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Postural instability precedes motion sickness

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ABSTRACT: We evaluated the hypothesis that postural instability precedes the onset of motion sickness. Subjects standing in a "moving room" were exposed to nearly global oscillating optical flow. In the experimental condition, the optical oscillations were a complex sum-of-sines between 0.1 and 0.3 Hz, with an excursion of 1.8 cm. This optical motion was of such low frequency and magnitude that it was sometimes not noticed by subjects. However, in two experiments, exposure to the moving room produced significant increases in scores on a standard motion sickness questionnaire. In addition, approximately half of subjects reported motion sickness. Analysis of postural motion during exposure to the moving room revealed increases in postural sway before the onset of subjective motion sickness symptoms. This confirms a key prediction of the postural instability theory of motion sickness. © 1999 Elsevier Science Inc.

KEY WORDS: Posture, Motor control, Optical flow, Instability, Sway.

INTRODUCTION

Motion sickness is an ancient problem, having afflicted humans for thousands of years. For most of human history it has been associated with transportation (ships and other vehicles). In this century, improvements in vehicle design and the switch from sea to air travel have combined to make sickness rare in transportation. Despite this, there is renewed interest in the cause and possible prevention of motion sickness. This is due to frequent reports of motion sickness among users of simulation technology. Specifically, sickness is common in systems that present optical depictions of inertial motion, such as flight and driving simulators, and many virtual environment (VE) systems [43,46]. Particularly frustrating for users and designers is the positive correlation between sickness incidence and simulation fidelity: improvements in simulation fidelity appear to increase the incidence of sickness [6]. The effectiveness of simulation and VE systems, and their acceptance by users, can be severely limited if they produce motion sickness [3]. This provides a strong practical motivation for understanding the malady. Prevention of motion sickness would be facilitated if we could develop objective measures to predict it and/or if we could identify and eliminate the factors that cause it.

Motion sickness is not limited to applied settings or to high-technology systems. To the surprise of researchers, it has occurred unbidden in laboratory experiments whose purpose was to study relations between vision and the perception and control of standing posture [33,34,45,47]. Generally, studies of posture and vision have not attempted to explain the occurrence of motion sickness or to assess its possible implications for theories that relate vision and posture [e.g., 8,34]. This suggests that researchers see motion sickness as having no theoretically important relation to postural control. By contrast, Riccio and Stoffregen [44] argued that there may be an intimate relation between motion sickness and the perception and control of bodily orientation.

Motion Frequency and Postural Sway

There is a strong empirical relation between the appearance of nausea and the frequency of imposed periodic motion. In operational (nonlaboratory) conditions, motion sickness occurs almost exclusively in the presence of imposed periodic motion at frequencies from 0.08 to 0.4 Hz [17,28–30]. Vibration at these frequencies is known to be characteristic of nauseogenic vehicles, such as ships, trains and aircraft [17,28–30]. Even prolonged motion (up to 12 h) at other frequencies leads to little or no sickness. The frequency data might suggest that motion in the 0.08 to 0.4-Hz range causes motion sickness. The relation between sickness and motion frequency might be accounted for within the sensory conflict theory by a filter that excluded high-frequency conflict from sickness-generating systems. A second possibility would be to argue that variations in transfer characteristics in different perceptual systems lead greater conflict when their outputs are in this range [52]. However, spontaneous (unperturbed) standing sway is concentrated between 0.1 and 0.4 Hz [2,35]. The fact that we are not sickened by our own postural sway indicates that vibration in this frequency range is not inherently nauseogenic.

The nonnauseogenic properties of spontaneous sway might be accounted for in the sensory conflict theory by positing a threshold for nauseogenic conflict. A threshold could be credible if postural sway were low in magnitude compared with the magnitudes of imposed vibration that are known to cause sickness [48]. This does not appear to be the case: Nausea has been caused by imposed optical oscillations that mimicked the amplitude and frequency of postural sway [34,47,48]. Thus, the occurrence of motion sickness in these situations is a problem for the sensory conflict theory.

Recently, a modification of the sensory conflict view has been presented [9,27]. The modification proposes that motion sickness results from perceptual-motor anomalies, not from changes in sensory input, but from *sensorimotor rearrangement*, which is defined as "rearrangements in the relationship between movement and sensorimotor feedback" [9, p. 320]. Although this view is promising, it has not yet been developed into a theory of motion

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sickness etiology. The concept of sensorimotor rearrangement is an "empirically based working hypothesis" [9, p. 322]. There has been no attempt to use either the sensory conflict theory or the sensorimotor view to explain the occurrence of motion sickness in experiments on posture and vision. The sensorimotor rearrangement view also has not addressed the empirical relation between motion sickness and the frequency of imposed motion [9].

Motion Sickness and Instability in the Control of Posture

The empirical relation between spontaneous postural sway, imposed periodic motion and motion sickness has been predicted, *a priori*, by a new theory of motion sickness. Riccio and Stoffregen [44] hypothesized that motion sickness is caused by instability in the control of the posture of the body and/or its segments. In the postural control literature, loss of stability generally is associated with a frank loss of control, such as falling. This is not the type of instability that was hypothesized by Riccio and Stoffregen [44] to be related to motion sickness. They defined postural stability as "the state in which uncontrolled movements of the perception and action systems are minimized." Postural instability, then, need not entail frank loss of control; stability may be degraded rather than lost outright. There can be variation in the magnitude of instability, and instability can persist over long periods of time without necessarily leading to loss of control.

One possible source of postural instability might be situations in which a person tried to control posture in the presence of imposed oscillations that had significant power between 0.1 and 0.3 Hz. This hypothesis is related to the concept of wave interference [49]. When independent waveforms interact, the results will be a function of their relative frequencies. Consider two independent systems, each of which oscillates within a narrow band of frequencies. Suppose that waveforms generated by these two systems interact. If the frequencies of the two systems are highly disparate, the waveforms will pass through each other with little effect. However, if the systems oscillate at similar frequencies, the interaction of the waveforms can lead to wave interference, including dramatic instabilities in both frequency and amplitude. A similar effect may underlie motion sickness in the presence of low-frequency imposed motion. The imposition of oscillations in the frequency range of spontaneous sway may destabilize the postural control system in frequency and/or amplitude through a wave interference effect. This would explain why, in the context of imposed periodic motion, sickness is observed only with imposed motion in the narrow band of frequencies that is spontaneously produced by postural sway.

The postural instability theory does not assert that wave interference is the only process that can induce postural instability or that imposed periodic motion is the sole cause of instability [44]. Wave interference between spontaneous and imposed periodic oscillations is only one of a variety of possible sources of postural instability. In the present study we discuss wave interference because the theory predicts that it should be especially provocative of sickness.

We view humans as adaptive nonlinear systems, a view that is gaining acceptance in the postural control literature [cf. 15]. Instability in such systems is not yet well defined [4,5]. For this reason, there is some uncertainty about how stability and instability should be measured; this is true not only for instability in posture but for instability in any adaptive nonlinear system. One consequence of this is that there is not yet a clear operationalization of the postural instability theory. Riccio and Stoffregen [44] suggested several parameters of postural motion in which instability might be observed. In the present study our purpose was not to evaluate all possible definitions of stability or even all those suggested by Riccio and Stoffregen [44]. Rather, because this was the first test of a new theory, our goal was more modest: to establish an empirical relation between postural instability (however measured) and motion sickness. For this reason, we evaluated only a subset of parameters of postural sway. Two of the more widely studied manifestations of instability are in amplitude and frequency [26].

There have been reports of postural instability after the onset of motion sickness symptoms [e.g., 18,21,36,45]. However, this is compatible with almost any theory of motion sickness etiology: It is no surprise if people are wobbly after becoming sick. Rather than being an effect of motion sickness, Riccio and Stoffregen [44] proposed that postural instability is a necessary precursor of motion sickness. Thus, a critical feature of the new theory is that postural instability should precede the appearance of motion sickness symptoms. In the present study our primary purpose was to evaluate this prediction. We did not evaluate the hypothesis that postural instability and motion sickness are related to the frequency of imposed motion, that is, we did not compare the relative nauseogenic properties of different frequencies of imposed motion. This will be addressed in future research.

Visually Induced Motion Sickness

We noted earlier that motion sickness can occur in the absence of imposed inertial motion when there is self-motion relative to the optic array [e.g., 7]. This *visually induced motion sickness* (VIMS) has been reported in fixed-base flight and automobile simulation [13,32,51] and in a variety of nonvehicular virtual environments [9,11,46]. Riccio and Stoffregen [44] predicted that optical oscillations created by human locomotion should induce sickness when viewed on videotape; this prediction has been confirmed [50]. In addition, sickness has been produced as an unintended consequence of exposure to simulations of optical flow fields that are created by spontaneous postural sway [33,34,47]. This effect has not been studied rigorously. For this reason our first purpose in the present research was to make a deliberate planned attempt to induce motion sickness with imposed optical flow mimicking the amplitude and frequency of spontaneous postural sway.

The postural instability theory applies to imposed optical motion and to imposed inertial motion. It should be possible for imposed optical oscillations to induce postural instability and motion sickness, provided that the oscillations are detected (i.e., information about the optical disturbance must be picked up by the observer). In previous research there has been no detailed analysis of postural motion during exposure to nauseogenic optical oscillations [18,21,34].

VIMS may be closely related to motion sickness that is associated with VE systems (cybersickness). Thus, one practical benefit of our study may be improved understanding of sickness that is associated with this emerging technology. A second advantage of studying VIMS is methodological. The use of optical perturbations to posture permitted us to measure sway during exposure to the nauseogenic stimulus without having to account for imposed inertial motion (which was absent). In addition, with our focus on standing postural sway there was no voluntary action on the part of subjects (e.g., locomotion or manipulation, as is common with head-mounted VE systems). For these reasons, body motion in our experiments should be generated solely by postural sway. Thus, a method involving VIMS and standing posture may be an ideal venue for an initial evaluation of the postural instability theory of motion sickness. Future tests in the context of inertial motion or voluntary motion will require the development of analysis techniques for subtracting out these motions from the postural data [37]. This may explain why previous research relating posture to

motion sickness has looked at postural motion only before and after exposure [e.g., 21].

EXPERIMENT 1

Standing subjects were exposed to large-field optical flow whose temporal and spatial properties closely resembled those of spontaneous postural sway. We measured subjects' postural motion and correlated it with the appearance of motion sickness symptoms. Because our hypothesis was that postural instability would precede symptom onset, it was critical for us to know whether symptoms began before or after any abnormal postural sway. To ensure reliable information about the timing of symptom onset, subjects were familiarized with common motion sickness symptoms before beginning the experiment. In addition, they were explicitly instructed to discontinue their participation in the experiment at the onset of any symptoms, however mild. We expected, as a result of this instruction, that only mild symptoms would be reported.

Riccio and Stoffregen [44] identified a wide variety of possible metrics for postural instability. In the present study we assessed instability using several metrics. The primary metric was variability of postural motion. We defined this operationally as the standard deviation (s) of head position in the anterior-posterior (AP) and lateral axes; we predicted an increase in s in one or both axes before symptom onset. Our prediction was qualitative rather than quantitative. Additional metrics are discussed below. The treatment of subjects in our experiments conformed to standards established by the American Psychological Association [1].

MATERIALS AND METHODS

Subjects

Twelve University of Cincinnati (UC) students (graduate and undergraduate) participated in this experiment, 7 men and 5 women ranging in age from 18 to 25 years with a mean age of 21.1 years. Subjects ranged in weight from 51.86 to 109.09 kg with a mean weight of 73.3 kg. Subjects ranged in height from 1.6 to 1.88 m with a mean height of 1.73 m. All subjects had normal or corrected to normal vision, reported no history of dizziness, recurrent falls or vestibular (inner ear) dysfunction and demonstrated that they could stand on one foot for 30 s with eyes open. Subjects were asked if they were currently in good health and were excused if they reported any current illness (subjects who were excused for ill health nevertheless received full credit for experimental participation; there was, thus, no motivation for falsely stating that they were in good health). Subjects were volunteers (graduate students) or received class credit (undergraduates drawn from the UC subject pool). When scheduling their participation, subjects were instructed not to eat anything for 4 h before the experimental session. Compliance with this instruction was verified at the beginning of the session.

Apparatus

We generated optical flow using a moving room [31,47], an enclosure consisting of a cubical frame, 2.4 m on a side, mounted on wheels and moving in one axis along rails (Fig. 1). Motion of the room was produced by a torque motor under computer control. The four vertical sides and the upper horizontal side of the frame were covered with rigid foam-plastic sheeting. The interior surfaces of these walls were covered with marble-pattern paper. Access was provided through an opening cut into the rear of the right wall. The opening was 0.5 m wide and was not visible to subjects during experimental trials. At the center of the front wall

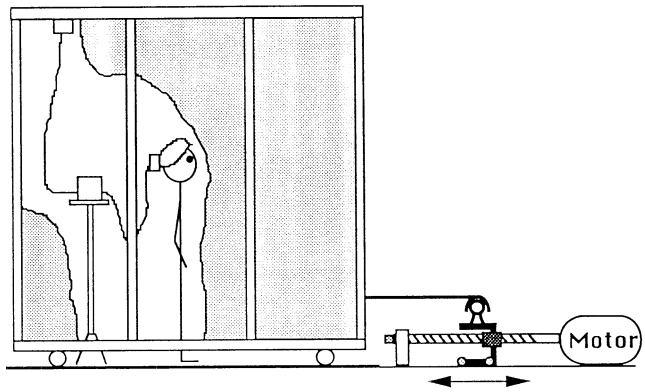

FIG. 1. The moving room. The subject wore a bicycle helmet to which was attached a receiver from the Flock of Birds tracking system. A second receiver was attached to the ceiling of the room. These were connected to the magnetic emitter, which rested on a stand behind the subject.

was placed a large detailed map of Ohio (96 × 106 cm; 32 × 34 degrees). Illumination was provided by a fluorescent fixture attached rigidly to the center of the ceiling of the moving room. The fixture extended several cm below the ceiling; this, combined with the flat walls, ensured that shadows were minimized. Subjects stood on the concrete laboratory floor, such that there is no imposed inertial motion.

Data on postural motion were collected using an electromagnetic tracking system (Flock of Birds, Ascension Technologies, Inc.). One receiver was attached to a bicycle helmet (which weighed 340 g) worn by the subject and another to the moving room. The transmitter was located behind the subject's head on a stand (Fig. 1). Six degree-of-freedom position data were collected from each receiver at 50 Hz and stored on disk for later analysis.

Procedure

Before the experiment, each subject completed a questionnaire on their motion sickness history. To assess their current level of symptoms and to ensure that they were familiar with motion sickness symptomology, subjects were also asked to complete the simulator sickness questionnaire (SSQ) [22]. The SSQ was selected for three reasons. It is a scale that has undergone extensive validation and testing [22] and it is widely used in both laboratory and field studies of motion sickness [e.g., 10,23–25,38]. Finally, the use of a measure of simulator sickness seemed appropriate because imposed global optical flow is widely interpreted as an optical simulation of self-motion or postural sway [e.g., 31,33,34]. Following Regan and Price [43], we used the SSQ to collect preexposure data, so as to establish a baseline against which postexposure data could be compared.

The room was driven using two functions (Fig. 2). One consisted of a simple 0.2-Hz oscillation, with an amplitude of 1.5 cm. The other was a sum of 10 sines, with frequencies of 0.0167, 0.0416, 0.0783, 0.1050, 0.1670, 0.1800, 0.1900, 0.2200, 0.2600 and 0.3100 Hz, each having an amplitude of 1.5 cm. The phase and amplitude of the component sines were adjusted so that the combined waveform had a maximum amplitude of 1.8 cm.

Subjects entered the moving room through the opening in the right wall and placed their heels on a marker on the floor so that they were facing along the line of motion. They were asked to keep their hands in their pockets and not to move their feet during trials [cf., 47]. There was not a single fixation point; subjects were asked

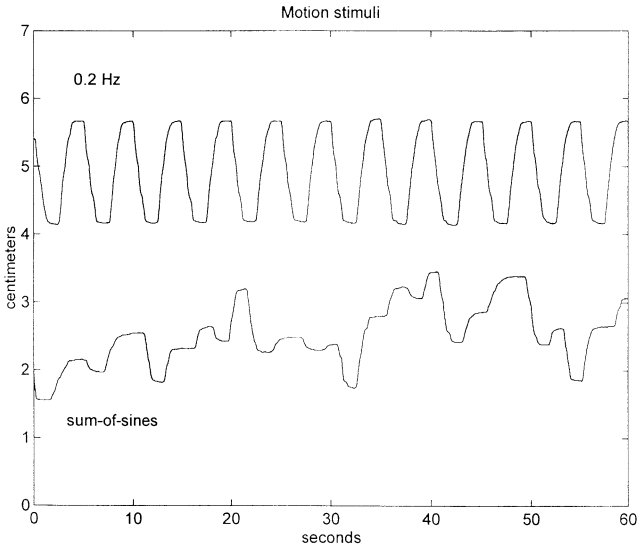

FIG. 2. Motion functions of the moving room. The upper trace shows the 0.2-Hz motion. The lower trace shows a portion of the sum-of-sines motion. The sum-of-sines function did not repeat but varied continuously over the 600-s trial duration.

to keep their gaze on the map on the front wall and to minimize head movements in varying their gaze. The sequence of trials is summarized in Table 1. We began by collecting data on spontaneous sway, with no room motion, for 20 s with eyes open and again with eyes closed. The purpose was to determine the extent of subjects' spontaneous sway before exposure to any imposed motion. This was followed by two 60-s exposures to the 0.2-Hz stimulus, one with eyes open and one with eyes closed. These trials were identical in duration and motion frequency to conditions used in previous research [47]. The eyes-open trial permitted us to assess subjects' responsiveness to imposed optical flow in a situation that was expected to be nonnauseogenic (due to the brevity of exposure). The eyes-closed trial allowed us to verify that induced postural motion was due solely to visual stimulation. These

TABLE 1

THE SEQUENCE OF TRIALS

Trial	Condition
1	20 s, eyes open, no imposed motion
2	20 s, eyes closed, no imposed motion
3	1-min, eyes open; room motion at 0.2 Hz, 1.5 cm amplitude (Fig. 2, top)
4	1-min, eyes closed; 0.2 Hz, 1.5 cm amplitude
5–8	10-min; eyes open, sum of 10 sines, 1.8 cm max. amplitude (Fig. 2, bottom)
9	1-min, eyes open; 0.2 Hz, 1.5 cm
10	20 s, eyes open, no imposed motion
11	20 s, eyes closed, no imposed motion

pretests were followed by four trials, each 10 min (600 s) long, using the sum-of-sines stimulus. Reports of perceived motion of the room and of the self were gathered at the end of each sum-of-sines trial. Subjects were asked to describe any experience of motion they had, and their verbatim reports were recorded. After exposure to the sum-of-sines motion, Trials 1, 2 and 3 were repeated. This was intended to permit us to evaluate pre and post differences in spontaneous sway and in responses to the simple 0.2-Hz imposed flow. While they were in the moving room, subjects were monitored continuously by an experimenter seated outside the opening. This was for their safety and to ensure compliance with instructions.

Subjects were warned that they might become ill and were instructed to discontinue the experiment immediately if they began to experience any noticeable symptoms. The time of discontinuation was recorded automatically. After discontinuation or the completion of four sum-of-sines trials, subjects were asked to fill out the SSQ a second time, after which those who felt well enough repeated three of the pretest trials. At the end of the session, subjects who had not yet reported any symptoms were asked to report on their motion sickness status over the next 24 h. They were given a brief questionnaire on which they indicated, on a yes/no basis, whether they developed motion sickness and if so, when. They were also given a printed copy of the SSQ, which they were asked to fill out at the time of symptom onset or after 24 h if no symptoms developed. Stoffregen [47] noted that symptom onset was sometimes delayed up to an hour after termination of exposure to a moving room. Similarly, Kennedy and Lilienthal [24] found that disorientation, dizziness and vertigo often are experienced only after leaving a simulator, sometimes up to 12 h later. It was for this reason that subjects who were asymptomatic at the end of the experimental session were asked to report their subjective state over the following 24 h.

RESULTS

In all cases, subjects complied with the instructions to not move their feet. In addition, there were no head turns or other head motions that were visible to the experimenter.

Motion sickness

Motion sickness history. Half of the subjects reported having been motion sick in the past. Of those subjects who did not become sick in our study, 43% reported some prior history of sickness. Of the subjects who did become sick in our study, 60% reported a prior history of sickness. Most sickness was reported in cars or boats, especially while reading. Subjects' ratings of their own sickness susceptibility had a mean of 3.43 (out of 10) for subjects who did not report sickness in our experiment and a mean rating of 5.2 for subjects who reported sickness. A *t*-test found no significant difference in ratings between the two groups.

Incidence of sickness and discontinuation. Subjects were divided into Sick and Well groups, with the Sick group containing all subjects who became sick during the experiment or up to 24 h after the experiment. There were seven subjects in the Well group and five (42%) in the Sick group.[1] Two of the Sick subjects developed symptoms after leaving the laboratory and three discontinued the experiment: ET discontinued after completing Trial 6, HM discontinued at 8:10 in Trial 7 and TS discontinued at 6:42 in Trial 5. Sickness reports (both oral and written) were unambiguous ("I

[1] The incidence of sickness was similar to previous studies in which subjects have been exposed to imposed optical flow in a moving room [47, cf. 45]. In the earlier work, subjects were not familiarized with the symptoms of motion sickness by the experimenters. Thus, it seems unlikely that self-reports of sickness in the present experiment were falsely elicited by the fact that subjects completed the SSQ before exposure to imposed optical flow.

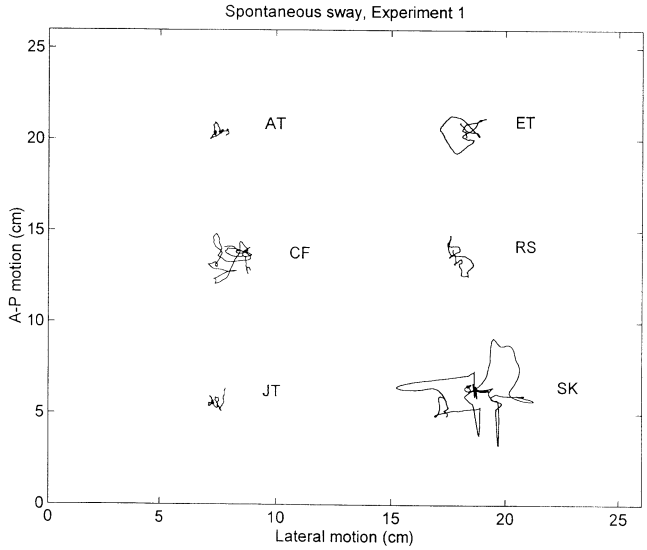

FIG. 3. Position data for representative individual trials, Trial 1 (eyes open, spontaneous sway); Experiment 1. Subjects are identified by their initials. Subject ET developed motion sickness later in the experiment, whereas RS and SK developed sickness after leaving the laboratory.

feel/felt sick," "I feel like I'm going to throw up," etc.). One subject who orally denied having symptoms was classified as sick on the basis of his postexperimental behavior and appearance. The experimenter made this classification before examining any postural data for this subject.

SSQ. Questionnaire scores for each subject were calculated in the recommended manner [22]. Due to the small sample size, we analyzed only the Total Severity Score. The scores were evaluated using a one-within (pretest vs. posttest), one between (Sick vs. Well groups) ANOVA. There were significant main effects for time of test (pretest vs. posttest), $F(1,10) = 15.58$, mean square (MS) = 7,654.61, $p < 0.05$, and for group (Sick vs. Well), $F(1,10) = 17.14$, MS = 12,201.45, $p < 0.05$. The interaction also reached significance: $F(1,10) = 13.795$, MS = 6,778.06; $p < 0.05$. The mean pretest scores were 20.2 (Sick) and 8.55 (Well).[2] The mean posttest scores were 90.51 (Sick) and 10.69 (Well). Post-hoc *t*-tests revealed that the pretest difference between Sick and Well groups was not statistically significant. The only significant difference was between the pretest and posttest scores for the Sick group.

Vection/perception of room motion. Four of seven Well subjects reported vection at some point during the sum-of-sines trials. Each of the five Sick subjects reported vection at some point. Subjects were also asked if they perceived the room as moving. Five Well subjects and four Sick subjects responded in the affirmative.

Postural Motion

Due to intermittent data acquisition problems, data on postural motion were not recorded for some of the sum-of-sines trials (Trials 5–8) and post-sum-of-sines spontaneous sway trials (Trials 9 and 10). Among subjects who developed motion sickness, four sum-of-sines trials were not recorded. Among subjects who did not develop motion sickness one sum-of-sines trial was not recorded and three post-sum-of-sines spontaneous sway trials were not recorded. Our statistical analyses of postural motion were based on those trials for which data were acquired. There was no corruption of any of the acquired data. Although the loss of data was undesirable, we did not regard it as a serious problem for the evaluation of our hypotheses, due to the large volume of data that was available for analysis.

Because of development of motion sickness, three subjects did not participate in the post-sum-of-sines trials (Trials 9–11). This meant that we had data for these trials for only two Sick subjects. For this reason, no statistical analyses were conducted on Trials 9, 10, and 11.

Spontaneous sway (Trials 1 and 2). Variability (*s*, in cm), range (cm) and velocity (cm/s) were calculated for each subject for both AP and lateral (LAT) motion. For each variable and axis of motion, separate one-within (vision: eyes open vs. eyes closed), one-between (groups: Sick vs. Well) ANOVAs were performed on the pretest trials (Trials 1 and 2). Position data for representative trials are presented in Fig. 3.

Variability: The analysis revealed a significant main effect of groups on variability in the AP axis ($F_{AP}(1,10) = 8.74$, MS = 1.26, $p < 0.05$), with the Sick group exhibiting significantly greater variability than the Well group. The main effect of vision and the group by vision interaction did not reach significance. There was no significant main effects or interactions for sway in the lateral axis. Means are presented in Table 2.

TABLE 2

MEAN (SE) VARIABILITY (*s*, in cm) OF SWAY FOR SICK AND WELL GROUPS IN AP AND LATERAL AXES FOR SPONTANEOUS SWAY, EXPERIMENT 1

	Trial 1 (eyes open)		Trial 2 (eyes closed)	
	AP	LAT	AP	LAT
Sick	0.74	0.98	1.12	0.90
	(0.20)	(0.48)	(0.27)	(0.31)
Well	0.33	0.25	0.60	0.23
	(0.06)	(0.05)	(0.13)	(0.05)

Velocity: There were also significant differences in sway velocity between Sick and Well groups, for both AP and lateral axes ($F_{AP}(1,10) = 7.92$, MS = 6.1, $p < 0.05$, $F_{LAT}(1,10) = 5.03$, MS = 2.54, $p < 0.05$). In both cases, velocities were higher for the Sick group. The main effects of vision and the group by vision interactions did not reach significance. Means are presented in Table 3.

Range: There was a significant difference between Sick and Well groups ($F_{AP}(1,10) = 9.47$, MS = 114.02, $p < 0.05$), with the Sick group exhibiting a greater range of AP sway. The main effect of vision and the group by vision interaction did not reach significance. The same outcome was observed in the lateral axis, with only the main effect of groups reaching significance

[2] For both the Sick and the Well groups, the pretest SSQ scores were higher than those typically obtained from persons who have not been exposed to nauseogenic stimulation. However, as is true of many standard motion sickness instruments, the SSQ was normed on a population of military personnel [22]. The unusually high pretest scores may therefore be related to differences between this military population and the civilian undergraduate subjects in the present study. Elevated scores might also result from preexisting illness (e.g., a head cold or hangover); however, none of our subjects reported any illness.

TABLE 3

MEAN (SE) VELOCITY (cm/s) OF SWAY FOR SICK AND WELL GROUPS IN AP AND LATERAL AXES FOR SPONTANEOUS SWAY, EXPERIMENT 1

	Trial 1 (eyes open)		Trial 2 (eyes closed)	
	AP	LAT	AP	LAT
Sick	1.45	1.03	1.51	0.91
	(0.66)	(0.47)	(0.26)	(0.25)
Well	0.38	0.32	0.53	0.30
	(0.09)	(0.10)	(0.06)	(0.05)

($F_{LAT}(1,10) = 5.42$, MS = 69.22; $p < 0.05$). Means are presented in Table 4.

1-min, 0.2-Hz stimulus (Trials 3 and 4). Variability, range and velocity data were calculated for each subject. We also conducted an analysis of gain between postural motion and room motion [8]. Finally, the postural motion of each subject was cross-correlated with the room motion. This analysis yielded the maximum correlation and the time lag at which this maximum correlation occurred. Separate analyses were conducted on the correlation and lag data. For each variable, the data were analyzed using one-within (vision), one-between (groups) ANOVAs. Analyses were conducted separately for each axis (AP, lateral). Position data for representative trials are presented in Fig. 4.

Variability: The analysis revealed significant differences between Sick and Well groups for both AP and lateral axes ($F_{AP}(1,10) = 8.40$, MS = 1.64; $p < 0.05$, $F_{LAT}(1,10) = 16.57$, MS = 1.78; $p < 0.05$). The Sick group exhibited greater sway for both visual conditions (eyes open, eyes closed). There was not a significant effect for vision or a significant interaction between vision and groups. The means are presented in Table 5.

Velocity: Significant differences were found between Sick and Well groups for both AP and lateral axes ($F_{AP}(1,10) = 5.32$, MS = 2.39; $p < 0.05$, $F_{LAT}(1,10) = 12.75$, MS = 1.00; $p < 0.05$). The Sick group exhibited greater sway velocity across visual conditions (eyes open, eyes closed). There was not a significant effect for vision or a significant interaction between vision and groups. The means are presented in Table 6.

Range: Significant differences were found between Sick and Well groups in the lateral axis ($F_{LAT}(1,10) = 20.54$, MS = 71.23; $p < 0.05$). The Sick group exhibited greater range across visual conditions. Again, neither the effect of vision nor the interaction reached significance. The means are presented in Table 7.

Cross Correlation: There was a significant main effect of vision, with coupling of postural motion with the room being

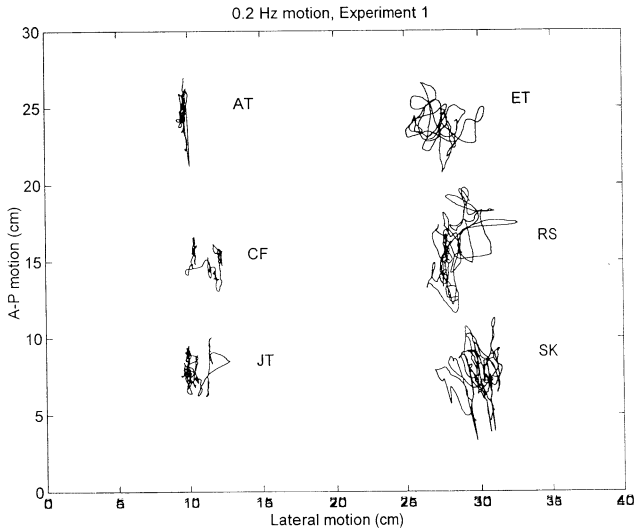

FIG. 4. Position data for representative individual trials, Trial 3 (eyes open, 0.2 Hz, 60-s room motion); Experiment 1. Subjects are identified by their initials. Subject ET developed motion sickness later in the experiment, whereas RS and SK developed sickness after leaving the laboratory.

significantly lower in the eyes closed trial; ($F_{vision}(1,10) = 18.41$, MS = 0.52, $p < 0.05$). This verifies that any effect of the moving room on posture was visual and shows that in both groups, postural control was influenced by the imposed optical flow. There was also a significant main effect of groups ($F_{groups}(1,10) = 9.92$, MS = 0.16, $p < 0.05$), with Sick subjects exhibiting stronger coupling with the room motion. The interaction was not significant. The analysis did not show any significant differences in time lag (the degree to which the subject was behind or ahead of the room). The mean correlations and lags for each group are presented in Table 8.

Gain: Gain is the ratio of the magnitude of the response to the magnitude of the stimulus. Gain was assessed only in the AP axis, because this was the axis of the imposed stimulus. There was a significant main effect of group (Sick vs. Well; $F(1,10) = 12.71$, MS = 1.61, $p < 0.05$) and a main effect for vision (eyes open vs. closed; $F(1,10) = 22.80$, MS = 1.12, $p < 0.05$). The interaction also achieved significance ($F(1,10) = 17.65$, MS = 0.86, $p < 0.05$). Post-hoc analyses revealed that Sick subjects exhibited higher gain but only with the eyes open (Trial 3). Means are presented in Table 9.

10-min, sum-of-sines stimulus (Trials 5–8). Variability, range

TABLE 4

MEAN (SE) RANGE (cm) OF SWAY FOR SICK AND WELL GROUPS IN AP AND LATERAL AXES FOR SPONTANEOUS SWAY, EXPERIMENT 1

	Trial 1 (eyes open)		Trial 2 (eyes closed)	
	AP	LAT	AP	LAT
Sick	5.48	4.55	7.52	4.25
	(2.32)	(2.29)	(1.51)	(1.41)
Well	1.39	0.94	7.52	0.95
	(0.27)	(0.20)	(0.86)	(0.19)

TABLE 5

MEAN (SE) VARIABILITY (*s*, in cm) OF SWAY FOR SICK AND WELL GROUPS IN AP AND LATERAL AXES FOR 0.2 Hz MOTION (60 s) TRIALS, FOR EXPERIMENT 1

	Trial 3 (eyes open)		Trial 4 (eyes closed)	
	AP	LAT	AP	LAT
Sick	1.38	0.75	1.51	0.81
	(0.32)	(0.22)	(0.43)	(0.19)
Well	0.75	0.41	0.93	0.38
	(0.06)	(0.10)	(0.08)	(0.04)

TABLE 6

MEAN (SE) VELOCITY (cm/s) OF SWAY FOR SICK AND WELL GROUPS IN AP AND LATERAL AXES FOR 0.2 Hz MOTION (60 s) TRIALS, FOR EXPERIMENT 1

	Trial 3 (eyes open)		Trial 4 (eyes closed)	
	AP	LAT	AP	LAT
Sick	1.46	0.65	1.32	0.68
	(0.64)	(0.22)	(0.49)	(0.24)
Well	0.51	0.29	0.58	0.31
	(0.04)	(0.03)	(0.03)	(0.05)

TABLE 7

MEAN (SE) RANGE (cm) OF SWAY FOR SICK AND WELL GROUPS IN AP AND LATERAL AXES FOR 0.2 Hz MOTION (60 s) TRIALS, FOR EXPERIMENT 1

	Trial 3 (eyes open)		Trial 4 (eyes closed)	
	AP	LAT	AP	LAT
Sick	7.39	4.19	8.41	4.98
	(2.11)	(1.27)	(2.46)	(1.43)
Well	4.79	2.06	4.65	1.90
	(1.48)	(0.33)	(0.38)	(0.30)

and velocity were calculated for each subject. In addition, simple correlations between subject and room motion were obtained. These data were analyzed using unpaired *t*-tests testing for Sick/Well differences across experimental trials for AP and lateral axes. We used *t*-tests because of missing data for some trials (due to subject discontinuation and unrecorded data); the resulting unequal *n*s violated an assumption of the ANOVA procedure. Position data for representative trials are presented in Fig. 5.

Variability: For the Sick group, the mean variability (*s*) of sway across the sum-of-sines trials was 2.17 cm (AP) and 2.36 cm (lateral). For the Well group, the means were 1.45 cm (AP) and 0.97 cm (lateral). There were significant differences between Sick and Well groups separately for the AP and lateral axes ($t_{AP(35)} = 3.50$; $p < 0.05$, MD = 0.75, $t_{LAT(35)} = 5.69$; $p < 0.05$, MD = 1.39).

Velocity: For the Sick group, the mean velocity of sway across the sum-of-sines trials was 1.58 cm/s (AP) and 1.00 cm/s (lateral). For the Well group, the means were 0.63 cm/s (AP) and 0.45 cm/s (lateral). There were significant differences between Sick and Well groups separately for the AP and lateral axes ($t_{AP(35)} = 7.88$; $p < 0.05$, MD = 0.95, $t_{LAT(35)} = 7.90$; $p < 0.05$, MD = 0.55).

Range: For the Sick group, the mean range of sway across the sum-of-sines trials was 17.46 cm (AP) and 16.40 cm (lateral). For the Well group, the means were 9.02 cm (AP) and 7.58 cm (lateral). There were significant differences between Sick and Well groups separately for the AP and lateral axes ($t_{AP(35)} = 5.69$; $p < 0.05$, MD = 8.44, $t_{LAT(35)} = 5.47$; $p < 0.05$, MD = 8.83).

Correlation: Simple times-series correlations were computed for each subject and trial. The correlations were normalized and then analyzed using an unpaired *t*-test. Unlike the 0.2-Hz trials, there was not a significant difference between Sick and Well groups.

Discussion

Sickness and optical flow. Motion sickness was induced by imposed optical flow having the same amplitude and frequency characteristics as the optical flow that results from spontaneous (unperturbed) standing postural sway. The maximum room excursion in any of our conditions was 1.8 cm and the maximum frequency of motion was 0.31 Hz. These motions were so mild that in some cases subjects did not perceive the room to be moving at all. This confirms previous reports [34,47] and verifies that VIMS is not limited to high amplitude motion [14]. It also confirms that VIMS can occur at the frequencies of motion that are associated with inertially induced sickness [17,28–30].

An additional effect was that vection was reported by each subject who became sick but by only four of seven subjects who did not become sick. This is consistent with the proposal that vection is a necessary condition for the development of VIMS [19].

Sickness and postural motion. There were numerous differences between Sick and Well subjects in postural motion. These differences existed before the onset of symptoms, confirming predictions of the postural instability theory of motion sickness [44]. There were significant differences between Sick and Well groups in all phases of the experiment on which statistical analyses were performed: spontaneous sway, responses to the simple sinusoid and responses to the complex sum-of-sines.

As predicted, during exposure to sum-of-sines optical flow, there were increases in several indices of postural sway among subjects who later developed motion sickness. Motion sickness was preceded by increases in the variability, range and velocity of postural sway. In several cases the amplitude of postural sway was an order of magnitude greater than the magnitude of the optical stimulus (Fig. 5). This was true in both the AP and lateral axes. The fact that these effects existed before the onset of motion sickness symptoms suggests that it may be possible to use objective real-time measures of postural motion to predict motion sickness in nauseogenic situations [cf., 20].

Two additional findings of interest concern postural sway be-

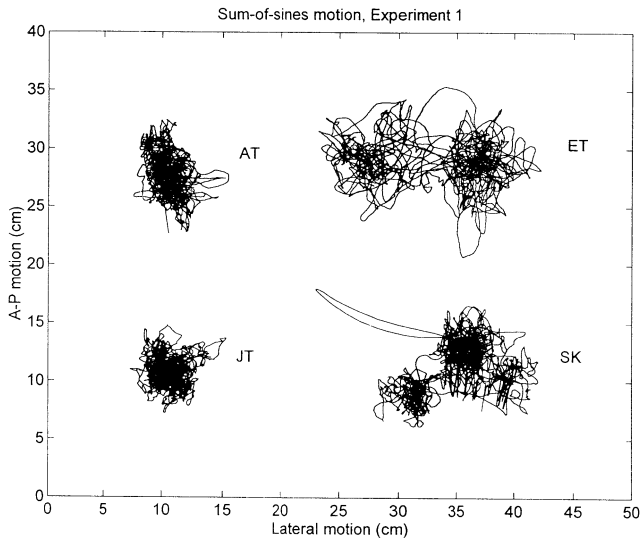

FIG. 5. Position data for representative individual sum-of-sines trials; Experiment 1. Subjects are identified by their initials. Subject ET developed motion sickness later in the experiment, and SK developed sickness after leaving the laboratory.

TABLE 8

MEAN (SE) CROSS-CORRELATION AND TIME-LAG OF SWAY FOR SICK AND WELL GROUPS FOR 0.2 Hz MOTION (60 s) TRIALS, FOR EXPERIMENT 1

	Trial 3 (eyes open)		Trial 4 (eyes closed)	
	r	lag	*r*	lag
Sick	0.52	−0.67	−0.02	−12.00
	(0.18)	(10.37)	(0.07)	(28.60)
Well	0.18	−14.71	0.01	−13.71
	(0.05)	(21.35)	(0.07)	(23.61)

fore subjects were exposed to the sum-of-sines stimulus. Sick and Well differed in their postural motion during exposure to the brief 0.2-Hz stimulus. Perhaps most remarkably, there were reliable differences between Sick and Well groups in spontaneous postural sway before exposure to any experimental motion.[3,4] This suggests general differences in postural motion between people who are susceptible to motion sickness and those who are not. If so, this could provide the basis for simple, objective, noninvasive, nonnauseogenic predictive tests for sickness susceptibility.

EXPERIMENT 2

Experiment 2 was conducted in an attempt to replicate the novel findings. The method and procedure were the same as in Experiment 1. There were no data management problems in Experiment 2; we were able to retain postural motion data for each trial in which subjects participated. As in Experiment 1, no statistical analyses were performed on postural motion data from Trials 9–11.

MATERIALS AND METHODS

Subjects

Eight UC students participated in this experiment, 5 men and 3 women ranging in age from 18 to 21 years with a mean age of 19.3 years. Subjects ranged in weight from 60.75 to 74.25 kg, with a mean weight of 70.48 kg. Subjects ranged in height from 1.63 to 1.80 m, with a mean height of 1.73 m.

Procedure

The procedure was identical to that used in Experiment 1.

RESULTS

One subject experienced a partial loss of consciousness and collapsed during Trial 5 (sum-of-sines motion). She reported no symptoms of motion sickness and no prior history of fainting. This subject was replaced. In all other cases subjects complied with the instructions to not move their feet. There were no head turns or other head motions that were visible to the experimenter.

Motion sickness

History. Sixty-three percent of the subjects reported being motion sick in the past (Well, 50%; Sick, 75%). Most subjects reported becoming sick in cars or boats, especially while attempting to read. Self-ratings of susceptibility to sickness produced a mean rating of 2.5 (out of 10) for Well subjects and a mean rating of 4.5 for Sick subjects. A *t*-test performed on these ratings did not reach significance.

TABLE 9

MEAN (SE) GAIN OF SWAY FOR SICK AND WELL GROUPS FOR 0.2 Hz MOTION (60 s) TRIALS, FOR EXPERIMENT 1

	Trial 3 (eyes open)	Trial 4 (eyes closed)
Sick	1.33 (0.48)	0.36 (0.12)
Well	0.27 (0.05)	0.16 (0.03)

Incidence of sickness and discontinuation data. Four subjects reported symptoms of motion sickness (50%). Three subjects who reported sickness discontinued the experiment: BS discontinued at 8:23 in Trial 7 (third sum of sines trial), NA discontinued at 6:21 in Trial 6 and TH discontinued at 1:08 in Trial 6. DP reported that symptoms developed after leaving the laboratory. Sickness reports (both oral and written) were again unambiguous.

SSQ. We again analyzed only the Total Severity Score of the SSQ. Mean scores were tested using a one-within (pretest/posttest), one-between (Sick/Well) ANOVA. The analysis revealed a significant main effect for time of test, with posttest scores being higher: $F(1,6) = 45.60$, MS = 2,644.53, $p < 0.05$. The main effect of groups was not significant: $F(1,6) = 3.20$, MS = 3,469.80, $p > 0.05$. However, the interaction was significant: $F(1,6) = 71.77$, MS = 4,162.19; $p < 0.05$. The mean pretest scores were 18.70 (Sick) and 21.51 (Well). The mean posttest scores were 76.67 (Sick) and 14.96 (Well). Post-hoc tests showed that posttest scores were significantly higher than pretest scores only for the Sick group.

Vection/perception of room motion. Two of four Well subjects reported vection at some point during the 10-min trials, whereas each of four Sick subjects reported vection. All subjects in both groups reported that they perceived the room to be moving.

Postural motion

Spontaneous sway (Trials 1 and 2). There were no significant differences between Sick and Well groups or between eyes-open and eyes-closed trials in the variability or range of postural motion. However, significant differences were obtained for velocity of sway in the AP axis. The mean velocity of AP sway for the Sick group (0.52 cm/s) was significantly greater than the mean for the Well group (0.38 cm/s): $F_{groups}(1,6) = 6.60$, MS = 0.08, $p < 0.05$. There was also a main effect of vision on AP velocity: $F_{vision}(1,6) = 11.96$, MS = 0.27, $p < 0.05$; mean (eyes open) = 0.32 cm/s; mean (eyes closed) = 0.58 cm/s. The interaction was not significant.

1-min, 0.2-Hz stimulus (Trials 3 and 4). There were no significant differences between Sick and Well groups or between eyes-open and eyes-closed trials for variability, velocity or range in either the AP or lateral axis.

Cross Correlation: Cross-correlations were significantly higher on eyes-open trials ($F(1,6) = 70.08$, MS = 0.27, $p < 0.05$), again confirming the visual basis of the moving room's effect on

[3] It is important to reiterate that although postural motion data were lost for some sum-of-sines trials, there were no such losses for Trials 1–4; the data sets for these trials are complete.

[4] In the spontaneous sway trials, postural motion in the Well group was comparable with that observed in previous studies of unperturbed stance [e.g., 39].

posture. There was not a significant main effect for group or a significant interaction. There were no significant effects in the analysis of time lag.

Gain: Gain was again assessed only in the AP axis. Gain for the eyes-closed trials was significantly lower than in the eyes-open trials ($F(1,6) = 8.81$, MS = 0.18, $p < 0.05$). Unlike Experiment 1, there was not a significant difference between Sick and Well groups.

10-min, sum-of-sines stimulus (Trials 5–8). Variability: There was not a significant difference between Sick and Well groups for sway in the AP axis. However, there was a significant difference between groups for sway in the lateral axis ($t_{LAT(25)} = 2.05$, $p < 0.05$, MD = 0.58), with Sick subjects (mean 1.34 cm) exhibiting significantly greater lateral sway than did the Well group (mean 0.76 cm).

There were no significant differences between Sick and Well for velocity, range of motion or simple correlation in either AP or lateral axes.

Discussion

Again, the moving room proved effective in inducing motion sickness. Also in replication, vection was reported by each subject who became sick but by only some subjects who did not.

Postural effects were less pronounced than in Experiment 1. This may have been due to the smaller sample size in Experiment 2 (this is borne out by the fact that mean differences between Sick and Well groups for measures of postural motion often differed by a factor of 2). There were no effects during exposure to the 0.2-Hz motion (Trials 3 and 4). Thus, the effects for these trials that were observed in Experiment 1 should be interpreted with caution. However, there was a significant difference between Sick and Well groups in AP sway velocity in the spontaneous motion trials (Trials 1 and 2), with higher sway velocity among subjects who would later become sick. In addition, there was a significant difference between Sick and Well groups in the variability of sway in the lateral axis during exposure to the sum-of-sines motion. Variability was greater among subjects who later developed motion sickness. These effects confirm the general finding of Experiment 1 that postural instability precedes motion sickness.

GENERAL DISCUSSION

In two experiments we measured several parameters of postural sway during unperturbed stance and while subjects were exposed to low-amplitude imposed optical flow having either a simple sinusoidal (0.2 Hz) or a complex sum-of-sines character. We found that motion sickness developed (either during exposure of after leaving the laboratory) in about half of the subjects. In both experiments, Sick and Well subjects differed in their postural motion, and these differences existed before the onset of motion sickness symptoms.

Motion Sickness

Motion sickness was observed in subjects who were exposed to imposed optical flow having amplitude and frequency characteristics that closely mimic those of spontaneous (unperturbed) sway. The amplitudes of imposed motion (never greater than 1.8 cm) were much smaller than motion amplitudes that typically used in research on motion sickness and were also much smaller than motion amplitudes that are associated with various forms of operational motion sickness (e.g., sea sickness, air sickness, simulator sickness). In addition, our subjects were not engaged in vigorous or challenging behaviors and were specifically instructed to minimize head rotations.

The nauseogenic properties of low-amplitude low-frequency imposed optical flow are an issue for sensory conflict theory. Motion sickness is a relatively rare phenomenon. By contrast, low-magnitude sensory conflict is believed to be a common if not constant feature of ordinary behavior [41,42]. If so, then the relative rarity of motion sickness must be explained by hypothesizing that sickness is caused only by relatively high levels of conflict (sensory conflict theories typically include a filter or threshold for conflict, such that low magnitudes of conflict will not lead to sickness). Any conflict that is created by imposed optical flow in the range of postural sway would seem to be of low magnitude relative to conflict that is believed to exist in vehicles, flight simulators and so on. Thus, any threshold whose purpose is to suppress conflict arising from ordinary behavior should have been effective in our experiments. For this reason it is not clear how a conflict-based theory [e.g., 40–42] would predict the consistent occurrence of motion sickness in the presence of low-magnitude low-frequency optical oscillation. We are not aware of any treatment of the sensory conflict theory that has predicted that motion sickness should result from exposure of standing subjects to imposed optical flow amplitude used in the present experiments. Such a prediction would not violate the logic of the sensory conflict theory and so might be developed. This may present a challenge for the sensory conflict theory. It would be necessary to explain how any conflict produced in our experiments would be greater in magnitude than conflict produced in other conditions of stance that do not elicit motion sickness.

It might be argued that subjects in our experiments did not actually experience motion sickness but that the increase in SSQ scores resulted from increased arousal, anxiety or other unrelated subjective experiences. We believe that this is very unlikely, for two reasons. The first is that in addition to SSQ scores, we collected subjects' direct, explicit reports that they felt themselves to be motion sick. The "arousal hypothesis" would require the assertion that these reports were false. Before participating in the experiments, subjects were required to read and sign an informed consent form that stated that "you are free to stop participating in this experiment at any time and for any reason. You will receive full credit for your participation." Thus, subjects who wished to terminate the experiment could do so without any reference (either false or true) to motion sickness. The second reason to believe that our Sick subjects experienced genuine motion sickness is the existence of previous independent reports of sickness in response to low-frequency low-amplitude imposed optical oscillations in other studies. Of particular relevance is the fact that subjects have reported motion sickness in studies that were not intended to produce sickness and for which the experimenters had no *a priori* hypothesis that sickness might occur [34,47].

Vection was reported by about half of the subjects who did not become sick. By contrast, vection was reported by every one of the subjects who did become sick. This provides support for the hypothesis that vection is a necessary prerequisite for the development of VIMS [19]. Future research should examine in greater detail the time course and magnitude of vection that is associated with VIMS.

The occurrence of vection in our experiments is remarkable for two reasons. First, most reports of vection are associated with amplitudes of optical displacement that are orders of magnitude greater than those used in the present experiments. Second, consider relations between simulated and real vehicular motion. Vection is a common feature of vehicular simulation, and this corresponds with the fact that people are consciously aware of their motion when they travel in real vehicles. By contrast, our subjects experienced vection despite the fact that the imposed optical flow mimicked postural sway, which does not normally give rise to a

subjective experience of self-motion. Why should a simulation give rise to vection when the simulated event does not? This may be an interesting topic for future research on vection.

Postural Sway

There were significant differences between the postural motion of Sick and Well groups, and these differences existed before the onset of motion sickness symptoms. This confirms a major prediction of the postural instability theory of motion sickness [44]. The small stimulus amplitudes serve to make our study a strong test of the postural instability theory; research on motion sickness typically involves imposed motions of much greater amplitude [12,18,28,50]. This effect should motivate further research on relations between postural control and motion sickness, including their temporal sequence. The appearance of postural instability before motion sickness has not been predicted by any theory of motion sickness that is based on the concept of sensory conflict [40–42]: The generation of sensory conflict is neutral with respect to whether the person is stable or unstable [44].

A particularly striking finding was that in both experiments, differences in the postural motion of Sick and Well groups existed before subjects were exposed to any experimental motion (i.e., during spontaneous, unperturbed sway). This finding, replicated in two experiments with small sample sizes, suggests general differences in postural control between persons who are susceptible to motion sickness and those who are not. This could be evaluated by examining the spontaneous postural sway of subjects before being exposed to a wide variety of nauseogenic situations, such as vehicles and vehicular simulations.

In both experiments there were significant increases in lateral sway (in the sum-of-sines trials) for subjects who later developed motion sickness. This is interesting because there was no imposed stimulation in the lateral axis. This result suggests that postural instability was not confined to the axis in which the destabilizing stimulus was presented. It appears that the instability propagated beyond this axis, so that it affected other—nominally unrelated—aspects of postural control. This is consistent with the idea of wave interference (which can occur outside the axis of wavemotion) and the more general idea of instability in adaptive nonlinear systems [16,44]. In future research it may be important to examine the possibility that instabilities in motor control may exist in other areas of behavior. One possibility might be that visually induced instabilities in posture might lead to concurrent instabilities in the stabilization of the visual system, such as fixation and eye movements [44]. This might be related to common anecdotal reports that motion sickness in vehicles is exacerbated by reading.

In the present experiments, measurements of postural motion were predictors of motion sickness. This predictive power was based on after-the-fact statistical analysis. However, it might be possible to develop systems that could use these and other measurements of postural motion to predict motion sickness on-line in the field (e.g., in vehicles or simulators). This could be used to advise susceptible individuals to terminate their activity, thus avoiding sickness. In the case of vehicular simulations, sensitivity to impending sickness might be used to modify the motion characteristics of the simulation (e.g., to suppress motion in the nauseogenic frequency range), so as to prevent sickness. Systems of these types might also be useful for the control of motion sickness in orbital flight, in which there are no imposed motions.

We have studied instability in standing posture, but the postural instability theory is not limited to stance. Instability can develop in the control of any part of the body. In many nauseogenic situations, the victims of motion sickness are seated. In such cases they may develop instabilities in controlling the posture of the head and neck. This prediction should be tested.

The postural instability theory assumes that postural control is an act carried out by the subject. An important prediction of the theory is that motion sickness should not occur when the subject is incapable of controlling body posture. One situation of this type occurs when the body is passively stable, that is, when it is restrained. If passive restraint were complete (e.g., if a person were attached to a litter with straps around the torso, limbs, neck and head), we would predict that stimuli of the type used in the present experiments would not produce motion sickness.

We noted earlier that there is not yet a clear definition for stability in adaptive nonlinear systems. It was, in part, for this reason that Riccio and Stoffregen [44] suggested a variety of possible metrics for evaluating postural motion in the context of motion sickness. Future studies should examine a wider range of metrics. This may provide more rigorous operational definitions for the postural instability theory of motion sickness and may aid in the broader program of defining stability and instability in general.

It might be argued that motion sickness began before the appearance of symptoms that reached the level of subjective awareness. If so, then these preconsciousness manifestations of motion sickness might have occurred simultaneous with or even before the onset of postural instability. This possibility would require the existence of reliable nonconscious precursors of subjective symptoms. Attempts have been made to identify anatomical, physiological and/or neural precursors to the subjective symptoms of motion sickness [20]. To date, this effort has not lead to any widely accepted definition of motion sickness that reliably differentiates sick from well subjects. In particular, the predictive power of autonomic and hormonal measures has been poor [20]. In future research it might be interesting to correlate the development of postural instability with changes in Galvanic Skin Response (GSR), heart rate and blood pressure, which have been shown to be related to susceptibility [20].

Sources of Instability

We motivated the present experiments by appealing to the concept of wave interference. However, the postural instability theory is not limited to instabilities that may be related to wave interference. The theory does not claim that motion sickness is caused only by imposed motion at 0.1–0.3 Hz. The theory relates postural instability to motion sickness but does not make restrictive claims about how instability may be induced, that is, about the nature of the motions that induce instability. It would be useful, in future research, to examine relations between postural stability and a wide variety of different kinds of imposed motions, having different frequencies, amplitudes, durations, axes and so on. Among other things, experiments of this kind would help to clarify which kinds of postural motion precede motion sickness and which do not.

CONCLUSION

The postural instability theory [44] is not a modification of the sensory conflict or sensorimotor rearrangement theories but differs from them in fundamental ways. The new theory is developed from fundamental assumptions about perception and action that are incompatible with the epistemology of previous theories. For example, in the epistemological assumptions that underlay the postural instability theory sensory conflict may not exist at all [44]. This is because discrepancies between the stimulation of different perceptual systems (which do exist) do not necessarily need to be interpreted as sensory conflict [48].

In two experiments we found that motion sickness was preceded by statistically significant increases in several indices of postural sway. This effect has not been predicted by theories of motion sickness that rely on the concept of sensory conflict. It has been predicted by a new theory of motion sickness that makes no appeal to sensory conflict but relates motion sickness to degradation in the ability actively to control the postural motion of the body and its parts. The findings create new challenges for our understanding of motion sickness and offer new possibilities for the prediction and prevention of motion sickness.

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