Review of Motion Sickness with Special Reference to Simulator Sickness

R. S. KENNEDY and L. H. FRANK

ABSTRACT

Simulator sickness has implications for training and safety because as many incidents of simulator sickness have been reported since 1980 as in all the previous time. The signs and symptoms, stimulus and response characteristics, anatomical structures, and susceptibility factors of simulator sickness are reviewed. The prevalent theories of the genesis of this malady are put forth and an integrating theory, which suggests that simulator sickness is a form of motion sickness and may be best understood as a special case of sensory conflict, is proposed. Sensory conflict is a useful principle in the study of simulator sickness because the malady is clearly polygenic and polysymptomatic. Therefore it may be argued that greater conflict leads to more severe and greater incidence of sickness. Evaluations that will lead to recommendations for preventing the problem, guidelines for predicting the outcome, and suggestions for future research may be planned.

INTRODUCTION

Although the self-propelled locomotive behavior of man (walking, running, jumping) does not induce motion sickness, transportation in some environments does. It is probably reasonable to assume that the history of motion sickness began with man's domestication of animals for transport. Riding camels or elephants, for example, can induce motion sickness; but, interestingly, riding horses does not (5). With the invention of the boat came sea sickness and the question of whether a seaman could perform his duties. In short, motion sickness became operationally significant. Thus the search for the causal factor or factors of motion sickness probably received its initial impetus from a practical concern about how to eliminate its debilitating effects.

STIMULUS CHARACTERISTICS

Many types of motion produce motion sickness. Among the most common places for sickness to occur are in ships, small boats, trains, gliders, zero-gravity...
aircraft, rotating rooms, chairs, vertical oscillators, horizontal sways, and moving-base or fixed-base flight simulators. In addition, tilted rooms and buildings and chimneys have been implicated.

Humans appear to be most susceptible to motion sickness when exposed to very low frequency vibrations in the range of 0.12 to 0.25 Hz (20), although the data are limited largely to those from swings and vertical oscillators. There does not appear to be a frequency-specific relationship when cross-coupled angular accelerations are employed (21). The normal locomotive behavior of man has a mean frequency of about 1.7 Hz (22,23) and, as previously mentioned, does not induce sickness.

Most of the power used in spectral density analyses of body sway is below 0.40 Hz (24), and perhaps platform stimuli in this range could be amplified at the head. Although acceleration of the environment is generally required, visual perception of motion alone is sufficient to produce sickness (25,26). The effects are usually limited to the period of exposure, but "postadaptation" effects are known to occur (4,17,27).

In a comprehensive review of low-frequency motion and human performance, Baker (28,p.2) comments that "there is virtually no pertinent, documented information regarding the effects of either motion sickness or motion upon human performance." The common finding is that task performance simply ceases when vomiting, the cardinal sign of motion sickness, begins. More subtle evidence of performance decrement before sickness has not been consistently found. An exception is the increased tracking error obtained when low-frequency motion causes direct biodynamic interference with the task.

In a series of studies on very-low-frequency vibrations (VLFV) conducted by G.R. Wendt and his associates during the 1940s (29-33), psychomotor performance tests were investigated, including an obstacle course, a 60-yd dash, cart throwing, and the Marshburn Complex Coordinator (a tracking device used in pilot selection). The subjects, mostly, U.S. Navy cadets, performed the psychomotor tests before and after a 20-min exposure to a motion of a vertical accelerometer similar to an elevator. Pretest and posttest performance scores of subjects who became motion sick were compared with scores of subjects who did not. Results showed virtually no effects of motion sickness on the performance tests (29,32).

A field study of motion sickness and performance was reported by Kennedy et al. (34). They measured performance on a counting and short-term memory task in three types of large aircraft undergoing severe turbulence during hurricane penetrations. The main finding was that performance decrements were directly related to the amount of turbulence experienced whereas the incidence of motion sickness appeared to be only partly correlated with turbulence and partly with the periodic frequency of the motion. On the other hand, Wiker, Pepper, and McCauley (35) showed that several psychomotor performances were degraded at sea in connection with seasickness.

**PHYSIOLOGICAL RESPONSES**

Table 1 (11) gives a summary of the physiological responses that occur as a function of motion sickness.

Humans are adaptable, and the effects of almost any environmental stressor on performance and physiology will change over time (duration of exposure). The nature of the change is usually diminution of the observed effect. These generalizations obviously have limits, because extremely intense stressors can cause injury or death (precluding adaptation). Predictions of performance decrements, ataxia, or other potential effects due to motion are difficult to make because the extent and time-course of adaptation are not known and may only be inferred from the literature on visual distortion (36).

There are both large individual differences in adaptation and large time-course variances within an individual's adaptation to differing motion environments. However, adaptation is a double-edged sword. It implies a modification of sensory processes to enable the individual to function more successfully in an altered environment. When the individual returns to his "normal" environment, however, the modified sensory processes most probably will not be optimal. Adaptation must occur in the opposite direction (readaptation) for the individual to function optimally in his normal environment. This type of adaptation and readaptation process has been well documented in the research literature under a variety of environmental influences such as optical distortion (37-39), weightlessness (40,41), rotation (42,43), and rectilinear motion (44). It is quite likely that adaptation, in the form of less symptomatology during repeated simulated exposure, will occur to the perceptual rearrangement found in flight simulators (whether visual or inertial). However, to rely on reduction or elimination of symptoms through adaptation misses the point of the requirement for minimum human factors engineering design criteria and may also affect safety of subsequent flying and other activities. The very adaptation that reduces the effects during exposure to the simulated environment may cause problems when the person returns to the normal environment. Furthermore, these effects may interact in peculiar ways, should the individual be transported in a conveyance be it under his own control (e.g., an automobile) or not.

**TABLE 1 Physiological Manifestations of Motion Sickness**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td>Changes in pulse rate or blood pressure, or both</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Altered respiration rate</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Sighing or yawning</td>
</tr>
<tr>
<td>Body Fluids</td>
<td>Changes in lactic dehydrogenase concentrations</td>
</tr>
<tr>
<td>Blood</td>
<td>Changes in lactic dehydrogenase concentrations</td>
</tr>
</tbody>
</table>

*Adapted from Konigswald and Parker (11).*
FLUID SHIFT THEORY

The idea that fluid shifts within the body may contribute to motion sickness is both recent (47) and old (48). Wallaston (48) claimed that motion sickness was caused by sloshing of the blood, which led to alternation engergment and anemia of the brain.

That fluid shift may be a possible explanation of space motion sickness accounts for the majority of research and interest in fluid shift theory. During space flight there is a cephalic shift of 1.5 to 2.0 L from the lower extremities (11). Calf girth correspondingly decreases about 30 percent. Mean resting heart rate and systolic blood pressure tend to increase, while diastolic pressure decreases.

According to space fluid shift theory, the rostral shift in body fluid alters cranial pressure and vestibular response. For example, altered fluid pressure in the labyrinth could result in a change in gain and phase shift (49). Graybiel and Lackner (50,51) have examined the evidence for this theory on earth by the use of head-down tilt to induce fluid shift. Their work has shown that fluid shift toward the head has no effect on susceptibility or causes a small decrease in susceptibility as the magnitude of the shift increases (41).

FEAR AND ANXIETY THEORY

Does anxiety or fear increase a person's susceptibility to motion sickness? According to Benson (52, p.486), "a definite correlation between susceptibility and psychometric measures of anxiety or neuroticism has not been established." It is not known for certain whether this is due to a true lack of relationship or, perhaps, to the lack of reliability in measures of anxiety (53) as well as to the already mentioned lack of reliability in measures of the motion sickness criterion. In any case, clear-cut evidence for the notion is hard to find.

BALANCE OF AUTONOMIC ACTIVITY POSTULATE

Waxing and waning of symptoms suggests competing processes (54). The symptoms of motion sickness resemble what might be associated with increased cholinergic (55) and decreased adrenergic activity, but the relationships are not clear-cut (56). Although the drugs which are effective in motion sickness are chiefly those that stimulate the sympathetic nervous system, or those that shut down the parasympathetic nervous system, the several exceptions (56) imply that this postulate should be considered as part of a larger theory.

TOXIC REACTION THEORY

Treisman asked, is the adaptive function of vomiting during motion sickness, and how does such a response contribute to the survival of the species? His answer was that the only adaptive significance vomiting could have is the expulsion of ingested toxins from the body. Hence, when the body vomits in response to motion sickness, it is interpreting the stimulus as if it were a poison. Wiker (58) has also made this point.

Normally, the sensory systems of the body complement each other. The eyes and the vestibular system are in harmony. When a toxin is ingested, it acts on the inner ear causing the vestibular signal to come in conflict with vision and other senses. This conflict signals to the body that it has ingested a poison and emesis occurs.

PERCEPTUAL CONFLICT THEORY

Perceptual conflict theory is known by several names: mismatch, neural mismatch, cue conflict, incongruity, and sensory rearrangement. The authors believe that perceptual conflict is the most descriptive term and, consequently, recommend its use.

In brief, the perceptual conflict theory posits a referencing function in which motion information, signaled by the eyes, vestibular apparatus, or proprioception, may be in conflict with expected values of these inputs based on a neural store (which reflects past experience) or with the way in which the system circuitry is wired. Kennedy (36) suggested, as have others (59,60), that perceptual conflict

ANATOMICAL STRUCTURES RELATED TO MOTION SICKNESS

Table 2 is a list of anatomical structures and their probable role in motion sickness. The interested reader is referred to the longer version of this paper (2) or to Money's and Wood's (45) excellent review of the neural mechanisms underlying the symptomatology of motion sickness on which it was based. Individual differences and other factors are given in Table 3.

TABLE 2 Anatomical Structures Related to Motion Sickness

<table>
<thead>
<tr>
<th>Structure</th>
<th>Role</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vestibular apparatus</td>
<td>Probably required</td>
</tr>
<tr>
<td>Visual apparatus</td>
<td>Importantly, not necessary</td>
</tr>
<tr>
<td>Viscera</td>
<td>Definitely not necessary</td>
</tr>
<tr>
<td>Proprioceptive afferents</td>
<td>Not known</td>
</tr>
<tr>
<td>Visual afferents</td>
<td>Not known</td>
</tr>
<tr>
<td>Peripheral afferents</td>
<td>Important, may not be necessary</td>
</tr>
<tr>
<td>Vestibular nuclei</td>
<td>Probably necessary</td>
</tr>
<tr>
<td>Chemoreceptor trigger zone</td>
<td>Necessity presently challenged</td>
</tr>
<tr>
<td>Cerebrum</td>
<td>Not necessary</td>
</tr>
<tr>
<td>Limbic</td>
<td>Speculatively related</td>
</tr>
</tbody>
</table>

TABLE 3 Individual Differences and Other Factors in Motion Sickness

<table>
<thead>
<tr>
<th>Factors</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>Men appear less susceptible</td>
</tr>
<tr>
<td>Age</td>
<td>Younger than 18 months—virtually immune</td>
</tr>
<tr>
<td></td>
<td>2 years to puberty—high</td>
</tr>
<tr>
<td></td>
<td>Older than 50—rapidly disappearing</td>
</tr>
<tr>
<td>Fluid independence</td>
<td>Appears to be related in several studies</td>
</tr>
<tr>
<td>Adaptation</td>
<td>Repeated exposures invariably result in lessening of symptoms</td>
</tr>
<tr>
<td>Head movement</td>
<td>The more complex the motion, the more sickness</td>
</tr>
<tr>
<td>Motion regularity</td>
<td></td>
</tr>
</tbody>
</table>

There is considerable evidence that overstimulation does not satisfactorily account for all incidences of motion sickness. As has been mentioned, vision alone is sufficient to induce sickness as demonstrated in the case of some fixed-base simulators (46). Motions that are difficult to consider oversaturating, such as slow rotation rooms and ship movement, can induce severe sickness.

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theory is based on a lack of correlation between appearance and reality. Under ordinary circumstances, there is a correspondence between what is sensed and the physical representation of the stimulus. The sensory systems report reality and, after periods of time, create reality in order that one may interact with it, spatially and temporally. The authors believe that central nervous system integration could be represented by a linear model (61). This version of the sensory conflict theory is described in greater detail in Kennedy and Frank (62) and Kennedy et al. (63).

TOWARD A UNIFIED THEORY

The signs and symptoms, stimuli and response characteristics, anatomical structures, susceptibility factors, and prevalent theories of motion sickness have been reviewed, and the authors believe that all are relevant for simulator sickness as well. It is evident from this review that motion sickness is both polysymptomatic and polygenic. It should also be evident from the number of corollaries, principles, postulates, and theories presented, and the examples proposed to explain the outcomes, that we are light years away from a proper understanding of motion sickness. But we may be closer to predicting its outcome and perhaps preventing its occurrence. With these provisos in mind, the following comments are offered.

The preceding theories need to be integrated into one. This is described in more detail elsewhere (63). The theories mentioned emphasize either the stimulus or the response characteristics that lead to motion sickness. However, it appears clear from the literature that the key to understanding motion sickness must include understanding of how the stimulus acts at the receptor level. It is the view of the authors that motion sickness is a result of decorrelated sensory channels. This premise, which is in concert with the perceptual conflict theory, states that any stimulus that causes a decorrelation to occur initiates the firing of the chemoreceptor trigger zone and motion sickness.

As is the case with the perceptual conflict theory, correlations between sensory receptors build up over time. Decorrelation occurs when inputs are not in accord with what is expected from the neural store or with the way in which that system is wired to respond. This causes "troubleshooting" to begin. The toxic reaction, overstimulation, and fluid shift theories of motion sickness are all compatible with this notion. Indeed, troubleshooting may be a hypothetical construct for a toxic reaction. Each theory implies that a modification occurs in which stimuli are integrated. Overstimulation modifies the receptor through sensitization, fluid shift through pressure changes, and poison through varied means.

The autonomic and fear theories of motion sickness are also compatible with the unified theory. The autonomic and fear theories, however, really address response to motion sickness, not causal factors.

Thus, as Money and Cheung (64) contend, Treisman is correct. Presumably, when inimical things happen to the organism, the central nervous system interprets those events to mean that it has been poisoned. In general, this interpretation occurs when real poisons are administered, but in those special cases in which altered and rearranged perceptions occur, if the vestibular system is implicated, the system interprets this to mean that the organism has been poisoned. Under some conditions, the body possesses resonances that, in the case of 0.20 Hz or so, the system also interprets as poison. It is wondered whether 0.20 Hz, or another resonance, would have an adverse (i.e., it's poison!) effect with visual stimuli alone. If so, such a finding would have strong heuristic value for simulator sickness.

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REFERENCES

16. A.R. Fregly. Vestibular Ataxia and Its Measure-


49. J.W. Wolfe, E.J. Engelken, and J.E. Olson. Vestibular Responses to Biothermal Caloric and Harmonic Patients with Meniere's Disease. In


50. A. Graybiel and J.R. Lackner. Comparison of Susceptibility to Motion Sickness During Rotation at 30 rpm in the Earth-Horizontal, 10° Head-Up, and 10° Head-Down Positions. Aviation, Space, and Environmental Medicine, Vol. 40, 1977, pp. 7-11.

51. A. Graybiel and J.R. Lackner. Rotation About the Z Axis After 6 Hours in the 10° Head-Down Position: Effect on Susceptibility to Motion Sickness. Aviation, Space, and Environmental Medicine, Vol. 50, No. 4, 1979, pp. 390-392.


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