Review of Motion Sickness with Special Reference to Simulator Sickness

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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.

Simulator sickness is not a new phenomenon for those who have worked around training devices and other optical devices that have dynamic characteristics. Moreover, to those who have experimented with perceptual adaptation to optical transformations, it is not surprising that sickness occurs. For those who perform experiments in vestibular science, simulator sickness may be considered the visual analogue of more traditional forms of motion sickness, including space sickness (1).

Simulator sickness, although not necessarily of epidemic proportions, is on the upswing. Almost as many incidents have been reported since 1980 as in all the years before then. A sourcebook (2) reports many relationships among causal factors, and a workshop has been held to discuss the implications of simulator sickness (3). Simulator usage is also increasing, and there is a question about whether the increase in observed frequency is due to the increased availability of simulators, increased kinematics available in simulated scenarios, or even individual differences of today's user population.

There have been five major (and many minor) reviews of the motion sickness literature (4-8) and all of these contain excellent accounts of what is known. Each has a comprehensive reference list. The following review has been prepared to emphasize the authors' bias that, as is the case with other forms of motion sickness [e.g., space adaptation syndrome (1,9-11)], simulator sickness may be best understood in the context of motion sickness as a special case of sensory rearrangement.

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 seasickness 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.

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MOTION SICKNESS--A DEFINITION

Motion sickness is a general term for a constellation of symptoms and signs, generally adverse, caused by exposure to abrupt, periodic, or unnatural accelerations. Overt manifestations (signs) are pallor, sweating, salivation, and vomiting (12-15). Drowsiness, dizziness, and nausea are the chief symptoms. Less frequently reported, but often present, are postural changes, or ataxia, sometimes referred to as "leans" or "staggers" (16,17). Other signs (5,18,19) include changes in cardiovascular, respiratory, gastrointestinal, biochemical, and temperature regulation functions. Other symptoms include general discomfort, apathy, dejection, headache, stomach awareness, disorientation, lack of appetite, desire for fresh air, weakness, fatigue, confusion, and, occasionally, incapacitation. The consequences for human performance and operational efficiency are decreased spontaneity, carelessness, and incoordination, particularly in manual control. Motion sickness is theoretically preventable, but that is not always practical. When symptoms are severe, the passage of time may be the only possible treatment.

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 swings, and moving-base or fixed-based 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 of 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, dart throwing, and the Mashburn 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 accelerator 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 (28,32).

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).

TABLE 1 Physiological Manifestations of Motion Sickness[a] *(11)*

	Manifestation
Cardiovascular	Changes in pulse rate or blood pressure, or both
	Increased tone of arterial portion of capillaries in the fingernail bed
	Decreased diameter of retinal vessels
	Decreased peripheral circulation, especially in the skin of the head
	Increased muscle blood flow
Respiratory	Alterations in respiration rate
	Sighing or yawning
	Air swallowing
Gastrointestinal	Inhibition of gastric intestinal tone and secretions
	Salivation
	Gas or belching
	Epigastric discomfort or awareness
	Sudden relief from symptoms after vomiting
Body fluids	Changes in lactic dehydrogen ASE concentrations
Blood	Increased hemoglobin concentration
	Increased pH and decreased $paCO_2$ levels in arterial blood, presumably from hyperventilation
	Decreased concentration of eosinophils
	Increased 17-hydroxycorticosteroids
	Increased plasma proteins
	Increased ADH
	Decreased glucose utilization
Urine	Increased 17-hydroxycorticosteroids
	Increased catecholamines
Temperature	Decreased body temperature
	Coldness of extremities
Visual system	Ocular imbalance
	Dilated pupils during emesis
	Small pupils
	Nystagmus

[a]Adapted from Nicogossian and Parker *(11)*.

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 simulator 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.

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

Structure	Role
Vestibular apparatus	Probably required
Visual apparatus	Important, not necessary
Viscera	Definitely not necessary
Proprioceptive afferents	Not known
Visual afferents	Not known
Peripheral afferents	Important, may not be necessary
Vestibular nuclei	Probably necessary
Chemoreceptor trigger zone	Necessity presently challenged
Cerebrum	Not necessary
Limbic	Speculatively related

TABLE 3 Individual Differences and Other Factors in Motion Sickness

Factors	Findings
Sex	Men appear less susceptible
Age	Younger than 18 months—virtually immune 2 years to puberty—high Puberty to 21—decreasing 21 to 50—declining Older than 50—rapidly disappearing
Field independence	Appears to be related in several studies
Adaptation	Repeated exposures invariably result in lessening of symptoms
Head movement	In most environments increases the symptoms
Motion regularity	The more complex the motion, the more sickness

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 overstimulating, such as slow rotation rooms and ship movement, can induce severe sickness.

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 alternate engorgement 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 (57) addressed the evolutionary significance of the emetic response to motion sickness. What, 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 explusion 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

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 a neural store of expectations. The expectations are also referenced to the sensory channel that delivered them and are stronger for more experiences and also in those ranges within which the channel is most sensitive. The purpose of information processing and perception functions is to predict 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 responses 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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