

# Visually Induced Motion Sickness Predicted by Postural Instability

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We investigated whether postural instability can predict motion sickness and studied relations among instability, motion sickness, and vection. Nine men and 4 women (mean age = 19.85 years) were exposed, while standing, to an optical simulation of body sway. Head motion was recorded using a magnetic tracking system. Postural instabilities were observed prior to the onset of motion sickness. Vection was reported by most participants, including all who became ill. A discriminant analysis revealed that parameters of postural motion accurately predicted motion sickness. The results confirm that postural instability precedes motion sickness and suggest that measures of postural motion may serve as reliable predictors of motion sickness. Potential applications of this research include the development of on-line diagnostic tools that will allow for the prevention of motion sickness in operational and training settings.

## INTRODUCTION

Motion sickness is a common by-product of exposure to optical depictions of inertial motion. This phenomenon, called *visually induced motion sickness* (VIMS), has been reported in a variety of virtual environments, such as fixed-base flight and automobile simulation (Frank, Casali, & Wierwille, 1988; Regan & Price, 1994; Yoo, Lee, & Jones, 1997) and in a variety of nonvehicular simulations (DiZio & Lackner, 1992; Ellis, 1991). Improvements in simulation fidelity are associated with increases in the likelihood of sickness (Crowley, 1987; McGuinness, Bouwman, & Forbes, 1981; Miller & Goodson, 1960). The effectiveness of virtual environments and simulation systems, and their acceptance by users, can be reduced if they produce motion sickness (Biocca, 1992). This is true especially if sickness in simulations occurs in situations in which it does not occur in the simulated system. This problem provides a practical motivation for understanding visually induced motion sickness. Prevention of

visually induced motion sickness would be facilitated if objective measures could be developed to predict it and if the factors that cause it could be identified and eliminated.

Explanations of motion sickness typically have been grounded in the concept of sensory conflict (e.g., Oman, 1982; Reason, 1978; Reason & Brand, 1975). However, the sensory conflict theory of motion sickness has low predictive validity (Draper, Viirre, Gawron, & Furness, 2001; Stoffregen & Riccio, 1991), which reduces the extent to which this theory can guide the design of simulators and other virtual environments. The present study does not attempt to evaluate the sensory conflict theory of motion sickness; rather, one of our goals was to evaluate a new, alternative theory of motion sickness etiology.

## Postural Sway and Imposed Vibration

The occurrence of motion sickness is influenced by the frequency of imposed oscillation. In laboratory studies, motion sickness occurs in the presence of imposed periodic motion at

frequencies from 0.08 to 0.40 Hz (Guignard & McCauley, 1990; Lawther & Griffin, 1988). Motion at other frequencies produces little or no sickness, even with long exposure durations (Guignard & McCauley, 1990). These data are consistent with what is known about operational vehicles that are associated with motion sickness: Vibration or oscillation in this frequency range is characteristic of ships, trains, aircraft, and vehicular ride (Guignard & McCauley, 1990; Lawther & Griffin, 1988). The consistency of the laboratory and operational data might suggest that motion sickness is caused by motion in the 0.08- to 0.40-Hz range. However, the spectral power of normal standing sway is concentrated between 0.1 and 0.4 Hz (Bensel & Dzendolet, 1968), yet people are not sickened by their own postural sway. Thus it cannot be the case that vibration in this frequency range is inherently nauseogenic.

### Destabilization of Posture

Riccio and Stoffregen (1991) suggested that motion sickness results from instability in control of the posture of the body or its segments. They defined postural stability as “the state in which uncontrolled movements of the perception and action systems are minimized” (p. 202). This means that stability may be degraded rather than lost outright; there can be variation in the magnitude of instability, and instability can persist over long periods without necessarily leading to frank loss of control.

What could cause postural stability to be degraded? Stoffregen and Smart (1998) suggested that instability might occur when posture is controlled in the presence of imposed oscillations of a frequency between 0.08 and 0.40 Hz through a form of wave interference (Tipler, 1987). When independently generated waveforms interact, the resulting waveform is a function of the relative frequencies of the components. If two systems oscillate at very different frequencies, the resulting waves will pass through each other with little effect. However, when two systems oscillate at similar frequencies, the interaction of the waveforms can lead to dramatic instabilities. This particular outcome, often described as *destructive interference*, usually occurs when the waveforms are similar but out of phase (Tipler, 1991).

Stoffregen and Smart argued that imposed oscillations in the frequency range of spontaneous sway might destabilize the postural control system, leading to abnormal patterns of body sway. Such effects would not be expected when the imposed vibration is not in the frequency range of body sway. If it gave rise to motion sickness, waveform interference could explain why sickness is associated with imposed motion in the narrow band of frequencies that are spontaneously produced by postural sway.

The postural instability theory of motion sickness (Riccio & Stoffregen, 1991) predicts that postural instability should precede the onset of motion sickness symptoms. Stoffregen and Smart (1998) tested this prediction by exposing standing participants to a wide-field optical simulation of body sway. The amplitude and frequency of imposed optical flow resembled the amplitude and frequency of body sway during natural stance. As predicted, during exposure to the imposed optical flow, participants who later became motion sick exhibited increases in postural sway. Increases were observed in the variability, velocity, and range of postural motion. Similar effects were observed by Stoffregen, Hettinger, Haas, Roe, and Smart (2000) for seated participants.

### Motion Sickness and Vection

Optical simulations of self-motion often give rise to the subjective experience of self-motion relative to the inertial environment, which is referred to as *vection*. Vection is common in vehicular simulators, wide field-of-view cinemas (e.g., IMAX), and head-mounted visual display systems. Hettinger and Riccio (1992) suggested that vection is a necessary precursor for the occurrence of visually induced motion sickness. In the present study, one of our goals was to test this hypothesis in the context of motion sickness elicited by an optical simulation of standing body sway. This type of test is important because in ordinary life, body sway does not commonly give rise to the subjective experience of self-motion. In the laboratory, optical simulations that mimic the amplitude and frequency of body sway give rise to a subjective experience of self-motion in some persons but not in others (e.g.,

Stoffregen, 1985); that is, some but not all persons experience vection. Yet body sway is almost always strongly coupled to the imposed optical oscillations, whether or not a person experiences vection.

The vection and sickness data lead to a simple question: Does motion sickness in this situation occur solely in participants who have experienced vection? If so, this would provide support for the hypothesis of Hettinger and Riccio (1992). Consistent with the prediction of Hettinger & Riccio, Stoffregen and Smart (1998) found that all participants who reported motion sickness also reported vection. However, their vection data were qualitative and were obtained only after termination of the experimental stimulus (see Lishman & Lee, 1973; Stoffregen, 1985). In the present study we obtained quantitative data about number and duration of vection episodes during exposure to imposed optical flow.

### **Quantitative Prediction of Motion Sickness**

Several factors make it difficult to predict the occurrence of motion sickness in a given individual in a given situation. Susceptibility to motion sickness varies from person to person, and within individuals it varies across situations and experience levels (Benson, 1984; Calkins, Reschke, Kennedy, & Dunlop, 1987; Miller & Graybiel, 1972; von Baumgarten, 1986; Yardley, 1992). In addition, many symptoms of motion sickness are not unique; they occur with numerous other maladies, and this overlap can lead to confusion (Griffin, 1990). Another factor is the possibility that motion sickness may have multiple causes (Kennedy & Fowlkes, 1992). A particularly difficult issue for prediction is the role of experience. When people are exposed to a simulation of a real-world event, the incidence of motion sickness is greater among those who have prior experience with that real-world event (Kennedy, Hettinger, & Lilienthal, 1990). This seems to suggest that reliable prediction of motion sickness would require knowledge of the individual's prior experience (Stoffregen & Riccio, 1991).

There remains a need to identify objective measures that can predict motion sickness. Riccio and Stoffregen (1991) argued that a

reliable predictor of motion sickness could be found by observing changes in postural control that occur during exposure to nauseogenic stimuli. The focus on posture during exposure differs from studies that have measured posture only before and after exposure (e.g., Anderson, Reschke, Homick, & Werness, 1986; Cobb, 1999; Hamilton, Kantor, & Magee, 1989; Kennedy & Stanney, 1996). Previous research (Stoffregen et al., 2000; Stoffregen & Smart, 1998) has provided support for a key hypothesis of the postural instability theory: that postural instability precedes motion sickness. Stoffregen and Smart, and Stoffregen et al., evaluated this hypothesis only in a qualitative fashion – that is, they did not attempt to predict motion sickness on the basis of the quantitative details of presickness postural sway. In the present study we attempted quantitatively to identify parameters of postural motion that could predict motion sickness.

### **The Current Study**

The current study resembles the study of Stoffregen and Smart (1998) in that standing participants were exposed to an optical simulation of body sway. It differs from the earlier study in three ways. First, participants gave reports of vection during exposure to the nauseogenic stimulus. Second, we attempted to generate predictive models based on the significant parameters of postural motion. Third, our analysis of body sway data included more axes of motion than were analyzed by Stoffregen and Smart.

During exposure to optical flow, participants used a handheld device to indicate when they experienced vection. We measured the number and duration of vection episodes. In addition, participants' postural motion was measured. We hypothesized that postural instability would precede symptom onset (Stoffregen et al., 2000; Stoffregen & Smart, 1998). To permit this hypothesis to be evaluated, participants were familiarized with motion sickness symptoms before the experiment began and were explicitly instructed to cease their participation in the experiment at the onset of symptoms, no matter how mild. Several parameters of postural motion were measured. The primary measures were variability (operationally defined as the

standard deviation of head position), velocity, and range (operationally defined as the absolute difference between maximum and minimum position of head motion) for each of three axes of translation and rotation. Additional measures will be detailed in the following section.

## METHOD

### Participants

Nine male and 4 female undergraduates volunteered to take part in the experiment. Participants were drawn from the participant pool in the Department of Psychology at the University of Cincinnati and received course credit for participating. Participants ranged in age from 18 to 23 years ( $M = 19.85$ ), in weight from 45.45 to 84.09 kg ( $M = 72.38$  kg), and in height from 1.63 to 1.88 m ( $M = 1.75$  m). All participants reported that they were in good health; they also reported normal or corrected-to-normal vision and no history of dizziness, recurrent falls, or vestibular dysfunction. Each participant demonstrated that he or she could stand on one foot for 30 s with their eyes open. Participants were treated in accordance with American Psychological Association ethical standards at all times (American Psychological Association, 1992) and were aware of the fact that the experiment was designed to induce

motion sickness. When scheduling their participation, participants were instructed not to eat anything for 4 h prior to the experimental session. Compliance with this instruction was verified at the beginning of the session.

### Apparatus

Optical flow was generated using a moving room (Lee & Lishman, 1975; Stoffregen & Smart, 1998), an enclosure consisting of a cubical frame, 2.4 m on a side, mounted on wheels and moving in one axis along rails (Figure 1). The room was moved by an electric motor under computer control. At the center of the front wall was a large map of Ohio ( $96 \times 106$  cm,  $32^\circ \times 34^\circ$ ). Participants stood on the concrete laboratory floor such that there was no imposed inertial motion.

The room was driven using two functions (Figure 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 sine waves were adjusted so that the combined waveform had a maximum amplitude of 1.8 cm.

Data on postural motion were collected using an electromagnetic tracking system (Flock

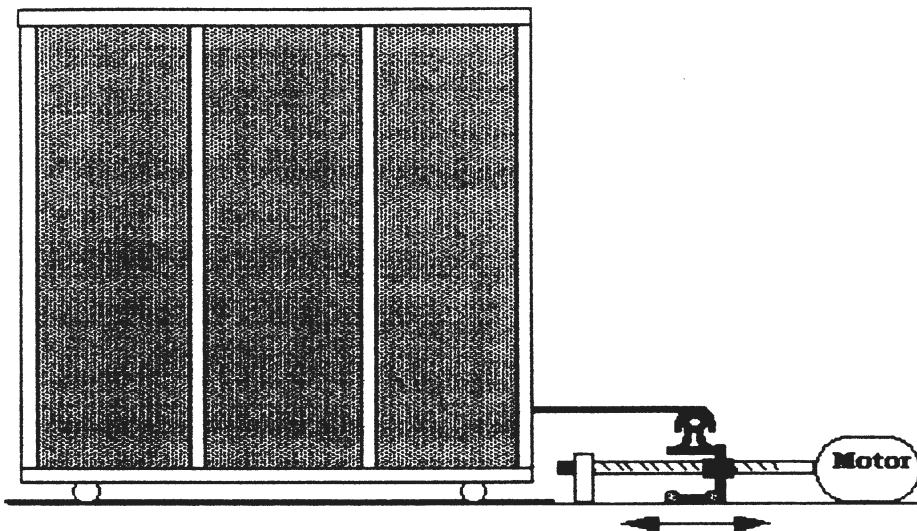


Figure 1. The moving room.

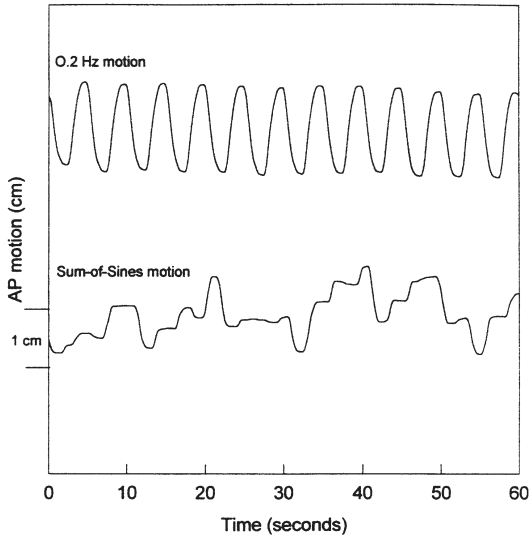


Figure 2. Motion functions of the moving room. The upper trace shows the 0.2-Hz motion. The lower trace shows a portion of the sum-of-sines motion. The sum-of-sines motion does not repeat over the 600-s period.

of Birds, Ascension Technologies, Inc., Burlington, VT). The transmitter was located on a stand behind the participant’s head. One receiver was attached to a bicycle helmet (weighing 0.34 kg) worn by the participant, and a second receiver was attached to the moving room. Six degree-of-freedom position/orientation data were collected from each receiver at 50 Hz and stored on disk for later analysis.

Participants were given a handheld button that sent binary signals to the computer and

was used to indicate vection experienced during the sum-of-sines trials.

**Procedure**

Prior to the experiment, participants completed a questionnaire on their motion sickness history. To assess their initial level of symptoms and to ensure that they were familiar with motion sickness symptoms, participants were asked to complete the simulator sickness questionnaire, or SSQ (Kennedy, Lane, Berbaum, & Lilienthal, 1993). Following Regan and Price (1994), these preexposure SSQ data were used to establish a baseline against which later SSQ data could be compared.

Participants 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 free hand (the one not holding the button) in their pocket and to not move their feet during trials. There was no single fixation point; participants were asked to keep their gaze on the map on the front wall and to minimize head movements while looking at the map.

The nature, number, and sequence of trials were the same as used in Stoffregen and Smart (1998) and are given in Table 1. During the sum-of-sines trials, participants held the button using their preferred hand, and they were instructed to press it whenever they experienced vection and to keep it depressed for as long as they experienced vection. *Vection* was defined as a feeling of self-movement, such as “the

**TABLE 1:** Sequence of Trials

Trial	Type of Motion	Duration
1	Spontaneous motion (eyes open)	20 s
2	Spontaneous motion (eyes closed)	20 s
3	Baseline motion 0.2 Hz (eyes open)	1 min
4	Baseline motion 0.2 Hz, (eyes closed)	1 min
5–8	Experimental motion 0.01–0.3 Hz	10 min
9	Spontaneous motion (eyes open)	20 s
10	Spontaneous motion (eyes closed)	20 s
11	Baseline motion 0.2 Hz (eyes open)	1 min

feeling you get when a car moves next to you and you mistake it for your own motion.” Verbal reports of perceived motion of the room and of the self were gathered at the end of each sum-of-sines trial. Participants were asked to describe any experience of motion that they had, and their verbatim reports were recorded. While in the moving room, participants were monitored continuously by an experimenter seated outside the door. This was for their safety as well as to ensure compliance with instructions.

Participants were warned that they might become ill, and they were instructed to discontinue the experiment immediately if they began to experience any noticeable symptoms of motion sickness. The time of discontinuation was recorded automatically. Following discontinuation or the completion of four sum-of-sines trials, participants were asked to fill out the SSQ a second time, after which those who felt well enough completed Trials 9 through 11. At the end of the session, participants 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 or not 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 (1985; see also Kennedy & Lilienthal, 1994) noted that symptom onset was sometimes delayed up to 1 h following termination of exposure to a moving room. It is for this reason that participants who may have been asymptomatic at the end of the experimental session were asked to report their subjective state over the following 24 h.

## RESULTS

### Motion Sickness History

Six of 13 participants (46%) reported being motion sick in the past; this included 40% of those who did not become sick in the present study (4/10) and 67% of those who did become sick (2/3). Sickness was reported in cars or boats, especially while reading. On a 0 to 10 scale, self-ratings of susceptibility to motion

had a mean of 2.7 ( $SD = 1.95$ ) for participants who did not report sickness in the present study and a mean rating of 5 ( $SD = 2.00$ ) for participants who reported sickness. These ratings did not differ,  $t(11) = 1.79, p > .05$ .

### Incidence of Sickness and Discontinuation

Participants were divided into *sick* and *well* groups, with the sick group containing all participants who became sick during the experiment or up to 24 h following the experiment. Participants were classified as sick by self-report. All 3 participants (23%) in the sick group were women. Participant JZ discontinued after completing Trial 7 (the third sum-of-sines trial). Participants MB and TB reported that symptoms occurred shortly after leaving the laboratory. As in previous experiments (Stoffregen et al., 2000; Stoffregen & Smart, 1998), reports of sickness were unambiguous (e.g., “I need to stop, I feel horrible”; “I felt really sick after leaving”). The remaining participants reported no symptoms and were placed in the well group.

As indicated, all of the participants who became motion sick in the present investigation were women. Gender has been implicated by some researchers as a factor that may influence susceptibility (e.g., Grunfeld & Gresty, 1998; Kennedy, Lanham, Massey, Drexler, & Lilienthal, 1995). However, no theory of motion sickness has predicted any gender effects involving postural instability. In addition, gender has not been found to be a factor in studies of postural control in the absence of motion sickness (e.g., Horak & Macpherson, 1996). Further, in the Stoffregen and Smart (1998) study, motion sickness was reported with approximately equal frequency by men (4) and women (5). Thus, although the occurrence of sickness solely in women is noteworthy, its significance is uncertain.

### SSQ Scores

Questionnaire scores for each participant were computed in the recommended manner (Kennedy et al., 1993). Because of the small sample size, only the total severity score was analyzed (Stoffregen & Smart, 1998). The mean pretest scores were 11.22 (sick:  $n = 3$ ) and 11.97 (well:  $n = 10$ ); these means did not differ,  $\chi^2(1) = 0.06, p > .05$ . The mean posttest scores

were 43.63 (sick) and 7.48 (well); these means differed significantly,  $\chi^2(1) = 6.27, p < .05$ .

### Vection

Twelve participants experienced vection during the sum-of-sines trials, including each of the 3 sick participants and 9 of the 10 well participants. The occurrence of vection in each member of the sick group is consistent with the hypothesis that vection is a necessary precursor for visually induced motion sickness (Hettinger & Riccio, 1992). For the sick group the mean number of vection episodes per trial was 14.4 ( $SD = 14.64$ ); for the well group the mean was 6.65 ( $SD = 8.82$ ). These means differed significantly,  $t(48) = 2.17, p < .05$ . For the sick group the mean duration of vection episodes was 67.46 s ( $SD = 118.23$ ), and for the well group the mean was 70.53 s ( $SD = 100.34$ ); these means did not differ,  $t(48) = 0.29, p > .05$ . All participants reported that they perceived the room to be moving at some point during the experiment.

### Postural Motion

Because of discontinuation, participant JZ did not complete Trials 9 through 11. This meant that for these trials postural data were collected from only 2 sick participants. For this reason, no analyses were performed on these trials. Because of an intermittent data acquisition problem, one sum-of-sines trial was not recorded for participant MB (Trial 7). No other data were affected. There was no corruption of any of the acquired data. All statistical analyses are based on those trials for which data were recorded.

Two types of analyses were applied to the data on postural motion: analysis of variance (ANOVA) and step-wise discriminant analysis (SDA). The purpose of the analyses of variance was to show that any effects of room motion on body sway were restricted to the visual consequence of room movements (i.e., optical flow rather than sound or other factors). For this reason, ANOVA was limited to comparison of eyes-open and eyes-closed trials (Trials 1–4). Separate two-factor mixed-model ANOVAs (vision: eyes open vs. eyes closed [within factor]; group: sick vs. well [between factor]) were performed for variability, veloci-

ty, and range in each axis of postural motion during the spontaneous sway and 0.2-Hz trials. Analyses were performed using SPSS version 8.0.0 for Windows (SPSS, Inc., Chicago, IL) using the general linear model procedure.

The analysis of primary interest was the SDA. This analysis (a form of regression) is used to determine which variables can be used to classify a given case (i.e., participant) into a particular group (in this case, sick vs. well). Like many traditional regression analyses, SDA yields an equation that maximizes the differences in a given variable (or variables) among groups based on maximum likelihood principles (Pedhazur, 1997). SDAs were performed only for trials on which the eyes were open. Separate analyses were performed for the spontaneous sway (Trial 1), 0.2-Hz motion (Trial 3), and sum-of-sines trials (Trials 5–8). Given that the goal was to identify postural parameters that predicted group membership, the criterion for entry into the analysis was that a variable must have accounted for a significant portion of unique variance. At each step, we selected the variable that minimized the sum of the unexplained variation for all pairs of groups. As part of the discriminant procedure, significance tests were performed on each variable individually to test for significant differences between sick and well groups; these tests are reported in the *Univariate results* section.

### Analysis of Variance

*Spontaneous sway (Trials 1 and 2).* Separate two-factor mixed-model ANOVAs (vision: eyes open vs. eyes closed [within factor]; group: sick vs. well [between factor]) were performed for variability, velocity, and range in each axis. Significant effects of vision were obtained in the anteroposterior (AP) axis for the variability, velocity, and range of motion:  $M_{Open} = 0.92$  cm,  $M_{Closed} = 1.17$  cm,  $F(1, 11) = 15.28, p < .05$ ;  $M_{Open} = 0.35$  cm/s,  $M_{Closed} = 0.63$  cm/s,  $F(1, 11) = 22.38, p < .05$ ; and  $M_{Open} = 1.67$  cm,  $M_{Closed} = 3.07$  cm,  $F(1, 11) = 11.38, p < .05$ , respectively. These results replicate the common finding that body sway is greater when the eyes are closed (e.g., Lee & Lishman, 1975). There were no significant effects of Group on any of the three variables. Representative data are plotted in Figure 3.

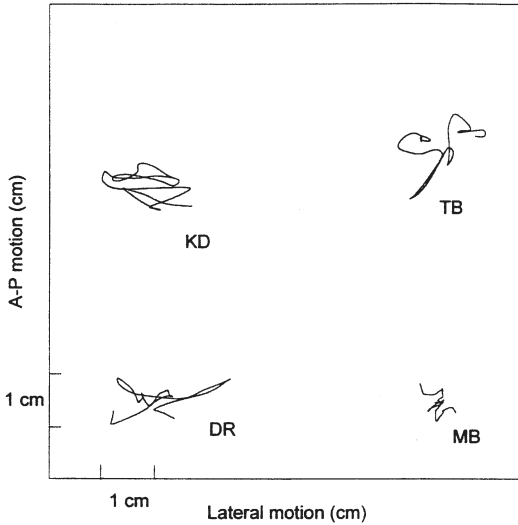


Figure 3. Representative spontaneous postural motion in the moving room (eyes open). Participants are identified by their initials. Those who *later* became sick are on the right.

*0.2-Hz motion (Trials 3 and 4).* Variability, velocity, and range were analyzed for these trials. In addition, we analyzed the coupling of body and room motion in terms of their cross-correlation, phase, and gain; this is common in research on relations between vision and posture (e.g., Warren, Kay, & Yilmaz, 1996). *Phase* is a measure that determines the amount of temporal coupling between the room and participant's motion, whereas *gain* is a measure of the relative amplitude of the response of the participant with respect to the stimulus. Separate, two-factor mixed-model ANOVAs (vision = within factor, group = between factor) were performed for each variable in each axis of motion with the exception of cross-correlation, phase, and gain, which were calculated only in the axis of stimulation (AP).

*Variability.* The main effects of vision and group on sway variability were not significant in any axis. In the roll axis there was a significant interaction between group and vision,  $F(1, 11) = 5.03, p < .05$ ; participants who would later become sick exhibited greater variability in the eyes-closed trial (Trial 4:  $M_{\text{Sick}} = 9.83^\circ, M_{\text{Well}} = 1.41^\circ$ ).

*Velocity.* There were no main effects of vision on the velocity of body sway. There was a significant effect of group on the velocity of

body sway in the lateral axis, vertical axis, and pitch axis:  $M_{\text{Sick}} = 0.29 \text{ cm/s}, M_{\text{Well}} = 0.14 \text{ cm/s}, F(1, 11) = 8.10, p < .05$ ;  $M_{\text{Sick}} = 0.15 \text{ cm/s}, M_{\text{Well}} = 0.08 \text{ cm/s}, F(1, 11) = 6.70, p < .05$ ; and  $M_{\text{Sick}} = 0.46^\circ/\text{s}, M_{\text{Well}} = 0.25^\circ/\text{s}, F(1, 11) = 8.80, p < .05$ , respectively. In each case velocity was greater in the sick group. There was a significant interaction between group and vision in the roll axis,  $F(1, 11) = 5.60, p < .05$ , with the sick group exhibiting greater velocity during the eyes-closed trial (Trial 4:  $M_{\text{Sick}} = 2.66^\circ/\text{s}, M_{\text{Well}} = 0.72^\circ/\text{s}$ ).

*Range.* A significant effect of group on the range of body sway was obtained in the lateral axis,  $M_{\text{Sick}} = 1.59 \text{ cm}, M_{\text{Well}} = 1.30 \text{ cm}, F(1, 11) = 7.80, p < .05$ , with the sick group exhibiting greater range of motion. In the vertical axis there was a significant main effect of vision,  $M_{\text{Open}} = 0.82 \text{ cm}, M_{\text{Closed}} = 1.14 \text{ cm}, F(1, 11) = 7.30, p < .05$ , with greater range when the eyes were closed. Significant Group  $\times$  Vision interactions were found in the vertical axis,  $F(1, 11) = 9.10, p < .05$ , the yaw axis,  $F(1, 11) = 6.10, p < .05$ , and the roll axis,  $F(1, 11) = 5.90, p < .05$ . In each case the well group exhibited greater range of motion when the eyes were open whereas the sick group exhibited greater range of motion when the eyes were closed. Representative data are plotted in Figure 4.

*Measures of coupling.* Cross-correlations were standardized ( $z$  transformed) for analysis. Cross-correlations were greater with the eyes open ( $M_{\text{Open}} = 0.14, SD = 0.23$ ) than with the eyes closed ( $M_{\text{Closed}} = -0.01, SD = 0.13$ ),  $F(1, 11) = 8.22, p < .05$ , indicating that body sway was influenced by the imposed optical flow. The sick and well groups did not differ, and there was no significant Group  $\times$  Vision interaction. Gain was greater with eyes open,  $M_{\text{Open}} = 0.47, M_{\text{Closed}} = 0.18, F(1, 11) = 14.74, p < .05$ . Gain was also greater for the sick group,  $M_{\text{Well}} = 0.20, M_{\text{Sick}} = 0.45, F(1, 11) = 6.05, p < .05$ . Phase was analyzed using circular statistics (Batschelet, 1981). With circular statistics, variance can be analyzed for only a single factor in each test (i.e., it is not possible to test for interactions). Accordingly, we used the Williams-Watson test, which is the circular analogue to a one-way ANOVA. Separate analyses were conducted to test for group differences in each

visual condition (eyes open and eyes closed) and for a general effect of vision. Differences between sick and well groups were not significant for either the eyes-open or eyes-closed condition. There was a general effect of vision on phase in that phase lag was decreased in the eyes-open trial,  $M_{Open} = 15^\circ$  ( $SD = 55.05^\circ$ ),  $M_{Closed} = 30.17^\circ$  ( $SD = 111.52^\circ$ ),  $F(1, 24) = 8.11$ ,  $p < .05$ .

Overall, the results of the 0.2-Hz trials replicate the common finding that body sway can be influenced by imposed optical flow (Lee & Lishman, 1975; Stoffregen, 1985; Stoffregen & Smart, 1998). When the eyes were open, gain, phase, and cross-correlation were each affected in the expected manner (i.e., relatively higher gain and cross-correlation and lower phase lag).

### DISCRIMINANT ANALYSIS

An important goal of the present study was to identify parameters of postural motion that can predict visually induced motion sickness. Because of the focus on vision, we excluded from the discriminant analysis Trials 2 and 4, in which the eyes were closed. Given the small sample size, it was important to limit the number of variables being tested. Prior to the analysis, all of the measures derived from the participants' motion were correlated. This was done in order to safeguard as much as possible against circularity (i.e., redundancy) in the analysis. It was expected that most variables would be at least moderately correlated with one another, as they are all derived from the same source and therefore cannot be truly independent. It was decided that variables that consistently exhibited strong correlations with other variables (i.e.,  $\geq .7$ ; Stevens, 1996) would be excluded from the analysis.

Based on these criteria, the range variable was excluded from the analysis as it correlated almost perfectly with variability (and to a slightly lesser degree with velocity). Further, motion in the yaw axis was omitted from the analysis. This was done because the yaw and roll axes were almost perfectly correlated in variability, velocity, and range (this is in part attributable to the structure of the cervical spine, which results in the coupling of motion

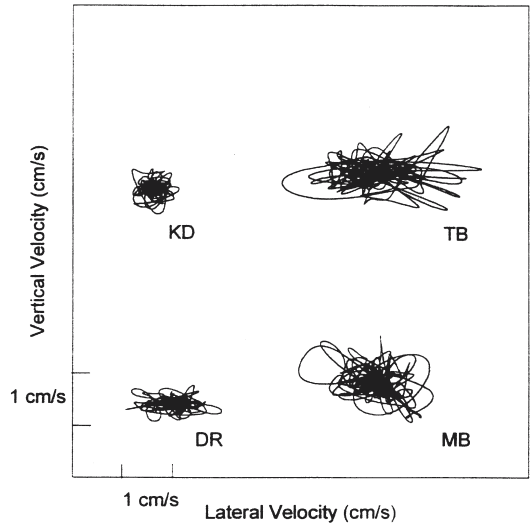


Figure 4. Velocity data for representative participants during the baseline (0.2-Hz) motion trial (eyes open). Participants who later became sick are on the right.

in these axes; White & Panjabi, 1990). The decision to keep the roll data rather than the yaw data was based on the lack of empirical evidence suggesting that motion in the yaw axis contributes to motion sickness (see Lawther & Griffin, 1987; Parker, 1998; Wertheim, Bos, & Bles, 1998). Although many of the remaining variables and axes exhibited moderate correlations with one another, it was felt that the discriminant analysis could be performed (though one should be cautious in trying to generalize beyond these data).

It was also necessary to determine the order in which variables were entered into the analysis. Variability in each remaining axis would be entered first, then velocity. The order of axes was AP, lateral, vertical, pitch, and roll. The choice of order was made in the following manner: The axes of translation were entered first, given that the stimulus was translational. Stoffregen and Smart (1998) found significant differences in the AP and lateral axes, so these axes were entered first. Vertical parameters were entered next based on the work by Lawther and Griffin (1987) as well as Guignard and McCauley (1990), which demonstrated a relation between the frequency of imposed motion and the incidence motion sickness, using vertical motion. Finally, research in sea

and space sickness (Parker, 1998; Wertheim et al., 1998) has suggested that motion in the pitch and roll axes can also contribute to motion sickness. Gain, phase, and cross-correlation data were entered last, as their relation to motion sickness has not been extensively studied. These precautions were taken because discriminant analysis is a form of regression procedure; however, it is not clear that these precautions affect the reliability of the analysis (Pedhazur, 1997; Stevens, 1996).

In the univariate and discriminant analyses to be reported, data were analyzed on the basis of individual trials rather than on the basis of participant means. This was done because instability usually develops over time; thus averaging across trials might mask emerging instability and hinder the ability to predict sickness.

### Spontaneous Sway and 0.2-Hz Trials

*Univariate results.* There were no significant effects in the analysis of sway in the eyes-open spontaneous sway trial (Trial 1). Significant group effects in the velocity of postural motion during the 0.2-Hz trial (Trial 3) occurred in the lateral, vertical, and pitch axes:  $F(1, 11) = 7.39, p < .05$ ;  $F(1, 11) = 7.71, p < .05$ ; and  $F(1, 11) = 16.38, p < .05$ , respectively. In each case, velocities were higher for the sick group. Gain was also significantly higher for the sick group,  $F(1, 11) = 5.96, p < .05$ .

*Discriminant results.* In the discriminant analysis, there were no significant effects in the analysis of sway in the eyes-open spontaneous sway trial (Trial 1). For the 0.2-Hz trial (Trial 3), the analysis yielded one significant discriminant function (only one is possible given that there were only two groups): Wilks's  $\lambda = 0.40$ ,  $\lambda^2(1) = 9.58, p < .05$ . Differences in pitch velocity classified participants into sick and well groups. This variable accounted for 60% of the variance. The resulting function was

$$y = 12.39 \times V_p - 3.68, \quad (1)$$

in which  $V_p$  is the mean velocity of pitch rotation for a given participant. The function accurately classified 12 participants (92%). The function was cross-validated by predicting group membership of a particular case based

on the function generated by using all the other cases, excluding the one being tested. Performing this analysis did not decrease the accuracy of the function.

Sickness was not predicted by any parameters of sway in the axis of stimulation (AP). However, pitch velocity exhibited a strong correlation with AP velocity ( $r = .77, p < .05$ ). Thus, although parameters of AP motion were not selected, a closely related axis was.

### Sum-of-Sines Trials

Our hypothesis was that postural instability would occur before the onset of motion sickness (Riccio & Stoffregen, 1991). Thus it was essential to analyze only those data that were collected before the onset of motion sickness symptoms. To ensure the satisfaction of this requirement, we analyzed only trials that were completed prior to reports of sickness. Representative trials are depicted in Figure 5.

*Univariate results.* Significant group effects in the variability and velocity of postural motion during the sum-of-sines trials were revealed. Sway velocity was greater for the sick group in the AP axis, the vertical axis, and the pitch axis:  $M_{\text{Sick}} = 0.91 \text{ cm/s}, M_{\text{Well}} = 0.61 \text{ cm/s}, F(1, 48) = 10.25, p < .05$ ;  $M_{\text{Sick}} = 0.31 \text{ cm/s}, M_{\text{Well}} = 0.17 \text{ cm/s}, F(1, 48) = 8.16, p < .05$ ; and  $M_{\text{Sick}}$

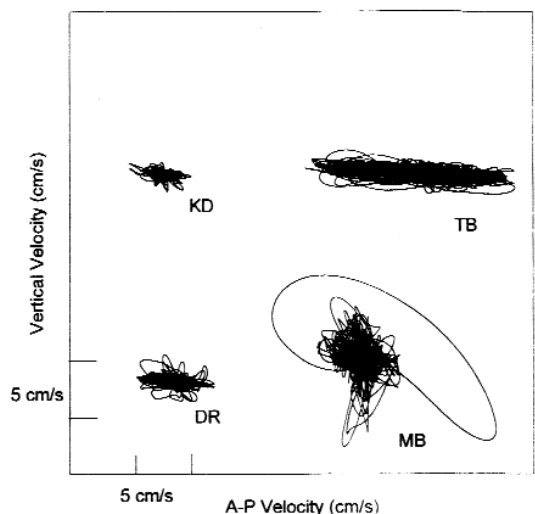


Figure 5. Velocity data for representative participants during the experimental (sum-of-sines) motion trial (eyes open). Participants who became sick are on the right.

= 0.66°/s,  $M_{\text{Well}} = 0.44^\circ/\text{s}$ ,  $F(1, 48) = 6.07$ ,  $p < .05$ , respectively. Sway variability was greater for the sick group in the vertical axis and in the pitch axis:  $M_{\text{Sick}} = 1.20$  cm,  $M_{\text{Well}} = 0.67$  cm,  $F(1, 48) = 21.23$ ,  $p < .05$ ; and  $M_{\text{Sick}} = 2.78^\circ$ ,  $M_{\text{Well}} = 1.63^\circ$ ,  $F(1, 48) = 19.53$ ,  $p < .05$ , respectively. There were no other significant results.

*Discriminant results.* The analysis yielded one significant discriminant function, Wilks's  $\lambda = 0.69$ ,  $\chi^2(1) = 17.40$ ,  $p < .05$ . Differences in vertical variability classified participants into sick and well groups. This variable accounted for 31% of the variance in the data. The resulting function was

$$y = 3.06 \times V_V - 2.63, \quad (2)$$

in which  $V_V$  is the mean variability in the vertical axis for a given participant. The function accurately classified 80% of the cases. Seven of the 40 well trials and 3 of the 10 sick trials were incorrectly classified (well trials and sick trials are the total number of trials completed by participants in the well and sick groups, respectively). The cross-validation utilized was the "leave one out" method described previously. Performing this test did not affect the accuracy of the classification. Interestingly, the incorrectly classified trials occurred later in the experimental sequence for the well group (Trial 7 or 8) and earlier for the sick group (Trial 5 or 6).

As in the 0.2-Hz trials, sickness was not predicted by parameters of sway in the axis of stimulation (AP). However, vertical variability and AP variability were correlated ( $r = .53$ ,  $p < .05$ ). Vertical variability also accounted for a larger proportion of the variance (40%) than did AP velocity (11%) causing it to be selected by the analysis. Similar to the 0.2-Hz trials, whereas motion in the axis of stimulation did not significantly predict sickness, a closely related axis did. The existence of significant destabilization outside the axis of stimulation is consistent the findings of Stoffregen and Smart (1998) and Stoffregen et al. (2000). The consistency of this finding may suggest a general phenomenon worthy of direct study: How is it that stimulation in one axis can cause instability in another axis in addition to or instead of the axis of stimulation?

## DISCUSSION

In this experiment, motion sickness was produced by exposure to low-frequency, low-amplitude optical flow that closely resembled the optical flow created by ordinary body sway. Vection was experienced by each of the sick participants, and vection episodes were more common in the sick group. Motion sickness was preceded by significant changes in postural motion. Prior to the onset of subjective motion sickness symptoms, increases in body sway were observed in the axis of stimulation and in other axes. Among participants who later became sick, increases in body sway were observed in the sum-of-sines trials. However, the sick group also exhibited postural instability in earlier trials involving simple sinusoidal motion. These postural sway findings replicate earlier studies (Stoffregen et al., 2000; Stoffregen & Smart, 1998). In the present study there were no differences in sway between the sick and well groups during spontaneous body sway (i.e., sway in the absence of imposed visual motion; Trials 1 and 2). Finally, the discriminant analysis identified parameters of body sway that reliably predicted the subsequent onset of motion sickness. These results are discussed in the remainder of this paper.

### Motion Sickness and Vection

Each of the participants who became sick reported experiencing some vection. However, all but one member of the well group also reported vection. The fact that each person who reported motion sickness also reported vection is consistent with the hypothesis that vection is a necessary precursor of visually induced motion sickness. However, the fact that vection was experienced by nearly all of the well participants indicates that vection is a weak predictor of sickness. We also found that participants who became sick reported more episodes of vection. Given that the mean duration of vection episodes did not differ for the sick and well groups, this finding means that the sick group experienced a greater overall duration of vection than did the well group.

Our results raise the issue of a link between vection and body sway. Such a link has been studied directly by Kuno, Kawakita, Kawakami,

Miyake, and Watanabe (1999), who measured postural motion while participants viewed a sinusoidally moving random-dot pattern via a head-mounted display (a sum of 10 sine waves). Participants used a joystick to indicate the experience of vection. Increases in sway were associated with increased experiences of vection, leading Kuno et al. to conclude that vection could be estimated empirically by examining postural motion. The fact that increased sway predicted vection was consistent with our findings. However, our results suggest strongly that the relation between postural instability and sway is asymmetrical: Body sway predicts vection (and motion sickness), but vection does not predict sway (or motion sickness).

### **Postural Instability Precedes Motion Sickness**

In this study, motion sickness was preceded by increases in several parameters of postural motion. This was true for body sway during exposure to the nominally nauseogenic sum-of-sines stimulus and for body sway during exposure to the nominally innocuous 0.2-Hz stimulus.

Stoffregen and Smart (1998) found that motion sickness was preceded by increases in the variability, range, and velocity of postural motion. Stoffregen et al. (2000) found that motion sickness was preceded by significant increases in the same parameters of body sway. In the present study we again found that motion sickness was preceded by significant increases in each of these parameters. Across these three studies, there has been some variation in the exact combination of parameters, trials, and axes of body motion in which effects have been observed. However, there has been considerable consistency in the overall pattern of results: Across the three studies, motion sickness has been reliably preceded by significant increases in objective, measurable properties of postural motion. This general finding confirms the central prediction of the postural instability theory of motion sickness (Riccio & Stoffregen, 1991). The fact that the precise pattern of significant effects has not been replicated exactly is not a problem for the postural instability theory, as Riccio and Stoffregen sug-

gested that instability might occur in any of a wide variety of parameters of postural motion.

With repeated replications of the general prediction it will become appropriate to expand the range of variables that are evaluated for instability that may predict motion sickness. For example, future research might evaluate the hypothesis that motion sickness might be preceded by instability in the coherence of body sway or in the relative phase of postural motion around the hip and ankle joints (Riccio & Stoffregen).

### **Postural Instability Predicts Motion Sickness**

In the 0.2-Hz trials, the discriminant analysis revealed that differences in pitch velocity classified participants into sick and well groups with 92% accuracy (i.e., 92% of the motion trials were identified correctly as belonging to a sick or well participant). In the sum-of-sines trials, the discriminant analysis revealed that differences in vertical variability classified participants into sick and well groups with 80% accuracy.

These findings show that differences in postural motion that exist prior to motion sickness can be used to predict who will get sick. This prediction can be done prospectively, rather than in a post hoc, retrospective manner. For example, using the equation generated by the discriminant analysis for the 0.2-Hz trials, a person exhibiting a pitch velocity of 0.2°/s would have a discriminant score of  $-1.2$ . This score could be compared with the group centroids (mean discriminant scores for each group: well =  $-0.62$ , sick =  $2.05$ ) to determine which group this person most closely resembled (this would be done by calculating the distance between the person's score and the centroids). The shortest distance suggests the group to which the person most likely belongs; in this case, the person would be classified as belonging to the well group (with a probability of 99%, given the person's discriminant score). The functions generated by the SDA accounted for more variance than did parameters that have typically been used to predict motion sickness, such as perceptual style, motion sickness history, and physiological activity (Kennedy, Dunlap, & Fowlkes, 1990; see also Stanney, Kennedy, Drexler, & Harm, 1999).

In the 0.2-Hz trials pitch velocity predicted sickness, whereas in the sum-of-sines trials sickness was predicted by vertical variability. It is interesting that neither of these predictors was in the axis of stimulation (AP). However, it is the case that the predictive variables are related to the axis of stimulation. Vertical motion occurs along a different axis than does AP motion, but jointly these axes define the sagittal plane of motion (Tortora & Grabowski, 1993). Pitch movement comprises motion in both the AP and vertical axes and therefore occurs in the sagittal plane as well. The ability of vertical motion to predict sickness is plausible, given that vertical (or heave) motion has been often implicated in studies of seasickness (e.g., Lawther & Griffin, 1987, 1988). Statistically, the predictive power of postural motion in the vertical axis is not surprising, given that variability in the AP and vertical axes was correlated.

Our analysis yielded a straightforward means of predicting sickness. However, our analysis has limitations that should be addressed with future research. First, it is unknown whether the present analysis can be applied to inertially induced motion sickness (e.g. sickness in cars, boats, or planes). Second, at present the predictions generated from this analysis do not address temporal issues; the models predict who will become sick but not when. Third, although postural instability has been found to precede motion sickness in both standing and seated participants and in both moving-room and flight-simulator environments (Stoffregen et al., 2000; Stoffregen & Smart, 1998), additional research is needed to determine the generalizability of our analysis across technologies and tasks. Fourth, the functions generated in this study were obtained with small sample sizes (13 in the study and 3 in the sick group).

It should be noted that quantitative studies of postural motion often utilize small sample sizes (e.g., Dijkstra, Schöner, & Gielen, 1994), in part because of the relatively high reliability of postural measures (increasing the sample size in these studies can increase statistical power but not substantive meaning). The amount of raw data collected from each participant was large (on the order of 120 000 data points), and the measures used in the analysis were derived from this abundant raw data.

Despite this, the issue of sample sizes remains real and suggests that caution should be used in generalizing the models beyond the current study. Additional data and analyses are needed in order to determine the extent to which the current results may be general. Replication will also aid in understanding the gender bias that we observed.

## CONCLUSION AND DESIGN IMPLICATIONS

The present study confirms the finding of Stoffregen and Smart (1998) and Stoffregen et al. (2000) that postural instability precedes visually induced motion sickness. Also in replication, this was found to be true not only for postural motion during exposure to nauseogenic stimuli but also for postural motion during exposure to innocuous visual motion. In addition, the current findings suggest that it is possible to use postural measures to predict future occurrences of motion sickness. If true, these measures could potentially be used to identify persons who are likely to be susceptible to visually induced motion sickness, allowing for proactive measures to be taken to prevent motion sickness.

Our findings suggest that it may be possible to use real-time data about postural motion to identify individuals at risk for motion sickness (Stoffregen et al., 2000). Such real-time measures of postural motion could be used to exclude such individuals from exposure to nauseogenic stimuli. In principle, such measures could also be used to make adjustments to simulator and virtual environment dynamics that could suppress postural instability and, in turn, prevent motion sickness. For example, display dynamics might be altered so as to prevent or suppress waveform interference. The practical utility of this strategy will be determined by further research.

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## REFERENCES

- American Psychological Association. (1992). Ethical principles of psychologists and code of conduct. *American Psychologist*, *47*, 1597–1611.
- Anderson, D. J., Reschke, M. F., Homick, J. E., & Werness, S. A. (1986). Dynamic posture analysis of Spacelab-1 crew members. *Experimental Brain Research*, *64*, 380–391.
- Batschelet, E. (1981). *Circular statistics in biology*. New York: Academic.
- Bensel, C. K., & Dzenolet, E. (1968). Power spectral density analysis of the standing sway of males. *Perception and Psychophysics*, *4*, 285–288.
- Benson, A. J. (1984). Motion sickness. In M. R. Dix & J. D. Hood (Eds.), *Vertigo* (pp. 391–426). New York: Wiley.
- Biocca, F. (1992). Will simulator sickness slow down the diffusion of virtual environment technology? *Presence: Teleoperators and Virtual Environments*, *1*, 334–345.
- Calkins, D. S., Reschke, M. F., Kennedy, R. S., & Dunlop, W. P. (1987). Reliability of provocative test of motion sickness susceptibility. *Aviation, Space, and Environmental Medicine* *58*(Suppl. 9), 50–54.
- Cobb, S. V. G. (1999). Measurement of postural instability before and after immersion in a virtual environment. *Applied Ergonomics*, *30*, 79–90.
- Crowley, J. S. (1987). Simulator sickness: A problem for army aviation. *Aviation, Space, and Environmental Medicine*, *58*, 355–357.
- Dijkstra, T. M. H., Schöner, G., & Gielen, C. C. A. M. (1994). Temporal stability of the action-perception cycle for postural control in a moving visual environment. *Experimental Brain Research*, *97*, 477–486.
- DiZio, P., & Lackner, J. R. (1992). Spatial orientation, adaptation, and motion sickness in real and virtual environments. *Presence: Teleoperators and Virtual Environments*, *1*, 319–328.
- Draper, M. H., Viirre, E. S., Gawron, V. J., & Furness, T. A. (2001). The effects of virtual image scale and system delay on simulator sickness within head-coupled virtual environments. *Human Factors*, *43*, 129–146.
- Ellis, S. R. (1991). Nature and origins of virtual environments: A bibliographic essay. *Computing Systems in Engineering*, *2*, 321–347.
- Frank, L. H., Casali, J. G., & Wierwille, W. W. (1988). Effects of visual display and motion system delays on operator performance and uneasiness in a driving simulator. *Human Factors*, *30*, 201–217.
- Griffin, M. J. (1990). *Handbook of human vibration*. London: Academic.
- Grunfeld, E., & Gresty, M. A. (1998). Relationship between motion sickness, migraine and menstruation in crew members of a "round the world" yacht race. *Brain Research Bulletin*, *47*, 433–436.
- Guignard, J. C., & McCauley, M. E. (1990). The accelerative stimulus for motion sickness. In G. H. Crampton (Ed.), *Motion and space sickness* (pp. 123–152). Boca Raton, FL: CRC.
- Hamilton, K. M., Kantor, L., & Magee, L. E. (1989). Limitations of postural equilibrium tests for examining simulator sickness. *Aviation, Space, and Environmental Medicine*, *59*, 246–251.
- Hettinger, L. J., & Riccio, G. E. (1992). Visually induced motion sickness in virtual environments. *Presence: Teleoperators and Virtual Environments*, *1*, 306–310.
- Horak, F. B., & Macpherson, J. M. (1996). Postural orientation and equilibrium. In L. B. Rowell & J. T. Shepherd (Eds.), *Handbook of physiology* (pp. 255–292). New York: Oxford University Press.
- Kennedy, R. S., Dunlap, W. P., & Fowlkes, J. E. (1990). Prediction of motion sickness susceptibility. In G. H. Crampton (Ed.), *Motion and space sickness* (pp. 179–216). Boca Raton, FL: CRC.
- Kennedy, R. S., & Fowlkes, J. E. (1992). Simulator sickness is polygenic and polysymptomatic: Implications for research. *International Journal of Aviation Psychology*, *2*, 23–38.
- Kennedy, R. S., Hettinger, L. J., & Lilienthal, M. G. (1990). Simulator sickness. In G. H. Crampton (Ed.), *Motion and space sickness* (pp. 317–342). Boca Raton, FL: CRC.
- Kennedy, R. S., Lane, N. E., Berbaum, K. S., & Lilienthal, M. G. (1993). Simulator sickness questionnaire: An enhanced method for quantifying simulator sickness. *International Journal of Aviation Psychology*, *3*, 203–220.
- Kennedy, R. S., Lanham, S. D., Massey, C. J., Drexler, J. M., & Lilienthal, M. G. (1995). Gender differences in simulator sickness incidence: Implications for military virtual reality systems. *Safe Journal*, *25*, 69–76.
- Kennedy, R. S., & Lilienthal, M. G. (1994). Measurement and control of motion sickness aftereffects from immersion in virtual reality. In *Proceedings of "Virtual reality and medicine: The cutting edge"* (pp. 111–119). New York: SIG – Advanced Applications.
- Kennedy, R. S., & Stanney, K. M. (1996). Postural instability induced by virtual reality exposure: Development of a certification protocol. *International Journal of Human-Computer Interaction*, *8*, 25–47.
- Kuno, S., Kawakita, T., Kawakami, O., Miyake, Y., & Watanabe, S. (1999). Postural adjustment response to depth direction moving patterns produced by virtual reality graphics. *Japanese Journal of Physiology*, *49*, 417–424.
- Lawther, A., & Griffin, M. J. (1987). Prediction of the incidence of motion sickness from the magnitude, frequency, and duration of vertical oscillation. *Journal of the Acoustical Society of America*, *82*, 957–966.
- Lawther, A., & Griffin, M. J. (1988). Motion sickness and motion characteristics of vessels at sea. *Ergonomics*, *31*, 1373–1394.
- Lee, D. N., & Lishman, J. R. (1975). Visual proprioceptive control of stance. *Journal of Human Movement Studies*, *1*, 87–95.
- Lishman, J. R., & Lee, D. N. (1973). The autonomy of visual kinesthesia. *Perception*, *2*, 287–294.
- McGuinness, J., Bouwman, J. H., & Forbes, J. M. (1981). *Simulator sickness occurrences in the 2E6 Air Combat Maneuvering Simulator (NAVTRAEQUIPCEN 80-C-0135-4500-1)*. Orlando, FL: Naval Training Equipment Center.
- Miller, J. W., & Goodson, J. E. (1960). Motion sickness in a helicopter simulator. *Aerospace Medicine*, *31*, 204–212.
- Miller, E. F., & Graybiel, A. (1972). Semicircular canals as a primary etiological factor in motion sickness. *Aerospace Medicine*, *43*, 1065–1074.
- Oman, C. M. (1982). A heuristic mathematical model for the dynamics of sensory conflict and motion sickness [Theoretical note]. *Acta Otolaryngologica*, *44*(Suppl. 392), 44.
- Parker, D. E. (1998). The relative roles of the otolith organs and semicircular canals in producing space motion sickness. *Journal of Vestibular Research*, *8*, 57–59.
- Pedhazur, E. J. (1997). *Multiple regression in behavioral research* (3rd ed.). Fort Worth, TX: Harcourt Brace College.
- Reason, J. T. (1978). Motion sickness adaptation: A neural mismatch model. *Proceedings of the Royal Society of Medicine*, *71*, 819–829.
- Reason, J. T., & Brand, J. J. (1975). *Motion sickness*. London: Academic.
- Regan, E. C., & Price, K. R. (1994). The frequency of occurrence and severity of side-effects of immersion virtual reality. *Aviation, Space, and Environmental Medicine*, *65*, 527–530.

- Riccio, G. E., & Stoffregen, T. A. (1991). An ecological theory of motion sickness and postural instability. *Ecological Psychology*, 3, 195–240.
- Stanney, K. M., Kennedy, R. S., Drexler, J. M., & Harm, D. L. (1999). Motion sickness and proprioceptive aftereffects following virtual environment exposure. *Applied Ergonomics*, 30, 27–38.
- Stevens, J. P. (1996). *Applied multivariate statistics for the social sciences* (3rd ed.). Mahwah, NJ: Erlbaum.
- Stoffregen, T. A. (1985). Flow structure versus retinal location in the optical control of stance. *Journal of Experimental Psychology: Human Perception and Performance*, 11, 554–565.
- Stoffregen, T. A., Hettinger, L. J., Haas, M. W., Roe, M., & Smart, L. J. (2000). Postural instability and motion sickness in a fixed-base flight simulator. *Human Factors*, 42, 458–469.
- Stoffregen, T. A., & Riccio, G. E. (1991). An ecological critique of the sensory conflict theory of motion sickness. *Ecological Psychology*, 3, 159–194.
- Stoffregen, T. A., & Smart, L. J. (1998). Postural instability precedes motion sickness. *Brain Research Bulletin*, 47, 437–448.
- Tipler, P. A. (1987). *College physics*. New York: Worth.
- Tipler, P. A. (1991). *Physics for scientists and engineers* (3rd ed.). New York: Worth.
- Tortora, G. J., & Grabowski, S. R. (1993). *Principles of anatomy and physiology* (7th ed.). New York: Harper-Collins.
- von Baumgarten, R. J. (1986). European vestibular experiments on the Spacelab-1 mission: 4. Overview. *Experimental Brain Research*, 64, 239–346.
- Warren, W. H., Kay, B., & Yilmaz, E. (1996). Visual control of posture during walking: Functional specificity. *Journal of Experimental Psychology: Human Perception and Performance*, 22, 818–858.
- Wertheim, A. H., Bos, J. E., & Bles, W. (1998). Contributions of roll and pitch to sea sickness. *Brain Research Bulletin*, 47, 517–524.
- White, A. A., & Panjabi, M. M. (1990). *Clinical biomechanics of the spine*. Philadelphia: Lippincott.
- Yardley, L. (1992). Motion sickness and perception: A reappraisal of the sensory conflict approach. *British Journal of Psychology*, 83, 449–471.
- Yoo, Y., Lee, G. C. H., & Jones, S. A. (1997). Vection, compensatory sway, and simulator sickness. In B. Dos & W. Karowski (Eds.), *Advances in occupational ergonomics and safety* (pp. 589–592). Amsterdam: IOS.

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