

# ‘Motion sickness preceded by unstable displacements of the center of pressure

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## Abstract

We exposed standing participants to optic flow in a moving room. Motion sickness was induced by motion that simulated the amplitude and frequency of standing sway. We identified instabilities in displacements of the center of pressure among participants who became sick; these instabilities occurred before the onset of subjective motion sickness symptoms. Postural differences between Sick and Well participants were observed before exposure to the nauseogenic stimulus. During exposure to the nauseogenic stimulus, sway increased for participants who became sick but also for those who did not. However, at every point during exposure sway was greater for participants who became motion sick. The results reveal that motion sickness is preceded by instabilities in displacements of the center of pressure.

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## 1. Introduction

Historically, motion sickness has plagued people who have been exposed to physical motion, such as occurs on ships. Recent decades have seen the advent of visually-induced motion sickness, in which nausea is induced by optical simulations of self-motion (e.g., Ellis, 1991; Lishman & Lee, 1973; Stoffregen, 1985). An area of particular concern is the simulation of vehicles (e.g., Frank, Casali, & Wierwille, 1988; Reagan & Price, 1994). A troubling feature of visually-induced motion sickness is that technological development appears to be making it worse: Improvements in simulation fidelity are associated with increases in the likelihood of sickness (Kennedy, Drexler, Compton, Stanney, & Harm, 2003; McGuiness, Bouwman, & Forbes, 1981; Miller & Goodson, 1960). This effect suggests that the understanding, prediction, and prevention of visually-induced motion sickness may not arise from improvements in technology, as such. These goals may be met through psychologically based theories of the interaction of simulation technologies with human behavior, that is, through theories of human–machine systems (e.g., Flach, Hancock, Caird, & Vicente, 1995). Such theories may aid in identifying behaviors that predict the incidence of motion sickness across various nauseogenic situations.

Theories of motion sickness typically have been derived from the concept of sensory conflict (e.g., Oman, 1982; Reason, 1978). Despite intense effort, theories based on the concept of sensory conflict have low predictive validity (Draper, Viirre, Gawron, & Furness, 2001; Stoffregen & Riccio, 1991) and so can offer little guidance in the design of simulators and other virtual environments. A second type of theory focuses directly on the behavioral interaction between the simulation and the user. The principal example is the postural instability theory of motion sickness (Riccio & Stoffregen, 1991). In this study, we did not directly contrast this theory with theories derived from the concept of sensory conflict. Rather, we pursued one of the main predictions made by the postural instability theory.

### 1.1. Destabilization of posture

The incidence of motion sickness is strongly related to the frequency of imposed periodic motion. Motion sickness is found almost exclusively when imposed periodic motion includes frequencies from 0.08 to 0.4 Hz (Guignard & McCauley, 1990). Vibration in this frequency range is characteristic of nauseogenic vehicles, such as ships, trains, and aircraft (Guignard & McCauley, 1990; Lawther & Griffin, 1986, 1987, 1988). Optical motion at these frequencies is sufficient to induce motion sickness in standing participants, even when the amplitude of oscillations is so small that many participants are not aware that anything is moving (Smart, Stoffregen, & Bardy, 2002; Stoffregen & Smart, 1998). These effects are peculiar because ordinary standing body sway is characterized by low amplitude oscillation between 0.1 and 0.3 Hz (Bensel & Dzendolet, 1968). We are not sickened by our own postural motion, but we can be sickened by a simulation of the optical consequences of body sway that is accurate in terms of frequency and amplitude. Why should this be so? We have hypothesized that the imposed optical simulation of body sway interacts with actual sway to produce unstable control of stance, through a process similar to destructive wave interference (Stoffregen & Smart, 1998). Our hypothesis suggests that unstable control of posture might be observed in persons who experience motion sickness while exposed to an accurate simulation of the optical consequences of body sway. The postural instability theory of motion sickness (Riccio & Stoffregen, 1991) predicts that postural

instability should precede the onset of motion sickness symptoms. This prediction has been confirmed by Stoffregen and Smart (1998) and Smart et al. (2002) in the context of stance, and by Stoffregen, Hettinger, Haas, Roe, and Smart (2000) for seated posture. During exposure to 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.

Duh, Parker, Philips, and Furness (2004) confirmed that postural instability is related to the frequency of imposed visual oscillation. Standing subjects were exposed to visual oscillation in the roll axis. Oscillation frequency varied between 0.05 and 0.8 Hz. Postural instability, measured in terms of motion of the center of pressure (COP), was inversely correlated with oscillation frequency: The greatest body sway was observed with 0.05 Hz visual oscillations. With respect to a possible causal role of postural instability in motion sickness, a limitation of this work is that Duh et al., did not attempt to use postural data to predict which subjects would become sick (cf. Smart et al., 2002): critically, postural motion and motion sickness were measured separately, in different experiments.

Our approach to motion sickness is centered on the stability of postural movements. In the context of animate movement, stability and instability are concepts of central importance. Formal definitions of these concepts have been offered. For example, in the context of theories of dynamic systems, stability can be defined with reference to properties of a limit cycle, a system's response to perturbations, hysteresis, and so on (Strogatz, 1993). We regard such criteria as being very important (e.g., Bardy, Marin, Stoffregen, & Bootsma, 1999; Bardy, Oullier, Bootsma, & Stoffregen, 2002) but we do not assume that they constitute absolute or universal definitions of stability for animate movement. As a practical matter, such definitions often require manipulations that do not fit comfortably with the phenomena of motion sickness (e.g., the introduction of a punctate perturbation) and require conditions on the data, such as stationarity and accurate determination of an embedding dimension (Abarbanel, 1996), that do not hold for postural sway (e.g., Riley, Balasubramaniam, & Turvey, 1999). Thus, we regard the definitions of stability and instability as being open questions, and we believe that our research relating motion sickness to postural movements may contribute to clarify the concept of postural instability.

### *1.2. The present study*

As presented by Riccio and Stoffregen (1991), the postural instability theory of motion sickness did not predict that instability would be found in or limited to any specific parameter of postural motion. Thus, it is important to look for signatures of instability in a variety of parameters of postural motion. This was the main purpose of the present study. We did this by conducting new types of analysis of postural motion data, and by collecting a different type of data. In our previous studies, data on postural motion were limited to displacements of the head and torso, as recorded using a magnetic tracking system (Smart et al., 2002; Stoffregen et al., 2000; Stoffregen & Smart, 1998). Stability and instability in postural control need not be limited to these displacements. Moreover, it cannot be assumed that there will be a 1:1 mapping between postural motions and the forces that underlie those motions (Bardy et al., 1999; Newell, van Emmerik, Lee, & Sprague, 1993; Riccio & Stoffregen, 1988). Kinetics and kinematics may be correlated under some conditions (e.g., in the laboratory), but under many normal circumstances relations between

these levels are equivocal and extremely complex. This is true not only with regard to postural control (Horak & Macpherson, 1996; Riccio & Stoffregen, 1988), but for movement, in general (Bernstein, 1967; Turvey, Fitch, & Tuller, 1982). Thus, the hypothesis that motion sickness may be preceded by instabilities in the kinetics of stance can be evaluated only by collecting data on these forces.

Our goals in the present study were (1) to determine whether differences in postural motion between Sick and Well participants previously identified in head and torso displacements would exist also in center of pressure data, (2) to examine new dependent variables that might reveal relations between postural stability and motion sickness and (3) to collect data on claustrophobia that will be relevant to future studies involving restraint.

We assessed the incidence and severity of motion sickness, and the incidence and severity of claustrophobia. We measured claustrophobia in part because our experimental apparatus is a small, enclosed space that might induce claustrophobia. However, our main motivation in assessing claustrophobia was to collect data that could be compared with similar data from future studies, in which we will assess both motion sickness and claustrophobia in participants who are physically restrained while in the moving room.

## 2. Method

### 2.1. Participants

Twenty-three students from the University of Minnesota volunteered to participate in this experiment, 9 males and 14 females ranging in age from 18 to 26 years with a mean age of 20 years. Participants ranged in weight from 45.81 kg to 87.09 kg with a mean of 65.07 kg, and in height from 1.55 m to 1.86 m with a mean of 1.70 m. All participants had normal or corrected to normal vision and reported no history of recurrent dizziness, recurrent falls, or vestibular (inner ear) dysfunction. All participants stated that they were in good health and were not pregnant. As part of the informed consent procedure, participants were informed that they could discontinue their participation at any time, for any reason, and that they would receive full credit for experimental participation regardless of whether they completed the experiment; there was, thus, no motivation for falsely stating that they were motion sick. When scheduling experimental sessions, participants were requested not to eat anything for 4 h before coming to the laboratory.

### 2.2. Apparatus

We generated optical flow using a moving room (Lee & Lishman, 1975; Smart et al., 2002), an enclosure consisting of a cubical frame, 2.44 m on a side, mounted on wheels and moving in one axis along rails (Fig. 1A). Motion of the room was produced by an electric motor under computer control. Rigid masonite sheets were attached to three sides and the top of the frame to create walls and a ceiling. The fourth (rear) side of the room was left open, providing access. The interior surfaces of the walls and ceiling were covered with blue and white marble-pattern adhesive paper. At the center of the front wall was placed a large, detailed map of the continental United States (53 × 80 cm; 19° × 28°). Illumination was provided by four incandescent floodlights mounted inside the room and oriented so that shadows were minimized. Participants stood on a force platform that rested on the concrete laboratory floor, such that there was no imposed inertial motion.

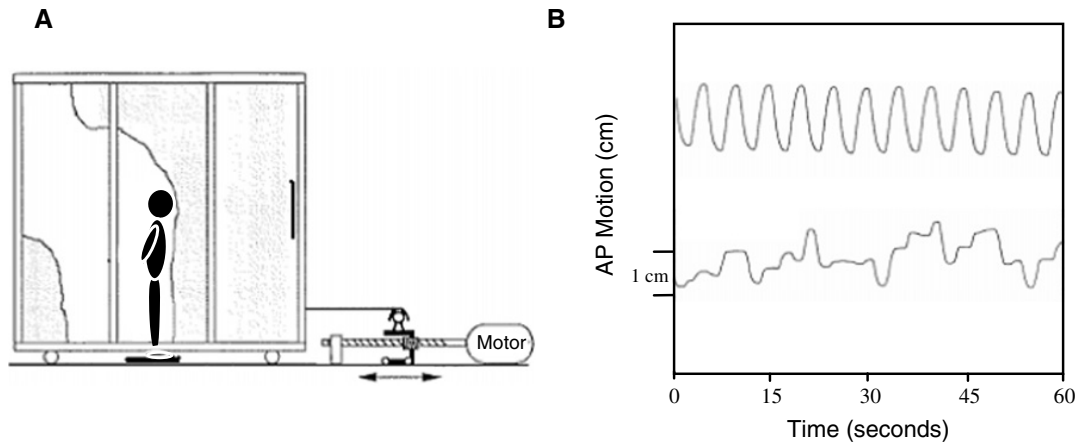


Fig. 1. (A) The moving room; (B) Motion functions used in the moving room. The upper trace shows the 0.2 Hz motion. The lower trace shows a portion of the sum-of-sines motion. The sum-of-sines function did not repeat but varied continuously over the 600 s trial duration.

Data on postural motion were collected using an AccuSwayPlus force platform (AMTI, Chicago). The position of the center of pressure was collected both in Antero-Posterior (AP) and Medio-Lateral (ML) axes at 50 Hz, and stored on disk for later analysis.

### 2.3. Procedure

To assess their current level of symptoms, and to ensure that they were familiar with motion sickness and claustrophobia symptomatology, participants were asked to complete the simulator sickness questionnaire, or SSQ (Kennedy, Lane, Berbaum, & Lilienthal, 1993), and the claustrophobia questionnaire, or CLQ (Radomsky, Rachman, Thordarson, McIsaac, & Teachman, 2001). We used the SSQ and CLQ to collect pre-exposure data, so as to establish a baseline against which post-exposure data could be compared (Reagan & Price, 1994; Smart et al., 2002).

The room was driven using two functions (Fig. 1B). One consisted of a simple, 0.2 Hz oscillation, with amplitude of 1.5 cm. The other was a sum of ten 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 amplitude of 1.5 cm. The phase and amplitude of the component sines were adjusted so that the combined wave form had maximum amplitude of 1.8 cm.

All participants successfully completed a pre-test in which they were asked to stand on one foot for 30 s. They then entered the moving room and stood on the force platform with their heels on a line marked on its surface. For the duration of each trial, they were asked to keep their hands in their pockets, or clasped behind or in front of them. They were free to change hand position across trials. Participants were asked not to move their feet during trials, but were not instructed to minimize postural motion, or to stand as still as possible. Participants who changed their hand position during trials, or who engaged in other obvious volitional adjustments, such as tossing their head or shrugging their shoulders, were excluded from our analyses, because such movements could not be reliably distinguished from postural motion in the center of pressure data. The decision to delete participants was immediate, that is, prior to the end of experimental sessions (and, therefore, prior to reports of motion sickness), though participants were permitted to complete the experimental session.

There was not a single fixation point; participants were asked to keep their gaze on the map on the front wall. 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. 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 (Smart et al., 2002; Stoffregen & Smart, 1998). In our earlier studies, Trials 1–4 were included as controls, that is, to document that participants exhibited normal sway (Trials 1 and 2) and that they exhibited normal coupling of body sway with imposed optic flow (Trials 3 and 4). However, our studies have revealed that sway during these trials (i.e., sway prior to any exposure to the nauseogenic stimulus) often differs for participants who later become sick, and those who do not. In the present study, we did not collect data about motion of the room, and so we could not evaluate hypotheses about coupling of postural activity to room motion. For these reasons, in the present study postural data from Trials 1–4 were analyzed primarily in the context of effects relating to motion sickness.

These pre-tests were followed by four trials, each 10 min (600 s) long, using the sum-of-sines stimulus. Following exposure to the sum-of-sines motion, Trials 1, 2, and 3 were repeated. This was intended to permit us to evaluate pre-post differences in spontaneous sway, and in responses to the simple, 0.2 Hz imposed flow. While they were in the moving room participants were monitored continuously by an experimenter seated outside. This was for their safety, and to ensure compliance with instructions.

Participants were warned that they might become ill, and were instructed to discontinue the experiment immediately if they began to experience any noticeable symptoms or fear. This warning was repeated after each of the sum-of-sines trials. Following discontinuation or the completion of four sum-of-sines trials participants were asked to fill out the SSQ and CLQ a second time, and to describe their symptoms. At the end of the session, participants who had not yet reported any symptoms were asked to report on their motion sickness and claustrophobia status over the next 24 h. They were asked to indicate on a yes/no basis, whether they developed motion sickness and/or claustrophobia, and to describe any symptoms. They were also given a printed copy of the SSQ and CLQ, which they were asked to fill out at the time of symptom onset, or after 24 h if no symptoms developed. Symptom onset is sometimes delayed up to an hour following termination of exposure to a moving room (Smart et al., 2002; Stoffregen, 1985; Stoffregen & Smart, 1998) or a flight simulator (Kennedy & Lilienthal, 1994).

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
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
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

## 2.4. Analysis of postural data

We conducted several analyses of postural motion before, during, and after exposure to the moving room. For all trials, we analyzed the positional variability of stance, as well as its velocity and range. The position of the COP in antero-posterior (AP) and medio-lateral (ML) axes was computed from the force and moment components measured by the force platform. The movements of the COP along the AP and ML axes were quantified by computing the variability (standard deviation of position), the range (difference between maximum and minimum positions), and the mean velocity for each trial.

We also conducted Detrended Fluctuation Analysis (DFA) on the time series of postural motion (Chen, Ivanov, Hu, & Stanley, 2002; Hausdorff, Peng, Ladin, Wei, & Goldberger, 1995; Hu, Ivanov, Chen, Carpena, & Stanley, 2001; Peng et al., 1994; Stanley et al., 1994). DFA is a time series method that describes the relation between the magnitude of fluctuations in postural motion and the time scale over which those fluctuations are measured. DFA is better suited for non-stationary signals (such as standing body sway; Carroll & Freedman, 1993; Riley et al., 1999) than traditional analyses, such as cross-correlation, because DFA involves local detrending. In conducting the DFA, the postural motion time series were first integrated, and then the integrated time series were divided into boxes of equal length,  $n$ . Next, a least-squares linear fit of the data in each box was calculated, representing the trend for that box. The integrated time series were then detrended by subtracting the local trend,  $y_n(k)$  (the  $y$ -coordinate of the linear fits), within each box. Finally, the root mean square fluctuation of the resulting integrated and detrended time series was calculated within each box, and that measure was averaged across boxes of the same size. These steps were repeated over different time scales (box sizes varying from 4 data points to a quarter of the time-series length) to characterize the relation between  $F(n)$ , the average fluctuation, and box size. In general, a log–log plot shows that  $F(n)$  increases linearly as box size increases. This linear relation indicates fractal scaling of the data. The slope of the line relating  $\log F(n)$  to  $\log n$ , is the scaling exponent  $\alpha$ , which describes the relation between postural motion variability and the time scale over which it is measured. The scaling exponent  $\alpha$  is an index of “memory” (long-range autocorrelation) in the data. White noise, which is uncorrelated, yields  $\alpha = 0.5$ . Long-memory correlations are indicated by  $\alpha > 0.5$ . Postural sway typically exhibits fractal scaling with exponents characteristic of fractional Brownian motion (cf. Collins & De Luca, 1993), although for prolonged, unconstrained standing DFA has suggested a pink ( $1/f$ ) noise structure (Duarte & Zatsiorsky, 2001). DFA is similar to stabilogram-diffusion analysis (Collins & De Luca, 1993) and rescaled range analysis (Duarte & Zatsiorsky, 2000), which also yield scaling exponents (Hurst exponents), but critically different in that it involves first integrating the time series (cf. Delignières, Deschamps, Legros, & Caillou, 2003).

The analyses were done separately for motion in the AP and ML axes. Some additional analyses were conducted only on data from the sum-of-sines trials; these are described below. For each significant main effect and interaction in our ANOVAs, we estimated the effect size using the partial  $\eta^2$  statistic.

## 3. Results

In all cases participants complied with the instructions to not move their feet. As noted above, during the experiment participants were under continuous direct surveillance. On

this basis, five participants were judged to have engaged in excessive voluntary movement (e.g., tossing of the head, or folding of the arms), and were deleted from the experiment on this basis. Of the deleted participants, one reported motion sickness. The remaining eighteen participants (eight men and ten women) 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.

### 3.1. Subjective reports

#### 3.1.1. Motion sickness and claustrophobia history

The data are summarized in Table 2. Sixty-seven percent of participants reported having been sick in the past. Of the eight participants in the Sick group, seven reported having been motion sick in the past, and one reported no previous experience of motion sickness. Of the 10 persons in the Well group, five reported that they had never been motion sick.

Five participants reported having been claustrophobic in the past. Four of the eight participants in the Sick group reported having experienced claustrophobia in the past, but in the Well group only one participant reported prior experience of claustrophobia.

#### 3.1.2. Incidence of sickness and discontinuation

Eight participants reported motion sickness (44% of our sample) and were placed in the Sick group. Of these, seven participants discontinued during the experiment, stating that they were motion sick. One person discontinued during Trial 5, three during Trial 6, one during Trial 7, and two during Trial 8. Each of the participants who discontinued stated that they were motion sick. One participant completed the experiment and became sick later that day, after leaving the laboratory. Sickness reports (both oral and written) were unambiguous (e.g., “I feel/felt sick”). Ten participants who completed the experiment and did not report motion sickness were placed in the Well group.

#### 3.1.3. Incidence of claustrophobia

One participant reported both sickness and claustrophobia when stopping the participation. This participant explained that she felt claustrophobic because she wanted to move her arms and legs but felt that she was unable to do so.

#### 3.1.4. Simulator sickness questionnaire

Questionnaire scores for each participant were calculated in the recommended manner (Kennedy et al., 1993), using the Total Severity Score. Mean SSQ scores are presented in Table 3. We used non-parametric tests, as the distribution of SSQ scores is known to be skewed (Kennedy et al., 1993). We used the Mann–Whitney *U*-test to compare the rank of scores between the Sick and Well groups, and the Wilcoxon Signed Rank Test to compare

Table 2  
History of motion sickness and claustrophobia

	Sick in the past	Not sick in the past	Claustrophobic in the past	Not claustrophobic in the past
Sick during the experiment	7	1	4	4
Well during the experiment	5	5	1	9

Table 3

Mean and standard deviation of scores on the simulator sickness questionnaire (the total severity score) and on the claustrophobia questionnaire (the global score) before (pre-test) and after (post-test) exposure to the moving room

Group	Simulator sickness questionnaire		Claustrophobia questionnaire	
	Pre-test	Post-test	Pre-test	Post-test
Well	17.58 (12.47)	19.45 (13.52)	21.40 (12.12)	21.40 (13.23)
Sick	14.02 (8.88)	78.07 (46.90)	30.62 (17.18)	30.12 (18.85)

the rank of pre-test and post-test scores within each group. We used the exact  $p$ -value for each tests and we set the criterion alpha level at .025 (2-tailed) because the SSQ data were used in two separate tests.

For pre-test scores, the Mann–Whitney  $U$ -test revealed no difference in rank between the Sick and Well groups,  $U = 33$ ,  $p > .025$ . For post-test scores, the difference in rank between the two groups was significant,  $U = 0$ ,  $p < .025$ . Scores for the Sick group (Mean Rank = 14.50) were higher than for the Well group (Mean Rank = 5.50).

For the Well group, the Wilcoxon Signed Rank Test revealed no difference between pre-test (Mean Rank = 3.70) and post-test (Mean Rank = 5.83) scores,  $z = -.071$ ,  $p > .025$ . For the Sick group, there was a significant difference between pre-test (Mean Rank = 0) and post-test (Mean Rank = 4.50) scores,  $z = -2.52$ ,  $p < .025$ , indicating that scores for the Sick group increased from pre-test to post-test.

The pre-test scores in our experiment were higher than pre-test scores obtained from the military personnel on which the SSQ was normed (Kennedy et al., 1993). However, the scores were comparable to pre-test scores that we have obtained with undergraduates in previous studies conducted in different laboratories (Smart et al., 2002; Stoffregen et al., 2000; Stoffregen & Smart, 1998).

### 3.1.5. Claustrophobia questionnaire

Questionnaire scores for each participant were calculated in the recommended manner (Radomsky et al., 2001). We used a global score, combining the restriction and suffocation scores. Mean scores are summarized in Table 3. The data were not skewed and variances were homogeneous. Accordingly, we used a repeated-measures ANOVA to test the factors Group (Sick versus Well) and Time (pre-test versus post-test). The alpha level was set at .05. There were no significant effects, indicating that exposure to the moving room did not produce any changes in rated claustrophobia.

## 3.2. Postural motion

Detrended fluctuations analyses revealed high linear fits for both AP and ML axes, with correlation coefficient values going from 0.91 to 1.00 for the AP axis (mean  $r^2 = 0.98$ ) and from 0.86 to 1.00 for the ML axis (mean  $r^2 = 0.98$ ).

### 3.2.1. Spontaneous sway (Trials 1 and 2)

The data are summarized in Table 4. For each dependent variable, we conducted a two-factor ANOVA on Vision (eyes open versus eyes closed)  $\times$  Group (Sick versus Well) with repeated measures on the first factor. In the AP axis, the main effects of Vision were

Table 4  
Significant main effects of vision on postural motion in Trials 1–2

	Variability AP (cm)		Velocity AP (cm s <sup>-1</sup> )		Range AP (cm)		Velocity ML (cm s <sup>-1</sup> )	
	Trial 1	Trial 2	Trial 1	Trial 2	Trial 1	Trial 2	Trial 1	Trial 2
Mean	.310	.488	.775	1.130	1.433	2.352	.564	.683
Standard deviation	.117	.163	.147	.333	.465	.729	.137	.198

For each dependent variable, the difference between Trial 1 (eyes open) and Trial 2 (eyes closed) was significant,  $p < .05$ .

significant for variability, velocity and range, each  $F(1, 16) > 29.53$ ,  $p < .05$ , partial  $\eta^2 = 0.67$ ,  $0.67$ , and  $0.65$ , respectively. In the ML axis, the main effect of Vision was significant for velocity,  $F(1, 16) = 25.98$ ,  $p < .05$ , partial  $\eta^2 = 0.62$ . Vision did not have significant effects on variability and range in the ML axis. The main effects of Group, and the Group  $\times$  Vision interactions were not significant for any of these variables.

A two-factor ANOVA on the scaling exponents ( $\alpha$ ) obtained from DFA revealed a significant effect of Vision for sway in the AP axis,  $F(1, 16) = 36.89$ ,  $p < .05$ , partial  $\eta = 0.71$ . Scaling exponents were larger when the eyes were open (Trial 1, mean  $\alpha = 1.50$ ) than when the eyes were closed (Trial 2, mean  $\alpha = 1.39$ ). In the DFA analyses main effect of Vision was not significant for sway in the ML axis. The main effects of Group and the Group  $\times$  Vision interactions also were not significant.

The significant effects of vision replicate and extend classical effects indicating that people tend to sway more when their eyes are closed. In some previous studies (Stoffregen et al., 2000; Stoffregen & Smart, 1998) we have found differences in spontaneous sway between participants who later became motion sick and those that did not. Group effects were not found in the present study.

### 3.2.2. 1-min, 0.2 Hz stimulus (Trials 3 and 4)

The data are summarized in Fig. 2. For each dependent variable, we conducted a two-factor ANOVA on Vision (eyes open versus eyes closed)  $\times$  Group (Sick versus Well) with repeated measures on the first factor. In the ML axis, the analyses revealed significant main effects of Vision on variability and range, each  $F(1, 16) > 7.91$ ,  $p < .05$ , partial  $\eta^2 = 0.40$  and  $0.33$ , respectively. For each significant effect, postural motion was greater when the eyes were closed. There were no other significant main effects or interactions for variability or range in the AP axis, or for velocity.

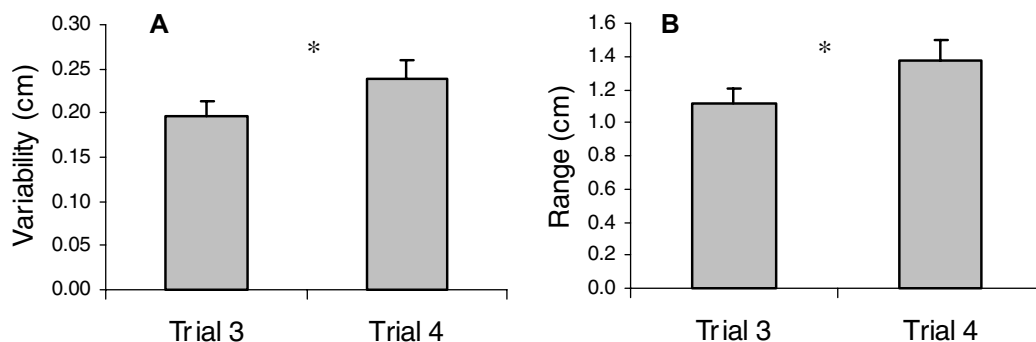


Fig. 2. Trials 3 and 4. Main effects of vision on postural motion in the ML axis. (A) variability, (B) range. \* $p < .05$ . The error bars represent standard error.

A two-factor ANOVA on the scaling exponents ( $\alpha$ ) obtained from DFA revealed a significant main effect of Group for sway in the ML axis,  $F(1, 16) = 4.66$ ,  $p < .05$ , partial  $\eta^2 = 0.23$ . Scaling exponents were larger for the Sick group (mean  $\alpha = 1.38$ ) than for the Well group (mean  $\alpha = 1.27$ ). The main effect of Vision and the Group  $\times$  Vision interaction were not significant. There were no significant effects in the AP axis.

### 3.2.3. 10-min, sum-of-sines stimulus (Trials 5–8)

Due to discontinuation, we did not have the same amount of postural data for each participant in Trials 5–8. Each participant in the Well group completed all of the sum-of-sines trials and, therefore, was exposed to the sum-of-sines stimulus for a total of 40 min. One participant in the Sick group completed the experiment, but the other seven discontinued without completing the full 40 min of sum-of-sines motion. In the Sick group, the mean duration of exposure to the sum-of-sines motion was 23 min, 23 s. We sought to ensure that our analyses did not include any postural motion that occurred after the onset of motion sickness symptoms. For this reason, in our analyses we included only data for trials that were completed, that is, trials in which the participant did not discontinue. For example, if a participant discontinued midway through Trial 7, we analyzed all of their data for Trials 5 and 6, but none of their data for Trial 7. The mean number of sum-of-sines trials completed by participants in the Sick group was two (Trials 5 and 6). One participant discontinued during the first sum-of-sines trial (Trial 5). Therefore, the following analyses include 17 participants.

**3.2.3.1. Overall sway.** Representative data from sum-of-sines trials for Sick and Well participants are presented in Fig. 3. To evaluate overall postural sway (and for comparability with our previous studies), we conducted unpaired  $t$ -tests comparing the Sick and Well groups, taking means across the sum-of-sines trials for each dependent variable. The differences between Sick and Well groups were significant in the AP axis for variability,  $t(15) = 2.64$ ,  $p < .05$ , Cohen's  $D = 1.23$ , and for range,  $t(15) = 2.19$ ,  $p < .05$ , Cohen's  $D = 1.04$  (Fig. 4). In each case, sway was greater in the Sick group, as predicted by Riccio and Stoffregen (1991). Analysis of the scaling exponents from DFA did not reveal any significant effects.

**3.2.3.2. Evolution of sway during exposure.** We evaluated the evolution of sway over the course of exposure to the sum-of-sines stimulus. To do this, we selected three windows from the data, each of which was 2 min in duration. For the Sick group, we choose the first, the middle, and the final two minutes for each participant, with the restriction that no window included a boundary between two trials (that is, each window included only continuous data from within a single trial). For example, if a participant discontinued after completing Trial 7, the first window was from 0 to 120 s of Trial 5, the middle window was from 241 s to 360 s of Trial 6, and the final window was from 481 s to 600 s of Trial 7. For the Well group, we took the first two minutes of Trial 5, the final two minutes of Trial 5, and the final two minutes of Trial 6. These windows were selected based on the mean exposure of participants in the Sick group, and ensured that the sway data for both groups corresponded to the same mean duration of exposure to the sum-of-sines stimulus. For each of the dependent variables, we conducted separate 2-factor ANOVAs Group (Sick versus Well)  $\times$  Window (first, middle, last) with repeated measures on the second factor.

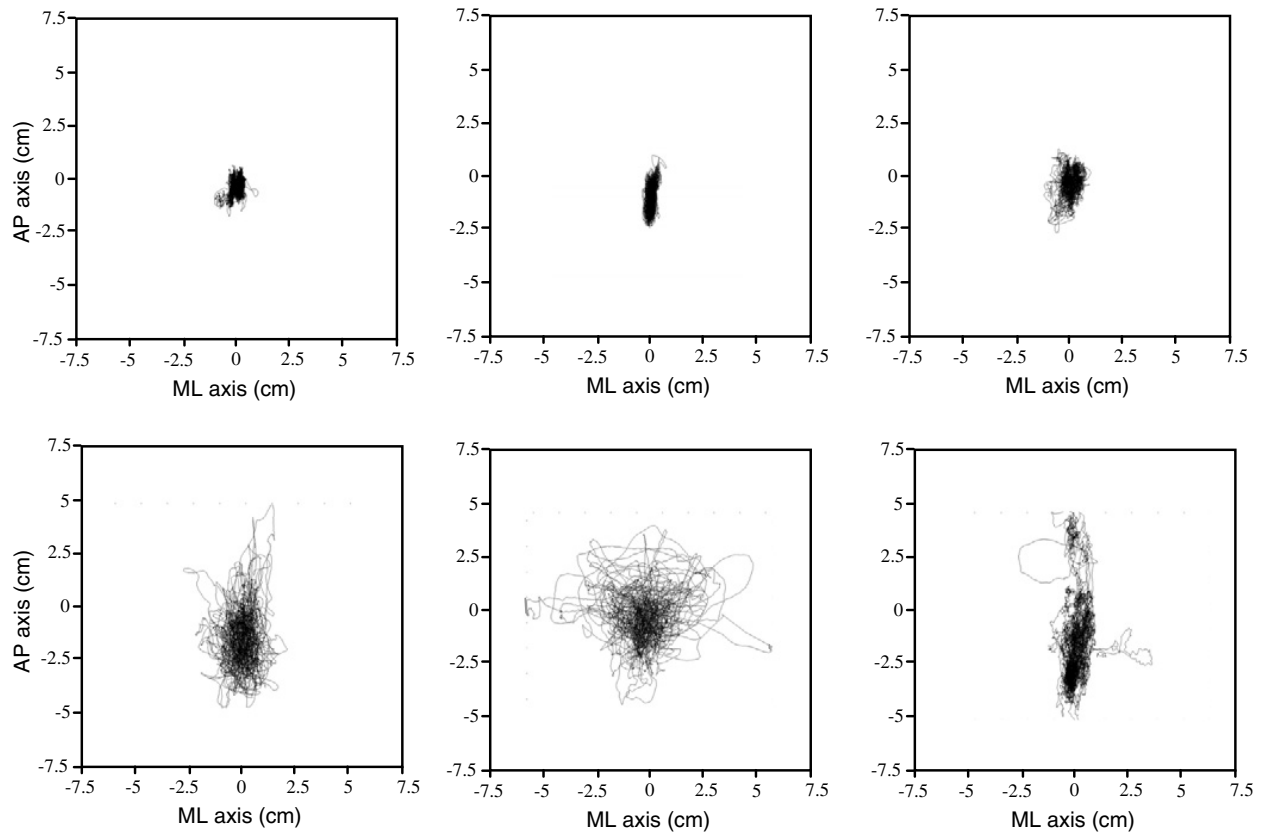


Fig. 3. Center of pressure data for representative trials in the sum-of-sines condition. Top panels: Participants who did not report motion sickness. Bottom panels: Participants who reported motion sickness (postural data were collected before sickness onset).

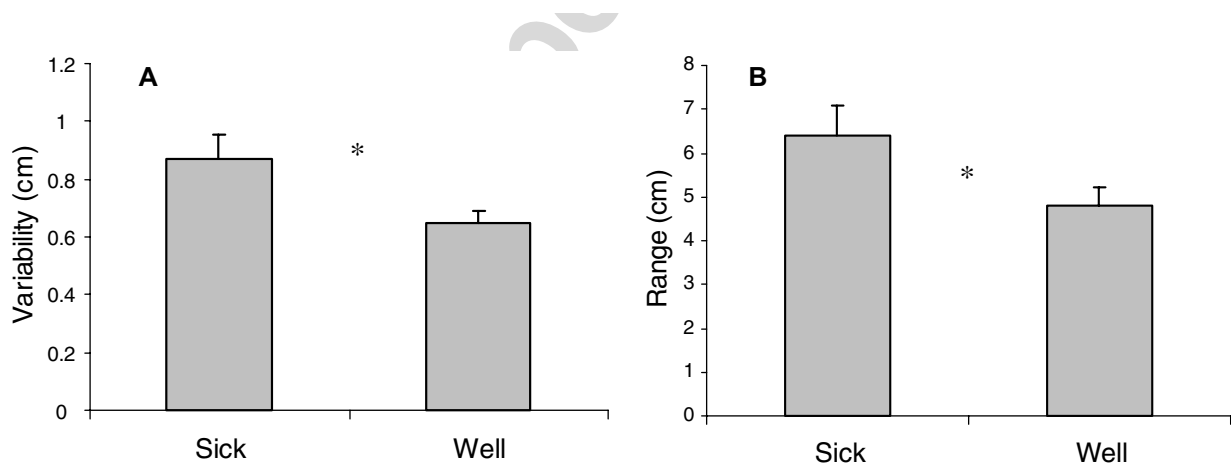


Fig. 4. Trials 5–8. Overall variability and range in the AP axis for the Sick and Well groups. \* $p < .05$ . The error bars represent standard error.

The ANOVAs revealed main effects of Window on variability in the AP axis (Fig. 5), on velocity in both the AP and ML axes (Fig. 6), and on range in the AP axis (Fig. 7), each  $F(2, 30) > 5.69$ ,  $p < .05$ , partial  $\eta^2 = 0.27$ , 0.35, and 0.29, respectively. There were significant main effects of Group on variability in the AP and ML axis (Fig. 5), and on range in the AP and ML axes (Fig. 7), each  $F(1, 15) > 4.57$ ,  $p < .05$ , partial  $\eta^2 = 0.40$ , 0.24, 0.40, 0.23, respectively. None of the Group  $\times$  Window interactions were significant, each  $F(2, 30) < 2.20$ ,  $p > .05$ .

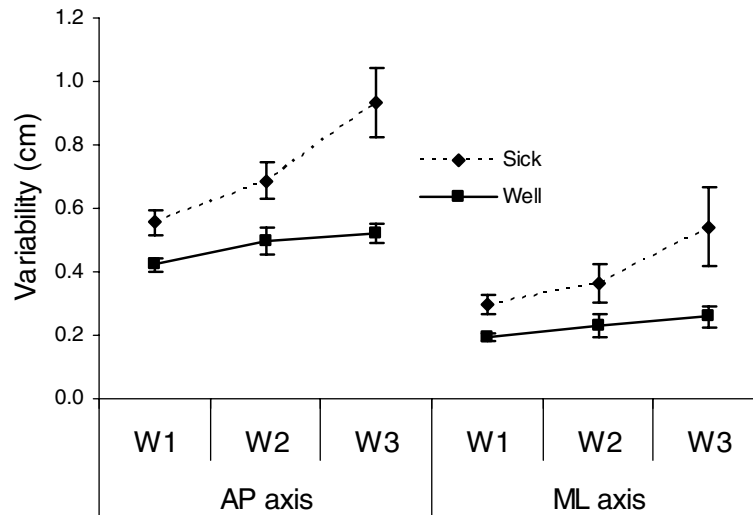


Fig. 5. Trials 5–8. Variability of the center of pressure in the AP and ML axes for windows (W1, W2, W3) and groups (Sick and Well). The error bars represent standard error.

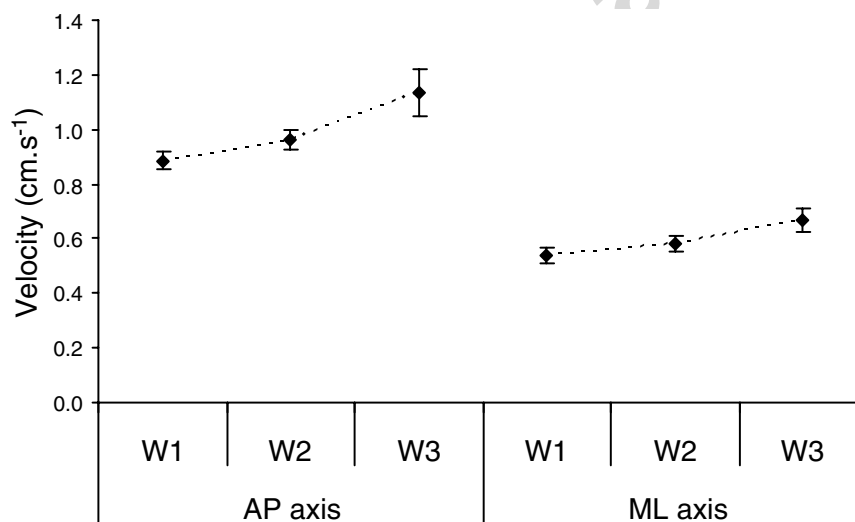


Fig. 6. Trials 5–8. Velocity of the center of pressure in the AP and ML axes as a function of windows. The error bars represent standard error.

Means and standard deviations for dependent variables involved in the post-hoc analyses are summarized in Table 5. The post-hoc analysis revealed significant differences between Window 1 and Window 3 on variability, velocity, and range in the AP axis, on velocity in the ML axis. There were also significant differences between Window 2 and Window 3 on velocity in the AP and ML axes. In each case, the mean in Window 3 was greater than in Window 1, or in Window 2. There were no differences between Window 1 and Window 2.

#### 3.2.4. Comparison of sway before and after the sum-of-sines stimulus

Because the experiment was interrupted immediately when participants reported motion sickness, Trials 9–11 were completed by only one participant in the Sick group. For this reason, we conducted statistical analyses including Trials 9–11 only for the Well

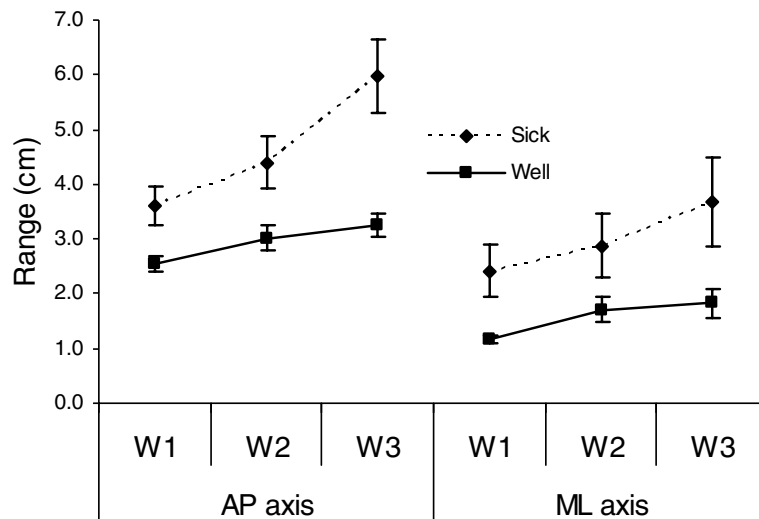


Fig. 7. Trials 5–8. Range of the center of pressure in the AP and ML axes as a function of windows (W1, W2, W3) and groups (Sick versus Well). The error bars represent standard error.

group. Accordingly, comparisons of sway before and after exposure to the sum-of-sines stimulus are not directly relevant to evaluation of the postural instability theory of motion sickness.

Table 5

Means and standard deviations on Windows 1, 2, and 3 for the dependent variables that had significant main effects of Window

	Window 1		Window 2		Window 3	
	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation
Variability AP	0.478	0.141	0.574	0.224	0.693	0.363
Velocity AP	0.886	0.145	0.963	0.159	1.138	0.363
Velocity ML	0.541	0.122	0.584	0.108	0.668	0.188
Range AP	2.979	1.165	3.586	1.575	4.371	2.300

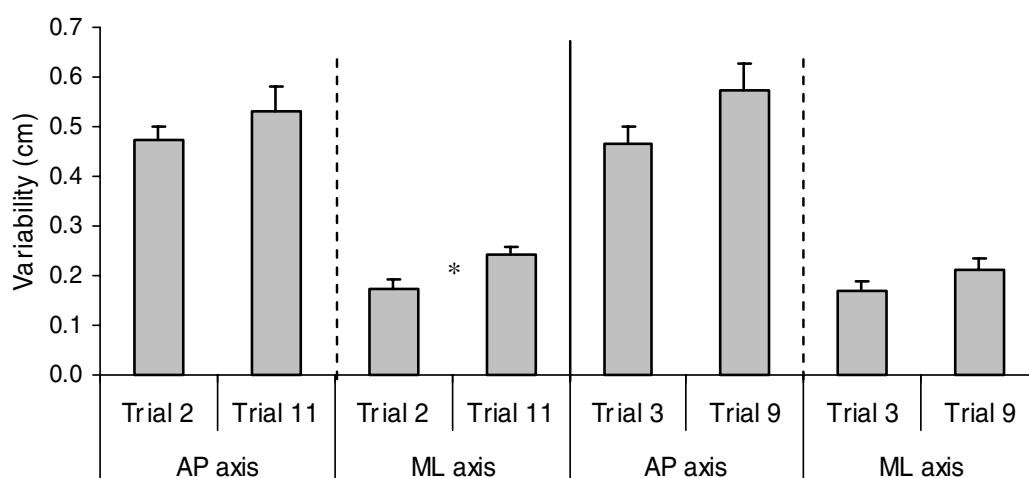


Fig. 8. Pre-post comparisons (Well group). Variability of the center of pressure in the AP and ML axes for Trials 2–11 and Trials 3–9. \* $p < .05$ . The error bars represent standard error.

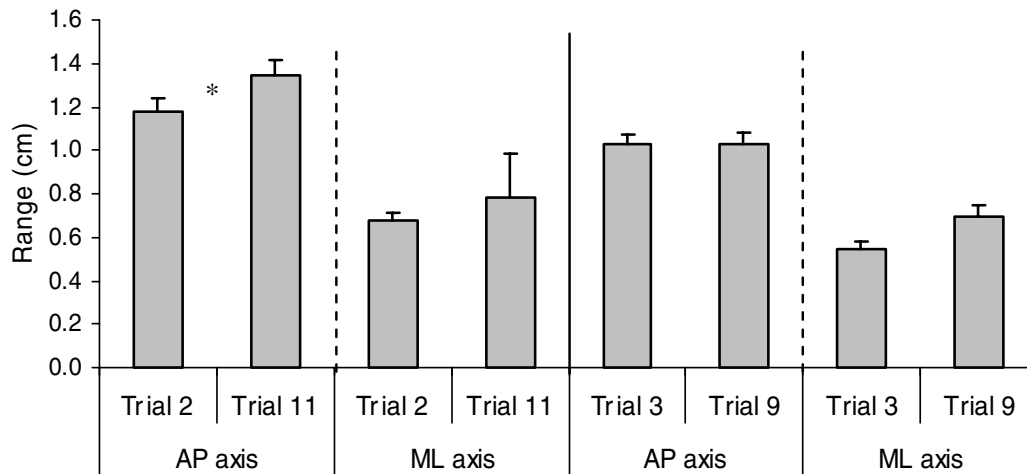


Fig. 9. Pre-post comparisons (Well group). Range of the center of pressure in the AP and ML axes for Trials 2–11 and Trials 3–9. \* $p < .05$ . The error bars represent standard error.

For the Well group, we compared postural motion during spontaneous sway with the eyes open (Trials 1 and 10), spontaneous sway with the eyes closed (Trials 2 and 11), and sway during exposure to the 0.2 Hz stimulus with the eyes open (Trials 3 and 9). For each of these comparisons, we conducted seven paired  $t$ -tests (one for each of our dependent variables). We found several significant differences between pre-test and post-test trials. In each case, sway was greater in the post-test trials.

**3.2.4.1. Spontaneous sway.** We found several significant pre-post differences during spontaneous sway with the eyes closed (Trial 2 versus Trial 11). There were significant effects in the ML axis for variability (Fig. 8) and range (Fig. 9),  $t(9) = 3.26$ ,  $p < .05$ , Cohen's  $D = 0.90$ , and  $t(9) = 3.03$ , Cohen's  $D = 0.89$ ,  $p < .05$ , respectively, and in the AP axis for velocity (Fig. 10),  $t(9) = 2.31$ ,  $p < .05$ , Cohen's  $D = 0.59$ . We did not find any pre-post significant differences during spontaneous sway with the eyes open (Trial 1 versus Trial 10).

**3.2.4.2. 0.2 Hz imposed motion.** We found a significant pre-post difference when participants were exposed to the 0.2 Hz motion with their eyes open (Trial 3 versus Trial 9) for

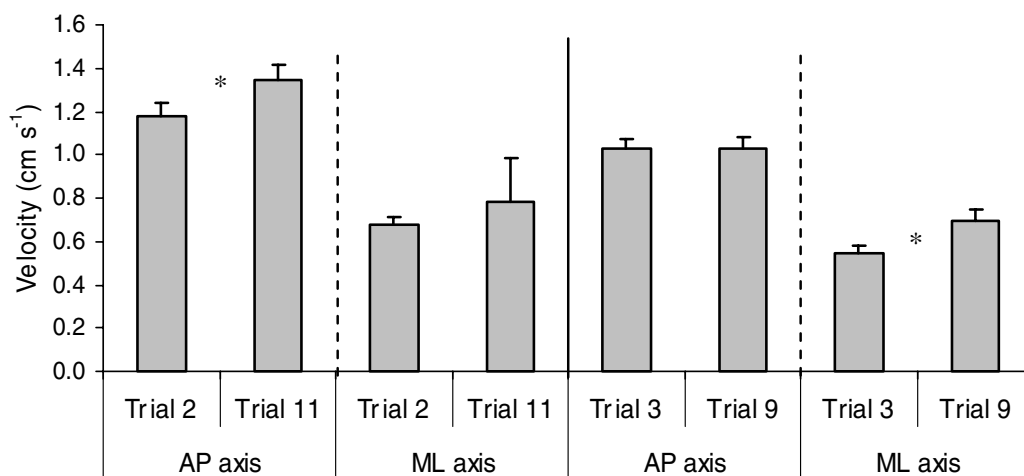


Fig. 10. Pre-post comparisons (Well group). Velocity of the center of pressure in the AP and ML axes for Trials 2–11 and Trials 3–9. \* $p < .05$ . The error bars represent standard error.

velocity in the ML axis,  $t(9) = 3.42$ ,  $p < .05$ , Cohen's  $D = 0.95$  (Fig. 10). All other comparisons were not significant,  $t(9) < 1.51$ , ns.

#### 4. Discussion

As in several previous studies, approximately half of our participants reported motion sickness after being exposed to an optical simulation of standing body sway. We found lots of interesting effects, which generally support the postural instability theory of motion sickness. They are discussed in turn.

##### 4.1. Motion sickness incidence and severity

An optical simulation of normal standing body sway produced motion sickness in 44% of our participants. The incidence and severity of motion sickness were closely similar to our previous studies (Smart et al., 2002; Stoffregen et al., 2000; Stoffregen & Smart, 1998). This is remarkable, given that people are not sickened by their own sway (for an exception, see Smart, Pagulayan, & Stoffregen, 1998), and given that people typically are not aware of the simulation, due to its low amplitude and frequency. Some approaches to motion sickness based on the concept of intersensory conflict (Ricci & Stoffregen, 1991) have attempted to explain the fact that sickness is associated with the same frequencies of oscillation that characterize stance (e.g., Duh et al., 2004). These hypotheses are post-hoc, that is, they cannot be derived from the concept of intersensory conflict, as such. Our prediction that sickness would occur was a priori, that is, it can be derived from the facts of oscillating systems, rather than from data about motion sickness incidence.

Another issue relates to the amplitude of simulated self-motion. Duh et al. (2004), predicted that motion sickness would occur preferentially with visual motion at 0.06 Hz, but the visual stimuli in their experiments moved with a peak velocity of  $70^\circ/\text{s}$ . By contrast, the linear room motions used in the present study (and by Smart et al., 2002 & Stoffregen & Smart, 1998) produced optical flow corresponding to a peak angular velocity (at the head) on the order of  $1.0^\circ/\text{s}$ . In order for the sensory conflict theory to explain the production of motion sickness by such low-amplitude stimulation, it would be necessary to explain how conflict produced in our experiments was greater in magnitude than conflict produced in other conditions of stance that do not elicit motion sickness. On this basis, we suggest that the crossover hypothesis proposed by Duh et al. cannot explain the finding that motion sickness is induced by optic flow that simulates body sway in both frequency and amplitude.

##### 4.2. Postural motion: Spontaneous sway

In two previous studies, we have found that motion sickness was predicted by postural motion before participants were exposed to any stimulus motion (Stoffregen et al., 2000; Stoffregen & Smart, 1998). In the present study, as in one other previous study (Smart et al., 2002) motion sickness has not been predicted by postural motion during unperturbed stance. The fragility of this effect may indicate that susceptibility to motion sickness is not reflected in spontaneous postural motion. An alternative possibility is that reliable, robust effects exist in variables other than those that have been evaluated to date, such as the variables provided by recurrence quantification analysis (e.g., Riley et al., 1999).

### 4.3. Postural motion: 0.2 Hz stimulus motion

Motion sickness was preceded by significant changes in postural motion (relative to participants who did not get sick) during exposure to a pattern of optic flow that was benign. Significant differences in postural motion between Sick and Well groups have been obtained in each of our previous studies (Smart et al., 2002; Stoffregen et al., 2000; Stoffregen & Smart, 1998). The consistency of this finding, across laboratories (University of Cincinnati versus University of Minnesota), across facilities (moving room versus flight simulator) and across postures (standing versus sitting) suggests that there is a powerful relation between motion sickness and postural responses to brief presentations of non-nauseogenic optic flow.

In our previous studies, we measured displacements of the head and torso (Smart et al., 2002; Stoffregen et al., 2000; Stoffregen & Smart, 1998). In each of these studies we found that motion sickness was preceded by postural motion prior to exposure to the nauseogenic stimulus (i.e., during Trials 1, 2, 3, and/or 4). In the present study, we found that motion sickness was preceded by displacements of the center of pressure during exposure to the 0.2 Hz stimulus motion. Using detrended fluctuation analysis we identified a significant difference between Sick and Well in the 0.2 Hz trials. Thus, we have once again found that motion sickness is preceded by postural motion prior to exposure to the nauseogenic stimulus. The consistency of this finding suggests a general phenomenon that may have considerable practical significance. Our results suggest that it may be possible to predict motion sickness susceptibility using objective data (as opposed to questionnaires, personal histories, or other self-reports) from test situations that are not themselves nauseogenic (Smart et al., 2002). This possibility contrasts with current practices in which susceptibility is not predicted, but directly tested by placing people into situations that are known to be nauseogenic (e.g., Kennedy, Dunlap, & Fowlkes, 1990).

Many of our dependent variables in this study imply an equation between postural instability and the “amount” of postural motion. Examples include variability and range. This is not true of the significant difference between the Sick and Well groups revealed by the detrended fluctuation analysis. The effect revealed by detrended fluctuation analysis does not imply that there was more motion in the Sick group. It means that the fluctuations were more strongly correlated with each other for the Sick group, and that the Sick group’s postural fluctuations tended more toward brown noise, while the Well group’s tended more towards pink noise (for definitions of these terms, see Schroeder, 1991). The results of the detrended fluctuation analysis underscore an important conceptual issue relating to the postural instability theory of motion sickness. Riccio and Stoffregen (1991) argued that motion sickness would be preceded by increases in postural instability. However, they did not claim that “postural instability” would always imply “more postural motion.” They defined *instability as uncontrolled movement*, and they stressed that there can be stable (and unstable) movements. Examples include leaning forward, rotating the eyes, head, and/or torso to track visually a moving object, and dancing. Each of these would register as “more movement,” relative to so-called “quiet stance,” but none of these would necessarily be unstable movements and, in fact, none of them are sufficient to cause motion sickness. A definition of instability in terms of uncontrolled movement challenges the common equation of “unstable movement” with “more movement”. Movement may be uncontrolled in terms of amplitude (e.g., our results in variability, velocity, and range), but it may also (or instead) be uncontrolled in other ways (e.g., our DFA results).

#### 4.4. Postural motion: *Sum-of-sines motion*

During exposure to the sum-of-sines stimulus, the variability and range of postural motion was greater for the Sick group than for the Well group. This result confirms findings from our previous studies, in which motion sickness has been preceded by instabilities in motion of the head and torso during exposure to the nauseogenic stimulus (Smart et al., 2002; Stoffregen et al., 2000; Stoffregen & Smart, 1998), and extends it to unstable motion of the center of pressure. The finding that motion sickness is preceded by unstable motion of the body during exposure to a nauseogenic stimulus confirms one of the central predictions of the postural instability theory of motion sickness (Riccio & Stoffregen, 1991).

A novel aspect of the present study was our analysis of the evolution of sway over time during exposure to the sum-of-sines. We found main effects of time (Windows), indicating that sway increased over time for all participants. We also found main effects of Group, indicating that over time sway was greater in the Sick group than in the Well group. This latter result confirms the differences between Sick and Well groups identified in our other analyses. There were no significant interactions between Group and Window. These effects, with the absence of interactions, indicate that participants in the Sick group responded differently to the sum-of-sines immediately, at the very beginning of exposure. This result is consistent with our numerous findings (here and in previous studies) of sway differences between Sick and Well before they are ever exposed to the sum-of-sines. We can conclude that sway did evolve over time during exposure to the sum-of-sines stimulus (it tended to increase in magnitude), but that the evolution of sway was not influenced by whether participants would or would not become motion sick.

Understanding the time course of sway during exposure to the sum-of-sines stimulus is important for both theoretical and practical reasons. We found that the Sick and Well groups differed in displacements of the center of pressure at the very beginning of exposure to the sum-of-sines stimulus. Thus, postural instability existed, on average, more than 20 min before the onset of subjective symptoms of motion sickness. This finding is theoretically important because it supports our claim that postural instability actually precedes the onset of motion sickness, rather than being an early concomitant of the subjective symptoms of the malady. A potentially significant practical implication of our findings on the evolution of instability during exposure to the nauseogenic stimulus is that it might be possible to predict the incidence of motion sickness using postural data from an exposure that is too brief to produce actual motion sickness (e.g., the first 2-min window from our analysis). When it is useful to test for motion sickness susceptibility through direct exposure to a specific nauseogenic stimulus, it may nevertheless be possible to predict susceptibility while avoiding the induction of actual symptoms.

Finally, our findings relating motion sickness to the evolution of sway during exposure to the nauseogenic stimulus suggest future research. It should be possible to monitor sway during exposure to optical simulations of self-motion, to identify individual participants who exhibit unstable responses to stimulus motion, and to terminate exposure immediately for these persons. Will motion sickness occur in persons who have been withdrawn from a nauseogenic situation after the onset of postural instability but before the onset of subjective symptoms? Previous findings suggest that this effect may be real: Many studies have found that motion sickness begins after termination of the nauseogenic stimulus; often several hours later (e.g., Kennedy & Lilienthal, 1994; Stoffregen, 1985; Stoffregen & Smart, 1998). However, in previous studies this effect has been adventitious; there has been no

controlled research in which experimenters have used measurements of postural motion as a basis for terminating exposure to a nauseogenic stimulus.

#### 4.5. Comparison of pre-exposure and post-exposure sway

Our pre-post analysis revealed that sway increased following exposure to the sum-of-sines stimulus. The pre-post analysis was limited to participants in the Well group, that is, the pre-post differences occurred among participants who did not get sick. This finding begs the question of the definition of instability: In the context of motion sickness (at least), more sway does not necessarily mean more instability. This result suggests a direct test, in which subjects engage in a deliberate movement task, so that minimal sway would mean poor performance. One option might be to ask participants in the moving room to sway deliberately back and forth so as to track the AP sum-of-sines motion of the room. Successful performance of the tracking task would yield large amounts of postural motion in irregular patterns. By contrast, participants who minimize sway would “fail” the tracking task. In such an experiment, how would the “amount” of sway and the accuracy of tracking performance be related to the incidence of motion sickness?

### 5. Conclusion

We found some of the same differences in sway between Sick and Well participants that have been reported in previous studies, confirming that similar effects occur for displacements of the COP and of the head and torso. We also identified differences between Sick and Well in new dependent variables and analyses, such as detrended fluctuation analysis. Overall, the results are consistent with predictions made by the postural instability theory of motion sickness (Ricci & Stoffregen, 1991), and support the hypothesis that instability in the control of stance is a necessary and sufficient precondition for the onset of motion sickness. One goal for future research will be the attempt to use data about postural motion to predict the incidence of motion sickness. The present study contributes to this goal by increasing the number of parameters that may be used in the development of predictive algorithms.

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