The Role of the Vestibular Organs in the Exploration of Space, NASA SP–77, 1965, pp. 43–56.
26. MEIRY, J. L.: The Vestibular System and Human Dynamic Space Orientation, Sc. D. thesis, Massachusetts Institute of Technology, 1965.
27. VAN DE CALSEYIDE, P.; AMPE, W.; AND DEPONDT, M.: Les Donées de l'Électronystagmographie dans l'Épreuve Rotatoire Sinusoidale Amortie. Acta Otorhinolaryngol., vol. 23, 1969, pp. 99–336.
28. JUNG, R.; AND TONNIES, J. F.: Die Registrierung und Auswertung des Drehnystagmus beim Menschen. Klin. Wchnschr., vol. 26, 1948, pp. 513–521.
29. ASCHAN, G.; AND BERGSTEDT, M.: The Genesis of Secondary Nystagmus Induced by Vestibular Stimuli. Acta Soc. Med. Upsaliensis, vol. 60, 1955, pp. 113–122.
30. KORNHUBER, H. H.: Physiologie und Klinik des Vestibularen Systems. Arch. Klin. Exp. Ohr. Nas. Kehlkopfheilk., vol. 194, 1969, pp. 111–148.

Use of Steinhausen's Model for Describing Periodic Coriolis Star Nystagmus

MÁXIMO VALENTINUZZI
Chicago College of Osteopathic Medicine

SUMMARY

Phase lag, maximal slow-phase velocity, and beat frequency were measured in periodic Coriolis star nystagmus. The results have been described by Steinhausen's model of the semicircular canal system. Estimates of the biophysical constants have been obtained. It is concluded that this model is a good functional approximation for describing, and also for interpreting, the behavior of the system.

INTRODUCTION

A sinusoidal Coriolis acceleration produced by combining two rotations can be used to stimulate the horizontal and the vertical semicircular canals simultaneously. The input received by each type of canal corresponds to a sine and a cosine function, respectively. The oculomotor response to this type of acceleration in the cat has been previously described and termed *periodic Coriolis star nystagmus* (ref. 1). It results from the composition of two periodic ocular displacements, perpendicular one to the other. The tip of the radius along which the center of the eye moves describes a locus that is, in general, an ellipse.

We have measured the phase lag, ϕ, between acceleratory input and nystagmic output; the maximal slow-phase velocity, $\dot{A}_{max}$; and the nystagmic frequency, f, in electronystagmograms from cats. The instrumental setup, material, and procedures used are described in references 1 and 2. The purposes of the present report are to summarize the experimental results and to show the usefulness of Steinhausen's model for describing, as well as for interpreting, these results.

EXPERIMENTAL RESULTS

Phase Lag

Figure 1 shows a portion of an electronystagmogram and the way in which the angle ϕ was measured. Figure 2 contains the pooled values of the average of ϕ_0 and ϕ_{180} obtained from several normal cats, using different directions of rotation. The lag is larger in the horizontal canals than in the vertical canals.

A series of experiments was performed using oscillations about the vertical axis (animal in ventral position) and about the binaural axis (animal in lateral position) in order to get independent responses from each type of semicircular canal. The phase lag was the same in both types of canals. The same cat was later subjected to oscillating Coriolis acceleration to produce periodic Coriolis star nystagmus, and values of ω_x were calculated for $\omega_x = 2\pi\nu$ (ν=frequency of the previously applied oscillation). The phase lag was different for the horizontal and vertical canals (fig. 3). In unilabyrinthectomized cats the lag behaved fundamentally as in the normal cats.

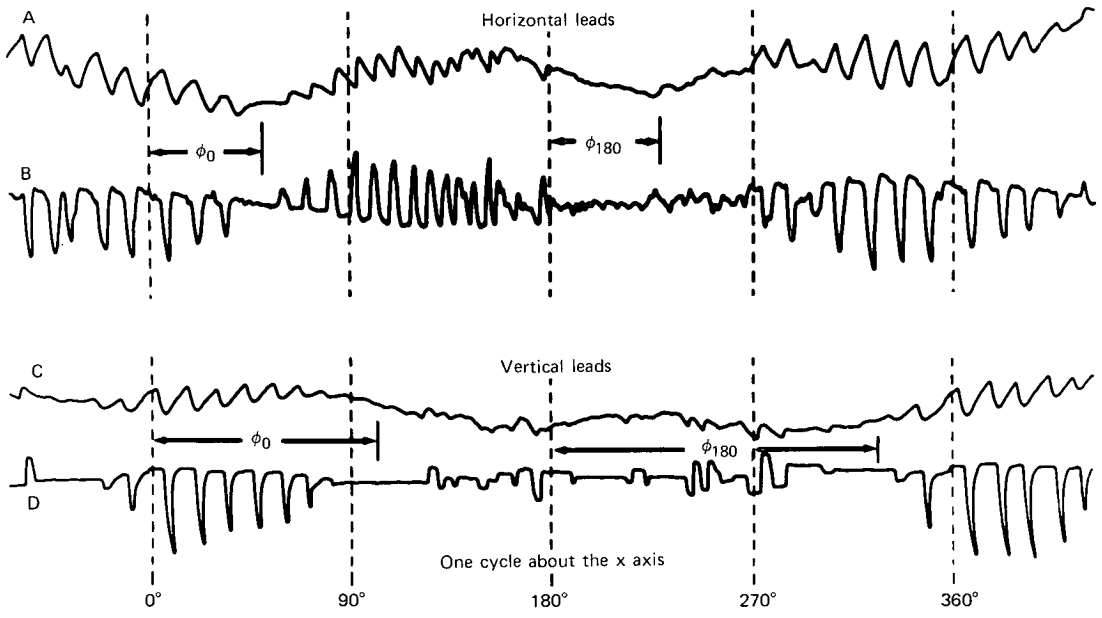


FIGURE 1.—*Measurement of phase lag in nystagmic Coriolis reaction in normal cat with implanted electrodes. A, C: direct recording. B, D: derivative recording (both phases). Stimulus: CW rotation at 80°/s about z axis; CW rotation at 20°/s about x axis. Duration of recording: 1 cycle about x axis. Horizontal leads: $\phi_0=50°$; $\phi_{180}=48°$. Vertical leads: $\phi_0=100°$; $\phi_{180}=145°$. In vertical leads, 90° has to be subtracted from ϕ to refer the lag to cosine. (from ref. 2)*

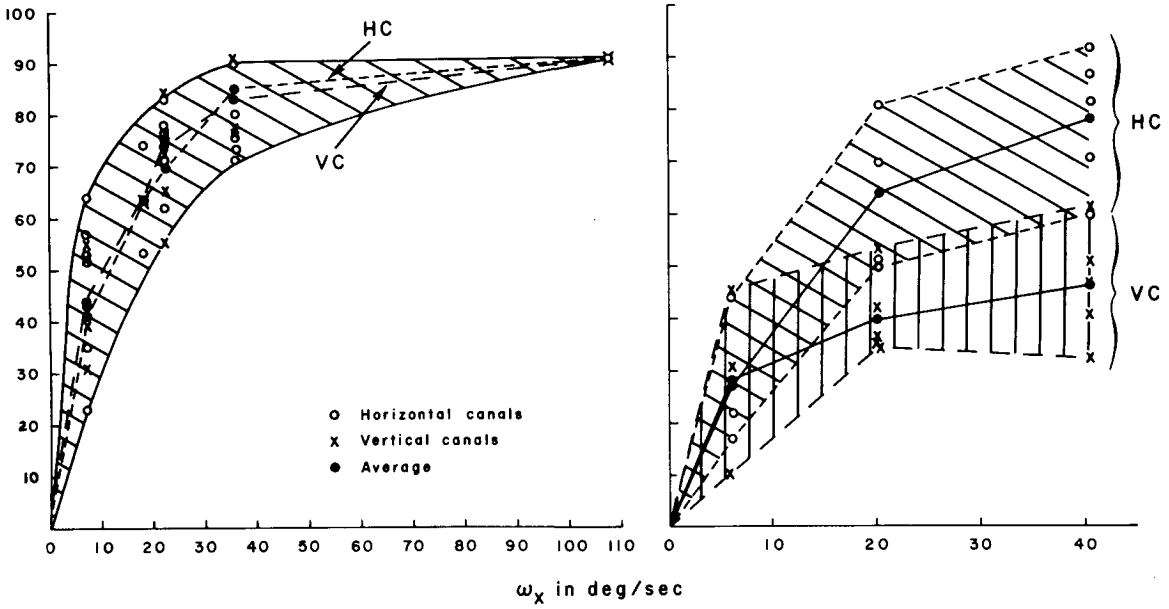


FIGURE 3.—*Separate stimulation of the horizontal and vertical canals of normal cats by a swing (left): The phase lag is the same for both types of canals. Simultaneous stimulation of the horizontal and the vertical canals by oscillating Coriolis acceleration (right): The phase lag is different for each type of canal. The individual values are contained in the shaded areas. Each individual value is the average of five to ten phase angles. The swing frequency, ν, has been transformed into ω by $\omega=2\pi\nu$. (from ref. 2)*

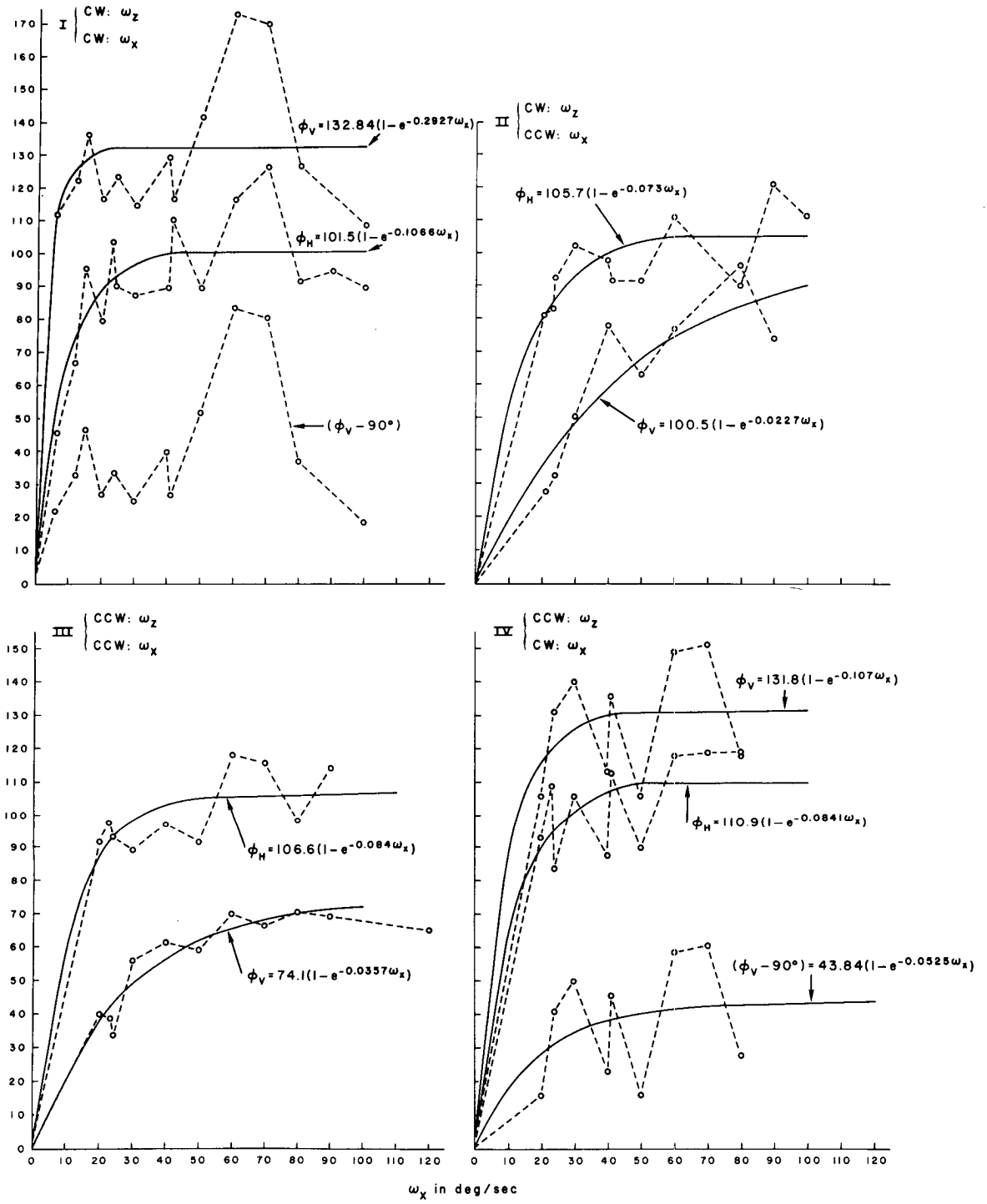


FIGURE 2.—*Pooled values of the phase lag in normal cats. A function* $\phi(\omega_x) = A(1-e^{-B\omega x})$ *has been used to smooth the values. I, II, III, IV correspond to the four combinations of rotations. In II and III,* ϕ_V *represents the real lag relative to cosine. The values* $(\phi_V - 90°)$ *in I were not suitable to calculate* $\phi(\omega_x)$*. Each circle is the mean value of six averaged phase angles, corresponding to different* ω_z *rotations between 17°/s and 160°/s. (from ref. 2)*

The phase lag as a function of the horizontal rotation was later reconstructed as shown in figure 4. Finally, ϕ was expressed as a function of the normalized frequency, ω_u, which is indicated in figure 5.

Maximal Slow-Phase Velocity and Frequency of Nystagmus

As shown in figure 1, there are two inversions in the direction of nystagmus for each cycle (360° rotation about the horizontal axis) in the horizontal and the vertical oculomotor output. The maximal amplitude A_{max}, the number of nystagmic beats n between inversions, and the temporal separation t of the inversions were measured. From these data the average jerk frequency $f=n/t$, the average duration of the jerk $\tau=t/n=1/f$, and the maximal slow-phase velocity $\dot{A}_{max}=A_{max}/\tau$ were estimated. The duration of the fast phase was neglected because it is small (0.1 to 0.2 s). The results of both cycle halves were averaged. Figures 6 and 7 contain the values of $\dot{A}_{max}$ and f for increasing ω_z. Their variation is linear with respect to ω_z.

In figures 8 and 9 the influence of the horizontal rotation, ω_x, in normal cats is illustrated. Figure 10 refers to cats with only one labyrinth. The values of A_{max} and f show a trend to increase for ω_x between 0°/s and 20°/s and to be constant beyond 20°/s.

Figure 11 shows the influence of both rotations on the slow-phase velocity and the nystagmic frequency.

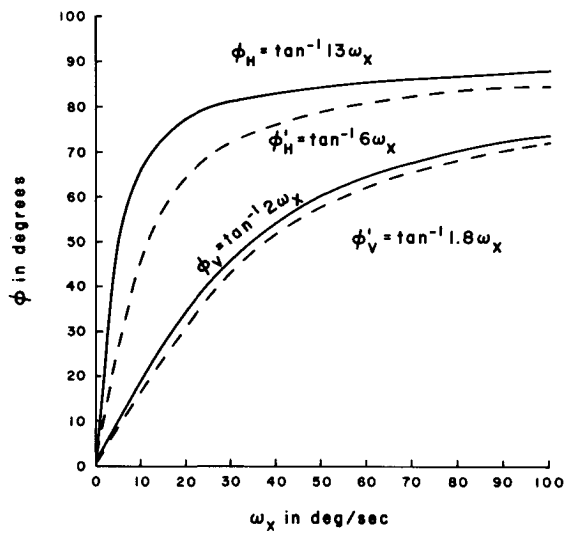


FIGURE 4.—*Values of ϕ obtained from $\phi=\tan^{-1}\omega_x T_1$ (eq. 3) using T_1 as given in table 1 for horizontal and vertical canals in normal cats (ϕ) and in unilabyrinthectomized cats (ϕ'). Compare these curves with those of figure 2. (from ref. 2)*

APPLICATION OF STEINHAUSEN'S MODEL

The differential equation of Steinhausen's model (refs. 3 and 4) with a sinusoidal driving stimulus yields the cupular deflection $\beta(t)$ as a sine function for the horizontal canals and as a cosine function for the vertical canals (ref. 2):

$$\beta(t)=\frac{4\omega_z\omega_x}{\sqrt{(\triangle/\Theta-\omega_x^2)^2+(\Pi\omega_x/\Theta)^2}}\begin{cases}\sin(\omega_x t-\phi_H) & \text{(horizontal canals)}\\ \sin\alpha\cos(\omega_x t-\phi_V) & \text{(vertical canals)}\end{cases} \quad (1)$$

where Π=endolymph viscosity, Θ=moment of inertia of the endolymph ring, $\triangle$=elasticity of the cupula-crista structure, α=angle between the vertical canal and the frontal plane of the skull, and ϕ=phase lag. The transient period (complementary solution) is omitted because it is short.

The phase lag ϕ between Coriolis input and cupular deflection (and therefore nystagmic oculomotor response) is obtained from

$$\phi=-\tan^{-1}\frac{(\Pi/\Theta)\omega_x}{(\triangle/\Theta)-\omega_x^2} \quad (2)$$

for both types of semicircular canals. This equation shows that ϕ varies only with ω_x. The pattern contained in figure 2 agrees with this prediction.

By applying the procedure used by other researchers (refs. 5 to 7), Π/Δ and Π/Θ can be estimated, so that the time constants are

$$T_1=\frac{\Pi}{\Delta}\approx\frac{\tan\phi}{\omega_x} \quad (3)$$

for $\omega_x^2 \ll \Delta/\Theta$; and

$$T_2=\frac{\Theta}{\Pi}\approx\frac{1}{\omega_x\tan\phi} \quad (4)$$

for $\omega_x^2 \gg \Delta/\Theta$.

Furthermore,

$$\omega_n=\sqrt{\left(\frac{\Delta}{\Pi}\right)\left(\frac{\Pi}{\Theta}\right)}=\sqrt{\frac{\Delta}{\Theta}} \quad (5)$$

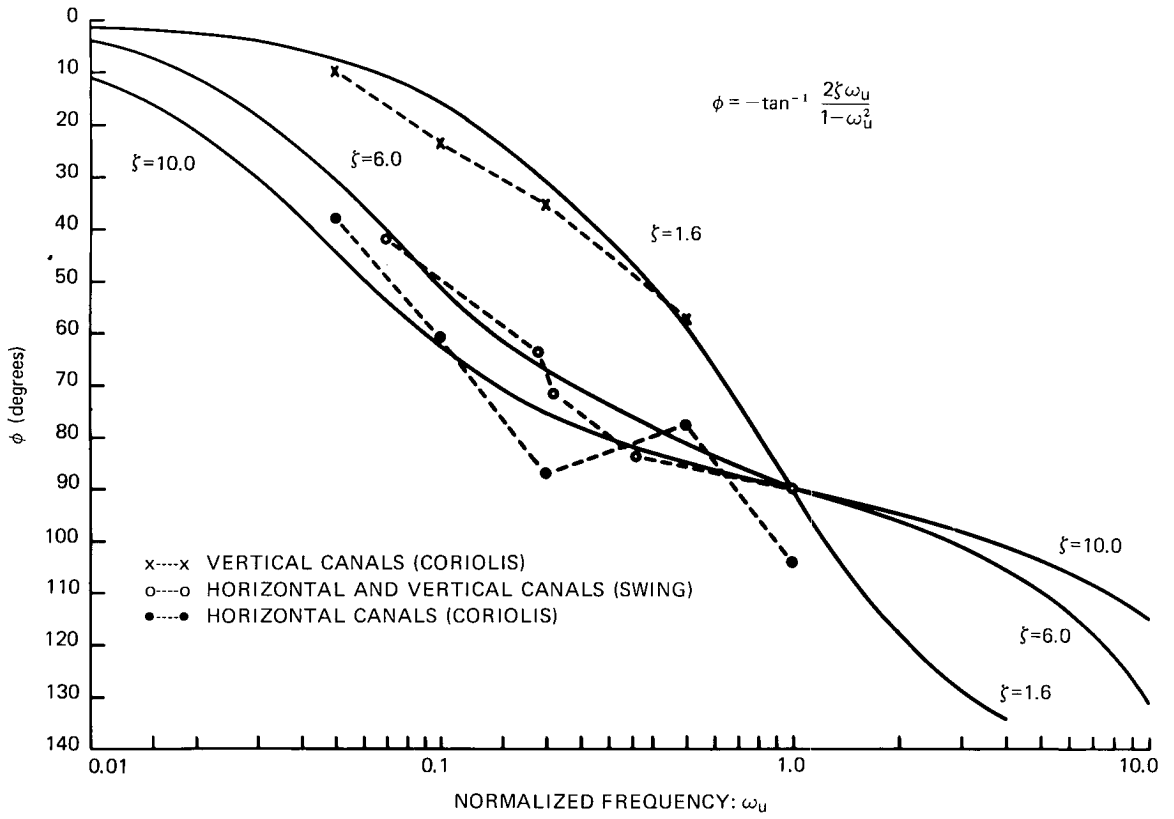


FIGURE 5.—*Phase lag, ϕ, versus normalized frequency, $\omega_u = \omega_x/\omega_n$ (equation 7) for normal cats. Solid lines: vertical canals ($\zeta = 1.6$) and horizontal canals ($\zeta = 10$) in simultaneous stimulation (table 1); both types of canal ($\zeta = 6.0$) in separate stimulation. Dashed lines correspond to averaged experimental values of ϕ with ω_x°/s transformed into ω_x rad/s and represented as $\log_{10}$ ω_u using ω_n of table 1. The experimental value $\phi = 61°$ for $\omega_u = 1$ in the vertical-canals curve was inadvertently omitted from the figure. The swing curve is based on figure 3. An average $T_1 = \Pi/\Delta \approx 7$ seconds was estimated from the pairs $\omega_x = 5°/s$, $\phi = 30°$; and $\omega_x = 25°/s$, $\phi = 72°$, using equation 3. With that value and $\omega_n^2 = (1.65)^2 = 2.72$, Π/Θ was estimated as 19 s, and therefore $T_2 \approx 0.05$ s and $\zeta = 6.0$. Each experimental value is based on the measurement of 10 phase angles. (from ref. 2)*

and

$$\zeta = \frac{1}{2}\sqrt{\left(\frac{\Pi}{\Delta}\right)\left(\frac{\Pi}{\Theta}\right)} \qquad (6)$$

The application of these estimate procedures to our experimental results gave the values contained in table 1.

The normalized frequency, $\omega_u = \omega_x/\omega_n$, and the damping factor, ζ, were introduced in equation 2 (ref. 6 and 8), so that

$$\phi = -\tan^{-1}\frac{2\zeta\omega_u}{1-\omega_u^2} \qquad (7)$$

Figure 5 shows curves obtained with equation 7 using $\log_{10}$ ω_u. For separate stimulation of the two types of canals the curves are practically the same. Since $\zeta_H \neq \zeta_V$ when the stimulation is simultaneous, the diagram gives two curves. The experimental values follow the calculated curves fairly well.

Now consider the behavior of the slow-phase velocity. From equation 1 we get the maximal cupular deflection when sine and cosine are equal to one:

$$\beta_{max} = \frac{4\omega_x\omega_x}{\sqrt{(\Delta/\Theta - \omega_x^2)^2 + (\Pi\omega_x/\Theta)^2}} \qquad (8)$$

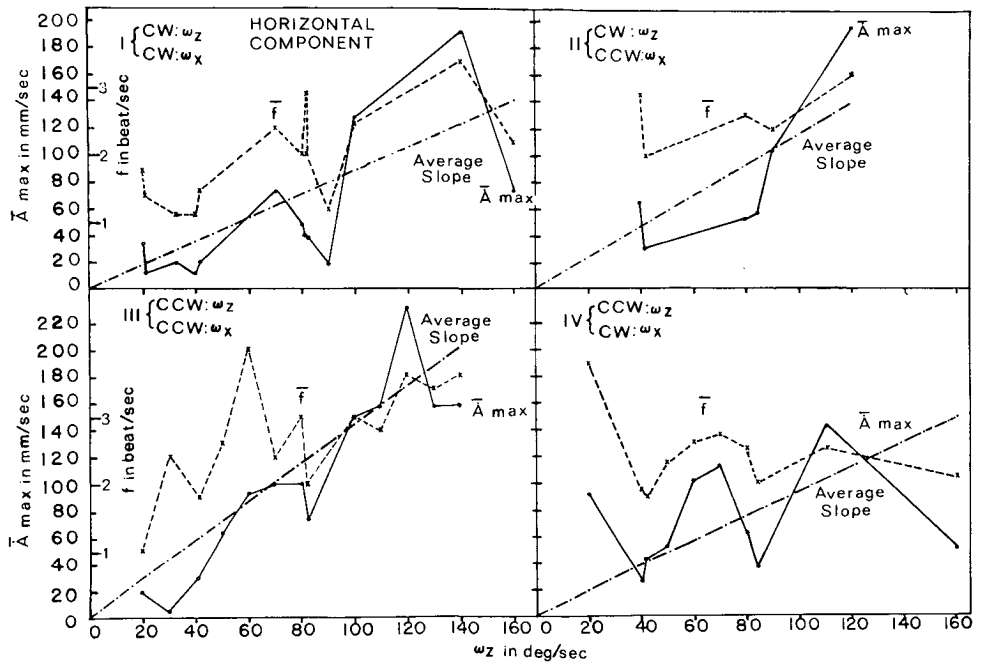


FIGURE 6.—*Pooled values of* $\dot{A}_{max}$ *and* f *against vertical rotation,* ω_z. *The values have been pooled for all values of* ω_x *on increasing* ω_z. *Horizontal component in the four combinations of rotations. The average slope is a rough estimate only.* $\dot{A}_{max}$ *and* f *vary linearly with* ω_z.

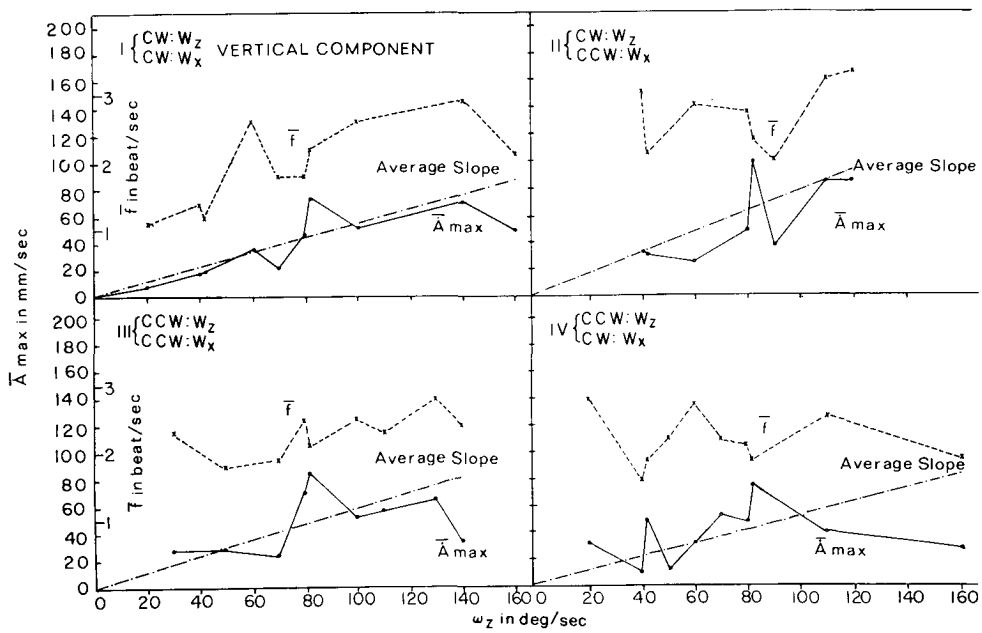


FIGURE 7.—*Pooled values as in figure 6, but for the vertical component.*

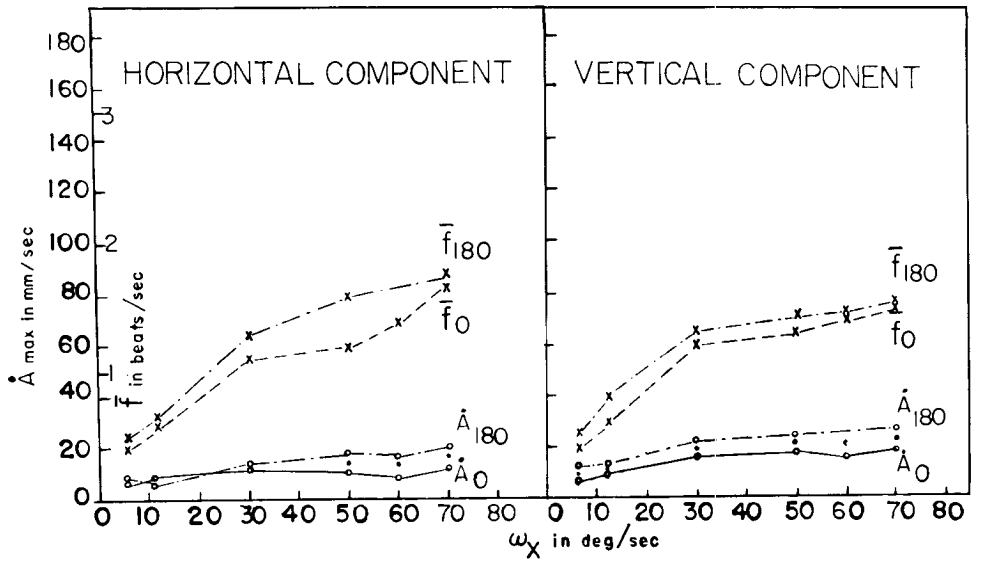


FIGURE 8.—*Effects of horizontal rotation on* $\dot{A}_{max}$ *and* f *in normal cat.* $\omega_z = 40°/s$, *CW (constant);* ω_x, *CW, increasing.*

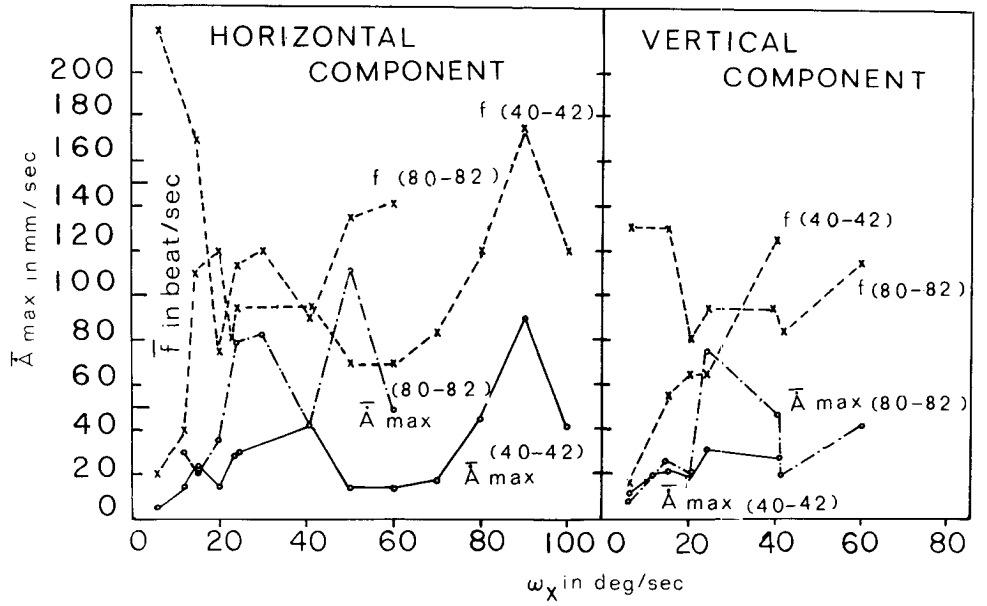


FIGURE 9.—*Pooled values of* $\dot{A}_{max}$ *and* f *versus horizontal rotation,* ω_x, *at two levels of* ω_z: *40 to 42 and 80 to 82°/s.*

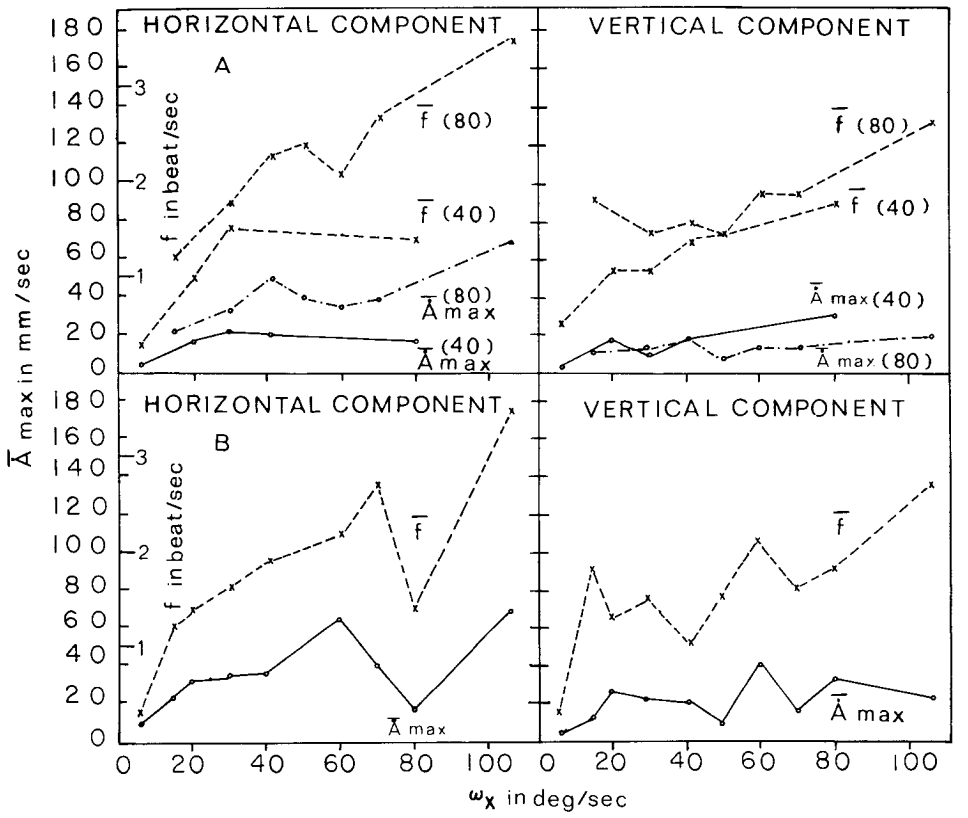


FIGURE 10.—*Effects on unilabyrinthectomized cats of all combinations of rotations. A: pooled values of* $\dot{A}_{max}$ *and* f *against* ω_x *at* $\omega_z = 40°/s$ *and* $80°/s$. *B: pooled values for all* ω_z *on increasing* ω_x.

TABLE 1.—*Estimated Values of* $\frac{\Pi}{\Delta}$, $\frac{\Pi}{\Theta}$, $\frac{\Theta}{\Pi}$, $\frac{\Delta}{\Theta}$, ω_n *and* ζ *(from ref. 2)*

Semicircular canals		$\frac{\Pi}{\Delta} = T_1$ (s)	$\frac{\Pi}{\Theta}$ (s^{-1})	$\frac{\Theta}{\Pi} = T_2$ (s)	$\frac{\Delta}{\Theta}$ (s^{-2})	$\omega_n = \sqrt{\frac{\Delta}{\Theta}}$ (rad/s)	$\zeta = \frac{\Pi}{2\sqrt{\Theta\Delta}}$
Horizontal	Two labyrinths	13.0	33.2	0.03	2.5	1.6 (≈91°/s) (≈0.25 Hz)	10
	One labyrinth	6.0	16.6	0.06	2.7	1.6	5
Vertical	Two labyrinths	2.0	5.7	0.17	2.8	1.7 (≈97°/s) (≈0.26 Hz)	1.6
	One labyrinth	1.8	5.4	0.18	3.0	1.7	1.6

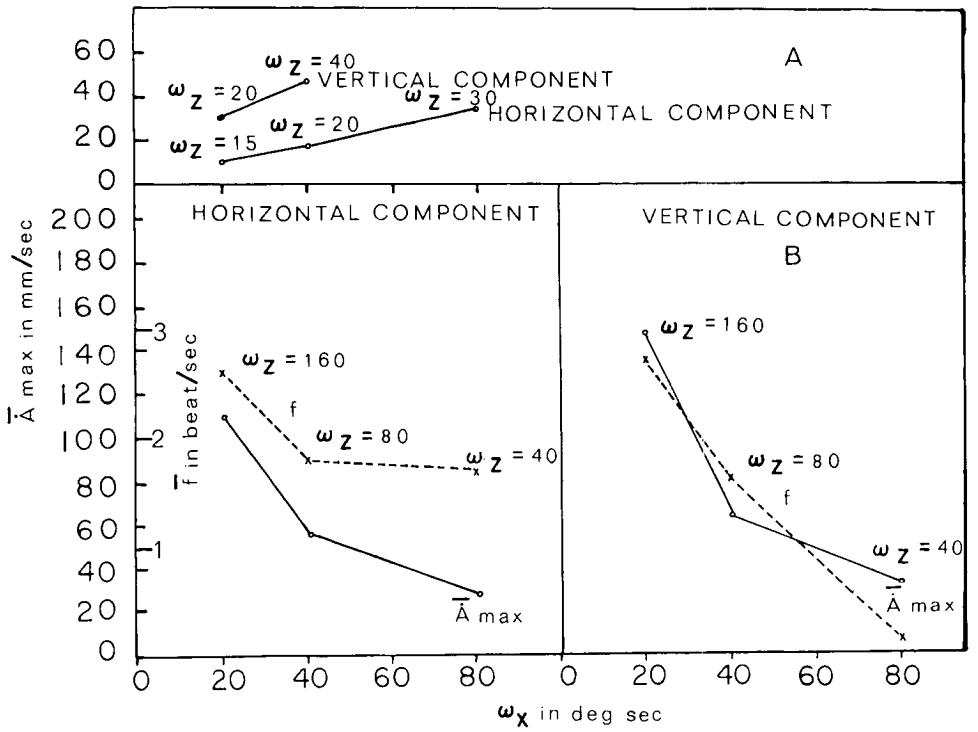


FIGURE 11.—*Effects on normal cat of horizontal and vertical rotations. A:* $\dot{A}_{max}$ *for increasing* ω_z *and* ω_x *(pooled values). B:* $\dot{A}_{max}$ *and* f *for constant product* $\omega_z\omega_x$.

This expression holds for the horizontal canals and is valid for the vertical canals after multiplication by sin α.

The assumption that the slow-phase velocity of the eye nystagmic displacement is proportional to the deflection of the cupula (ref. 6) may be expressed by

$$\dot{\psi}(t)=a\beta(t) \tag{9}$$

where $[a]=s^{-1}$ and $[\beta(t)]=$rad.

The maximal value, $\dot{\psi}_{max}$, is obtained from equations 8 and 9. The result predicts that $\dot{\psi}_{max}$ varies linearly with ω_z for constant ω_x. When ω_z remains constant, $\dot{\psi}_{max}$ varies with ω_x in a more complicated way. Our experimental information fits these predictions.

In order to plot $\dot{\psi}_{max}$ against ω_x, the denominator of equation 8 was transformed into the form

$$(x^2+\alpha x+\beta)=(x+\delta)\ (x+\xi) \tag{10}$$

with $\omega_x^2=X$. This gave

$$\alpha=(\xi+\delta)=\left[\left(\frac{\Pi}{\Theta}\right)-2\frac{\Delta}{\Theta}\right] \tag{11}$$

and

$$\beta=\xi\delta=\left(\frac{\Delta}{\Theta}\right)^2 \tag{12}$$

A family of curves is produced by varying ω_z. They can be framed by three tangents. The corner values are

$$\omega c'=\sqrt{\xi} \tag{13}$$

and

$$\omega c''=\sqrt{\delta} \tag{14}$$

Figure 12 shows some curves obtained in this way. Experimental values have been added.

The nystagmic frequency, f, has been treated as follows. If the duration of a nystagmic response to an impulse, step, or oscillating acceleration is divided into equal intervals, and if the jerk frequency for each interval is measured, the values

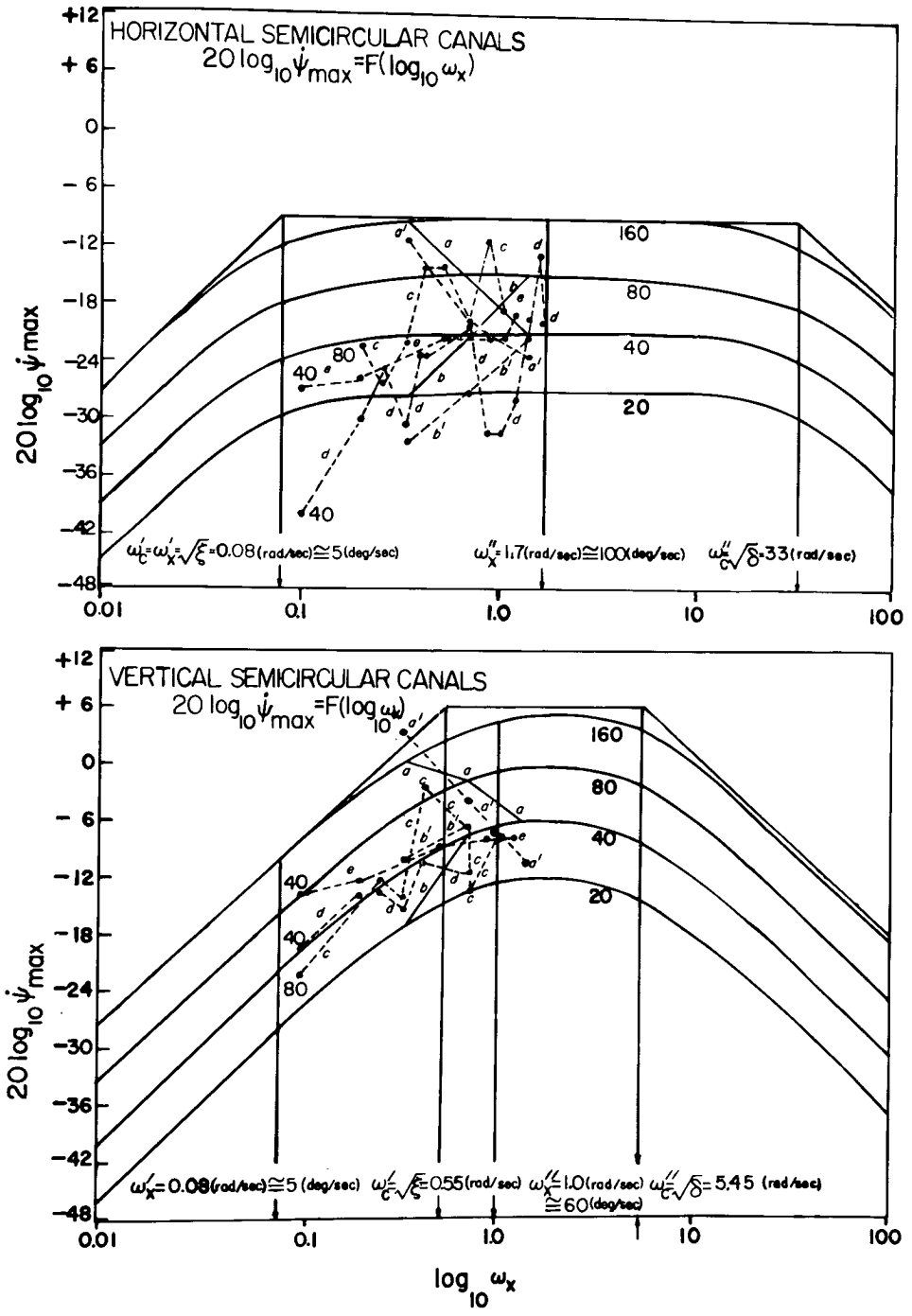


FIGURE 12.—Graph *of equation 9 (with eqs. 8 and 10) as a function of* ω_x *for four values (levels) of* ω_z *(20, 40, 80, and 160°/s) in the form of* $20 \log_{10} \dot{\psi}_{max} = F(\log_{10} \omega_x)$. *The curves can be framed by three tangents whose intersections correspond to the corner values* $\sqrt{\xi}$, $\sqrt{\delta}$ *(eqs. 13 and 14). Horizontal semicircular canals: a: diagram for* $\omega_z = 160, 80, 40°/s$ *and* $\omega_x = 20, 40, 80°/s$. *a': from figure 11 (B, HC), with* $k = 0.0025$. *b: diagram for* $\omega_z = 20, 40, 80°/s$ *and* $\omega_x = 20, 40, 80°/s$. *b': from figure 11 (A, HC), with* $k = 0.0025$ *(*$\omega_z = 15, 20, 30°/s$*). c: from figure 9 (80–82, HC), with* $k = 0.0025$. *d: from figure 9 (40–42, HC), with* $k = 0.0025$. *e: from figure 8 (HC), with*

appear distributed as though they followed the deflection of the cupula (fig. 13). Let $f(t)$ express the frequency as a function of time. On the basis of the above observation, it seems plausible to formulate a proportionality between $f(t)$ and $\beta(t)$; that is,

$$f(t)=b\beta(t) \qquad (15)$$

From equation 9,

$$f(t)=\frac{b}{a}\dot{\psi}(t)=C\dot{\psi}(t) \qquad (16)$$

The mean frequency, $\bar{f}$, was estimated in the electronystagmograms. It differs from f_{max} by the factor 0.62. This relationship derives from integrating equation 16 between 0 and $T/4$ and dividing by $T/4$, where T is the period of the acceleratory oscillation.

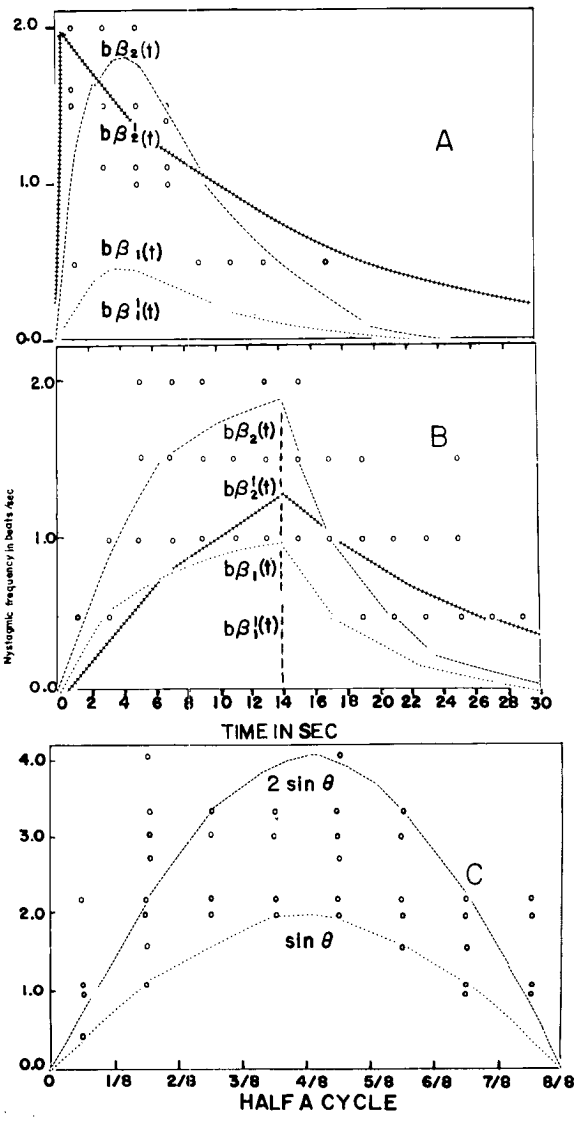


FIGURE 13.—*Distribution of nystagmic frequency values in beats per second from several nyastagmograms. A*—Impulse stimulation. *Circles: frequency* f *within 2-second intervals. The lines* $b\beta(t)$ *illustrate the theoretical behavior of the nystagmic frequency according to equation 15 for an impulse acceleration of two different intensities. B*—Step stimulation. *Circles and lines as in A. The input ends at t=14 s; from this moment on the recovery movement of the cupula takes place. In A and B the time constants of the horizontal canal* ($T_1=13$ *s;* $T_2=0.03$ *s) have been used for* $\beta_1'(t)$ *and* $\beta_2'(t)$. *To cover the circle better,* $\beta(t)$ *was calculated with* $T_1=5$ *s and* $T_2=0.3$ *s. C*—Sinusoidal stimulation. *Circles: frequency* f *within every eighth of a half cycle. The lines represent theoretical frequencies as proportional to sin θ and 2 sin θ; that is, to two different oscillaton amplitudes (eqs. 1 and 15). The pattern of the* f *values in A, B, and C suggests a proportionality relationship between* f(t) *and* β (t) *(eq. 15).*

k=*0.0065. Vertical semicircular canals: a: diagram for* $\omega_z=160,80,40°/s$ *and* $\omega_x=20,40,80°/s$. *a': from figure 11 (B, VC) with* k=*0.01. b: diagram for* $\omega_z=20$ *and* $40°/s$ *and* $\omega_x=20$ *and* $40°/s$. *b': from figure 11 (A, VC), with* k=*0.01. c: from figure 9 (80–82 VC), with* k=*0.01. d: from figure 9 (40–42 VC), with* k=*0.01. e: from figure 8 (VC), with* k=*0.02. In equation 9 the constant* a= *sin* x *for vertical canals was arbitrarily taken as unity. The factor* k *(rad/mm) relates* ψ *with* A*(amplitude), and it was estimated by trial and error.*

DISCUSSION

Steinhausen's model is useful for describing as well as for interpreting periodic Coriolis star nystagmus. Since this oculomotor output is given by a system of canals (two horizontal and four vertical canals), the model does not refer to just one of them, but it represents an idealized system functionally equivalent to the real system. Here we use a concept defined by Lorente de Nó and Berens (ref. 9): "For all practical purposes it may be assumed that there is in the head a single ideal canal always placed in the plane of rotation."

This model is a first approach. It does not cover other features of the entire labyrinthine system. Nonlinearities may exist, and changes due to adaptation and habituation appear. The contribution of other incoming signals, for example otolith signals, is overlooked.

CONCLUSIONS

1. The biomechanical model originated by Steinhausen is useful, as an approximation, for describing and also for interpreting the periodic Coriolis star nystagmus.

2. This model, as applied to periodic Coriolis star nystagmus, permits an estimate of the biophysical characteristics of the canal-endolymph-cupula-crista system.

3. The plot of the phase angle against the logarithm of the horizontal rotation, as referred to the natural frequency, gives a comprehensive understanding of the labyrinthine system.

4. The behavior of the maximal slow-phase velocity and frequency of nystagmus fits the conditions of Steinhausen's model.

ACKNOWLEDGMENT

The procedure for smoothing the values of figure 2 and the construction of the curve family of figure 12 were programed and performed by Dr. Dwight Aplevich of the University of Chicago, to whom I express my thanks.

REFERENCES

1. Fernández, C.; and Valentinuzzi, M.: Nystagmic Coriolis Reaction in the Cat. I. Preliminary Report. Acta Oto-Laryngol., vol. 65, 1968, pp. 186–199.
2. Valentinuzzi, M.: Phase Lag in Periodic Coriolis Star Nystagmus. Med. & Biol. Eng., vol. 8, 1970, pp. 559–574.
3. Steinhausen, W.: Über die Beobachtung der Cupula in den Bogengangsampullen des Labyrinths des Lebenden Hechts. Pflügers Arch. ges. Physiol., vol. 232, 1933, pp. 500–512.
4. Van Egmond, A. A. J.; and Jongkees, L. B. W.: The Mechanics of the Semicircular Canals. J. Physiol., vol. 110, 1949, pp. 1–17.
5. Groen, J. J.; Lowenstein, 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., vol. 117, 1952, pp. 329–346.
6. 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. NSAM–458. Naval School of Aviation Medicine, Pensacola, Fla., 1961.
7. Niven, J. I.; and Hixson, W. C.: Effects of Exposure to Continuous Rotation on Nystagmus Phase Shift. NSAM–870. Naval School of Aviation Medicine, Pensacola, Fla., 1963.
8. Milsum, J. H.: *Biological Control Systems Analysis*. McGraw-Hill, 1966, 466 pp.
9. Lorente de Nó, R.; and Berens, C.: Nystagmus. The Cyclopedia of Medicine, IX, Piersol and Bortz, eds., F. A. Davis, Philadelphia, 1959, pp. 793–812.

Use of Lorente de Nó's Neuron Circuit Model for Describing Acceleratory Nystagmus

MÁXIMO VALENTINUZZI
Chicago College of Osteopathic Medicine

SUMMARY

Results of a previous metric analysis and an electronic simulation of acceleratory nystagmus are given. On this basis, a tentative mathematical model for describing acceleratory nystagmus is reported. The essential content of the model is Lorente de Nó's neuron circuit, to which the two-factor theory of excitation has been applied.

INTRODUCTION

As is well known, the oculomotor apparatus (eye globe, extraocular muscles, orbit) yields either a periodic or a pseudoperiodic output when the semicircular canals are stimulated by an angular acceleration. Such a response is formed by a sequence of jerks (beats), each having a slow phase and a fast phase (fig. 1). Our purpose has been to simulate by an electronic model and to describe by a mathematical model this type of biomechanical output.

In order to obtain precise empirical numerical information about the characteristics of acceleratory nystagmus, corresponding to impulse angular accelerations, a series of electronystagmograms from cats was measured point by point (ref. 1). The quantitative behavior of the amplitude, the duration of the slow phase and the fast phase, and the value of the time constant of both phases were determined.

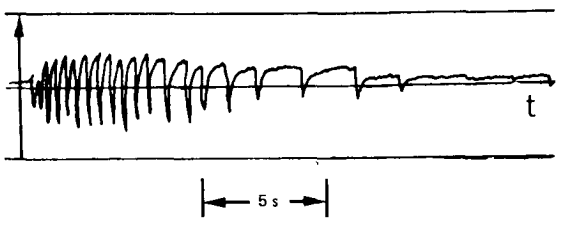


FIGURE 1.—*Horizontal electronystagmogram. Normal cat subjected to an impulse angular acceleration perpendicular to the horizontal semicircular canals ($\dot{\omega}=220°/s^2$). Note sequence of jerks (beats), each having a slow phase and a fast phase. (from ref. 1)*

Summarizing these results, we can say that the envelope of the beat amplitudes follows an exponential pattern and that both phases of the beat correspond also to exponential curves, whose time constants show variations that are somehow erratic. The beat duration (both phases taken together) increases exponentially (table 1; figs. 2 to 5).

TABLE 1.—*Values of Duration of Slow Phase, τ_i; Time of Beat Occurrence, t_i; and Amplitude, A_i (from an electronystagmogram similar to that of fig. 1, as measured by Santos and Pascual)**

Beat No.	τ_i(s)	t_i(s)	A_i(mm)
III	—	1.5	18.5
IV	0.7	2.2	17.5
V	0.9	3.1	16.0
VI	0.9	4.0	14.2
VII	0.9	4.9	16.5
VIII	1.0	5.9	12.2
IX	1.1	7.0	10.0
X	1.6	8.6	8.5

* Actually τ_i includes fast-phase duration, σ_i, which is small ($\approx$0.1 to 0.2 s). (See fig. 2.)

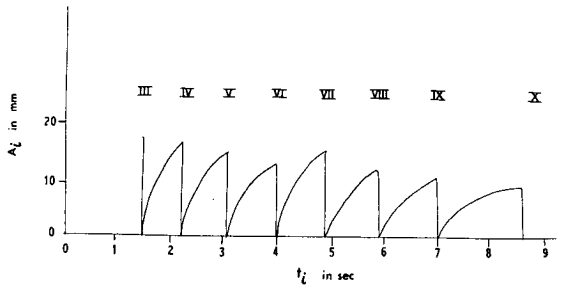


FIGURE 2.—*Reconstruction of a portion of a nystagmogram normalized relative to the baseline (from table 1). The phases are approximate. The original recording was similar to figure 1.*

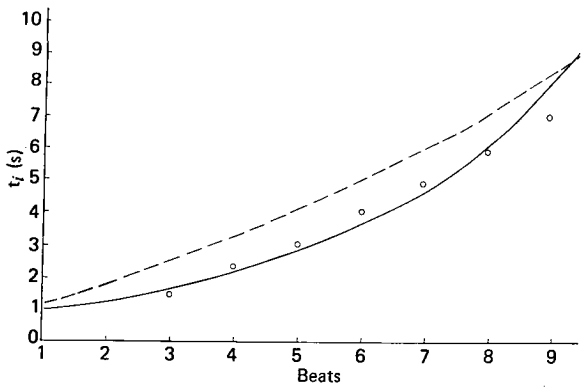


FIGURE 3.—*Time of occurrence of beats. The experimental values (circles) have been approximated by the geometric progression term* $t_i=1.3^{i-1}$, *where* $i=1, 2, 3, \ldots, 10$ *(solid line). The dashed line corresponds to the values of* t_i *as given in table 2.*

In the present paper the following basic notation is used (other symbols are introduced in the text):

t	nystagmogram time, with origin 0
t_i	value of nystagmogram time at which beat number i ends (beat occurrence time)
t'	beat time, variable of the slow and fast phases, with origin 0′ or 0″
τ_i	duration of slow phase of beat number i, with origin 0′
σ_i	duration of fast phase of beat number i, with origin 0″
A_i	maximal amplitude of recorded beat number i
ψ_i	maximal eye deviation of beat number i

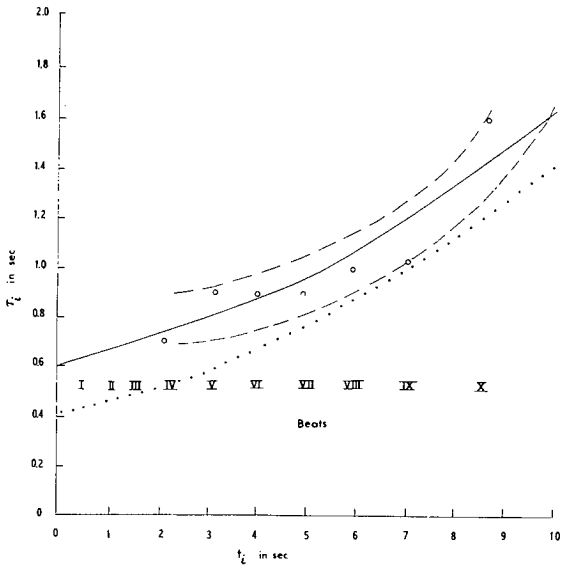


FIGURE 4.—*Plot of* τ_i *versus* t_i *(from table 1). The scattered values (between dashed lines) have been approximated by* $\tau_i=0.6e^{0.1t_i}$ *(solid line). The dotted line has been obtained from the solid line by subtracting* $\sigma=0.2$ *s.*

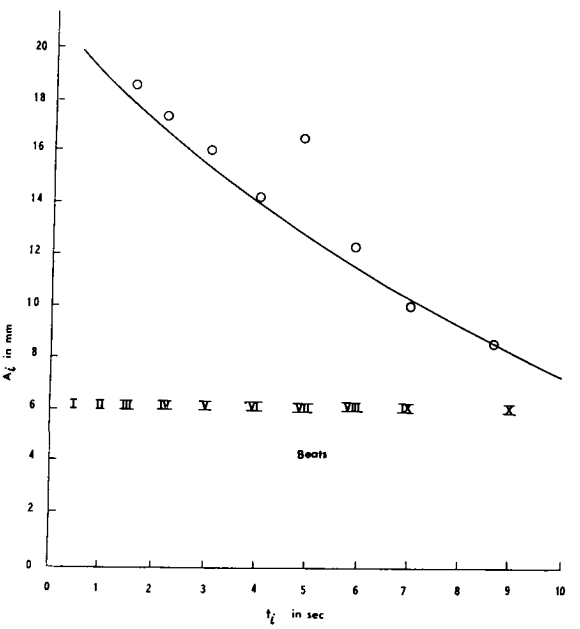


FIGURE 5.—*Plot of* A_i *versus* t_i *(from table 1). The experimental values (circles) have been approximated by* $A_i=21e^{-0.1t_i}$ *(solid line). Notice that the amplitude decline has a time constant of the same order of magnitude as the cupular recover* ($\Delta/\Pi\approx0.1$ *s).*

NEURON CIRCUIT MODEL

Lorente de Nó proposed in 132 (ref. 2) the neuron circuit indicated in figure 6 (f_3, i_1) to explain qualitatively the generation of ocular nystagmus. This neuron circuit is assumed to exist in the medullar reticular formation. Spiegel and Sommer (ref. 3) expanded on this theory, considering several different nervous structures which may be involved in the rhythmic response. The fundamental concept is that of the reciprocal excitation-inhibition process.

We have taken Lorente de Nó's neuron circuit as a starting point for designing the neuron network of figure 6 to describe the behavior of the system formed by the semicircular canals, the nervous pathways and nuclei, and the oculomotor apparatus when an angular acceleration is applied to the canals.

*ELECTRONIC SIMULATION**

The simulation of the semicircular system was based on the following equation (refs. 1 and 4):

$$\Theta\dot{\beta}(t)+\Pi\beta(t)+\Delta\int\beta(t)\,dt=\Theta\alpha \qquad (1)$$

where Θ is the moment of inertia of the endolymph ring, Π is the endolymph friction coefficient, Δ is the elasticity coefficient of the cupula-crista structure, $\beta(t)$ is the deflection of the cupula, and $\dot{\alpha}$ is the angular velocity. This is Steinhausen's equation (ref. 5) subjected to one integration. Comparing equation 1 to the well-known electrical equation (refs. 6 and 7),

$$\dot{L}i+Ri+\frac{1}{C}\int i\,dt=e \qquad (2)$$

yields the equivalences $\Theta\doteqdot L$ (self-induction coefficient), $\Pi\doteqdot R$ (electrical resistance), $\Delta\doteqdot 1/C$ (reciprocal of capacitance), $\beta(t)\doteqdot i(t)$ (electrical current), and $\alpha\doteqdot e$ (voltage), which form the basis of the electronic simulation (fig. 7).

The central nervous part in figure 6 was simulated by the transistor setup of figure 8. Each pathway represents a complex fascicular structure with a

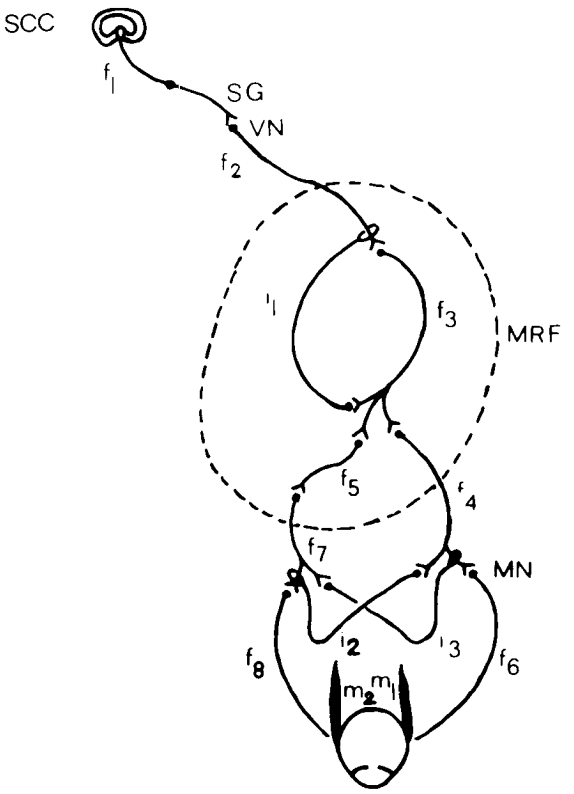


FIGURE 6.—*Neuron circuit model.* SCC, *semicircular canal;* SG, *scarpa ganglion;* VN, *vestibular nuclei;* MRF, *mesencephalic and pontine reticular formation;* MN, *motor nuclei;* f, *facilitatory pathway;* i, *inhibitory pathway;* m, *muscle (from ref. 1). Neurons* i, f_5 *are assumed to have high threshold. Neurons* f_3, i_1 *correspond to Lorente de Nó's loop.*

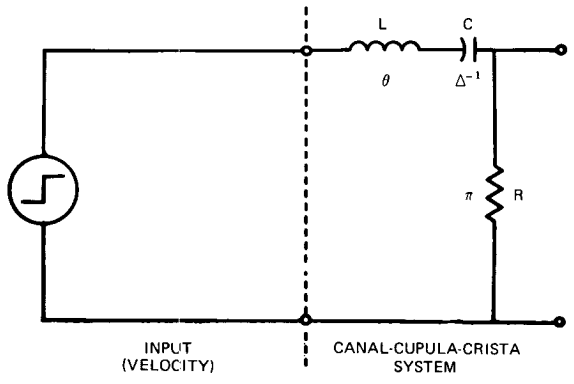


FIGURE 7.—*Simulation of the canal-cupula-crista system according to the mechanoelectrical analogies given in the text (from ref. 1).* L: *Self-induction coefficient.* C: *capacitance.* Δ: *Elasticity coefficient of cupula-crista structure.* R: *Electrical resistance.* Π: *Endolymph friction coefficient.*

* The content of this section is the result of a cooperative work with Professors Jorge Santos and Manuel Pascual, Universidad Nacional del Sur, Depto. de Electrotecnia, Bahía Blanca, Argentina (refs. 1 and 4).

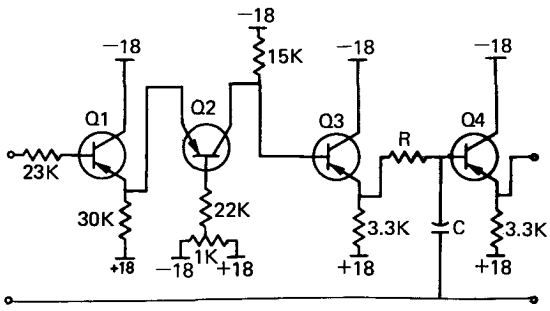


FIGURE 8.—*Simulation of an afferent fasciculum. R and C determine the time constant of the recruiting process; Q1, Q2, and Q3 are 2N4059 transistors to amplify power. (from ref. 1)*

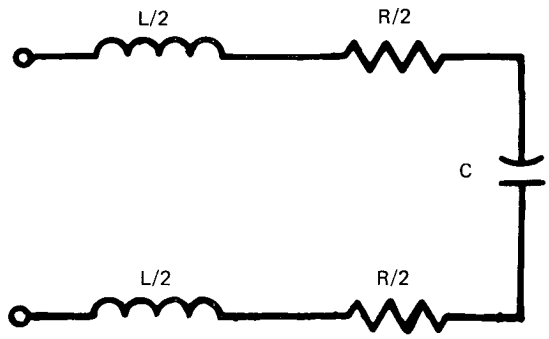


FIGURE 10.—*Simulation of the oculomotor output. R, L, and C produce a transfer function of the type determined by Robinson (ref. 8). Voltage corresponds to torque; velocity corresponds to electrical current. Voltage from the capacitor corresponds to eye deviation. (from ref. 1)*

certain recruiting-process time constant. The sequential facilitation may be gradual or abrupt. If abrupt, the facilitation occurs when a certain threshold is reached (which corresponds to the inhibitory pathway i and the facilitatory pathway f_5 of fig. 6). The time constant of the recruiting process is determined by the values of R and C in figure 8. Strong facilitation is obtained by inverting the Q output waveform (fig. 9).

The oculomotor system was simulated by the circuit of figure 10. R, L, and C produce a transfer function of the type determined in reference 8. The torque exerted on the globe follows linearly the nervous excitation. Voltages appearing across the capacitor are equivalent to eye angular displacement.

Figure 11 shows a recording of a response of the

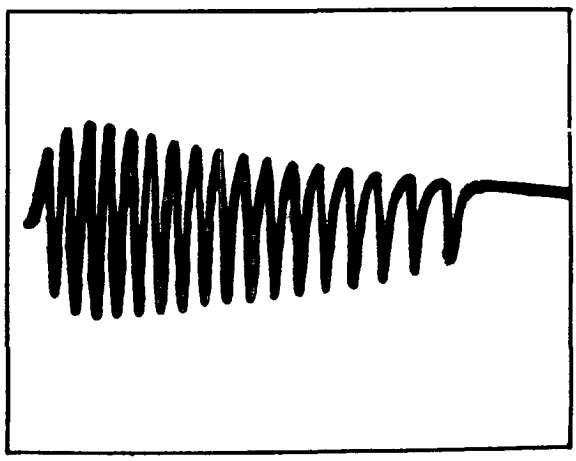


FIGURE 11.—*Simulated nystagmogram (response of simulator to impulse input). The time constants of the beats are invariable. (from ref. 1)*

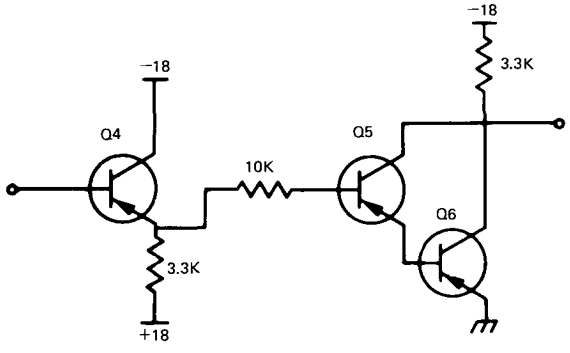


FIGURE 9.—*Simulation of the mechanism of abrupt inhibition. Strong facilitation is obtained by inverting the Q6 output waveform. Transistor Q4 provides power gain (compensation for loss in R). Q5 is an inverting transistor, with feedback between Q5 and Q6. (from ref. 1)*

simulator to an impulse input (equivalent to an impulse angular acceleration). The exponential decline of the beat amplitudes (whose envelope corresponds to the curve of cupular recovery), the lengthening of the duration of the slow phase, and the overshooting relative to the baseline are well reproduced. Step and sine inputs have not been tried.

The results obtained are encouraging. Of course, similar patterns do not imply similar mechanisms. However, these results serve as a guide in elaborating a mathematical model that gives a phenomen-

ological description of the system and some general insight into its biodynamics.

*MATHEMATICAL MODEL**

Differential Equations and Their Solutions

We have outlined a mathematical description of the operation of the neuron network (f_1, f_2, f_3, i_1) contained in figure 6. For the pair (f_1, f_2) the sequential excitation is formulated by the differential equations

$$\frac{df_1(t)}{dt} = A_{f1}\beta(t) - B_{f1}f_1(t) \tag{3}$$

and

$$\frac{df_2(t)}{dt} = A_{f2}f_1(t) - B_{f2}f_2(t) \tag{4}$$

These and the following equations are based on Rashevsky's theory of excitation (ref. 9). Neuron f_1 is stimulated by the crista receptors (hair cells), which are activated by the deflection of the cupula. With the simplifying assumptions that the constants in equations 3 and 4 are equal ($A_{f1}=B_{f1}=A_{f2}=B_{f2}$) and that the excitation in f_1 is linearly related to the deflection $\beta(t)$ of the cupula, equation 4 becomes

$$\frac{df_2(t)}{dt} = A_{f2}\beta(t) - B_{f2}f_2(t) \tag{5}$$

$$\therefore \quad f_2(t) = \beta(t)\left(1 - e^{-B_{f2}t}\right) \tag{6}$$

The behavior of the excitation $f_2(t)$ is determined by $\beta(t)$. Neuron f_2 is considered as the source of interrupted step inputs acting on neuron f_3. The interruptions originate in the inhibitory neuron i_1, which becomes excited by f_3 when this reaches the threshold of i_1. Therefore, each nystagmic beat is produced by a step function, $\bar{E}$, received from f_2 by f_3 (fig. 12). Designating the nystagmic beats by i=I, II, III,...,N (order of their occurrence as time t elapses from 0 to the end of the response), we write

$$\bar{E}_i = f_2(t_i - \sigma_i) \tag{7}$$

$\bar{E}_i$ becomes stopped when the slow phase ends; that is, for f_2 (τ_i). Using the beat time, t', we put

$$\left.\begin{aligned} &\bar{E}_i \cong f_2(t') \quad \text{for } 0' \leqslant t' < (\tau_i - \delta) \\ &\bar{E}_i = 0 \quad \text{for } \begin{cases} t' < 0' \\ t' \geqslant \tau_i \end{cases} \end{aligned}\right\} \tag{8}$$

with $\delta \to 0$.

Actually equation 8 implies reversal of the step function (eq. 7); in other words, neuron f_3 receives two successive opposite step inputs (or a square wave).

For the pair (f_3,i_1) we have

$$\frac{df_3(t')}{dt'} = A_{f3}\bar{E}_i - B_{f3}f_3(t') \tag{9}$$

and

$$\frac{di_1(t')}{dt'} = 0 \tag{10}$$

for $0' \leqslant t' \leqslant (\tau_i - \delta)$, with $\delta \to 0$. Therefore,

$$f_3(t') = \frac{A_{f3}\bar{E}_i}{B_{f3}}\left(1 - e^{-B_{f3}t'}\right) \tag{11}$$

and

$$i_1(t') = \text{constant (or eventually zero)} \tag{12}$$

Neuron i_1 is in the resting state during $0' \leqslant t' \leqslant \tau_i$. It has a threshold, h_{i1} and the excitation of f_3 attains this threshold at $t' = \tau_i$, so that when

$$f_3(\tau_i) = h_{i1} \tag{13}$$

neuron i_1 fires and inhibits f_3. This inhibition goes on until the fast phase ends, as t' runs from $0''$ to σ_i (for $t' > \tau_i$).

As soon as f_3 becomes inhibited, its own decline starts according to

$$f_3(t') = h_{i1}e^{-B_{f3}t'} \text{ for } t' > 0'' \tag{14}$$

The excitation in i_1 (inhibitory action) is described by

$$\frac{di_1(t')}{dt'} = A_{i1}h_{i1} - B_{i1}i_1(t') \tag{15}$$

for $t' \geqslant 0''$. The input originated in neuron f_3 (eq. 13) acts on neuron i_1 during a very short time, $\xi < \sigma_i$, and it is equivalent to an impulse, the area of which is $I = h_{i1}\xi$. This impulse raises the state of i_1 from its resting-state level quasi-instantaneously to an excitation level of value $f_3(\tau_i) = h_{i1}$ (eq. 13); that is, exactly to the threshold level of neuron i_1 and not above it, because at that instant neuron f_3

*The fundamental ideas contained in this section are the result of a cooperative work with Dr. Michael A. Fox, begun in 1967 at the University of Chicago.

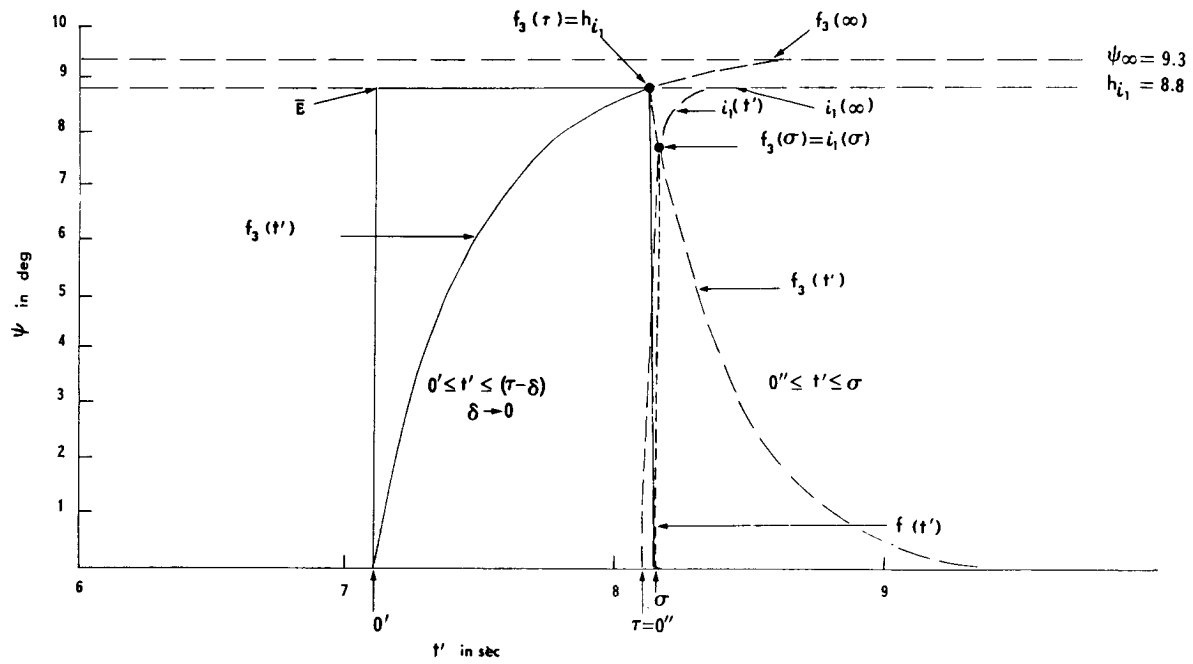


FIGURE 12.—*Composition of beat curve.* $\bar{E}$, *input determining slow phase* $f_3(t')$; h_{i_1}, *threshold of inhibitory pathway, corresponding to maximal eye deviation;* $f_3(t')$*(dashed line), spontaneous decay of excitation;* $i_1(t')$, *inhibitory action;* $f(t')$, *fast phase. The intersection* $f_3(\sigma)=i_1(\sigma)$ *determines the duration of the fast phase* ($\sigma<0.2$ *s). This figure corresponds to beat* i=*IX of figure 13.*

becomes inhibited by i_1. From then on the excitation state of i_1 decays exponentially; i.e.,

$$i_1(t') = A_{i_1} h_{i_1} e^{-B_{i_1} t'} \qquad (16)$$

for $t' > \tau_i = 0''$. In addition, equation 16 has to be shifted upward by h_{i_1} (fig. 12) so that

$$i_1(t') = h_{i_1} - h_{i_1} A_{i_1} e^{-B_{i_1} t'} \qquad (17)$$

Since equation 17 must be zero at $t' = \tau_i = 0''$, A_{i_1} turns out to be unity. For $t' \to \infty$, equation 17 tends to h_{i_1} (fig. 12). For $t' > 0''$, the *negative effect* described by

$$i_1(t') = h_{i_1} \left(1 - e^{-B_{i_1} t'} \right) \qquad (18)$$

has to be added to the spontaneous decay of neuron f_3 as given by equation 14. Therefore the output of the neuron circuit (f_3,i_1) results, for $t' \geqslant 0''$, as the difference between equations 14 and 18:

$$f(t') = h_{i_1} \left[e^{-B_{f3} t'} - \left(1 - e^{-B_{i_1} t'} \right) \right] \qquad (19)$$

The neuron activity can be thought of as given in voltage or impulse frequency (refs. 9 to 11).

Duration of the Slow Phase and the Fast Phase

The duration of the slow phase, τ_i, is a function of the intensity of excitation $\bar{E}_i$ in neuron f_2 and of the threshold h_{i_1}. An approximate expression for τ_i can be obtained as follows. Take equation 11 at $t' = \tau_i$:

$$f_3(\tau_i) = h_{i_1} = \frac{A_{f3} E_i}{B_{f3}} \left(1 - e^{-B_{f3} \tau_i} \right) \qquad (20)$$

$$\therefore \quad e^{-B_{f3} \tau_i} = 1 - \frac{B_{f3} h_{i_1}}{A_{f3} E_i} \qquad (21)$$

Since τ_i is usually small,

$$1 - B_{f3} \tau_i \cong 1 - \frac{B_{f3} h_{i_1}}{A_{f3} E_i} \qquad (22)$$

$$\therefore \quad \tau_i \cong \frac{h_{i_1}}{A_{f3} E_i} \qquad (23)$$

From equations 6 and 8,

$$\tau_i \cong \frac{h_{i1}}{A_{f3}\beta(t_i - \sigma_i)} \tag{24}$$

In other words, the duration of the slow phase increases with increasing values of the threshold h_{i1} and decreases as the deflection of the cupula, $\beta(t)$, increases; and vice versa. Consider, as an example, the return of the cupula to its resting position after an impulse angular acceleration; that is,

$$\beta(t_i - \sigma_i = \beta_o e^{-a(t_i - \sigma_i)} \tag{25}$$

Then

$$\tau_i \cong \frac{h_{i1}}{A_{f3}\beta_o} e^{a(t_i - \sigma_i)} \tag{26}$$

This is the behavior of $\tau_i (i = \text{I, II, III,...,}N)$ experimentally observed (figs. 1 and 2). The result (eq. 26) would be different in the case of step or sine angular acceleration.

The nystagmic beat is completed for $t' = \sigma_i$, with $f(t') = 0$. In consequence, from equation 19,

$$e^{-B_{f3}\sigma_i} = \left(1 - e^{-B_{i1}\sigma_i}\right) \tag{27}$$

Since σ_i is quite small ($\sigma_i < 0.2$ s), we write

$$e^{-B_{f3}\sigma_i} \approx 1 - B_{f3}\sigma_i \tag{28}$$

$$\therefore B_{i1} + B_{f3} \approx \frac{1}{\sigma_i} \tag{29}$$

Equation 29 gives a rough estimate of the order of magnitude of the time constant of the fast phase. We may formulate an approximate description of the fast phase equivalent to equation 19; i.e.,

$$f(t') \approx h_{i1} e^{-(B_{f3} + B_{i1})t'} = h_{i1} e^{\frac{t'}{\sigma_i}} \tag{30}$$

for $t' > \tau_i$.

The time t_i corresponding to a given beat i can be expressed as follows:

$$t_i = \sum_{\nu=1}^{\nu=i} \tau_\nu + \sum_{\nu=1}^{\nu=1} \sigma_\nu \tag{31}$$

If σ_i is approximately constant, then

$$t_i \approx i\sigma + \sum_{\nu=1}^{\nu=i} \tau_\nu \tag{32}$$

With geometric terms, so that $r = \tau_i / \tau_{i-1}$, we get the approximations

$$\tau_i \approx \tau_1 r^{i-1} \tag{33}$$

and

$$t_i \approx i\sigma + \tau_1 \frac{r^i - 1}{r - 1} \tag{34}$$

Actually σ_i varies as τ_i changes. Koike (ref. 12) has found a strong positive correlation between τ_i and σ_i.

Behavior of the Maximal Amplitude of the Beats

The amplitude of the beat, $A_i(t')$, corresponds to the eye deviation, $\psi_i(t')$. The maximal amplitude, $A_i(\tau_i)$, expresses maximal eye deviation, $\psi_i(\tau_i)$. The eye deviation follows the excitation and the inhibition of the neuron network of figure 6. From equations 6, 11, 13, 19, and 30, we write, using conversion factors G (gain),

$$A_i(t') = G_\psi \psi_i(t') = G_f \frac{A_{f3}\beta(t_i - \sigma_i)}{B_{f3}} \left(1 - e^{-B_{f3}t'}\right) \tag{35}$$

for $0' \leqslant t' \leqslant \tau_i$;

$$A_i(t') = G_\psi \psi_i(t') \approx G_f h_{i1} e^{-\frac{t'}{\sigma_i}} \tag{36}$$

for $t' > \tau_i$; and

$$A_i(\tau_i) = G_\psi \psi_i(\tau_i) \approx G_f \frac{A_{f3}}{B_{f3}} \beta(t_i - \sigma_i) \tag{37}$$

In the case of equation 25, $A_i(\tau_i)$ declines exponentially, as found in experiments. In general, the maximal eye deflections in a nystagmic response follow the deflection of the cupula. Equation 37 implies that the threshold of the excitatory pathway, i_1, varies as the deflection of the cupula, a point which demands more critical thinking.

DESCRIPTION OF NYSTAGMOGRAM IN TERMS OF THE SUCCESSION ORDER OF THE BEATS: NUMERICAL ILLUSTRATION

For describing a complete nystagmic response, equations 35 to 37 have to be applied to each beat of the sequence. The beat time intervals and the maximal eye deflection depend upon the succession order, i, of the beat.

Let us choose a response to an impulse angular acceleration in the cat. Using the well-known equation (ref. 13)

$$\beta(t)=\gamma\frac{\Theta}{\pi}\left(e^{-\frac{\Delta}{\pi}t}-e^{-\frac{\pi}{\Theta}t}\right) \qquad (38)$$

where γ is the initial angular velocity, Θ is the moment of inertia of the endolymph ring, and π is the friction coefficient of the endolymph, the envelope of the maximal nystagmic amplitudes A_i can be calculated.

Suppose that an angular acceleration $\dot{\omega}=250°/s^2$ is applied during time $t=0.6$ s. Then $\gamma=\dot{\omega}t=150°/s$. Using this value along with $\Theta/\pi=0.03$ s and $\Delta/\pi=0.1$ s for the horizontal canal (ref. 14), equation 38 becomes

$$\beta(t)=4.5(e^{-0.1t}-e^{-33t})° \qquad (39)$$

Referred to beat order, it is

$$\beta_i(t_i-\sigma_i)=4.5[e^{-0.1(t_i-\sigma_i)}-e^{-33(t_i-\sigma_i)}]° \qquad (40)$$

For advanced i (t_i long enough), the second exponential term can be neglected, so that

$$\beta_i(t_i-\sigma_i)=4.5e^{-0.1\ (t_i-\sigma_i)}\ \text{degrees} \qquad (41)$$

We choose arbitrarily $G_\psi^\wedge=1$, this factor being nonessential for the purpose of the illustration. From equation 37,

$$A_i=\psi_i\approx G_f\frac{A_{f3}}{B_{f3}}\beta(t_i-\sigma_i) \qquad (42)$$

On the basis of equations 41 and 42 and table 2, we get

$$\frac{A_i}{\beta_i}=G_f\frac{A_{f3}}{B_{f3}}\approx 4.6 \qquad (43)$$

The values of t_i in table 2 have been used to calculate the decreasing ψ_i of figure 13. For practical reasons, σ_i has been taken as constant and equal to 0.2 s in the figure, but in the calculation of ψ_i it has been neglected because this does not introduce an important error. Three initial maximal amplitudes (0, 0′, 0″) were estimated with equation 40.

The slow phases have been described by equation 11. From measurements performed by Santos and Pascual,

$$B_{f3}\approx 3\ s^{-1} \qquad (44)$$

The factor $\frac{A_{f3}E_i}{B_{f3}}$, which corresponds to $\psi_i\ (\infty)$ (table 2), was obtained from

TABLE 2.—*Values of* $\tau_i=0.6e^{0.1t}-0.2$ *(see fig. 4)**

Beat No.	τ_i(s)	σ_i(s)	t_i(s)	ψ_i(°)	$\psi_i(\infty)$(°)
0	0.08	0.01	0.09	19.0	86.3
0′	0.10	0.01	0.20	20.2	77.6
0″	0.20	0.20	0.60	19.8	44.1
I	0.43	0.20	1.23	18.4	26.2
II	0.46	0.20	1.89	17.2	23.0
III	0.49	0.20	2.58	16.0	20.5
IV	0.53	0.20	3.31	15.0	18.7
V	0.60	0.20	4.11	13.8	16.4
VI	0.69	0.20	5.00	12.6	14.3
VII	0.78	0.20	5.98	11.4	12.2
VIII	0.99	0.20	7.17	10.0	10.5
IX	1.00	0.20	8.37	8.8	9.3
X	1.13	0.20	9.70	7.6	8.0

* Values of σ_i are arbitrarily chosen between 0 and 2. The t_i values were calculated by equation 31. $\psi_i=21e^{-0.1t_i}$. (See fig. 5.) The $\psi_i(\infty)$ values were obtained from equation 45.

$$\psi_i\approx\psi_i(\infty)(1-e^{-3\tau_i}) \qquad (45)$$

using ψ_i and τ_i from table 2.

The fast phases have been described by equation 30, where $h_{i1}=\psi_i$. By trial and error, we obtained the estimate (eq. 29)

$$B_{f3}+B_{i1}=46.5\ s^{-1} \qquad (46)$$

$$T_{i1}\approx 0.023\ s \qquad (47)$$

If the intersection of $e^{-3t'}$ and $1-e^{-43.5t'}$ (B_{i1} obtained from eq. 46) is looked for graphically, the result is $\sigma_i\approx 0.045$ s.

Figure 12 contains the beat i=IX of figure 13, showing its geometrical composition.

DISCUSSION

The results obtained from the numerical analysis of experimental recordings and the electronic simulation of acceleratory nystagmus have been useful for attempting a mathematical model of the mechanism of nystagmus. The model outlined in this paper is essentially based on the two-factor theory of excitation and Lorente de Nó's neuron circuit. The model covers several characteristics of the system behavior, although it misses some features; for example, the overshooting of the eye displacement at the end of the fast phase as it is observed in motion pictures and recordings. The implication

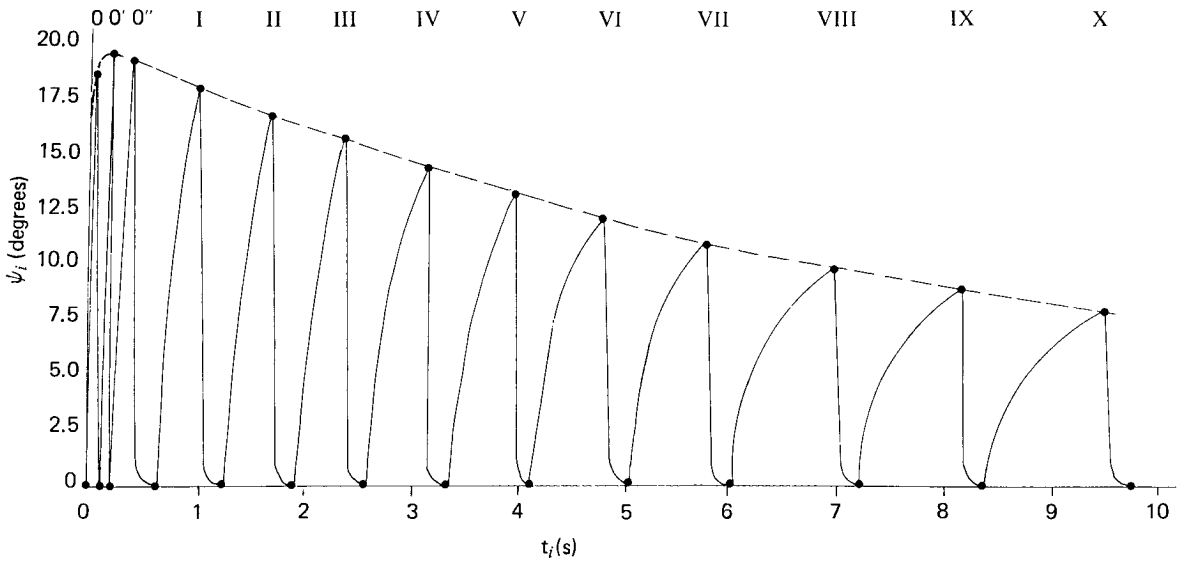


FIGURE 13.—*Point-by-point constructed nystagmogram using equations 35 to 37 ($B_{i_3}=3\ s^{-1}$; $B_{i_1}=43.5\ s^{-1}$; t_i, τ_i as given in table 2). Beats 0, 0', and 0" are estimated as explained in the text. Figure 1 and table 1 were used as guides.*

that the threshold of the inhibitory pathway depends upon the deflection of the cupula is one of the weak points of the model. It is an advantage to take the time order of the beat as a variable for the rhythmic response, from which the parameters of the beat itself can be deduced.

For horizontal nystagmus this model has to be thought of as functionally equivalent to the real system corresponding to both horizontal semicircular canals. If it were applied to vertical nystagmus, it should be assumed to be equivalent to the system of the four vertical semicircular canals.

This model may be utilized as a first approach for a digital computer simulation of the accelerator nystagmus, with different types of inputs (impulse, step, and sine inputs).

CONCLUSIONS

1. A metric analysis of experimental recordings and an electronic simulation of acceleratory nystagmus have helped to devise a tentative mathematical model of the labyrinth-oculomotor system.

2. The two-factor theory, as applied to Lorente de Nó's neuron circuit model, gives encouraging results for future modeling of the labyrinth-oculomotor system.

REFERENCES

1. SANTOS, J.; PASCUAL, M.; AND VALENTINUZZI, M.: On the Electronic Simulation of Acceleratory Nystagmus. Seventh Annual Meeting, Engineering Science in Biomedicine, Society of Engineering Science, Inc., Washington University, St. Louis, Mo., 1969.
2. LORENTE DE NÓ, R.: Researches on Labyrinth Reflexes. Trans. Amer. Otol. Soc., vol. 22, 1932, pp. 287–303.
3. SPIEGEL, E. A.; AND SOMMER, I.: *Neurology of the Eye, Ear, Nose and Throat.* Grune and Stratton, 1944.
4. SANTOS, J.; PASCUAL, M.; AND VALENTINUZZI, M.: Nota Preliminar sobre un Simulador Electrónico del Nistagmo Aceleratorio. Reuniones de la Sección Buenos Aires del Institute of Electrical and Electronic Engineers, 1968.
5. STEINHAUSEN, W.: Über die Beobachtung der Cupula in den Bogengangsampullen des Labyrinths des Lebenden Hechts. Pflügers Arch. ges. Physiol., vol. 232, 1933, pp. 500–512.
6. BROPHY, J. J.: *Basic Electronics for Scientists.* McGraw-Hill, 1966.

7. JAEGER, J. C.: *An Introduction to Applied Mathematics.* Clarendon Press, 1963.
8. ROBINSON, D. A.: The Oculomotor Control System: A Review. Proc. IEEE, vol. 56, 1968, pp. 1032–1048.
9. RASHEVSKY, N.: *Mathematical Biophysics.* Dover Publications, 1960.
10. MORALES, M. F.: Asynchrony of Labrynthine Receptors as a Physical Factor in Motion Sickness. Bull. Math. Biophys., vol. 8, 1946, pp. 147–157.
11. SHIMAZU, H.; AND PRECHT, W.: Tonic and Kinetic Responses of Cat's Vestibular Neurons to Horizontal Angular Acceleration. J. Neurophysiol., vol. 28, 1965, pp. 991–1013.
12. KOIKE, Y.: An Observation of the Eye Speed of the Nystagmus. Acta Oto-Laryngol., vol. 50, 1959, pp. 337–390.
13. VAN EGMOND, A. A. J.; GROEN, J. J.; AND JONGKEES, L. B. W.: The Mechanics of the Semicircular Canal. J. Physiol., vol. 110, 1949, pp. 1–17.
14. VALENTINUZZI, M.: Phase Lag in Periodic Coriolis Star Nystagmus. Med. & Biol. Eng., vol. 8, 1970, pp. 559–574.

PARTICIPANTS

Visitors

ADES, HARLOW W.
Department of Electrical Engineering
University of Illinois
Urbana, Illinois 61801

ANDERSON, DAVID J.
Kresgè Hearing Research Institute
Ann Arbor, Michigan 48103

ANLIKER, JAMES
NASA Electronics Research Center
575 Technology Square
Cambridge, Massachusetts 02139

ANLIKER, MAX
Department of Aeronautics and Astronautics
Stanford University
Palo Alto, California 94305

BARNES, GRAHAM R.
RAF Institute of Aviation Medicine
Farnborough, Hants, England

BARRETT, BERNARD
1600 North Palafox Street
Pensacola, Florida 32501

BENSON, ALAN J.
RAF Institute of Aviation Medicine
Farnborough, Hants, England

BERRY, CHARLES A.
NASA Headquarters, Code MM
Washington, D. C. 20546

BILLINGHAM, JOHN
NASA Ames Research Center
Moffett Field, California 94035

BROWN, JAMES H.
National Science Foundation
1800 G Street N.W.
Washington, D. C. 20550

BROWN, RAY D.
6570th AMRL (MREE)
Wright-Patterson AFB, Ohio 45433

CHENG, MICHAEL
Douglas Hall, McGill University
Montreal, Quebec, Canada

CLARK, BRANT
Department of Psychology
San Jose State College
San Jose, California 95114

CLARKE, MARTIN
University of Iowa
Department of Physiology and Biophysics
Iowa City, Iowa 52240

COLLINS, WILLIAM E.
Civil Aeromedical Institute
FAA Aeronautical Center, AC-118
P. O. Box 25082
Oklahoma City, Oklahoma 73125

CRAMER, ROBERT L.
USAF School of Aerospace Medicine
USAFSAM (SMKEV)
Brooks AFB, Texas 78235

CRAMPTON, GEORGE H.
Medical Research Laboratory
Edgewood Arsenal, Maryland 21010

DAY, M. AGNITA CLAIRE (SISTER)
St. Louis University
School of Nursing and Allied Health Professions
1401 S. Grand Blvd.
St. Louis, Missouri 63104

DIETLEIN, LAWRENCE F.
Chief, Biomedical Research Office
Code DB
NASA Manned Spacecraft Center
Houston, Texas 77058

DURFEY, JOHN Q.
Professor Anesthesiology
Medical College of Virginia
Richmond, Virginia 23219

EDWARDS, RICHARD G.
Wenner-Gren Research Laboratory
University of Kentucky
Lexington, Kentucky 40506

FERNÁNDEZ, CÉSAR
Department of Surgery (ENT)
University of Chicago
950 East 59th Street
Chicago, Illinois 60637

FLANAGAN, JAMES L.
CHABA Executive Council
Bell Telephone Laboratories
Murray Hill, New Jersey 07971

GONZALEZ, GERARDO
Department of Otolaryngology
Tulane Medical School
1430 Tulane Avenue
New Orleans, Louisiana 70112

HARRIS, RANDALL
NASA
Langley Air Force Base, Virginia 23365

HOMICK, JERRY L.
Neurophysiology Laboratory
NASA Manned Spacecraft Center
Houston, Texas 79058

HUMPHREYS, J. W., JR.
American Board of Surgery
1617 J. F. Kennedy Boulevard
Philadelphia, Pennsylvania 19103

INGE, WALTER H.
ADTC (ADTT)
Eglin AFB, Florida 32542

JOHNSON, WALTER H.
Department of Otolaryngology
University of Toronto
92 College Street
Toronto 5, Ontario, Canada

JONES, WALTON L.
NASA Headquarters, Code MMD
Washington, D. C. 20546

JUNKER, ANDREW M.
6570 AMRL (MREE)
Wright-Patterson AFB
Dayton, Ohio 45433

KELLER, GEORGE A.
Code PD-SS, Marshall Space Flight Center
Huntsville, Alabama 35812

KELLOGG, ROBERT S.
6570th AMRL
Wright-Patterson AFB, Ohio 45433

KENNEDY, ROBERT S.
Behavioral Sciences Department
Naval Medical Research Institute
Bethesda, Maryland 20014

LANDOLT, JACK P.
Canadian Defence Research Board
DRET, P.O. Box 2000
Downsview, Ontario, Canada

LINDEMAN, HENRIK H.
Department of Electrical Engineering
University of Illinois
Urbana, Illinois 61801

LONG, G. E.
McDonnell Douglas Astronautics Company
Huntington Beach, California 92646

LORD, DOUGLAS R.
NASA Headquarters, Code MF
Washington, D. C. 20546

MARAMAN, GRADY
NASA Langley Research Center
Hampton, Virginia 23365

MARTIN, ANDREW S.
USAARL
Ft. Rucker, Alabama 36360

MCCLURE, J. A.
Room 7308
Medical Science Building
University of Toronto
Toronto, Ontario, Canada

MILBURN, WANDA
Research (Audiologist)
1202 Granger Avenue
Ann Arbor, Michigan 48104

MONEY, KENNETH E.
Canadian Defence Research Board
DRET, P. O. Box 2000
Downsview, Ontario, Canada

ODELL, PATRICK L.
Texas Technological University
Department of Mathematics
Lubbock, Texas 79409

OMAN, C. M.
Department of Aeronautics and Astronautics
Massachusetts Institute of Technology
Cambridge, Massachusetts 02139

OOSTERVELD, W. J.
University of Amsterdam
Vestibular Department
Wilhelmina Gasthuis
Helmersstraat
Amsterdam, The Netherlands

OUTERBRIDGE, J. S.
O. T. L. Research Labs
Royal Victoria Hospital
Pine Avenue West
Montreal 112, Quebec, Canada

PARKER, DONALD E.
Psychology Department
Miami University
Oxford, Ohio 45056

PARKER, JAMES F., JR.
Biotechnology, Inc.
3027 Rosemary Lane
Falls Church, Virginia 22042

PILAND, WILLIAM M.
NASA Langley Research Center
Hampton Station, Virginia 23365

PRECHT, WOLFGANG
Deutschordenstr. 46
6 Frankfurt-Niederrad
Germany

REASON, JAMES T.
University of Leicester
Department of Psychology
Leicestershire, LE1 7RH, England

SANDERS, DANIEL T.
Army Research and Development Command
Office of the Surgeon General MEDDH-RPA
Washington, D. C. 20314

SCHMID, ROBERTO
Istituto di Elettrotecnica ed Elettronica
Politecnico di Milano
20133 Milano, Italy

SCHUPPE, C.
Martin Company
Denver, Colorado 80201

SCHWARZ, DIETRICH
University of Toronto
92 College Street
Toronto, Ontario, Canada

SEKITANI, TORU
Medical Research Center
University of Iowa
Iowa City, Iowa 52240

STAPP, J. P.
Department of Transportation
3817 Harrison St., N. W.
Washington, D. C. 20015

STEELE, JACK E.
6570th AMRL (MRBM)
Wright-Patterson AFB, Ohio

STEWART, JOHN D.
NASA Ames Research Center
Moffett Field, California 94035

STONE, RALPH W.
NASA Langley Research Center
Hampton, Virginia 23365

THOMPSON, ALLEN B.
General Electric Company—Apollo Systems
P. O. Box 58408
Houston, Texas 77058

TOLHURST, GILBERT
Office of Naval Research, Code 454
Navy Department
Washington, D. C. 20360

TYLER, PAUL E.
Bureau of Medicine and Surgery
Code 7113
Navy Department
Washington, D. C. 20390

VALENTINUZZI, MÁXIMO
Chicago College of Osteopathic Medicine
Department of Physiology and Pharmacology
1122 East 53rd Street
Chicago, Illinois 60615

VAN BUSKIRK, WILLIAM C.
Department of Orthopedic Surgery
Tulane Medical School
New Orleans, Louisiana 70118

VON BAUMGARTEN, R. J.
MHRI, Medical School
University of Michigan
Ann Arbor, Michigan 48108

WARD, W. DIXON
Box 461 Mayo
University of Minnesota
Minneapolis, Minnesota 55455

WEISS, ALFRED D.
Director of Otoneurology
Massachusetts Eye & Ear Infirmary
243 Charles Street
Boston, Massachusetts 02114

WHITCOMB, MILTON A.
CHABA
2101 Constitution, N. W.
Washington, D. C. 20418

WOLFE, JAMES W.
USAF School of Aerospace Medicine
USAFSAM (SMKEV)
Brooks AFB, Texas 78235

WOOD, CHARLES D.
LSU Medical School Shreveport
Louisiana State University
501 E. Stoner Avenue
Shreveport, Louisiana 71101

YESSENOW, MELVYN D.
Human Factors Psychologist
Grumman Aerospace Corporation
Bethpage, L. I., New York 11691

YOUNG, JACK H.
Kresge Hearing Research Institute
1301 Ann Street
Ann Arbor, Michigan 48104

YOUNG, LAURENCE R.
Department of Aeronautics
Massachusetts Institute of Technology
Cambridge, Massachusetts 02139

ZECHMAN, FRED W.
University of Kentucky Medical Center
Lexington, Kentucky 40506

Staff
Naval Aerospace Medical Research Laboratory,
Naval Aerospace Medical Institute, and
Naval Aerospace Medical Center
Pensacola, Florida 32512

Allebach, Newton W.
Beischer, Dietrich E.
Courtney, Marvin D.
Cramer, D. Bryant
De Santis, Mark
Fregly, Alfred R.
Gernandt, Bo E.
Gilson, Richard D.
Gray, Oscar, Jr.
Graybiel, Ashton
Grissett, James D.
Guedry, Fred E., Jr.
Hixson, W. Carroll
Knepton, James C., Jr.
Miller, Earl F. II
Molina, Efrain A.
Niven, Jorma I.
Norman, Joel W.
Oetken, Frances
Rafaelly, Nicholas
Reno, Vernon R.
Ricks, Edward L., Jr.
Schaefer, Herman J.
Smith, Margaret J.
Stockwell, Charles W.
Tang, P. C.
Trimble, Theron L.
Turnipseed, Gene T.
Walker, Michael L.
Williams, Carl E.

☆ U.S. GOVERNMENT PRINTING OFFICE : 1974 O—498-390

NATIONAL AERONAUTICS AND SPACE ADMINISTRATION
WASHINGTON, D.C. 20546

OFFICIAL BUSINESS
PENALTY FOR PRIVATE USE $300

SPECIAL FOURTH-CLASS RATE BOOK

POSTAGE AND FEES PAID
NATIONAL AERONAUTICS AND SPACE ADMINISTRATION
451

POSTMASTER: If Undeliverable (Section 158 Postal Manual) Do Not Return

"The aeronautical and space activities of the United States shall be conducted so as to contribute . . . to the expansion of human knowledge of phenomena in the atmosphere and space. The Administration shall provide for the widest practicable and appropriate dissemination of information concerning its activities and the results thereof."

—NATIONAL AERONAUTICS AND SPACE ACT OF 1958

NASA SCIENTIFIC AND TECHNICAL PUBLICATIONS

TECHNICAL REPORTS: Scientific and technical information considered important, complete, and a lasting contribution to existing knowledge.

TECHNICAL NOTES: Information less broad in scope but nevertheless of importance as a contribution to existing knowledge.

TECHNICAL MEMORANDUMS: Information receiving limited distribution because of preliminary data, security classification, or other reasons. Also includes conference proceedings with either limited or unlimited distribution.

CONTRACTOR REPORTS: Scientific and technical information generated under a NASA contract or grant and considered an important contribution to existing knowledge.

TECHNICAL TRANSLATIONS: Information published in a foreign language considered to merit NASA distribution in English.

SPECIAL PUBLICATIONS: Information derived from or of value to NASA activities. Publications include final reports of major projects, monographs, data compilations, handbooks, sourcebooks, and special bibliographies.

TECHNOLOGY UTILIZATION PUBLICATIONS: Information on technology used by NASA that may be of particular interest in commercial and other non-aerospace applications. Publications include Tech Briefs, Technology Utilization Reports and Technology Surveys.

Details on the availability of these publications may be obtained from:

SCIENTIFIC AND TECHNICAL INFORMATION OFFICE

NATIONAL AERONAUTICS AND SPACE ADMINISTRATION

Washington, D.C. 20546