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SUMMARY PAGE

THE PROBLEM

A comparison between blind and normally sighted persons was made to investigate the role of vision in the genesis of motion sickness.

FINDINGS

A group of twelve persons selected only on the basis of their visual defects were exposed to stressful Coriolis accelerations under standardized conditions. All demonstrated differences in susceptibility to acute motion sickness that bore no relation to their rank order of visual deprivation. Insofar as comparison with a group of normal subjects was made possible, no significant differences in susceptibility were demonstrable.

It was concluded that vision is not an essential but rather a secondary etiologic factor in the genesis of motion sickness. This is not incompatible with the fact that symptoms characteristic of motion sickness may be visually induced in the absence of "motion."

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INTRODUCTION

This report deals with the role of vision in the genesis of motion sickness based on a comparison between blind and normally sighted persons who were exposed to stressful Coriolis accelerations under standardized conditions. Statements to the effect that blind persons are susceptible to motion sickness appear in the scientific literature (1,2,13), but we have not found any reference to systematic observations conducted on blind but otherwise normal persons. A reason for this neglect is found in the ease with which the visual environment can be controlled in sighted subjects; yet, important differences exist especially between persons born blind and sighted persons with eyes covered. These include: 1) the anatomical and functional organization in the central nervous system, 2) past conditioning based on visual experience, and 3) any immediate influences based on visual memory.

PROCEDURE

SUBJECTS

Twelve blind and sixteen normal persons participated in this experiment. All received a thorough medical examination, the details of which will be published elsewhere, and were in a satisfactory state of health, although the physical fitness of the blind was below that of the normal group.

The normal subjects, six women and ten men, were between 16 and 20 years of age. All had normal visual acuity. Two had a history of middle-ear infection without sequellae. All had normal hearing except for four men who had a slight high-frequency loss. The threshold caloric test (8) values and ocular counterrolling indices (10) were within the normal range, indicating normal function of the canalicular and otolithic systems, respectively.

The blind subjects, five women and seven men, were between 16 and 44 years of age. The important optic findings of the twelve, along with their past history of exposure to motion and subsequent symptomatology, are shown in Table I. All except one (AL) were born with their visual affliction. Three subgroups were identified: no light perception in either eye (two subjects); no light perception in one eye and minimal light perception with (one subject) and without (two subjects) projection in the other; and seven who were partially sighted and could perceive horizontal and vertical elements in a well-structured visual environment.

Functional evaluation of the vestibular organs in these blind subjects posed difficulties in some instances due to the presence of spontaneous eye movements and inability to fixate satisfactorily. Nystagmography was impossible in one (SL) because the oculi bulbae did not generate an electrical field; visual observation of nystagmus, however, indicated he had a normal threshold of response to angular acceleration and a good response to thermal stimulation. In the remaining eleven, nystagmography revealed that the threshold nystagmus response to thermal stimulation of the canals was

normal in three and slightly above normal in eight. The ocular counterrolling index was well within the normal range in ten and within the normal range in two. If a small allowance is made for the difficulties encountered in carrying out the vestibular tests, all of the results may be regarded as within the normal range.

The twelve blind and fourteen of the sixteen normal subjects had no experience in the slow rotation room (SRR) prior to the present experiment. The remaining two subjects (BRO and GR) had had much experience in the room.

APPARATUS

The new Pensacola slow rotation room, described elsewhere in detail (3), is a windowless circular structure without any central supports, 20 feet in diameter and 10 feet high. Its excellent operational characteristics and instrumentation were not taxed in the present experiment.

THE PROVOCATIVE TEST

A slight modification of the Dial Test (6) was used. The Coriolis accelerations were generated by simultaneous rotations of room and subject. In this experiment velocity of the room was either 7.5 or 20 rpm. The head movements (H-M) were carried out according to taped instructions in sequences of five, requiring 25 seconds, followed by a rest period of 6 seconds. These movements involved flexion of the head (and, to some extent, the upper trunk) and return to the upright: 1) forward, 2) leftward, 3) backward, 4) rightward, and 5) forward. Past experience has indicated that, with few exceptions, even the highly insusceptible will experience symptoms at the higher angular velocity. Although a cut-off at 300 H-M's has often been employed, only 100 H-M's were used in this experiment.

The severity of the symptoms was graded according to the diagnostic criteria summarized in Table II (4). It should be mentioned that these criteria include the reliable responses; some are unreliable, others are covert. Stated differently, the evidence used in making a diagnosis may be less than the total symptomatology. Due to the cumulative effects of the stressful accelerations, subjects tend to underestimate rather than overestimate the severity of symptoms until these become prominent; hence, the endpoint may be overshot. Past experience (11) has indicated that when subjects are acutely stressed, M IIA is as reliable a criterion as M III, and even M IIB is highly reliable. M I, however, is unreliable as an endpoint even though it may be an accurate assessment of an overt symptom. The designated endpoint in this study was either "severe malaise" (M III) or 20 sequences (100 H-M's).

METHOD

Prior to each test an open-ended questionnaire termed "subject's preexperimentation interview" was completed to ensure that the subject was fit at the time of testing. Then he was secured in the chair, the room was brought up to the desired velocity, and,

after 60 seconds at constant velocity, the taped instructions were played and the subject began the head movements.

Each subject was tested individually, and at least one day separated consecutive experimental trials. The blind subjects were tested with eyes open, first at 7.5 rpm, then at 20 rpm. Subject GI, who experienced frank motion sickness (S) at 7.5 rpm after 70 H-M's, was not exposed to 20 rpm. The sighted subjects were tested first with eyes covered by an opaque patch, then with eyes open, both at 7.5 and 20 rpm; a few did not participate in all four experimental trials.

RESULTS

The symptomatology was quite similar in subjects with and those without visual defects. The principal findings are summarized in Table III. Among the twelve blind subjects eight reached or exceeded the designated endpoint M III; one each experienced M IIA and M I, and two were symptom free. Of the two who were "totally blind" one reached the endpoint at 7.5, indicating high susceptibility, and the other at 20 rpm. Of the three who were "virtually blind" none reached the designated endpoint; one each reached M IIA and M I, and the third was symptom free. Of the seven who were partially sighted six reached the designated endpoint and one was symptom free.

When the sixteen normal subjects were tested with eyes covered, six reached the designated endpoint, three reached M IIA, one reached M IIB, three reached M I, and three were symptom free. Only thirteen of the sixteen were properly tested with eyes open; eleven reached the designated endpoint, and one each, M IIB and M I. Of the remaining three, one did not participate, subject TI reached M IIB at 7.5 but was not exposed at 20 rpm, GR was symptom free at 7.5 and was exposed at 20 rpm on an earlier (reaching the designated endpoint) but not on the present occasion.

In Table IV the findings on the two groups are compared, using not only the designated but also a reliable endpoint described above under Procedure. With the designated endpoint, it is seen that, as a group, the normal subjects with eyes covered were less susceptible than the blind, but not with eyes open when there was a tendency toward lower susceptibility in the blind subjects. The same conclusions were demonstrated using the reliable endpoint.

A comparison between the virtually blind and the normal persons with eyes covered and between the partially sighted and the normals with eyes open revealed insignificant differences in susceptibility.

DISCUSSION

The fact that the susceptibility of the two subjects with total absence of light perception since birth was similar to that of the six most susceptible normal subjects with eyes covered is the best evidence that vision is not essential in the genesis of motion sickness. A person completely blind since birth would, in all likelihood, suffer

morphological changes and functional deterioration along the visual pathways similar to those observed in kittens deprived of sight at birth (14). Such abnormalities would reduce the optic connections in the central nervous system, rendering it less complex and, conceivably, less likely to be "disturbed" by unusual vestibular inputs. In any event, functionally there would be no visual contribution to the intersensory organization in the CNS, and visual-nonvisual "conflicts" or interactions would be impossible. If this were generally applicable, the corollary could be drawn that sighted persons with eyes covered have the same innate susceptibility to motion sickness as those completely blind. A sighted person on closing his eyes, however, still has visual memories, and he may respond to visually conditioned stimuli.

The great variation in susceptibility to motion sickness among unselected persons with normal function of the vestibular organs prevents firm conclusions to be drawn from comparisons between small groups. Individual differences must account for the much lower susceptibility in the three subjects who were virtually blind compared with the two who did not have any light perception and the seven who were partially sighted.

There is no doubt but that the normal group was more susceptible with eyes open compared with eyes covered. There were only two exceptions to this generality, and in one instance the difference was not substantial. This finding is in accord with previous experience in an SRR (7). The small differences noted when the five totally or almost totally blind were compared in susceptibility to the sighted with eyes covered and when the seven partially sighted were compared to the sighted with eyes open are partly fortuitous but nevertheless point up the relatively great role of the vestibular organs compared with vision in the genesis of motion sickness.

This study did not touch on the many other aspects of vision that directly or indirectly might influence susceptibility to motion sickness in particular people or under particular circumstances. Our findings are in no way incompatible, however, with the fact that symptoms of motion sickness may be visually induced in the absence of "motion," (12) or that vision, by locking onto an Earth reference for example, may reduce susceptibility to motion sickness (5,9).

REFERENCES

1. Chinn, H. I., and Smith, P. K., Motion sickness. Pharmacol. Rev., 7:33-82, 1955. (Especially p. 48.)

2. Desnoes, P. H., Seasickness. J. Amer. Med. Asso., 86:319-324, 1926. (Especially p. 320.)

3. Graybiel, A., and Wood, C. D., Rapid vestibular adaptation in a rotating environment by means of controlled head movements. Aerospace Med., 40:638-643, 1969.

4. 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., 39:453-455, 1968.

5. Johnson, W. H., and Taylor, N.B.G., Some experiments on the relative effectiveness of various types of accelerations on motion sickness. J. aviat. Med., 32:205-208, 1961.

6. Kennedy, R. S., and Graybiel, A., The Dial Test: A standardized procedure for the experimental production of canal sickness symptomatology in a rotating environment. NSAM-930. NASA Order R-93. Pensacola, Fla.: Naval School of Aviation Medicine, 1965.

7. Kennedy, R. S., Tolhurst, G. C., and Graybiel, A., The effects of visual deprivation on adaptation to a rotating environment. NSAM-918. NASA Order R-93. Pensacola, Fla.: Naval Aerospace Medical Institute, 1965.

8. McLeod, M. E., and Meek, J. C., A threshold caloric test: Results in normal subjects. NSAM-834. NASA Order R-47. Pensacola, Fla.: Naval School of Aviation Medicine, 1962.

9. Manning, G. W., and Stewart, W. G., Effect of body position on incidence of motion sickness. J. appl. Physiol., 1:619-628, 1949.

10. Miller, E. F. II, Evaluation of otolith organ function by means of ocular counterrolling measurements. In: Stahle, J. (Ed.), Vestibular Function on Earth and in Space. Oxford, England: Pergamon Press, 1970. Pp. 97-107.

11. Miller, E. F. II, and Graybiel, A., Comparison of five levels of motion sickness severity as the basis for grading susceptibility. NAMI-1098. NASA Orders T-81633 and R-93. Pensacola, Fla.: Naval Aerospace Medical Institute, 1970.

12. Miller, J. W., and Goodson, J. E., A note concerning "motion sickness" in the 2-FH-2 Hoover trainer. NSAM-519. Pensacola, Fla.: Naval School of Aviation Medicine, 1958.

13. Tyler, D. B., and Bard, P., Motion sickness. Physiol. Rev., 29:311-369, 1949. (Especially p. 325.)

14. Wiesel, T. N., and Hubel, D. H., Extent of recovery from the effects of visual deprivation in kittens. J. Neurophysiol., 28:1060-1072, 1965.

Table I

Summary of Optic Findings and History of Motion Exposure in the Twelve Blind Subjects*

Subject	Sex	Age (Yr)	Eye Right	Eye Left	Etiology	Age Onset	History of Motion Sickness Symptomatology: Conveyance	History of Motion Sickness Symptomatology: Symptoms
FO	M	43	No L-P	No L-P	?Cong. glaucoma	Birth	C, C-D, S	C-D, S
SI	M	17	No L-P	No L-P	Cong. glaucoma	Birth: L-P + Proj. Age 5: No L-P	C	None
JO	F	17	L-P	No L-P	Phthisis bulbae	Birth: Prog. loss Min. L-P, OD	C, C-D	None
SL	M	16	No L-P	Min. L-P	Retrolental fibroplasia	Birth	C, C-D	None
REV	F	18	No L-P	L-P poor + Proj.	Phthisis bulbae	Birth	C, C-D	None
AL	M	44	10/200	10/200	Retinitis	13	C, C-D, S	S
GI	F	16	L-P + Proj.	Ct. Fn.	Cong. cataracts	Birth	C, C-D, S	S
GR	M	20	L-P + Proj.	20/100	Cong. cataracts	Birth	C, C-D	C
HU	M	18	20/200	20/200	Optic atrophy	Birth: Prog. loss	C, C-D	None
MD	M	18	20/200	No L-P	Cong. cataracts	Birth	A, C, C-D	None
SM	F	16	No L-P	5/200	Retrolental fibroplasia	Birth	C, C-D, S	S
REY	F	27	20/200	20/200	Albinism	Birth	C, C-D	C, C-D

* Abbreviations used in table:

Cong: Congenital
Ct. Fn.: Count fingers
L-P: Light perception
OD: Oculodextro
Proj: Light projection
Prog: Progressive

A: Aircraft
C: Car
C-D: Carnival devices
S: Sea

Table II

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	Vomiting or retching	Nausea+ II, III	Nausea I	Epigastric discomfort	Epigastric awareness
Skin		Pallor III	Pallor II	Pallor I	Flushing/Subjective warmth ≥ II
Cold sweating		III	II	I	
Increased salivation		III	II	I	
Drowsiness		III	II	I	
Pain					Headache ≥ II
Central nervous system					Dizziness
					Eyes closed ≥ II
					Eyes open III

Levels of Severity Identified by Total Points Scored

Frank Sickness	Severe Malaise	Moderate Malaise A	Moderate Malaise B	Slight Malaise
(S)	(M III)	(M IIA)	(M IIB)	(M I)
≥ 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.

Table III

Manifestations of Motion Sickness in Twelve Blind and Sixteen Normal Subjects Exposed to Coriolis Accelerations in a Room Rotating Either at 7.5 or 20 RPM

Blind Subjects	7.5 rpm H-M*	7.5 rpm Symptom Level	20 rpm H-M	20 rpm Symptom Level
No light perception:				
FO	100	M III	55	S
SI	100	M I	70	S
No light perception one eye; minimal or poor light perception other eye, one with projection:				
JO	100	0	100	M IIA
SL	100	0	100	M I
REV	100	0	100	0
Partially sighted:				
AL	100	M I	55	S
GI	70	S	(Not exposed)	
GR	100	M I	55	S
HU	100	0	18	M III
MD	100	0	100	0
SM	100	M IIA	100	M III
REY	100	0	100	S

Normal Subjects	Eyes Covered 7.5 rpm H-M	Eyes Covered 7.5 rpm Symptom Level	Eyes Covered 20 rpm H-M	Eyes Covered 20 rpm Symptom Level	Eyes Open 7.5 rpm H-M	Eyes Open 7.5 rpm Symptom Level	Eyes Open 20 rpm H-M	Eyes Open 20 rpm Symptom Level
BRA	100	0	83	S	100	0	50	S
BRO	100	0	45	S	100	0	25	S
DA	100	0	100	0	100	0	100	S
DU	100	M I	100	M IIA	100	M IIB	80	M III
GR	100	M I	100	0	100	0	85	S#
HI	100	0	100	M I	100	M IIB	100	M IIB
JU	100	0	100	M IIA				
LE	100	M I	100	M IIB	100	M I	100	M I
MY	100	0	100	0	100	0	100	M III
PE	100	0	100	0	100	0	95	S
PD	100	0	35	S	100	M IIB	20	S
SA	100	0	100	M IIA	100	0	43	M III
TH	100	0	100	M I	100	M IIB	35	S
TI	100	M I	50	M III	100	M IIB		
UP	100	M I	45	S	75	S		
WE	100	M III	60	S	100	M IIA	70	M III

*H-M = Number of head movements to symptom level (see text).
#Exposed on earlier occasion.

Table IV

Comparison of Susceptibility to Acute Motion Sickness Among Subjects

Number Failing to Reach Criterion Endpoint (M III)						Number Failing to Reach a Reliable Endpoint (M IIB)					
Blind Subjects			Normal Subjects			Blind Subjects			Normal Subjects		
	rpm			rpm			rpm			rpm	
	7.5	20.0		7.5	20.0		7.5	20.0		7.5	20.0
Virtually blind:			Eyes covered:			Virtually blind:			Eyes covered:		
N = 5	4	3	N = 17	15	10	N = 5	4	2	N = 16	15	6
Partially sighted:			Eyes open:			Partially sighted:			Eyes open:		
N = 7	6	1*	N = 15	14	2	N = 7	5	1	N = 13+	8	1

* One subject not tested at 20 rpm because of frank sickness at 7.5 rpm.

+ One subject reached designated endpoint at 7.5 rpm; two subjects not available.

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13. ABSTRACT

A group of twelve persons selected only on the basis of their visual defects were exposed to stressful Coriolis accelerations under standardized conditions. All demonstrated differences in susceptibility to acute motion sickness that bore no relation to their rank order of visual deprivation. Insofar as comparison with a group of normal subjects was made possible, no significant differences in susceptibility were demonstrable.

It was concluded that vision is not an essential but rather a secondary etiologic factor in the genesis of motion sickness. This is not incompatible with the fact that symptoms characteristic of motion sickness may be visually induced in the absence of "motion."

14. KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
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Role of vision in motion sickness						
Diagnostic criteria of motion sickness						
Coriolis-acceleration-stress effects						