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Large interindividual differences among 74 normal subjects in the change in susceptibility to motion sickness with effective lifting of the normal g-load by parabolic flight maneuvers were recorded with high test-retest reliability. Most subjects, who were required to make standardized head movements while seated in a chair rotating at a constant speed, demonstrated either a substantial increase or a decrease in susceptibility, in confirmation of a previous study, while a few appeared to be more or less unaffected by the 1 g to 0 g gravitational change. A similar test procedure conducted with eighteen of the subjects at lunar- and Martian-gravity levels revealed further interindividual differences in susceptibility as a function of g-level. The subjects with gravity-dependent susceptibility revealed: 1) a progressive change in susceptibility as a function of g-load in either the positive or negative direction that was characteristic of the individual, 2) a susceptibility level that appeared to be maintained at the fractional g-load, and 3) immunity to motion sickness at all g-levels tested below the Earth standard. The case history as well as ground-based functional and provocative tests of normal subjects proved to be inadequate in predicting susceptibility to motion sickness under subgravity conditions.

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ALTERED SUSCEPTIBILITY TO MOTION SICKNESS AS A FUNCTION OF SUBGRAVITY LEVEL

Earl F. Miller II, and Ashton Graybiel

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SUMMARY PAGE

THE PROBLEM

1) To determine the value of functional and provocative ground-based vestibular tests in predicting susceptibility to motion sickness under reduced g-loads.

2) To measure motion sickness susceptibility as a function of g-load.

FINDINGS

The motion sickness susceptibility of 74 healthy subjects who made standardized head movements while seated in a constantly rotating chair was measured in the laboratory and during the sequentially arranged weightless phases of parabolic flights. Twenty-one of these subjects were tested in zero g on more than one occasion and eighteen were tested additionally at lunar (0.17 g) and Martian (0.38 g) gravity levels. Large interindividual quantitative differences in the change in susceptibility, which had high test-retest reliability, were recorded with the effective lifting of the normal g-load. Most subjects demonstrated either a substantial increase or decrease in susceptibility, in confirmation of a previous study, while a few appeared to be more or less unaffected by the 1 g to 0 g change. Similar measurements at lunar and Martian gravity levels revealed further interindividual differences in susceptibility as a function of g-level. The gravity-dependent subjects revealed either 1) a progressive change in susceptibility as a function of g-load, 2) a given susceptibility level that appeared to be maintained at the fractional g-load, or 3) immunity to motion sickness at all g-levels tested below the Earth standard. The case history as well as ground-based functional and provocative tests of these normal subjects proved to be inadequate in predicting susceptibility to motion sickness under subgravity conditions.

ACKNOWLEDGMENT

Our sincere appreciation is expressed to Mr. Donald Griggs, chief of the zero-g section, and to crew members of the KC-135 at the Wright-Patterson Air Force Base, Ohio, who continuously provided order and precision for our experimental work under very difficult parabolic flight conditions. The subjects are commended for their full cooperation in spite of repeated exposures to forces that often evoked severe symptoms of motion sickness.

INTRODUCTION

In a recent report (15) dealing with susceptibility to motion sickness in the weightless phase of parabolic flight, the findings indicated that about one half of the subjects were less susceptible aloft than under ground-based conditions and that the others were more susceptible. There was also some indication that susceptibility under ground-based conditions might have value in predicting susceptibility aloft, although this point was not emphasized since the differentiation based upon this criterion was far from clearcut and the number of subjects was small. The present study represents an extension of the earlier study, in part by augmenting the number of subjects exposed in weightlessness and in part by increasing its scope to include exposing subjects to fractional g-levels.

PROCEDURE

SUBJECTS

A total of 74 normal male volunteer subjects, whose ages ranged from 19 to 39 years (60 were between 19 and 25 years), were tested under terrestrial and weightless conditions; twenty-one of the subjects were tested in zero g on more than one occasion and eighteen were tested additionally at two other subgravity levels. The group was comprised of 43 military pilots or pilot-trainees, 13 nonaviator officers, 13 enlisted men, and 5 civilians. Each subject was physically qualified for flying and in good health on each day tested. Furthermore, tests of the specific vestibular organs (5, 11, 12, 14) revealed functional responses that were within normal limits.

METHOD

Weightlessness (zero g) and two other subgravity levels (0.17 g and 0.38 g) were generated by parabolic trajectory flights of a jet aircraft (USAF KC-135) that had been specially equipped and modified as an airborne laboratory. As many as three standard rotating chairs, modified to operate under flight stresses, were bolted to the aircraft frame as near as possible to the aircraft's center of gravity. The computer-directed flight trajectories typically yielded approximately 22 seconds of zero g, 27 seconds of lunar gravity (0.17 g), and 48 seconds of Martian gravity (0.38 g) within an accuracy of ± 0.01 g.

The method for measuring motion sickness susceptibility at subgravity and terrestrial levels was similar and essentially as described fully in another report (15). The subject, with eyes covered and seated in the chair rotating at a constant velocity, made standardized 90-degree head movements (Figure 1) in response to tape-recorded instructions until he manifested a pattern of symptoms that quantitatively indicated that he had reached the endpoint of moderate malaise (M IIA) (Table I) (7), or was asymptomatic upon reaching 175 head movements, or when aloft, the flight was terminated prematurely. The rotational velocity individually preselected for evoking malaise IIA in a gradual, controllable manner was in most cases predicted from the subject's Motion Experience

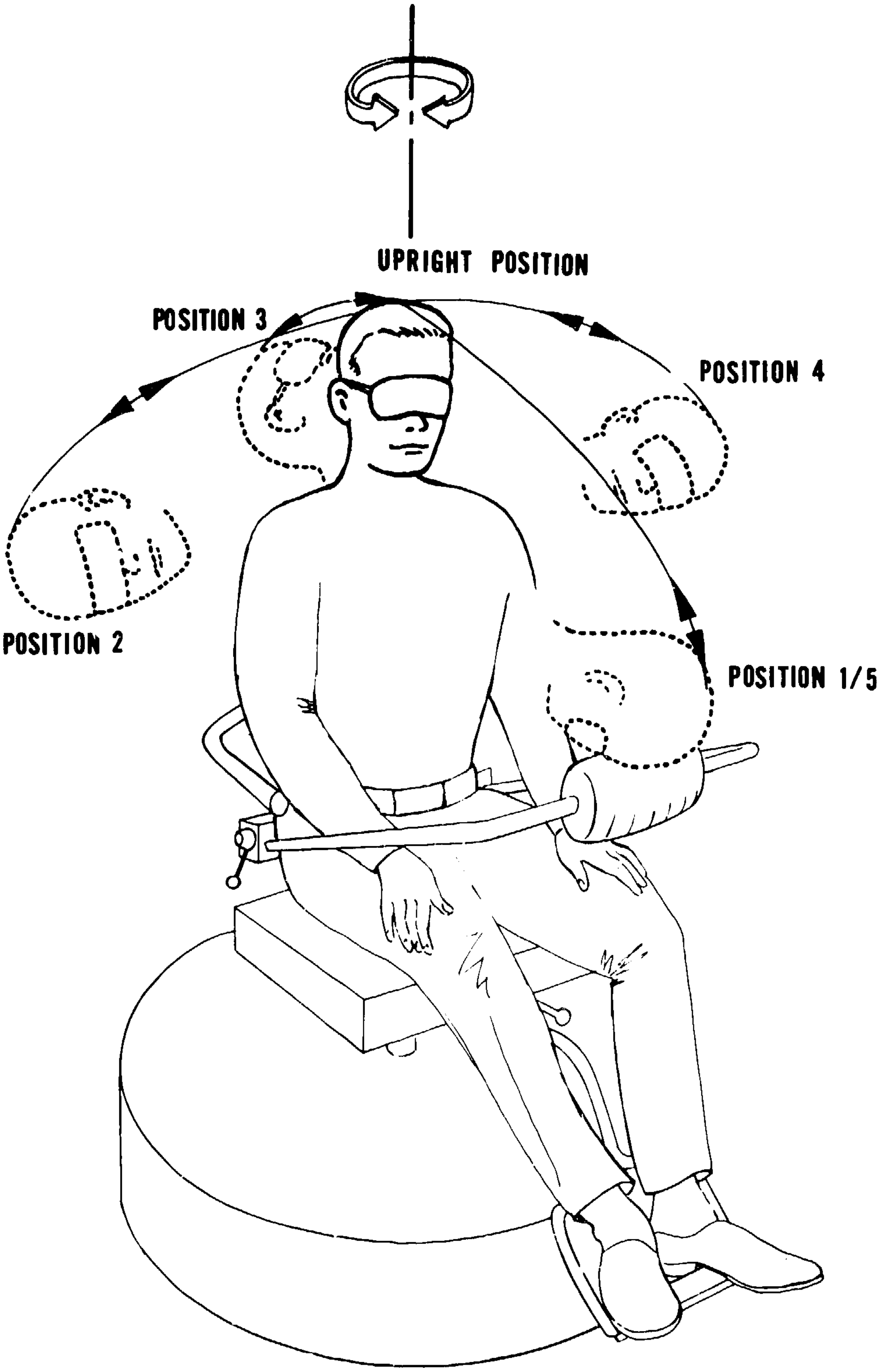

Figure 1

Diagram of standardized procedure for making each sequence of head movements to and from tilt position 1 through 5 during chair rotation.

Table I

Diagnostic Categorization of Different Levels of Severity of Acute Motion Sickness

Category	Pathognomonic 16 points	Major 8 points	Minor 4 points	Minimal 2 points	AQS* 1 point
Nausea syndrome	Nausea III†, retching or vomiting	Nausea II	Nausea I	Epigastric discomfort	Epigastric awareness
Skin		Pallor III	Pallor II	Pallor I	Flushing/Subjective warmth $\geq$ II
Cold sweating		III	II	I	
Increased salivation		III	II	I	
Drowsiness		III	II	I	
Pain					Persistent Headache $\geq$ II
Central nervous system					Persistent Dizziness: Eyes closed $\geq$ II Eyes open III

Levels of Severity Identified by Total Points Scored

Frank Sickness	Severe Malaise	Moderate Malaise A	Moderate Malaise B	Slight Malaise
(FS)	(M III)	(M IIA)	(M IIB)	(M I)
$\geq$ 16 points	8 - 15 points	5 - 7 points	3 - 4 points	1 - 2 points

*AQS - Additional qualifying symptoms.

†III - severe or marked, II - moderate, I - slight.

Questionnaire data as well as his level of motion sickness manifested during the prerequisite indoctrination flight. This flight consisted of several parabolic maneuvers that generated typical transient periods of weightlessness preceded and followed by hypergravic (~ 2 g) periods of approximately 15 seconds' duration. During each maneuver the subject was restrained in an aircraft seat and his head was immobilized by an orthopedic collar device.

Testing under 1-g conditions involved repeating the sequence of five head movements (frontward, upright, pause, rightward, upright, pause, backward, upright, pause, leftward, upright, pause, frontward, and upright) executed over a 14-second period followed by 20 seconds in which the head was kept in the terminal upright position. This sequence of test events simulated those necessarily introduced by the subgravity and the normal/hypergravic phases of the parabolic maneuver, respectively, but there were certain procedural differences. In order to take full advantage of each maneuver, head movements were continued in the described clockwise sequence throughout the subgravity phase only, which allowed usually eight head movements in 0 g, 11 in 0.17 g, and 17 in 0.38 g per parabola. Allowing greater numbers of head movements in a series without pause would tend, in theory at least, to increase slightly the difficulty in preventing overshoot of the M IIA endpoint, but in practice, diagnosis and endpoint control were readily accomplished. The rest periods between head movement sequences, which occurred during the recovery phase of the parabola and the aircraft turnaround maneuvers, averaged approximately 40 seconds at all subgravity levels. This was about double the rest period of the ground-based test version, which was found to be long enough to allow symptoms to develop and be manifested but short enough to prevent any significant recovery from the symptoms. Inflight, symptom recovery during normal test conditions was undetectable and must have been small. When unusual flight requirements introduced long intratest delays, the data were rejected and the subject was retested on another day. The reliability of the method has been demonstrated by the similarity of results obtained in repeated tests of the same subject in this study and by a test-retest evaluation in a previous study (16).

The term "equivalent head movements" was introduced in this study to allow direct comparisons of the number of head movements executed at various g-levels and rotational velocities of the chair. The chair velocity often had to be changed from that used to calibrate a subject on the ground in order to fit the test situation as well as to elicit a proper growth rate of symptoms when an upward or downward shift in susceptibility occurred with a change in gravitational loading. The data were reduced to the common denominator of equivalent head movements that served as an inverse measure of susceptibility by utilizing vestibular stressor effect (E-factor) values associated with each of the test velocities (2.5 to 30.0 rpm) as determined in a previous study (17) and presented in Table II. The most frequently used rotational velocity for each individual served as the comparison base; equivalent numbers of head movements at other velocities were found by multiplying the number of head movements executed in reaching malaise IIA times the average stressor value of each head movement assigned to each of these velocities. Ten head movements at 5 rpm, for example, are calculated as approximately equivalent in terms of provocative effect to one head movement at 20 rpm.

Table II

Relative Stressor Effect (E-Factor) of a Single Head Movement Designed to Evoke Malaise IIA as a Function of Rotational Velocity

Rotational Velocity rpm	E-Factor
30.0	0.67
25.0	0.48
20.0	0.33
15.0	0.205
12.5	0.15
10.0	0.105
7.5	0.064
5.0	0.032
2.5	0.01

Change in susceptibility with the alteration of the acting g-force was determined by subtracting the number of equivalent head movements executed under subgravity conditions from the average number executed on the ground (before and after testing in parabolic flight) as expressed by the following equation:

$$\Delta EHM = \overline{EHM}_{1\ g} - EHM_{sub\ g}$$

Positive Δ EHM resultant values indicate an ability to make greater numbers of head movements in 1 g as compared to the subgravity level; in other words, that susceptibility is an inverse function of g level. Negative values state the opposite.

RESULTS

The zero-g data obtained on 74 subjects are shown in Figure 2 and arranged vertically in descending order from the maximum positive to maximum negative Δ EHM's scaled on the x-axis. The results of repeated testing of a single subject connected in the figure by a vertical bar are chronologically arranged in the same descending order. This arrangement forms a continuum of Δ EHM values which pass through zero; grouped around this fulcrum of the distribution are a few subjects who demonstrated little or no essential change in susceptibility with the complete lifting of the g-load. For most subjects a greater number of head movements was required to reach the endpoint when the test was repeated in weightlessness.

Figure 3 illustrates the motion sickness susceptibility in terms of equivalent head movements of 18 subjects measured as a function of g-load. Not all of the four test g-levels are represented for every subject due to the unavailability of the aircraft or of the subject in a given test series. The data are arranged in rows A through D and columns 1 through 5 and grouped according to similar responses. Susceptibility as a continuous function of g-level is suggested by the dashed line connecting the bar values of the graphs. This representation of the data should be considered as an approximation of this function since a large data gap exists between 0.38 g and 1 g. The three general types of responses found in the larger group of subjects were by chance demonstrated among this smaller unselected group of subjects, as illustrated in Figure 3 by those with: 1) increased (A row), 2) essentially unchanged (B row), and 3) decreased susceptibility to motion sickness (C, D rows). However, within the two groups that revealed susceptibility changes as the g-load was reduced (A, C, D), a further subdivision was possible by grouping those who demonstrated 1) some type of progressive change in either the plus (C1, C2, C3) or minus (A1, A2, A3) direction; 2) a susceptibility level that was apparently maintained by a fractional g-load (A4, A5, C4, C5); and 3) (D 1-5) immunity to motion sickness at all g-levels tested below the Earth standard.

Figure 4 illustrates the distributions of the basic (1 g) susceptibility of all 74 subjects, expressed as the Coriolis sickness susceptibility index (16) and coded according to the three general types of altered responses revealed under the weightless condition (susceptibility in 0 g > 1 g, 0 g ~ 1 g, 0 g < 1 g).

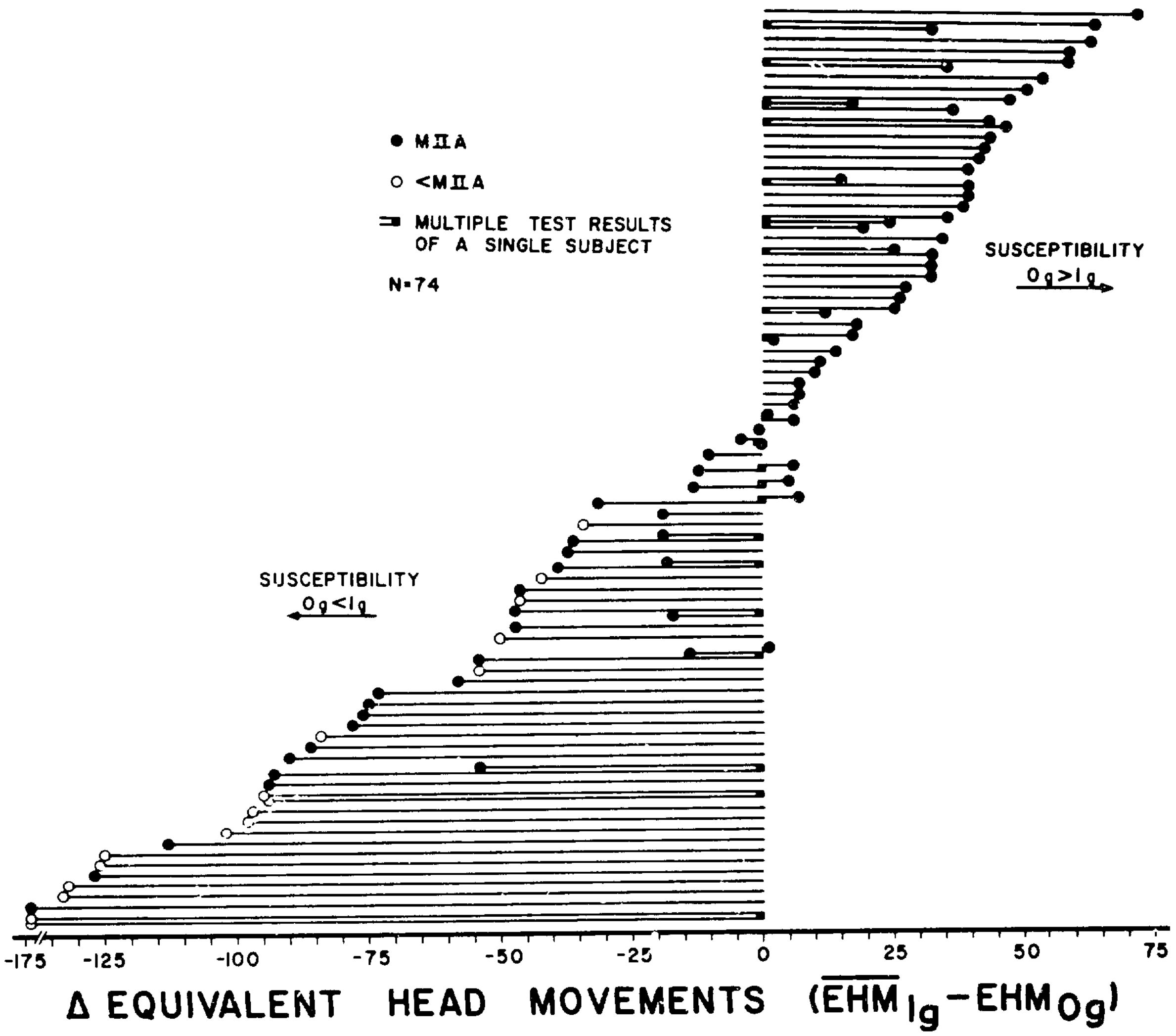

Figure 2

Range of altered susceptibility among 74 subjects tested in zero g as generated by parabolic flight. Their susceptibility change is expressed as the difference between the number of equivalent head movements executed under zero- and Earth-gravity conditions, reaching the endpoint of malaise IIA, or a limit of head movements imposed by the test conditions.

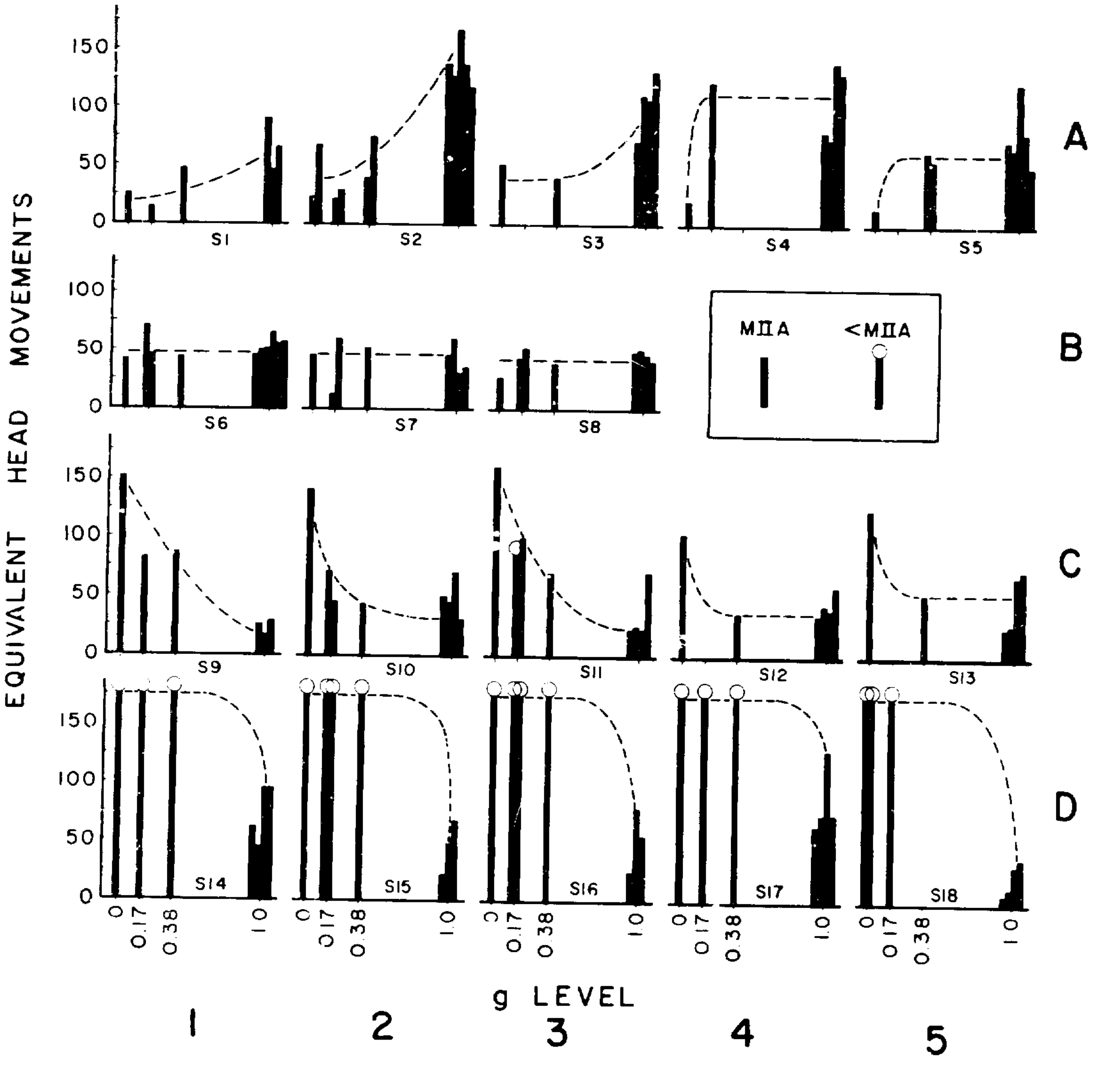

Figure 3

Motion sickness susceptibility of 18 subjects (S1 - S18) measured on the ground (1.0 g) and during parabolic flight maneuvers that simulated zero, lunar (0.17 g) and Martian (0.38 g) gravities. Dashed line connecting bars indicates approximate susceptibility as a continuous function of g-level. See text for explanation of 1 to 5 and A to D.

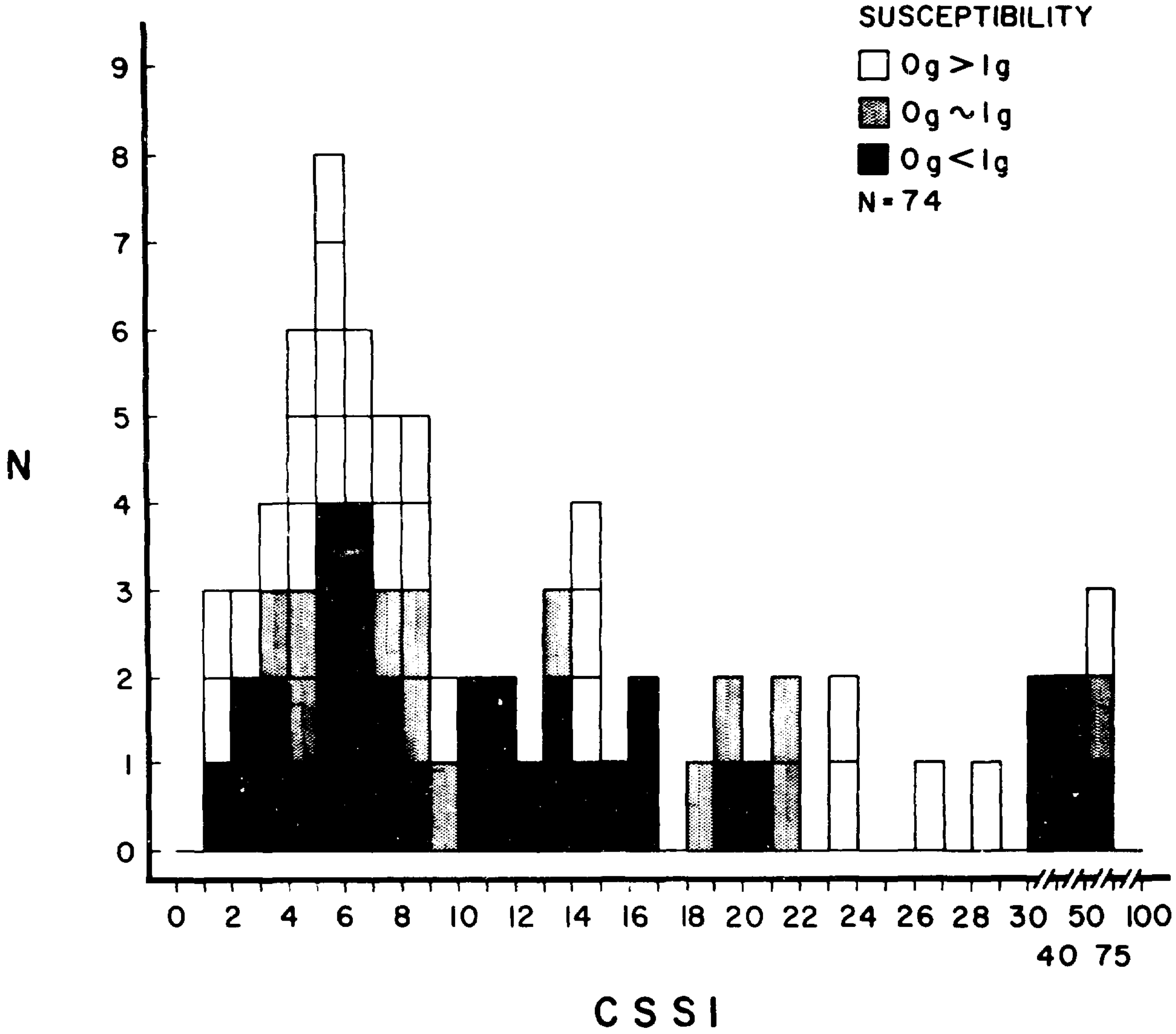

Figure 4

Frequency distribution of the ground-based Coriolis Sickness Susceptibility Index (CSSI) of 74 subjects coded according to the three general types of altered susceptibility in zero g (susceptibility: 0 g > 1 g, 0 g ~ 1 g, 0 g < 1 g).

DISCUSSION

The data confirm the positive or negative change in susceptibility with lifting the g-load but not the use of ground-based measured susceptibility in predicting these changes as was suggested by evidence previously reported (15). In addition, the present study identified a small percentage of the subjects who had essentially gravity-independent susceptibility. When the susceptibility change occurred,it was often so dramatic that the subject could readily recognize the difference in the stressor effect between tilting his head under normal and under weightless conditions. Head-tilt movement is a simple technique of changing the stimulus to the vestibular organs, the essential mechanism in the etiology of motion sickness. If the resultant angular and linear accelerative stimulus pattern is bizarre in terms of everyday experience, afferent vestibular impulses may trigger a complex chain of neural events, causing in part the manifestation of neurovegetative symptoms.

It is generally accepted that either of the specific labyrinthine organs, the semicircular canals and otoliths, can act as the primary cause of motion sickness (6, 18, 19). However, the contribution of each in the alteration of the susceptibility level with the lifting of the g-load can be differentiated at least on a theoretical basis. The physical properties of the cupulo-endolymph system render it essentially independent of gravitoinertial influence within the bony labyrinth, whereas the cilio-otolith system comprises the specialized gravireceptor organs. The latter system is physiologically deafferented in weightlessness, and apparently responsive to g-loading in accordance with Fechner's law (13). Under subgravity conditions, therefore, otolithic afferent impulses would be expected to be totally different from those normally generated by head movements. These explanations, however, do not rule out the provocative effects of canalicular afferent impulses modified at some level by interaction with altered otolithic inputs. Canal response as measured by various functional tests, for example, has been shown to be gravity dependent (1, 10, 20, 21).

The parabolic flight method of simulating weightlessness is often criticized on the basis that the hypergravic phases of the maneuver introduce variables that influence and perhaps even invalidate the results obtained during the relatively short intermediate subgravity periods. We recognize well the potential impact of this factor on our data, particularly in the study of motion sickness since the parabolic flight maneuvers can in themselves evoke this malady. For this reason every attempt was made in this study to reduce or determine the relative contribution of any provocative effect of the porpoising maneuvers. Subjects who demonstrated severe susceptibility during their indoctrination flight were excused from further study. Individuals who demonstrated parabolic flight susceptibility were tested first in each mission in order that their tests could be completed well within the number of parabolas that first provoked significant symptoms without head movements. Tolerance to the trajectories generally increased with each flight. No apparent relationship was found between the level of susceptibility of porpoising maneuvers, per se, and the altered response to cross-coupled angular accelerations during the weightless phase of these movements. Furthermore, the three general types of altered responses were found among those subjects with extensive experience with zero-g flight maneuvers or immunity to their provocative effect.

Another factor that would support a claim that, under the conditions of our study, the stressor stimulation derived from active head movements was more important than that from the porpoising maneuvers was the fact that, following the test procedure, the evoked symptoms of the diagnostic endpoint did not increase and very often gradually decreased in severity if the subject restricted his movement as the aircraft maneuvers were continued. It had also been demonstrated in a previous study (15) that certain subjects remained symptomless when their heads were immobilized during the weightless phase of parabolic flight and developed severe symptoms only when head movements were made under similar conditions (Figure 5). If the parabolic maneuver contributed significantly to the production of motion sickness in either study, it is reasonable to assume that our data always would be biased toward yielding higher susceptibility in 0 g as compared to 1 g. This could account for the results in certain subjects who might have been adversely affected by porpoising, but not for those whose susceptibility was less in weightlessness. Nor would this explain why several astronauts and cosmonauts have experienced various levels of motion sickness in space but not during their extensive Earth-bound simulation training for these missions (3,4). When space sickness occurred, it was associated with head (body) movement in the near weightless environment. One crew member, for example, reported that he could evoke marked stomach awareness by purposely moving his head on at least three separate occasions during a space mission (2,3). In view of this evidence, the qualitative changes in susceptibility measured in parabolic flight would appear valid; magnitudinal differences, on the other hand, might well have been colored by this method of simulating g zero.

The pilots who experienced space sickness might fall into our 0 g > 1 g susceptibility group since this categorization was at least valid for one astronaut who was tested following his experience of severe space sickness (3). Further validation of comparison between parabolic- and space-flight results may be possible in conjunction with the proposed Skylab vestibular experiment which will measure susceptibility as a function of length of exposure to weightlessness. Since the routine case history, as well as ground-based functional and provocative test results, of normal subjects is presently inadequate in predicting susceptibility to motion sickness under subgravity conditions, susceptibility tests in parabolic flight remain the only presently available means* of identifying crewmen who may be adversely affected by normal activity in the weightless spacecraft. These individuals could then be trained prior to flight to increase their zero-g tolerance.

One means that has been suggested to protect crewmen against space sickness is to generate by spacecraft rotation some level of artificial gravity. The susceptibility data presented herein as a function of g-level suggest guarded optimism in this approach

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*Soviet investigators have claimed that tolerance to swinging in a four-bar Khilov swing is related to reactions in parabolic flight (9).

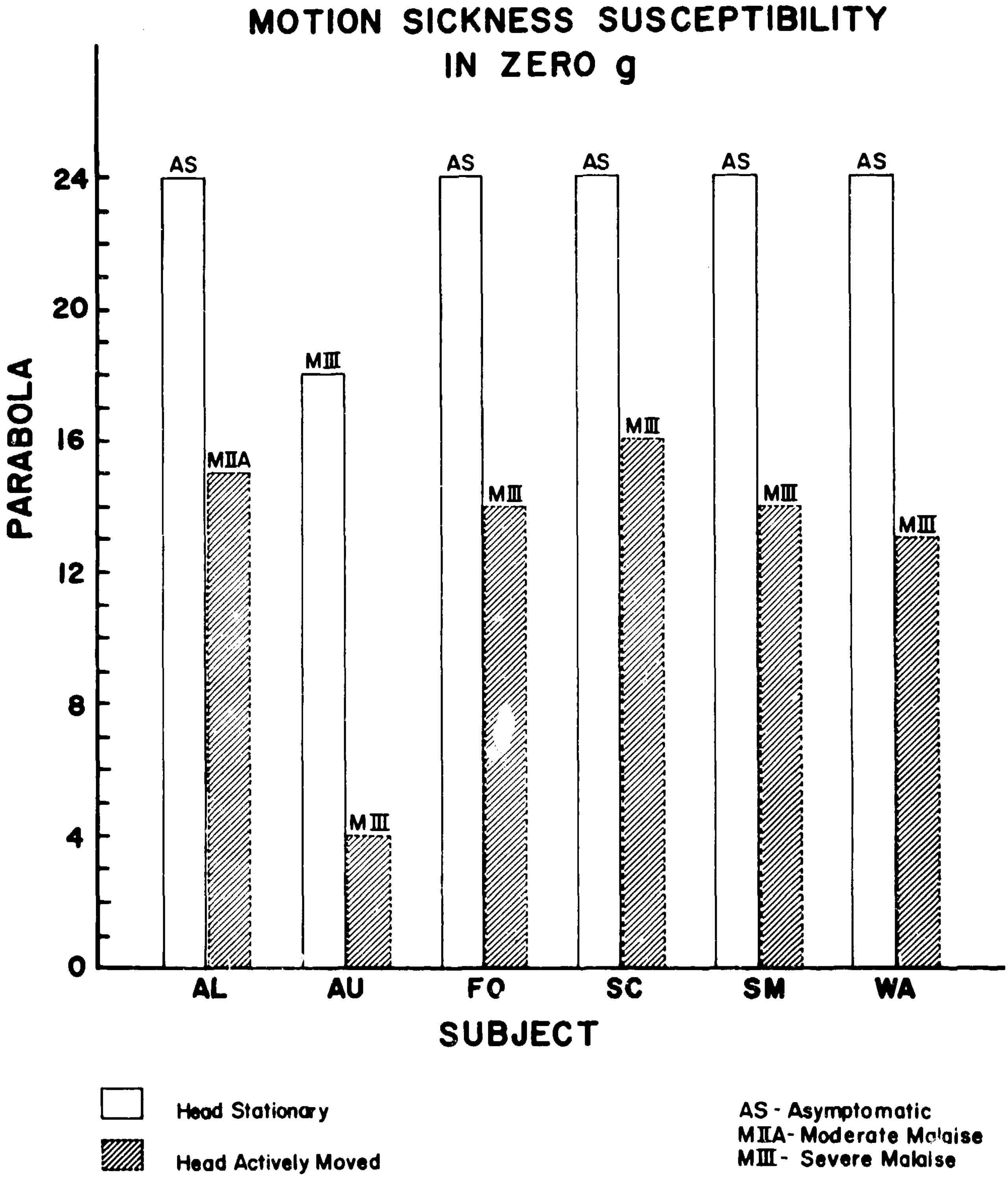

Figure 5

Effect among six susceptible subjects (ref. 15) of active head movements relative to the restrained condition upon motion sickness susceptibility measured in terms of the number of parabolas required to provoke malaise III.

as a remedial measure since several subjects with 0 g > 1 g susceptibility were aided by increasing the g-load; however, the benefit derived by other subjects (e.g., A1, A2, A3, in Figure 3) was slight and perhaps inadequate for habitability in space without supplemental preflight or inflight procedures to increase protection.

The procedure of repeated testing of motion sickness susceptibility, even though designed to avoid adaptation insofar as possible, appeared to decrease the susceptibility of certain persons. The training effect in these cases seemed to be nonspecific as to g-loading, but more systematic studies of adaptation transfer from one g-level to another are required to assess this factor. It is possible (8) that crew requirements for evaluating hardware and flight procedures in the airborne laboratory prior to flight may be incidentally acting to increase tolerance for subsequent activity in the weightless spacecraft.

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