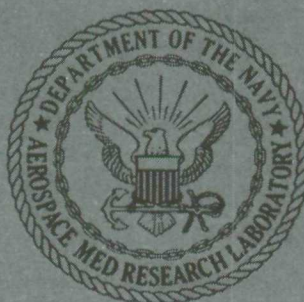


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DIRECTION-SPECIFIC ADAPTATION EFFECTS ACQUIRED
IN A SLOW ROTATION ROOM

Ashton Graybiel and James Knepton



NAVAL AEROSPACE MEDICAL RESEARCH LABORATORY

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SUMMARY PAGE

THE PROBLEM

Two stress profiles were used to elucidate the characteristics of direction-specific adaptation effects acquired during an incremental adaptation schedule in a slowly rotating room. The characteristics would be revealed by symptoms of motion sickness of the subject during the execution of head movements. Thirty-eight subjects were required to execute 120 head movements in a slow rotation room at each 1-rpm increase in velocity of the room between 0 and 6 rpm and, after a single-step gradual return to zero velocity, execute 120 head movements either immediately after the return or after delay periods varying from 1 to 24 hours unless, at any time, more than mild symptoms of motion sickness were elicited. A second stress profile differed by the sequential addition of an incremental adaptation schedule (identical to the first) in which the direction of rotation was reversed.

FINDINGS

The experimental findings demonstrated the acquisition of direction-specific adaptation effects that underwent spontaneous decay with a short time constant (hours). With their disappearance a nondirection-specific adaptation was revealed with a long time constant (days). Speculations are presented which could account for the simultaneous acquisition of short-term and long-term adaptation effects. The findings support the theory that motion sickness, although a consequence of vestibular stimulation, has its immediate origin in nonvestibular systems, implying a "facultative" or temporary linkage between the vestibular and nonvestibular systems.

ACKNOWLEDGEMENTS

We gratefully acknowledge the participation of the subjects, the technical assistance of Mr. Roy Garlock, and the constructive criticism of our associates.

INTRODUCTION

In a slowly rotating room the vestibular organs can be stimulated in an abnormal manner by the stressful accelerations generated when the subject rotates his head out of the plane of the room's rotation. Thus, using "standardized" head movements, the experimenter can control the intensity of the stimulation by varying the angular velocity of the room; additional manipulations include "head fixation" and reversing the direction of rotation. If typically normal subjects are selected, the responses elicited are mainly of vestibular origin and tend to fall into two categories (3). The first comprises reflex responses such as "sensations," illusions, and nystagmus that are revealed through effector systems that normally articulate with the vestibular system. The responses in the second category comprise an epiphenomenon, superimposed on any manifestations of the first category, best known under the general term motion sickness. Motion sickness, although a consequence of vestibular stimulation, has its immediate origin in nonvestibular systems, suggesting a "facultative" or temporary linkage between the vestibular and nonvestibular systems, where first-order responses have their origin.

It has been demonstrated that subjects, executing a sufficient number of head movements at 1-rpm increments in rate of rotation, may reach otherwise intolerable angular velocities either symptom free or nearly so (4), implying that the acquisition of adaptation to rotation involves the vestibular system proper and not the nonvestibular systems involved in the genesis of motion sickness. It has also been demonstrated (10, 11) that subjects exposed to an "incremental adaptation schedule" are not (similarly) affected when head movements are executed after a single step return to zero velocity; if symptoms are experienced before return to zero velocity, they are nearly always aggravated, but if symptoms are not experienced, they may or may not be elicited after return. The present study represents an attempt to elucidate the characteristics of the direction-specific adaptation effects acquired during an incremental adaptation schedule and revealed by symptoms of motion sickness during the execution of head movements after either a one-step return to zero velocity or a reversal of direction of rotation.

PROCEDURE

SUBJECTS AND APPARATUS

Thirty-eight Navy ensigns, 22 and 23 years of age, served as subjects. They were chosen from a group of 51 tested volunteers (awaiting flight training), mainly on the basis that they did not become frankly sick and "fail" the first experimental test and that they would be available for subsequent testing. All were in excellent health, and medical evaluation revealed that they were fit for duty involving flying. All except two (subjects 19 and 26) were measured on one or more tests of canalicular (8, 1), otolithic (9), and "vestibular" (2) function, and with three exceptions (subjects 4, 37 and

35) measures in all categories were obtained. All of the test results were within the normal range.

A large rotating room, situated in an air-conditioned laboratory complex, was used. The room could be rotated in a clockwise (CW) or counterclockwise (CCW) direction, and its performance characteristics and instrumentation were not taxed in this experiment.

METHOD

The Stress Profiles

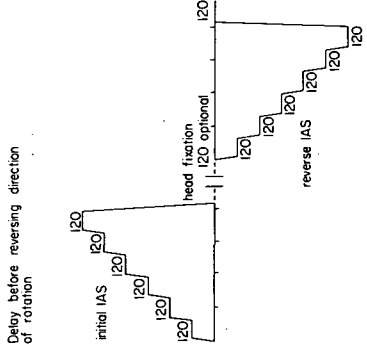
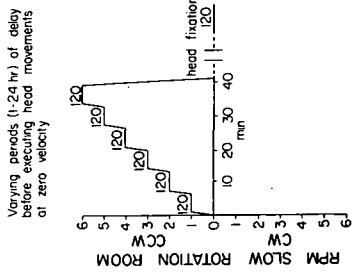
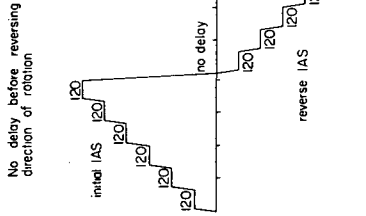
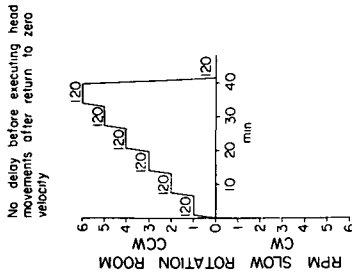
Abnormal accelerations were generated by the active rotation of the subject's head (and body) out of the plane of the room's rotation; the distance between the center of the subject's head (seated upright) and the center of the room was approximately 46 inches. The head movements were executed while the subject was seated on a specially designed chair that had adjustable pads (front, back, left, and right) acting as "stops" limiting the head movements in the four quadrants — in this experiment, rotation through 90 degrees. The eight movements ("over" and "back" in the four directions) were randomized and a taped recording set the cadence at one movement every 3 seconds. The eight movements involved were called a "sequence," and the subject was always required to execute 120 head movements (15 sequences) unless the endpoint was reached.

Two "standard" stress profiles were used. One required the execution of 120 head movements at each 1-rpm increase in rotation (CW or CCW) between 0 and 6 rpm and the execution of 120 head movements either immediately after a single-step gradual return to zero velocity ("no delay") or after delay periods varying from 1 to 24 hours (Figure 1A). About 1 minute was taken between the 1-rpm increases in velocity and 2 minutes between 6 rpm and the single-step return to zero velocity. The other standard stress profile differed from the first by the addition of a second incremental-adaptation schedule (IAS) in which the direction of rotation was reversed either immediately after return to zero velocity or after delay periods measured in hours (Figure 1A). The terms "initial IAS" and "reverse IAS" are used because the initial direction of rotation was semirandom. Head movements at zero velocity prior to initiating the reverse IAS were optional. Any variations from the standard stress patterns are always indicated.

Scoring the Severity of Motion Sickness

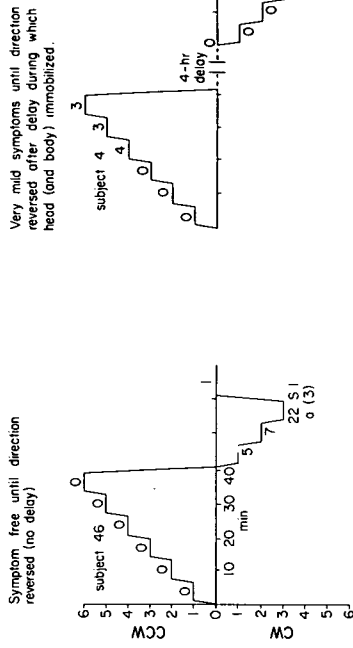
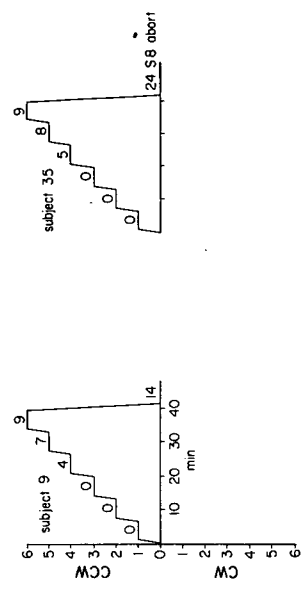
Two observers in collaboration with the subject estimated the levels of severity of the vestibular side effects. After each discrete head movement the subject signalled ("Yes" or "No") whether he detected a "sensation" of movement, an apparent visual

A. "STANDARD" STRESS PROFILES AND MANIPULATIONS INVOLVING CW AND CCW ROTATION AND ZERO VELOCITY



B. ACTUAL EXAMPLES OF SCORING SYMPTOMS OF MOTION SICKNESS IN POINTS OR SUSCEPTIBILITY TO MOTION SICKNESS IN TERMS OF LEVELS OF STRESS (AND POINTS) WHEN TESTING WAS ABORTED

Typical responses



Very mild symptoms until direction reversed after delay during which head (and body) immobilized.

Keys to figures:
 IAS = incremental adaptation schedule
 S = Head-movement sequence
 o = Abort
 () = RPW at time of abort

Numbers on line in
 A: Number of head movements
 B: Motion-sickness point score

Figure 1

A. "Standard" stress profiles and manipulations involving CW and CCW rotation and zero velocity. B. Actual examples of scoring symptoms of motion sickness in points or susceptibility to motion sickness in terms of levels of stress (and points) when testing was aborted.

illusion, or a tendency to be deflected from the plane in which the movement was carried out. The severity of motion sickness symptoms was given a numerical score, according to the diagnostic criteria described elsewhere (6) and summarized in Table 1. In brief, 16 points and above comprised the range of "frank motion sickness" and fewer points, the range of "mild motion sickness." There was always adequate time after execution of the head-movement sequences to make the estimates, and "rating sheets" facilitated keeping scores. Vomiting was avoided (with rare exception) by discontinuing the head movements with the appearance of nausea.

This report deals only with motion sickness. Very low scores raise a question of validity; the investigator must decide either to use a score of even one point, as we have done here, or otherwise to define what constitutes "motion sickness." Figure 1B, which summarizes actual experimental findings, helps to make clear how the data were handled. Subject 9 manifested mild symptoms of motion sickness during steps 4, 5, and 6 of the IAS and reached a typical endpoint on completing the head movements after a one-step return to zero velocity. The measurements for subject 35 were similar to those for subject 9 during the IAS, but testing was aborted after he executed 64 head movements (8 sequences) after return to zero velocity. Subject 46 was symptom free during the initial IAS, but testing was aborted during the reverse IAS (without delay) after eight head movements had been executed at 3 RPM. A 4-hour delay separated the initial and reverse IAS in testing subject 4; testing was aborted after he completed the head movements at 4 rpm.

Operational Plan

The ensigns rapidly became familiar with the equipment, the execution of standardized head movements, and the method of scoring responses. Prior to every experimental trial the investigator (with the aid of a questionnaire) made sure that the subject was fit and explained the nature of the task for that day. In tests in which the delay at zero velocity was relatively brief, the subject sometimes remained in the slow rotation room (SRR) with head fixed. Usually the subject remained in an adjacent ward under constant surveillance to ensure that his head was always fixed, at least relative to the thorax, and that his needs were met with minimal moving about.

The problem of each subject serving as his own control (inter-test adaptation) in a series of experimental tests was met in part by the long period between test sessions (usually 5-9 days), in part by the use of decreasing as well as increasing periods of delay at zero velocity, and in part by the introduction of a test with "no delay" which could be compared with earlier experimental tests.

The results to be reported fall into three parts, and some additional details concerning experimental procedures will be given in the appropriate sections.

Table I

Scoring Severity of Acute Motion Sickness

Category	16 points	8 points	4 points	2 points	1 point
Nausea syndrome	Vomiting or retching	Nausea [†] II, III	Nausea I	Epigastric discomfort	Epigastric awareness
Skin color		Pallor III	Pallor II	Pallor I	Flushing
Cold sweating		III	II	I	
Increased salivation		III	II	I	
Drowsiness		III	II	I	
Pain					
Central nervous system					Headache
					Dizziness:
					Eyes closed \geq II
					Eyes open III

[†]III = severe or marked, II = moderate, I = slight.

RESULTS AND DISCUSSION

PART I. EVIDENCE FOR DIRECTION-SPECIFIC ADAPTATION EFFECTS

Table II A summarizes the findings in 23 subjects chosen because they were symptom free or nearly symptom free on completion (6 rpm) of the initial IAS in their first experimental test. Inasmuch as head movements at zero velocity generate normal vestibular stimuli, any increase in the susceptibility-to-motion-sickness score on return to zero velocity compared with that at 6 rpm is a measure of adaptation to rotation and consequent loss of adaptation to the stationary environment.

As shown in Table II A, the subjects fall into three categories. Seven of the 23 subjects were completely symptom free during the initial IAS, and four of these seven were symptom free when exposed to the provocative challenge at zero velocity, the other three manifesting mild symptoms. Six additional subjects were symptom free at 6 rpm but not during 1 rpm to 5 rpm; none of this group was symptom free during the challenge at zero velocity and one failed to complete the 15 head-movement sequences. The remaining ten subjects experienced very mild responses at 6 rpm and, with the exception of one, during 1 rpm to 5 rpm as well. On return to zero velocity, tests in five of the ten subjects had to be aborted; one subject was symptom free.

Table II B presents the scores of the 16 subjects exposed in subsequent tests to a reverse IAS immediately after completion of an initial IAS. Head movements during the reverse IAS generate abnormal vestibular stimuli; hence, the provocative challenge even at 1 rpm is greater than at zero velocity. Scores obtained during the reverse IAS that substantially exceed those elicited during an initial IAS indicate the previous acquisition of direction-specific adaptation effects. Ten of the 16 subjects failed to complete the entire test; the aborts during the reverse IAS occurred at a velocity varying from 1 rpm to 4 rpm. Among the six who completed the reverse IAS none reached the endpoint and one was completely symptom free throughout.

It is of interest to compare the scores in Table II A and B. Among the seven subjects who were completely symptom free during the first experimental test (first seven entries in the table), very mild responses were elicited in three subjects during the initial IAS in the second test, suggesting borderline susceptibility at this level of stress. Testing of all three was aborted during the subsequent reverse IAS. Among the other four (completely symptom free during the first test and the initial IAS in the second test) two reverse IAS tests were aborted, one subject manifested mild symptoms, and one was symptom free.

Results from only eight of the 16 subjects who were not completely symptom free in experimental test 1 appear in Table II B. In four of those eight subjects this was their second test in the experimental series. During the reverse IAS, testing was aborted in one of those four at 2 rpm and three manifested very mild responses. In another three

Table 11

Change in Motion-Sickness-Susceptibility Score in 23 Normal Male Subjects When Head Movements Were Executed Either Immediately After a One-Step Return From a Six-Step Incremental-Adaptation Schedule (IAS) (A), or During Exposure Without Delay to a Reverse IAS (B)

Subject Number	Experimental Test Number	A		Score on Return to Zero Velocity	Experimental-test Series Number	B		Reverse IAS Score 1-5 rpm	Reverse IAS Score 6 rpm
		Initial IAS Score 1-5 rpm*	Initial IAS Score 6 rpm			Initial IAS Score 1-5 rpm	Initial IAS Score 6 rpm		
2	1	0	0	6	2	2	3	$\alpha(3-4)^+$	4
3	1	0	0	0	2	0	0	9	0
21	1	0	0	0	2	0	0	0	0
22	1	0	0	4	2	1	3	$\alpha(1)S5$	0
31	1	0	0	0	2	2	0	$\alpha(1-2)$	0
33	1	0	0	0	2	0	0	$\alpha(1)S13$	0
39	1	0	0	5	2	0	0	$\alpha(3)S2$	2
11	1	1	0	4	5	0	0	2	0
10	1	2	0	1	2	0	0	1	1
16	1	2	0	$\alpha(S9)26$					
43	1	2	0	3					
44	1	2	0	7	2	4	4	$\alpha(2)S4$	0
38	1	2	0	12					
18	1	6	0	1	2	1	1	$\alpha(2)S3$	0
28	1	1	1	1					
37	1	0	2	$\alpha(S13)22$	5	0	0	$\alpha(4)S6$	0
13	1	2	2	$\alpha(S8)15$					
19	1	2	2	$\alpha(S6)10$					
25	1	2	2	$\alpha(S4)12$					
42	1	2	2	6	3	0	2	$\alpha(1)S12$	3
34	1	2	2	2	2	0	0	3	0
15	1	4	2	0	2	2	2	4	0
46	1	4	2	$\alpha(S2)14$	5	0	0	$\alpha(3)S1$	0
		4	2	8					

* Maximum motion-sickness score.

+ α , abort; numbers in parentheses, rpm at abort.

S, sequence-of-head-movement number.

of the eight subjects it was their fifth experimental-series test; all were symptom free during the initial IAS, but during the reverse IAS testing was aborted in two of those three and one manifested very mild symptoms. For the remaining subject it was the third test in the series, and although he was nearly symptom free during the initial IAS, his testing was aborted at 1 rpm during the reverse IAS.

Discussion

The experiments demonstrate that a high degree of direction-specific adaptation can be acquired by subjects in whom symptoms of motion sickness are either absent or very mild during an initial incremental adaptation schedule. That the degree of direction-specific adaptation acquired varies among subjects is shown by the fact that, for some, the challenge immediately after a one-step return to zero velocity is sufficient to elicit responses, while in others the far more severe challenge of reversing rotation is required. In all cases, however, the acquisition of direction-specific effects in subjects symptom free or nearly so simplifies the investigator's task in that he can deal with symptoms elicited by the provocative challenges and avoid the need to take into account the time course of recovery from symptoms of motion sickness.

Absence of motion sickness during an initial IAS reflects either a failure of the abnormal vestibular impulses significantly to disturb the stability of the vestibular system or implies that the disturbance of stability has not reached the point at which vestibular influences escape their normal bounds to elicit symptoms of motion sickness. The failure is illustrated by the measurements in subject 21 (Figure 2a) who was symptom free during the challenge at zero velocity (test 1) and during a reverse IAS carried out 7 days later (test 2). Subject 33 (Figure 2b) was symptom free during the challenge of a one-step return to a zero velocity (test 1) but not during the reverse IAS (test 2) 6 days later, when testing was aborted at 1 rpm. An abort at 1 rpm must be attributed to antecedent events. The stimulus intensity during the initial IAS of test 2 was sufficient to disturb the stability of the vestibular system, yet not to a degree that would allow the escape of vestibular influences and elicit motion sickness. In other words, the vestibular system was coping with the disturbance within the range of its functional reserve and, presumably, was aided by the acquisition of adaptation effects. The far greater challenge during a reverse IAS compared to that at zero velocity is demonstrated by the failure of subject 33 to complete the 15 head-movements sequences during CW rotation at 1 rpm. The diagrams of Figure 2 (c and d) illustrate the elicitation of symptoms during the challenge after a one-step return to zero velocity in two subjects symptom free during the initial IAS; subject 2 (6 points) manifested mild responses and subject 32 (serial test 4) represented the only instance when testing was aborted during that procedure.

When symptoms are elicited during an initial IAS, they indicate not only that homeostasis in the vestibular system has failed but also that adaptation is being acquired. It is not uncommon for symptoms to appear then disappear by the time terminal velocity is reached; seven such instances are shown in Table IIA. This

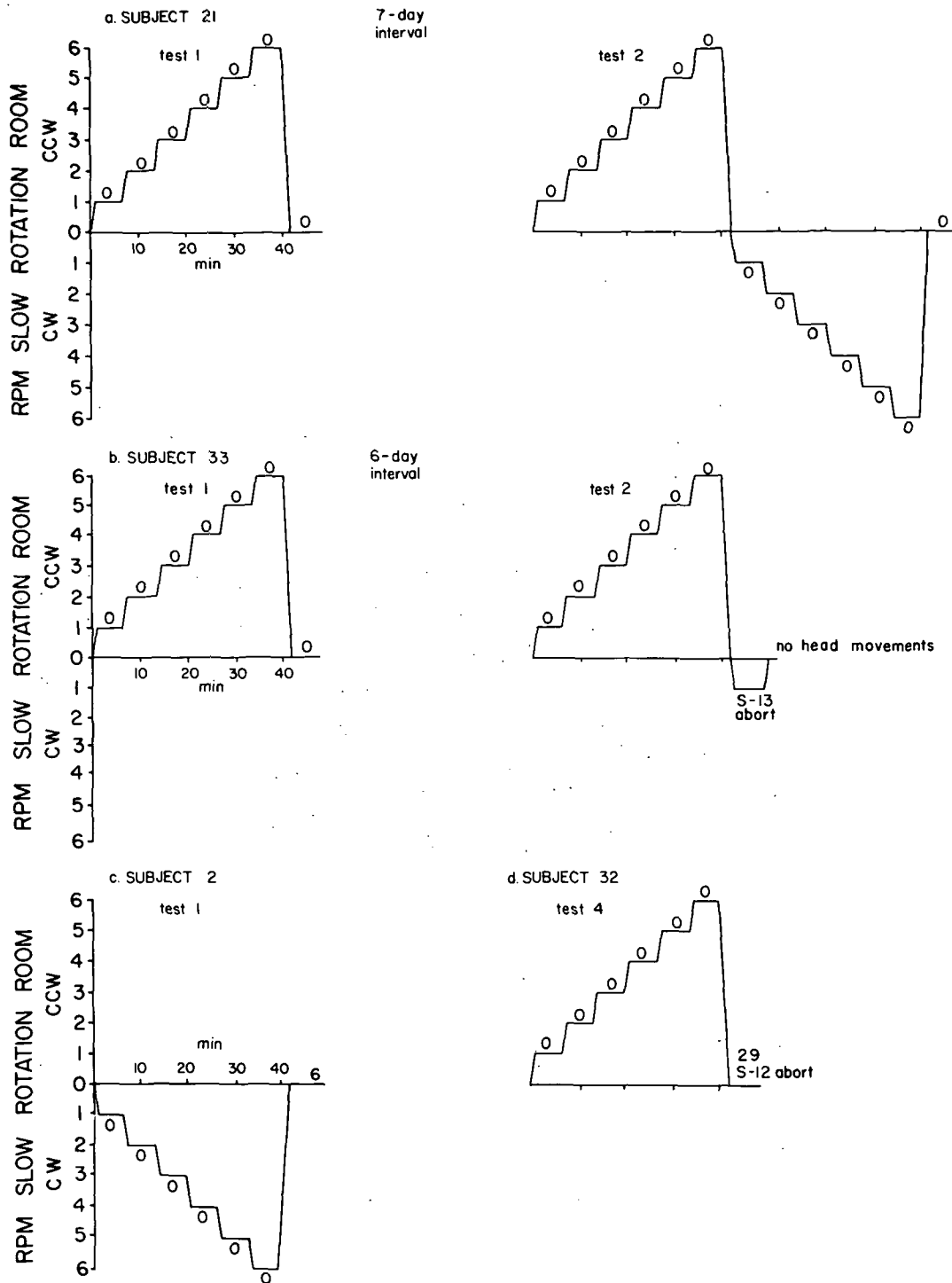


Figure 2

Differences in response among subjects symptom free during the initial IAS when challenged either immediately after a one-step return to zero velocity (execution of 120 head movements) or during a reverse IAS. a) Insusceptible to motion sickness during both challenges, b) insusceptible during the first but highly susceptible during the second (more severe) challenge. Mild (c) and severe (d) responses when challenged after a one-step return to zero velocity.

phenomenon implies either that the rate of adaptation to rotation eventually exceeds the incremental increases in stress or that secondary etiological factors, of a temporary nature, contribute to the appearance of symptoms. In either event, both the time frame within which symptoms may appear and disappear and the delicate balance of etiologic factors (primarily stressful accelerations) with which the vestibular system can or cannot cope are demonstrated. Detailed experimental studies would be required to determine the relation between severity of symptoms and the acquisition of adaptation to rotation. Our findings suggest that the severity of symptoms provides an imperfect guide to the amount of adaptation acquired and that the reason may reside in differences in susceptibility of nonvestibular cell groups (concerned in the genesis of motion sickness) to nonsystem-bound vestibular influences.

The demonstration that signals of opposite sign may be generated in the vestibular system is not new although studies showing this were concerned with reflex vestibular effects and not with motion sickness. In one (previous) series of experiments subjects were exposed to repeated stimulation by flexing the head toward the left shoulder (and return) while rotating counterclockwise in the SRR under conditions favorable for perception of the oculogyral illusion (5). Initially, during the "down" movement the illusion was perceived as an apparent upward movement of a dimly lighted target in darkness, and the reverse (apparent) movement occurred on return to the upright. After a large number of head movements the illusion was either no longer perceived or greatly reduced in the practiced direction (toward the left) but readily perceived if the head was moved toward the right shoulder. On cessation of rotation, movement of the head in the practiced direction elicited an illusory movement but one opposite in direction to the illusion initially perceived during rotation. Movement of the head in the unpracticed direction did not elicit an illusion. Essentially the same findings were obtained using nystagmus as the indicator; the nystagmic response declined with practice during rotation, and the direction of the beat reversed after cessation of rotation (7). These data demonstrate that adaptation effects acquired by executing head movements limited to one quadrant not only are direction specific but also do not readily transfer to the opposite quadrant.

PART II. SPONTANEOUS DECAY OF DIRECTION-SPECIFIC ADAPTATION EFFECTS

In this part of the experimental series the interval between the initial IAS and the provocative challenge either at zero velocity or during the reverse IAS varied from "no delay" to 24 hours. It is convenient to describe the results of the two provocative challenges separately.

Rate of Spontaneous Decay as Indicated by the Challenge at Zero Velocity

A series of experimental tests with subjects serving as their own controls was needed to plot the decay of direction-specific adaptation as a function of delay at

zero velocity. The chief procedural problem here involved long-term (inter-test) adaptation effects. Subjects were selected from among the 38 on the basis that, in repeated tests, long-term adaptation effects were either not evident or demonstrably small; the initial IAS score was always available and used as the main guide in estimating the acquisition of long-term adaptation.

Another problem was the elicitation of frank motion sickness during the initial IAS and the possibility that even after the disappearance of overt symptoms, return to normal stability would be incomplete. This problem fortunately was seldom encountered and, at worst, results in a scoring error involving tests wherein a reversal of direction occurred without delay at zero velocity.

The findings in 22 subjects are summarized in Table III. The subjects are ranked in terms of their susceptibility to motion sickness as revealed by the measurements in their experimental test 1, primarily the response at 6 rpm. The no-delay abort scores reflect the head-movement sequence during which the test was terminated. With one exception (subject 14) all aborts occurred immediately after return to zero velocity; testing in this subject was aborted both at no-delay and after a 2-hour delay at zero velocity. All subjects experienced symptoms in the no-delay tests, but subject 14 became symptom free after varying periods of delay at zero velocity; in subjects 44 and 25 the decay in direction-specific adaptation effects was fairly rapid and in subjects 29 and 32 it was moderately rapid. Subject 14, who invariably chose to exceed the usual endpoint, manifested the slowest decay, between 16 and 24 hours. Not shown in Table III is his response on reversing rotation after a 24-hour delay at zero velocity; testing then was aborted at 3 rpm.

Figure 3 represents an attempt to plot the data of Table III. The first two points on the curves represent motion-sickness-susceptibility scores in the first experimental test; that is, the score at the 6 rpm step of the initial IAS and the score after return to zero velocity, respectively. When an abort occurred at zero velocity, two points were added to the motion-sickness score for every uncompleted sequence, hence reflecting levels of hypersusceptibility rather than actual motion-sickness scores. Each subsequent point in the plot was obtained in a subsequent test conducted after a delay (1-24 hours) at zero velocity. The general configuration of the individual curves suggests that the decay in direction-specific effects occurs exponentially. It is seen at a glance that the decay in direction-specific effects for most of the 22 subjects took place chiefly or completely within 4 hours.

Rate of Spontaneous Decay as Indicated by the Challenge of Reversing the Direction of Rotation

Long-term inter-test adaptation presented a procedural problem and limited both the number of subjects and the number of experimental tests in a series that were

Table III

Rate of Spontaneous Decay (By Head Fixation) in Direction-Specific Adaptation Effects Acquired During Exposure of 22 Subjects to an Incremental-Adaptation Schedule (IAS) in a Rotating Room and Revealed by Executing Head Movements After Return to Zero Velocity

Subject No.	Initial IAS Score 1-5 rpm*	6 rpm	Scores Return to Zero Velocity, No Delay	Serial Test Numbers and Scores on Return to Zero Velocity After Delays Measured in Hours (1-24)																	
				1	2	3	4	5	6	8	16	24									
22	0	0	4	4	11	3	0														
16	2	0	a26, S9 ⁺																	2	1
44	2	0	7					4	0												
38	6	0	12			2	2														
28	1	1	a22, S13			2	5														
37	0	2	a15, S8			2	4													4	0
13	2	2	a10, S6					3	2												
25	2	2	6			2	0														
46	4	2	8																		
15	4	2	a14, S2			2	9														
17	3	3	8			2	1														
4	5	6	a16, S5																		
7	6	6	8			2	2														
29	3	8	a24, S2			6	0														
40	11	8	10			2	3		3	1											
9	7	9	14			2	18														
35	8	9	a24, S8																		
41	6	9	a14, S3																		
26	10	10	18																		
32	7	14	a32, S3																		
14	11	18	a28, S1			2	a56														
36	13	19	a19, S5			2	2														

* Maximum motion-sickness score.

+ a, abort; S, sequence-of-head-movement number.

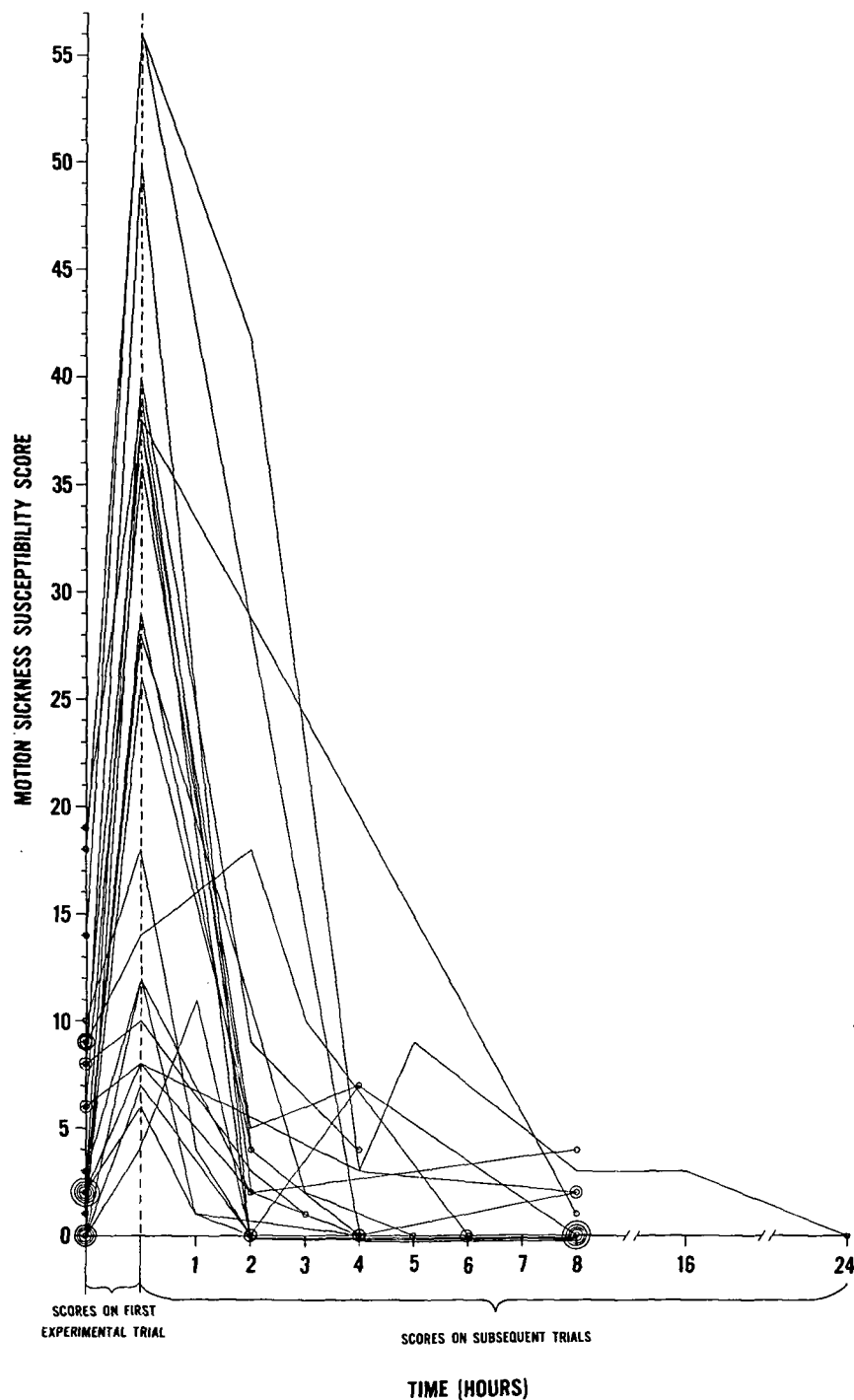


Figure 3

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

suitable for analysis. A second problem involved scoring and interpreting tests that were aborted during the reverse IAS. The more important figure in these abort scores is the rpm at which the abort occurs and not the points indicating the severity of motion sickness. The severe provocative challenge during the reverse IAS minimized the problem presented in maintaining head fixation over long periods for the reason that a few head movements (at zero velocity) would scarcely affect the reverse IAS challenge.

Table IV summarizes the findings in 27 subjects ranked primarily in terms of completeness of disappearance of direction-specific adaptation effects (reverse IAS scores shown on the right) and secondarily in terms of the number of hours (delay-time) required. The left side of the table shows two no-delay scores which serve as baselines for measuring the decay in direction-specific adaptation effects. Challenge at zero velocity scores greater than 4 points correctly foretold, in our experience, that testing would be aborted during a reverse IAS (no delay) in a subsequent test. In test 1 the measurements in 24 of the 27 subjects (11 aborts and 12 scores of 5 points or more) indicated that testing would be aborted during a no-delay reverse IAS. This use of test 1 scores minimized inter-test adaptation when test 2 was a reverse IAS after delay, often after long-delay periods. The no-delay reverse IAS scores were measured in 11 subjects; 6 of these 11 were undergoing test 2. When the data in the two no-delay tests were combined, it was found that an abort had occurred or could have been predicted in test 2 during a no-delay reverse IAS in all except subject 3.

The sequential ordering of the tests represents the attempt to minimize or at least to reveal the effect of inter-test (long-term) adaptation. In measuring 8 subjects the longest delay time (2 to 8 hours) occurred in the second test in the series, thereby reducing to a minimum inter-trial adaptation; at that time 2 were symptom free, 4 experienced mild or very mild symptoms, and testing was aborted in the remaining 2 subjects. These findings are fairly representative of the entire group.

The first subject (25) in Table IV illustrates rapid decay of direction-specific adaptation effects. In test 2 of the experimental series (2-hour delay) he was symptom free, yet in test 3 (no delay) testing was aborted at 1 rpm during the reverse IAS, indicating that he had acquired a high level of these adaptation effects during the initial IAS. Moreover, inter-test adaptation was minimal because in test 4 (1-hour delay) testing was aborted at 1 rpm. Subject 14 illustrates the slowest decay of direction-specific adaptation among those 27 subjects. Measurements in subject 37 illustrate adequate handling of inter-test adaptation and in subject 32 the difficulty in handling inter-test adaptation. Measurements in subject 3 (the most insusceptible among the entire group) reveal similar scores during the reverse IAS with no delay and with delay periods of 4 and 6 hours. Drowsiness was the symptom mainly responsible for the scores, and this response, in some persons at least, may be elicited at low levels of stress as the only manifestation of motion sickness (4).

Table IV

Spontaneous Decay (by head fixation) in Direction-Specific Adaptation Effects as a Function of Delay at Zero Velocity Between the Initial and Reverse Incremental Adaptation Schedule. The 27 Subjects are Ranked Primarily According to Increasing Susceptibility to Motion-Sickness During a Reverse IAS and Secondarily According to Increasing Intervals (1 to 24 hours) Between the Initial IAS and the Reverse IAS. The Scores Obtained after a One-Step Return to Zero Velocity in the Experimental Test 1 have "Baseline" Value.

		No Delay Scores																	
		Subsequent Tests Involving Change in Direction of Rotation					Serial Test Numbers and Scores During Reverse IAS After a Delay Measured in Hours (1-24)												
Subject No.	Experimental Test 1	Initial IAS	Reverse IAS	Serial Test No.	6 rpm Score	6 rpm Score	Serial Test Numbers and Scores During Reverse IAS After a Delay Measured in Hours (1-24)												
							1	2	3	4	5	6	8	16	24				
Score on Re-turn to Zero Velocity				No. Sc.	No. Sc.	No. Sc.	No. Sc.	No. Sc.	No. Sc.	No. Sc.	No. Sc.	No. Sc.	No. Sc.	No. Sc.	No. Sc.	No. Sc.			
25	6	3	6	6	α(1)*	4	α(1)	2	0										
31	0	2	0	0	α(1-2)				4	0									
37	αS8	5	0	0	α(4)		2	α(5)	3	2	4	0							
16	αS9													2	0				
17	8						2	α(2)	3	α(5)				4	0				
13	αS6								3	7		2	2						
7	8	3	7	7	α(1)		2	3											
3	0	2	0	4					3	4		4	3						
23	5								2	8		3	3						
46	8	5	0	α(3)					3	4				2	3				
35	αS8						3	α(4-5)	2	5									
38	12						2	α(4)	3	6									
40	10						2	α(4)	3	α(3)	7	α(4-5)		5	10	9	6		
28	αS13						2	α(5-6)	3	7									
36	αS5						4	α(4-5)	2	7				3	7				
30	6								2	9									
24	7	2	15	α(3-4)					3	9									
44	7	2	4	α(2)					4	5									
22	4	2	3	α(1)					3	α(5-6)									
32	αS3	9	0	α(1)			11	5											
29	αS2						6	α(4-5)	3	α(5)				2	α(5)				
33	0	2	0	α(1)			3	α(3)	4	α(4)		7	2						
4	αS5						8	α(6)	2	α(4-5)				4	α(3)				
14	αS1						3	α(2-3)	2	α(3-4)				6	α(2)	7	α(2)	8	α(3-4)
26	18								2	α(1-2)									
15	αS2								3	α(2-3)									
9	14													4	α(2)				

*α, abort; number in parentheses, rpm at abort.

Discussion

The findings in this part of the study did not confirm the experimenters' hypothesis that direction-specific adaptation effects acquired in the slow rotation room would "tend to be preserved" as long as the head remained fixed. Rather, it was discovered that these effects disappeared spontaneously and fairly quickly. The differences between the results of a provocative challenge at zero velocity and with a reverse IAS were in all likelihood quantitative and not qualitative. Our data suggest that central vestibular repatterning was involved and that this new patterning had dynamic characteristics favoring rapid disappearance under conditions of minimal vestibular stimulation (head fixation).

PART III. EVIDENCE OF SIMULTANEOUS ACQUISITION OF SHORT-TERM (DIRECTION-SPECIFIC) AND LONG-TERM (NONDIRECTION-SPECIFIC) ADAPTATION EFFECTS

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

Figure 4b shows the measurements obtained on subject 25 during his first four tests. In the second test he was completely symptom free except for a score of 1 point during the reverse (IAS) after a 2-hour delay. In test 1 the score of 6 points on return to zero velocity (a relatively weak challenge) suggests that testing would have been aborted during exposure to a reverse IAS if it had been done at that time, which was more than confirmed in tests 3 and 4. The only reasonable explanation for the virtual absence of symptoms during the reverse IAS in test 2 and during the challenge after return to zero velocity is that the 2-hour delay was sufficient for the direction-specific effects acquired during the initial IAS to disappear, revealing the nondirection-specific adaptation acquired during that initial IAS and, probably, during the IAS in test 1.

Subject 7 (Figure 4c) demonstrated susceptibility to motion sickness during the initial IAS in tests 1 and 2 but demonstrated only very mild symptoms during his first exposure, after a delay of 2 hours, to a reverse IAS (test 2). The score of 1 point on final return to zero velocity suggests that less central vestibular repatterning occurred than in test 1 (8 points). Based on our experience, only the delay of 2 hours prevented an abort in subject 7 and only prior acquisition of nondirection-specific adaptation could account for the less severe symptoms during the first reverse IAS (and return to zero of test 2) compared with the initial IAS of test 1 and of test 2.

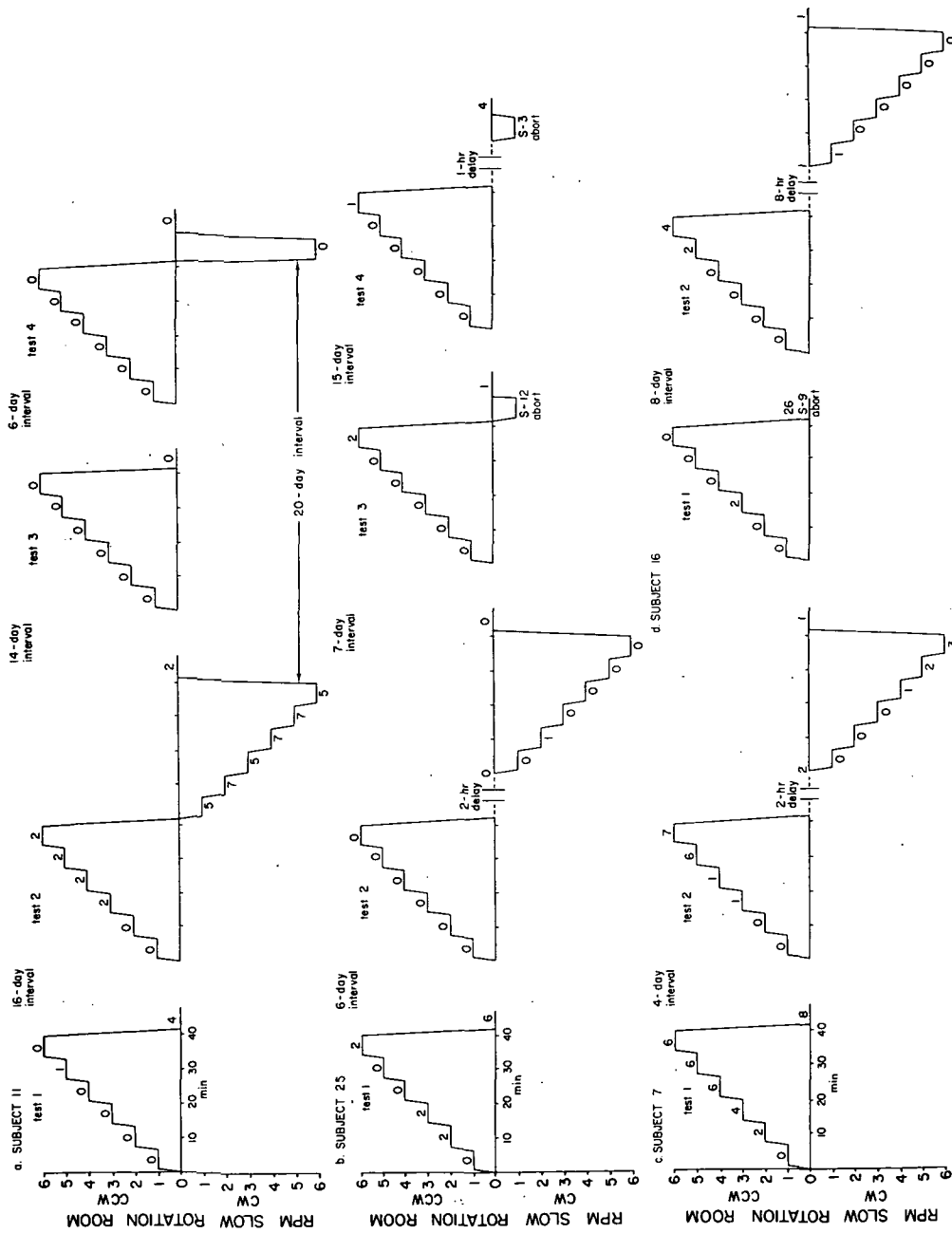


Figure 4

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

Figure 4d shows that for subject 16 in test 1, testing was aborted during the challenge after return to zero velocity, implying that testing would have been aborted during a reverse IAS if this had been measured at that time. Symptoms during the initial IAS in test 2 were more prominent than in test 1, implying that central vestibular repatterning was taking place and that, in the light of test 1 results, testing would have been aborted during a no-delay reverse IAS had this been done. The 8-hour delay allowed nearly all of the direction-specific effects to disappear, thereby revealing prior acquisition of nondirection-specific adaptation that accounted both for the virtual absence of symptoms during the reverse IAS and, more importantly, for the 1-point score on final return to zero velocity.

The findings just described imply that with the disappearance of direction-specific adaptation, with a short time constant of decay, there is revealed nondirection-specific adaptation with a long time constant of decay. The question arises, are the two types to be regarded as *sui generis*, or is one, in any respect, a consequence of the other? There is a possibility at least that the short time constant is not unique but in the nature of an artifact. This is based on two assumptions: First, the complex acceleration patterns generated in the rotating room comprise patterns that are specific for the direction of rotation and patterns that are similar whether the room rotates clockwise or counterclockwise. In consequence, acquisition of nondirection-specific effects would be continually overtaking but always lagging behind the acquisition of direction-specific adaptation effects. The second assumption is based on the dynamic ephemeral character of the early stage of acquisition of adaptation to rotation, including spontaneous reversibility.

A second possibility would amount to the continual conversion (or transfer) of direction-specific effects (acquired by one part of the vestibular system) to generalized adaptation effects through efferent (feedback) activity.

Still a third possibility is that we are dealing with two distinct types of adaptation, each with its own mechanism and time constant of decay.

CONCLUSIONS

Three conclusions may be drawn from the present study.

First, a procedure has been described that extends the scope of motion sickness studies which can be conducted using rotating devices. The procedure involves: 1) the selection of typically normal healthy subjects, 2) manipulation of the stimulus through control over the device (rpm and direction of rotation) and the subject (head movements or head fixation), and 3) a reliable means of measuring responses. The possibility of coupling and uncoupling the linkage between the vestibular system proper and nonvestibular systems immediately concerned with the genesis of motion sickness was demonstrated, but the procedure is open to greater exploitation than was attempted in

the present experiment. For example, by using predetermined endpoints instead of predetermined terminal velocities, it may be possible to generate similar levels of hypersusceptibility either on different occasions in the same subject or in different subjects, thus minimizing the effects of inter-test adaptation and permitting a rank ordering in terms of the rpm required to achieve the desired level of hypersusceptibility. An incremental-adaptation schedule is also a provocative incremental-test schedule; by varying the number of head movements at step increases one may increase "adaptation" or increase "provocation."

Second, both short-term (direction-specific) and long-term (nondirection-specific) adaptation effects can be elicited during a standardized incremental adaptation schedule. The decay time of the short-term effects was exponential and measured in hours. Some possible relationships between the two types of adaptations were mentioned. These relationships can be explored in systematic fashion by simple variations in experimental design.

Third, the present findings have practical significance. In space missions involving the generation of artificial gravity by rotation of a portion of the spacecraft, the need to make sudden transitions between rotating and nonrotating portions of the spacecraft have been regarded as posing the chief vestibular problem, and knowledge of direction-specific adaptation effects will aid in the prevention of motion sickness during such transitions. Heretofore unexplained occurrences of motion sickness on sudden cessation of rotation are now predictable, hence preventable. Present findings may serve as a point of departure for the systematic exploration of vestibular disturbances other than those acquired in rotating devices.

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13. ABSTRACT <p>Thirty-eight subjects were required to execute 120 head movements in a slow rotation room at each 1-rpm increase in velocity of the room between 0 and 6 rpm and, after a single-step gradual return to zero velocity, execute 120 head movements either immediately after the return or after delay periods varying from 1 to 24 hours unless, at any time, more than mild symptoms of motion sickness were elicited. A second stress profile differed by the sequential addition of an incremental adaptation schedule (identical to the first) in which the direction of rotation was reversed. The experimental findings demonstrated the acquisition of direction-specific adaptation effects that underwent spontaneous decay with a short time constant (hours). With their disappearance a nondirection-specific adaptation was revealed with a long time constant (days). Speculations are presented which could account for the simultaneous acquisition of short-term and long-term adaptation effects. The findings support the theory that motion sickness, although a consequence of vestibular stimulation, has its immediate origin in nonvestibular systems, implying a "facultative" or temporary linkage between the vestibular and nonvestibular systems.</p>			

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