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TITLE

Importance of the Vestibular System in Visually Induced Nausea and Self-Vection

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Importance of the vestibular system in visually induced nausea and self-vection

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The objective of this study was to determine the importance, if any, of the non-auditory labyrinth of the inner ear in visually induced nausea and self-vection in subjects exposed to a moving visual field with and without concomitant pitching head movements. Subjects teated were 15 normals, 18 unilateral labyrinthectomies and 6 bilateral labyrinthectomies. The findings show a higher incidence of pseudo-Coriolis induced nausea in normal subjects compared to unilateral and bilateral labyrinthectomized subjects. When the subjects were exposed to the moving visual field only (no head movement), pronounced self-vection occurred in all subjects, but with earlier onset in the bilateral labyrinthine defective subjects as compared to normal and unilateral defective subjects. The subjective intensities of self-vections reported by labyrinth-defectives were much more pronounced as compared to normal subjects, and it is apparent that visual input in these subjects achieves much more importance in maintaining compensatory eye movements, and the gain of neck reflexes is enhanced. The findings that visual stimulation is more effective in producing the disabling effects after labyrinthine destruction could possibly be explained by enhancement of vision after loss of labyrinthine sensory input, and the gain in neck reflexes is also enhanced after labyrinthectomy.

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1. Introduction

It has been well established that nodding head movements concomitant with body rotation around the vertical axis results in intense stimulation of the vestibular receptors of the inner ear (Coriolis effects), thereby readily inducing motion sickness through appropriate CNS connections [3,5].

Athough it is known that motion sickness can also be induced when the stationary subject nods his head while viewing a moving visual field which produces a sensation of self-motion (vections) in the opposite direction, the pseudo-Coriolis effect [1,2], the relative importance of the vestibular receptors in such circumstances is not fully understood. It was the object of this study to clarify this question by exposing both normal and labyrinthine defective subjects to pseudo-Coriolis stimulation and compare the sensory effects thereby produced.

2. Methods

The subjects sat in a specially constructed arm chair located centrally in a darkened cylindrical booth 8 feet in diameter. Moving spots of white light were projected onto the entire wall facing the subject as reflected from a rotating assembly of mirrors suspended from the ceiling (Fig. 1). A variable speed electric motor controlled the rate of horizontal movement of the light spots, which was maintained at 10 r.p.m.

When the normal stationary subject viewed the rotating visual field, a definite sensation of self-rotation (vection) in the opposite direction was always experienced. Times for onset of self-vection and degrees

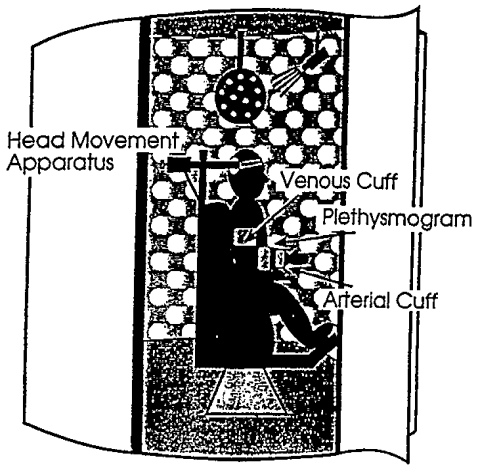

Fig. 1. Cylindrical room used to produce pseudo-Coriolis effects in normal and labrynthine defective subjects by exposure to rotating visual field and controlled pitching head movements.

of subjective intensity, whether "slight", "moderate", or "strong" were determined verbally by intercom. Psuedo-Coriolis effects resulted from controlled patterns of pitching head movements (Fig. 2) concomitant with the sensations of self-vection. These head movements at a velocity of 5°/s for 6 s were controlled by means of an electrically driven head rest with pads held in position by means of Velcro straps around the forehead. A small D.C. electric motor actuated nodding head movements through an arc of 30° and induced an increased sensation of spatial disorientation often accompanied by nausea. The test procedure was divided into 6 phases (numbered 1 to 6) each lasting for 5 minutes. The activity involved in each phase was as follows:

Phase 1: Control period. Light spots stationary. Head stationary.
Phase 2: Light spots moving. Head stationary.
Phase 3: Light spots moving. Intermittent pitching head movement.
Phase 4: Light spots moving. Continuous pitching head movement; used if phase 3 ineffective for nausea.
Phase 5: Light spots moving. Head stationary.
Phase 6: Light spots stationary. Head stationary.

The intensity of motion sickness was assessed using the following index: MS0 = no symptoms; 1 = slight dizziness and disorientation; 2 = slight gastrointestinal discomfort, sweatiness; 3 = nausea; 4 = severe nausea; 5 = request termination because of nausea; 6 = emesis imminent. Blood pressure and heart rate were automatically recorded by a programmed electrosphygmomanometer (Narco PE300) attached to the right arm [4].

Three types of subjects were used:

- Normals with no history of vesfibular disease (Group A).
- Subjects with unilateral vestibular function only (Group B, unilateral labyrinthectomies).
- Subjects with bilateral nonfunctional labyrinths (Group C, bilateral labyrinthectomies).

In Groups A and B, the subjects were requested to respond verbally through an intercom system to questions at regular intervals regarding symptoms (self-vection, headache, perspiration, nausea, etc.). Group C subjects were all completely deaf and had been instructed in writing to announce verbally if any symptoms were experienced throughout all phases of the experiment.

A total of 39 subjects (male and female) were tested, ranging in age from 21 to 70 years (mean: 38 years). For controls, 15 of these were normal healthy subjects with no history of vestibular disorder. The remainder have undergone either unilateral (15 subjects) or bilateral (6 subjects) labyrinthectomies, all for the removal of acoustic neuromas with no other neurological defects. The surgery resulted in deafness and lack of response to cold water irrigation on the side or sides involved. All subjects were fully informed in advance of any possible discomfort and signed appropriate consent forms.

After instrumentation was completed, the subject was allowed to stabilize. They were instructed to keep the eyes open throughout the test. Two patterns of moderate intensity of pitching head movement were used as follows: the first was of a square wave pattern at 6-s intervals, followed by a second series consisting of continuous head movements of stronger intensity (Fig. 2). The experiment was terminated when the entire cycle was completed or when the subject complained of severe nausea; the head movement was stopped and the subject was requested to close his or her eyes.

3. Results and discussion

There were no consistent or significant changes in blood pressure or heart rate during pseudo-Coriolis stimulation of the labyrinthine defective subjects, as was also previously reported for normal healthy subjects [4]. In the 15 normal subjects, no symptoms of motion sickness occurred with moving visual fields alone; however, when head movement was superim-

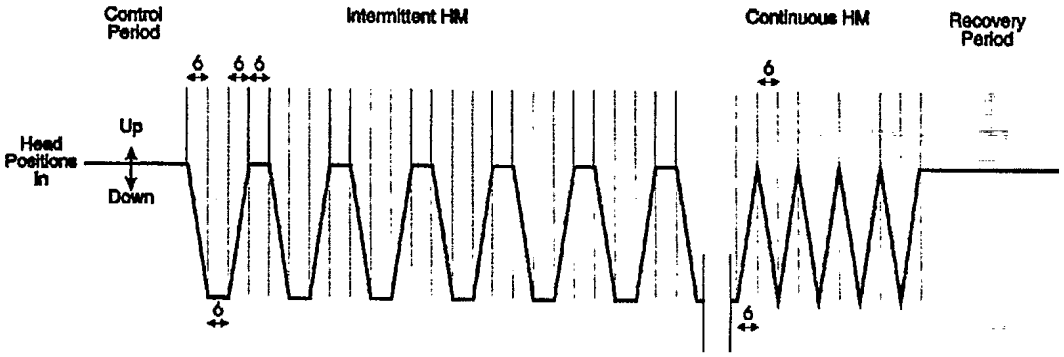

Fig. 2. Pattern of induced pitching head movements used to produce pseudo-Coriolis effects. Numbers indicate times in seconds.

Table 1
Comparing motion sickness/index intensity and time of self-vection onset during pseudo-coriolis stimulation (pitching head movements and moving visual field)

	Nomal ($n = 15$)		Unilateral ($n = 18$)		Bilateral ($n = 6$)	
Motion sickness index	No. of subjects (% of total) $n = 15$	Onset self-vection (s)	No. of subjects (% of total) $n = 18$	Onset self-vection (s)	No. of subjects (% of total) $n = 6$	Onset self-vection (s)
0	4 (27%)	36	8 (44%)	36	2	17
1	4 (27%)	40	3 (17%)	38	(33%)	34
2	2 (13%)	48	4 (22%)	25	1(17%)	20
3	4 (27%)	24	1 (6%)	36	2(33%)	20
4+	1 (6%)	28	2 (11%)	62	1(17%)	

n = number of subjects.
s = time in seconds.

posed (Fig. 3B), 73% developed symptoms ranging from 1 to 4+ on the MS scale (Table 1). Of the 18 unilateral defective subjects similarly exposed to pseudo-Coriolis stimulation, 56% showed symptoms of motion sickness ranging from 1 to 4+ on the MS scale (Fig. 2). Of the six bilateral defective subjects, (67%) developed some degree of motion sickness ranging from 1 to 3 on the MS scale (Fig. 3C). It is interesting to note that in unilateral and bilateral categories, 83% of the series had MS of 2 or less, whereas 67% of the normal subjects had MS of 2 or less. These findings indicate that motion sickness can indeed by induced in both unilateral and bilateral defective subjects in certain circumstances (moving visual field concomitant with head movement in a different spatial plane and with eyes open).

When the subjects were exposed to the moving visual field only (no head movement), pronounced self-vection occurred in all subjects, but sometimes with earlier onset in the bilateral defective subjects as compared to normal and unilateral defective subjects (Table 2). The descriptions of the subjective intensity of the selfvection reported in labyrinthine defective subjects were much more pronounced (whether "slight", "moderate" or "strong") than when described by normal subjects. The times for onsets of self-vection, however, were independent of the degree of nausea.

Table 2
Onset of self-vection times (subjects stationary and exposed to moving visual field)

Subjects	Onset of self-vection (in $s \pm$ SE)
Normal $n = 15$	37.2 ± 6.98
Unilaterals $n = 18$	47.6 ± 7.67
Bilaterals $n = 6$	20.2 ± 4.24

n = number of subjects in the series.
s = time in seconds.

In interpreting the significance of these results it is indicated that visual input achieves much more importance in maintaining compensatory eye movements and the gain of neck reflexes is enhanced after labyrinthectomy.

4. Conclusions

It was established by this study that nausea and self-vection induced by pseudo-Coriolis stimulation (subject stationary with pitching head movements concomitant with a moving visual field) could develop not normally in healthy subjects but also in both unilateral and

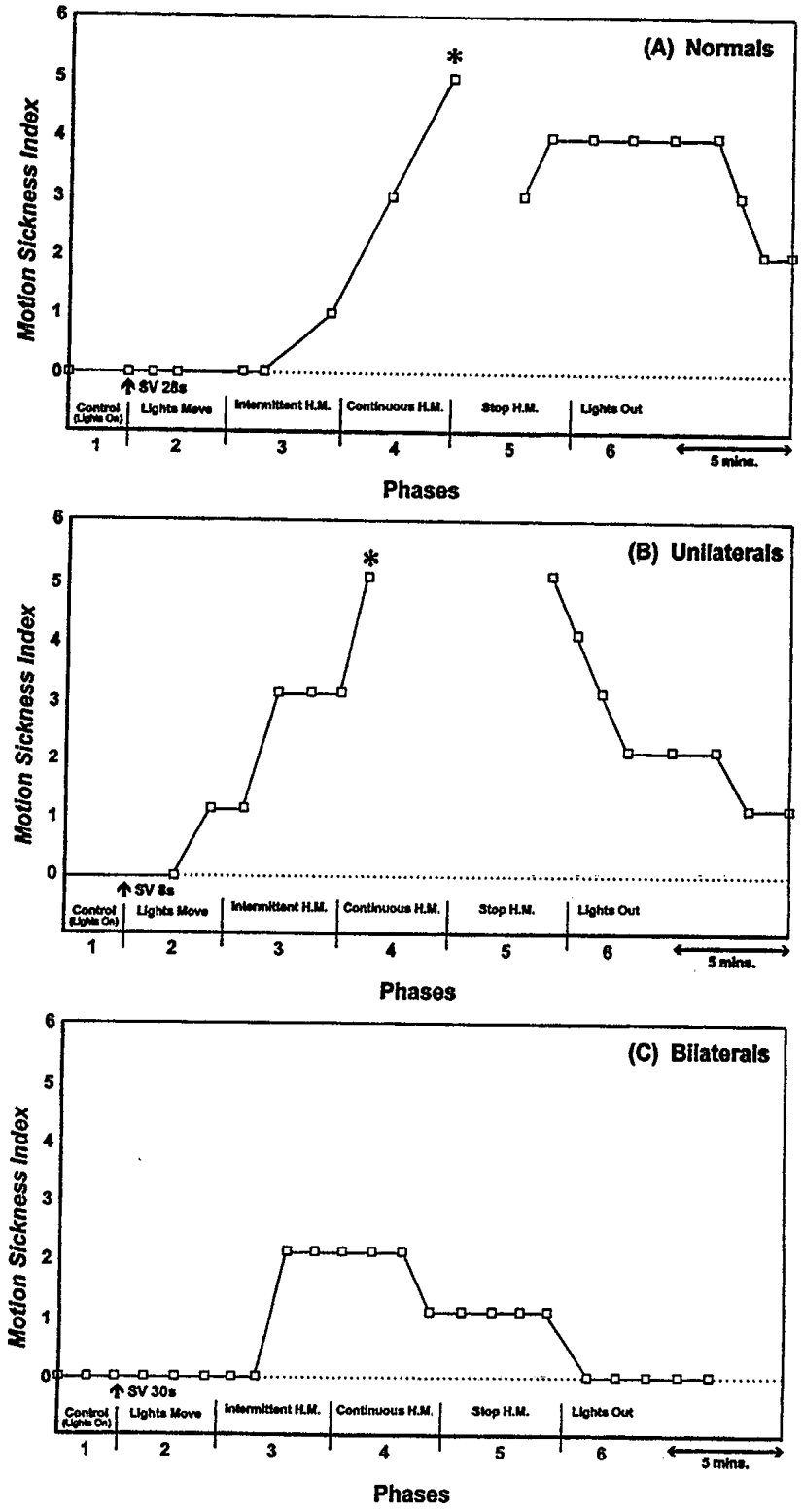

Fig. 3. Effects of pseudo-Coriolis stimulation on Motion Sickness Index Scores showing mean values of normals (A) and labyrinthine-defective subjects (B and C). Phase 1: control period. Phase 2: light spots moving, head stationary. Phase 3: light spots moving, intermittent head movement, Phase 4: light spots moving, continuous head movement. Phase 5: light spots moving, head stationary. Phase 6: light still, head stationary. Asterisk denotes when the subject requested termination of head movement. Arrow denotes onset of self-vection and the time of its occurrence.

bilateral ($P > 0.1$) labyrinthectomized subjects. The findings indicate no significant differences in the degree of nausea from pseudo-Coriolis stimulation among the unilateral and bilateral defective subjects compared to the normal subjects.

However, the observation that the intensity of subjective self-vection was more pronounced in the labyrinthine-defective subjects would suggest that the vestibular receptors in normal subjects may modulate induced self-vection. It has been well established by several authors that motion sickness, as the term is generally used (sea sickness, air sickness etc.), does not occur in labyrinthine defective subjects [6] and, when present, it is due to conflicting sensory inputs. It may be considered surprising, therefore, to find that nausea developed in our labyrinthine defectives (LD) when exposed to pseudo-Coriolis stimulation. However, it should be pointed out that even LD subjects were also exposed to conflicting sensory input in that our experimental conditions produced conflict of visual sensory input (subjective horizontal self-vection superimposed on vertical eye movements). In this regard, it was recently established by Cheung et al. [1] that no visually induced sickness occurred in any of their 6 bilaterally defective subjects who were seated in a chair, the head immobilized, and exposed to a moving visual field. Normal subjects with healthy vestibular responses, however, did suffer nausea of varying intensity when exposed to the same conditions. Our findings therefore are of particular importance in showing that nausea can indeed by induced in subjects who have diminished or no vestibular component when they are exposed to a moving visual field concomitant with head movement which causes conflict of visual input (horizontal self-vection interrupted by vertical eye movements).

With regard to nausea induced by pseudo-Coriolis exposure, our results indicate that although nausea can occasionally develop to some degree by visual stimulation alone, it is more intensely initiated when accompanied by vestibular stimulation (head movement), as seen when pitch head movements accompany yaw vection [8]. It should be stressed, however, that after labyrinthectomy, visual input achieves much more importance maintaining compensatory eye movements, and the gain of neck reflexes is enhanced. Therefore, the finding that visual stimulation is more effective after labyrinthectomy could be explained by an enhancement of visual effects after the loss of labyrinthine sensation.

It should be pointed out that the nausea that developed in the unilateral and bilateral labyrinthectomy subjects after pseudo-Coriolis stimulation may also result from enhancement of the cervico-ocular reflex that accompanies such defects.

Acknowledgments

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