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RESEARCH ARTICLE

Motion sickness, body movement, and claustrophobia during passive restraint

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Abstract Standing participants were passively restrained and exposed to oscillating visual motion. Thirty-nine percent of participants reported motion sickness. Despite passive restraint, participants exhibited displacements of the center of pressure, and prior to the onset of motion sickness the evolution of these displacements differed between participants who later became sick and those who did not. Claustrophobia occurred during restraint, but only among participants who became motion sick. The results are consistent with the postural instability theory of motion sickness. We discuss the possible relation between claustrophobia symptoms, postural movements and motion sickness incidence.

Keywords Motionsickness · Posture · Physical restrain · Claustrophobia

Introduction

Theories of motion sickness etiology have typically been based on the concept of sensory conflict (e.g., Duh et al. 2004; Oman 1982; Reason 1978). The essential idea of this approach is that motion sickness situations are characterized by patterns of perceptual stimulation that differ from patterns expected on the basis of past experience (Stoffregen and Riccio 1991). These differences constitute sensory conflict, which is alleged to produce motion sickness. However, theories based on this concept have low predictive validity (Draper et al. 2001), and some researchers have argued that such theories may not be scientifically falsifiable (e.g., Ebenholtz et al. 1994). In the present study, we sought to evaluate an alternative theory of motion sickness etiology, the postural instability theory of motion sickness (Riccio and Stoffregen 1991).

The postural instability theory of motion sickness is not based on the concept of sensory conflict but, rather, on relations between perception and the control of action. Unlike the sensory conflict theory, this approach considers the activity of the individual as fundamental in motion sickness etiology (Riccio and Stoffregen 1991). The postural instability theory predicts that motion sickness will be preceded and predicted by instabilities in control of the posture.[1]

One of the most consistent findings in the motion sickness literature is a strong relationship between motion sickness incidence and the frequency of

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[1] In the mathematics of dynamic systems, instability has a precise definition related to a system's response to a change in initial conditions or to a perturbation (e.g., Strogatz 1993). Such conceptions of instability may be related to postural instability, but there is no a priori reason to define postural instability in such a specific sense. Thus, we regard the definitions of postural stability and instability as being open questions, and we believe that our research relating motion sickness to postural movements may contribute to clarifying the concept of postural instability.

imposed oscillatory stimuli. Laboratory experiments and field studies have made it clear that sickness is associated with imposed oscillations within a narrow band of frequencies, 0.08–0.4 Hz (e.g., Golding et al. 1997; Lawther and Griffin 1988; O'Hanlon and McCauley 1974; for a review, see Guignard and McCauley 1990). Motion sickness is reduced or absent with imposed motion at other frequencies.

Imposed motion between 0.08 and 0.4 Hz is characteristic of many operational situations that are associated with motion sickness, such as vehicles that operate on land, on water, and in the air (Guignard and McCauley 1990). Stoffregen and Riccio (1991) noted that the range 0.08–0.4 Hz also contains much of the spectral power of human postural sway. The coincidence in frequency ranges between postural sway and nauseogenic motion has motivated research relating postural motion to imposed optical oscillations (Bonnet et al. 2006; Smart et al. 2002; Stoffregen and Smart 1998). In these studies, standing participants have been exposed to experimentally generated optic flow that reproduced the amplitude and frequency characteristics of stance (Bensel and Dzendolet 1968). Optic flow was created by physical displacement of the visible surroundings in a moving room that oscillated along the line of sight. Motion of the room consisted of the sum of 10 sine waves between 0.016 and 0.31 Hz, with maximum peak-to-peak amplitude of 1.8 cm. These motions were of such low frequency and amplitude that many participants were not aware of any motion. Across studies, there were two important results. First, the optic flow induced motion sickness in approximately 40% of participants. Second, prior to the onset of any subjective symptoms of motion sickness, there were significant differences in the postural motion of participants who eventually became sick, relative to those who did not. As predicted by the postural instability theory (Riccio and Stoffregen 1991), motion sickness was preceded by instabilities in control of the posture. These instabilities were observed in the amplitude of postural sway, as well as in the dynamics of sway (Bonnet et al. 2006; Smart et al. 2002; Stoffregen and Smart 1998).

Motion sickness and passive restraint

The consistency of these effects motivated us to test two other predictions of the theory. Riccio and Stoffregen (1991) argued that people can be posturally unstable only when they have active control of their posture, that is, when posture is maintained through the person's own muscular activity. If motion sickness is related to unstable control of posture, then motion sickness should occur only in the context of active attempts to control posture. Under full passive restraint (e.g., when strapped down) a person should be unable to move. It should be impossible for such persons to become unstable, and so the postural instability theory of motion sickness predicts that they should be immune to motion sickness. Several studies have shown that passive restraint can reduce the incidence of motion sickness (e.g., Fox et al. 1982; Johnson and Mayne 1953; Johnson and Taylor 1961; Lackner et al. 1991; Mills and Griffin 2000). However, in other studies, motion sickness has been observed among persons subjected to passive restraint (e.g., Graybiel and Miller 1970; Warwick-Evans et al. 1998). The occurrence of motion sickness during passive restraint may not invalidate the postural instability theory of motion sickness. It is very difficult to eliminate completely the possibility of any movement, and any residual motion could be relevant to the etiology of motion sickness. For this reason, "it may be possible to produce motion sickness in not-quite-fully restrained animals" (Riccio and Stoffregen 1991, p. 223). This possibility provides a strong motivation to measure any movements that might occur during passive restraint. Previous studies relating motion sickness to passive restraint have not included measurements of body movement. In our previous studies relating motion sickness to postural instability in stance, participants have not been subjected to any form of restraint (Bonnet et al. 2006; Smart et al. 2002; Stoffregen and Smart 1998). In the present study, we measured body movement while passively restrained participants were exposed to visual motion that is known to induce motion sickness in unrestrained persons (Bonnet et al.; Smart et al.; Stoffregen and Smart). We predicted either (1) that motion sickness would not occur, or (2) that during passive restraint movements of participants who became motion sick would differ from those of participants who did not, and that such differences would precede the subjective symptoms of motion sickness.

Passive restraint and claustrophobia

The restriction of movement that characterizes passive restraint might elicit feelings of claustrophobia, which is a form of anxiety. Anxiety has been related to the incidence of motion sickness (e.g., Owen et al. 1998) but empirical results have been inconsistent (e.g., Fox and Arnon 1988; Money 1970). The largest significant correlation coefficient that has been found between subjective reports of the intensity of state anxiety and motion sickness[2] is 0.37 (Fox and Arnon 1988; Tucker and

[2] The studies cited here did not assess persons with anxiety disorders.

Reinhardt 1967); correlations between motion sickness severity and trait anxiety are lower (Fox and Arnon 1988; Owen et al. 1998). In research relating motion sickness to anxiety, participants have not been restrained; presumably, it is for this reason that researchers have not assessed claustrophobia as such, and have instead focused on anxiety, in general. There have been no attempts specifically to relate claustrophobia to motion sickness. We measured claustrophobia before and after passively restrained participants viewed the nauseogenic stimulus. This permitted us to determine whether our restraint system elicited feelings of claustrophobia, and it permitted us to assess possible relations between claustrophobia and motion sickness.

The present study

We subjected standing persons to passive restraint during exposure to visual motion that is known to induce motion sickness among standing persons who are not restrained. We measured displacements of the center of pressure before and during exposure. We assessed motion sickness incidence through direct, yes or no statements, and we quantified the severity of symptoms using a standard questionnaire. We also assessed potential subjective side effects of passive restraint, using yes/no statements and a standard claustrophobia questionnaire. For movement data during passive restraint, we conducted separate analyses of overall patterns (i.e., means over trials), and of changes in movement patterns during the period of restraint. We expected that passive restraint would confer immunity to motion sickness or that prior to the onset of motion sickness, bodily motion would differ between participants who became sick and those who did not.

Method

Participants

Eighteen students (eight males and ten females) from the University of Minnesota participated after giving written informed consent. Participants were volunteers or received course credit. They ranged in age from 18 to 23 years (mean = 20.4 years), in weight from 55.80 to 88.50 kg (mean = 70.03 kg), and in height from 1.56 to 1.92 m (mean = 1.71 m). Participants had normal or corrected-to-normal vision, were not pregnant, and reported no history of recurrent dizziness, recurrent falls, or vestibular (inner ear) dysfunction. None of the participants had participated in any other study of motion sickness in our laboratory. 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. This ensured that participants had no motivation for falsely stating that they were motion sick or claustrophobic. When scheduling experimental sessions, participants were requested not to eat anything for 4 h before coming to the laboratory. The procedure used in this study was approved by the Institutional Review Board of the University of Minnesota.

Apparatus

We generated optical flow using a moving room (Lee and Lishman 1975; Smart et al. 2002) consisting of a cubical frame (2.44 m on a side) mounted on wheels and moving in one axis along rails (Fig. 1a). Rigid masonite sheets covered with blue and white marble-pattern paper were attached to three sides and the top of the frame to create walls and a ceiling. The fourth (rear) side of the room provided access. At the center of the front wall was a large, detailed map of the continental United States (53 × 80 cm; 19° × 28°). Illumination was provided by four incandescent floodlights oriented so that shadows were minimized. Participants stood on a force platform (AccuSwayPlus, AMTI, Chicago, USA) collecting data on displacements of the center of pressure at a sampling rate of 50 Hz. The force platform rested on the carpeted concrete laboratory floor, such that there was no imposed inertial motion. Restraint was achieved by strapping participants to a tilt table (801 Electronic Tilt Table; ActiveAid, Inc., Redwood Falls, MN, USA), that was rotated so that the table surface was locked in its vertical position.

Procedure

To assess participants' initial level of symptoms, and to ensure that they were familiar with motion sickness and claustrophobia symptomologies, participants were asked to complete two questionnaires at the beginning of the experiment: the simulator sickness questionnaire, or SSQ (Kennedy et al. 1993), and the claustrophobia questionnaire, or CLQ (Radomsky et al. 2001).

Each participant successfully completed a pre-test in which they were asked to stand on one foot for 30 s. They then stood on the force platform with their heels on a line marked on its surface, such that they were about 1.5 m from the front wall. For the duration of each trial, participants were asked not to move their feet and to keep their hands in their pockets, or clasped

behind or in front of them. They were asked to keep their gaze on the map on the front wall (there was no fixation point). Participants were warned that they might become ill, and were instructed to discontinue the experiment immediately if they began to experience any noticeable symptoms of motion sickness or claustrophobia.

The sequence of trials is summarized in Table 1. We began by collecting data on spontaneous postural sway, with no room motion, for 20 s with eyes open (Trial 1), and again with eyes closed (Trial 2). Next, the room moved 1.5 cm (peak-to-peak) at 0.2 Hz (Fig. 1b) for 60 s with their eyes open (Trial 3) and for 60 s with the eyes closed (Trial 4). In Trials 1–4, participants were not restrained. These trials were followed by four trials, each of which was 10 min (600 s) in duration, during which the room moved with a sum-of-sines pattern (Trials 5–8). The sum-of-sines comprised ten sine waves, 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 combined waveform had maximum peak-to-peak amplitude of 1.8 cm (Fig. 1b). During exposure to the sum-of-sines, standing participants were restrained by being strapped to the vertical tilt table using elastic bands at the head, shoulders, hips, and knees. The bands were adjusted by the experimenter so that participants felt them to be tight but not uncomfortable. Participants' body weight was not supported by the tilt table. Participants were released from restraint for a few minutes at the end of each sum-of-sines trial. Following exposure to the sum-of-sines motion, the conditions of trials 1, 2, and 3 were repeated (Trials 9–11) to evaluate pre-post differences in sway. Participants were monitored continuously by an experimenter stationed outside the moving room.

At the end of exposure to imposed optic flow (either through discontinuation or after the completion of all trials) participants were asked whether they felt motion sick and/or claustrophobic, and were asked to describe any symptoms. Participants who stated that they were motion sick or claustrophobic were asked to fill out the SSQ and CLQ directly. Participants who stated that they were not motion sick 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 (e.g., Stoffregen 1985) or a flight simulator (e.g., Kennedy and Lilienthal 1994).

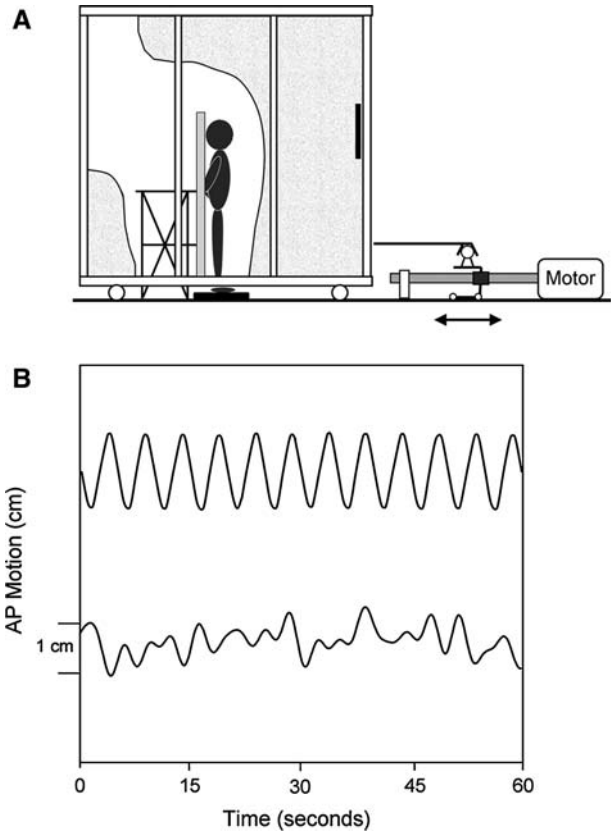

Fig. 1 **a** The moving room and the tilt table. **b** Motion patterns of the moving room. Upper trace: Motion at 0.2 Hz with amplitude 1.5 cm and duration 60 s. Lower trace: A portion of the sum-of-sines stimulus, which continued for 600 s without repeating. The maximum amplitude of the sum-of-sines was 1.8 cm

Table 1 The sequence of trials

Trial	Condition
1	20 s, eyes open, no imposed motion, unrestrained
2	20 s, eyes closed, no imposed motion, unrestrained
3	1 min, eyes open, room motion at 0.2 Hz, 1.5 cm amplitude, unrestrain
4	1 min, eyes closed, 0.2 Hz, 1.5 cm amplitude, unrestrained
5–8	10 min, eyes open, sum of 10 sines, 1.8 cm max amplitude, restrained
9	1 min, eyes open, 0.2 Hz, 1.5 cm, unrestrained
10	20 s, eyes open, no imposed motion, unrestrained
11	20 s, eyes closed, no imposed motion, unrestrained

Movement data analysis

We quantified displacements of the COP along the antero-posterior (AP) and medio-lateral (ML) axes by computing the variability (standard deviation of COP position) and the range (difference between maximum and minimum COP positions), and also computed the mean velocity for each trial. Analyses of movements during exposure to the sum-of-sines stimulus were based on criteria derived from the data and, therefore, are described below. For each significant effect in our ANOVAs, we estimated the effect size using partial eta squared (partial η^2).

Results

Subjective reports

Incidence and discontinuation

Seven participants (four females, three males) stated that they were motion sick and were placed in the Sick Group. Six participants discontinued (mean time of discontinuation = 22 min), and one reported sickness after completing all trials. Eleven participants stated that they were not sick and were placed in the Well Group; none of them discontinued.

No participants reported claustrophobia as a reason for discontinuation. However, one participant who discontinued reported both claustrophobia and sickness. Another sick participant reported anxiety but not claustrophobia at the time of discontinuation.

Motion sickness and claustrophobia history

All of the participants in the Sick group reported having been motion sick in the past, as opposed to only 27% of participants in the Well group. In the Sick group, 28.5% reported having been claustrophobic in the past, compared with 45.5% of participants in the Well group.

Simulator sickness questionnaire

We computed the Total Severity score for each participant in the recommended manner (Kennedy et al. 1993). The distribution of post-test SSQ scores was positively skewed, so we used Mann–Whitney *U* tests, and Wilcoxon Signed Rank Tests to compare unpaired and paired samples, respectively. Because each data set was used twice, we adjusted the alpha criterion to 0.025. For pre-test scores, the Sick ($M = 12.29$, $SD = 18.03$) and Well ($M = 9.86$, $SD = 10.35$) groups did not differ, $U = 37$, $P > 0.025$. Differences between pre-test and post-test scores were significant for the Well group, $z = -2.51$, $P < 0.025$, and for the Sick group, $z = -2.37$, $P < 0.025$. Finally, SSQ scores at post-test were significantly higher for the Sick group ($M = 121.82$, $SD = 58.37$) than for the Well group ($M = 27.20$, $SD = 32.52$), $U = 5.50$, $P < 0.025$.

Claustrophobia questionnaire

Following Radomsky et al. (2001), we computed overall pre-test and post-test claustrophobia scores for each participant. The data distributions were not skewed and the variances were homogeneous. Thus, we conducted a two-factor Time (pre-test vs. post-test) × Group (Sick vs. Well) ANOVA with repeated measures on the first factor. For the Sick group, mean claustrophobia scores were 25.43 ($SD = 8.87$) at pre-test and 46.14 ($SD = 14.55$) at post-test. For the Well group, mean claustrophobia scores were 17.45 ($SD = 11.67$) at pre-test and 12.55 ($SD = 8.98$) at post-test. The ANOVA revealed significant main effects for Time and for Group, each $F(1, 16) > 10.31$, $P < 0.05$, partial $\eta^2 = 0.39$ and 0.54, respectively. The Time × Group interaction was also significant, $F(1, 16) = 27.10$, $P < 0.05$, partial $\eta^2 = 0.63$, revealing that passive restraint was not inherently aversive, and that claustrophobia increased only among the Sick group.

Relations between claustrophobia and motion sickness

We evaluated the possibility that scores on the SSQ and CLQ might be correlated among all participants. The correlation between SSQ and CLQ scores was not significant at pre-test, Spearman's rho, $rs(16) = 0.18$, *ns* (Fig. 2a), but was significant at post-test (Fig. 2b), Spearman's rho, $rs(16) = 0.80$, $P < 0.05$. In previous research, the strongest correlation between motion sickness and anxiety has been $r = 0.37$, for ratings of motion sickness severity and anxiety (Fox and Arnon 1988). Our strong post-test correlation raises questions about causal relations between motion sickness and claustrophobia, which will be addressed in the Discussion.

The influence of restraint on subjective reports

Our use of passive restraint might have lead to subjective symptoms independent of the occurrence of motion sickness. To assess this possibility we compared subjective reports from the present experiment with reports from a study in which participants were not

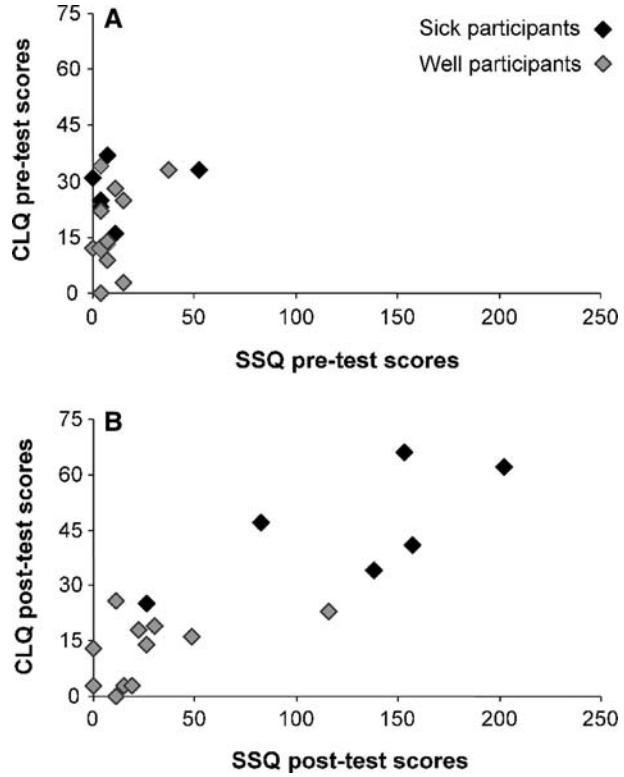

Fig. 2 Correlation between motion sickness and claustrophobia scores for **a** the pre-test, $r(16) = 0.18$, $P > 0.05$, and for **b** the post-test, $r(16) = 0.80$, $P < 0.05$. *Black*: Sick participants. *Gray*: Well participants

restrained (Bonnet et al. 2006). The incidence of motion sickness was the same in the two studies, $\chi^2(1) = 0.22$, *ns*. For the questionnaire data, we computed difference scores, by subtracting pre-test scores from post-test scores. Pre-post differences in SSQ scores were the same in the restrained and the unrestrained experiments, $U = 120.5$, *ns*. This result indicates that motion sickness severity was not influenced by the presence of restraint. By contrast, for claustrophobia, an Experiment (Restrained vs. Unrestrained) × Group (Sick vs. Well) ANOVA on the CLQ difference scores revealed a significant main effect of Experiment, $F(1, 32) = 7.37$, $P < 0.05$, partial $\eta^2 = 0.19$, showing that pre-post changes in claustrophobia severity were higher in the present, restrained experiment than in the study of Bonnet et al., in which participants were not restrained. The main effect of Group and the Experiment × Group interaction were also significant, each $F(1, 32) > 17.50$, $P < 0.05$, partial $\eta^2 = 0.35$ and 0.37, respectively. The interaction effect indicates that claustrophobia was related to restraint only among participants who became motion sick (Fig. 3). This result also confirms that motion sickness can occur in the absence of claustrophobia

Movement data

Spontaneous sway, unrestrained (Trials 1 and 2)

For each dependent variable, we performed two-factor Vision (eyes open vs. eyes closed) × Group (Sick vs. Well) ANOVAs with repeated measures on the first factor. The analyses revealed significant main effects of Vision on variability, velocity, and range in the AP axis, each $F(1, 16) > 5.78$, $P < 0.05$, partial $\eta^2 = 0.27$, 0.65, and 0.42, respectively. The main effect of Vision was also significant for variability and range in the ML axis, each $F(1, 16) > 5.05$, $P < 0.05$, partial $\eta^2 = 0.25$ and 0.24, respectively. For each significant effect, motion was greater when the eyes were closed (see Table 2). There were no significant main effects of Group, and none of the Group × Vision interactions were significant.

0.2 Hz stimulus, unrestrained (Trials 3 and 4)

Because of technical problems, we were unable to record postural data during Trial 4 for three participants (one from the Sick group, and two from the Well group). The following analyses include the remaining 15 participants.

For each dependent variable, we conducted a two-factor ANOVA on Vision (eyes open vs. eyes closed) × Group (Sick vs. Well), with repeated measures on the first factor. There were significant main effects of Vision on the variability and range of motion in the AP axis; both were greater when the eyes were open (Trial 3), than when they were closed (Trial 4), each $F(1, 13) > 6.95$, $P < 0.05$, partial $\eta^2 = 0.45$ and 0.35, respectively. The main effect of Vision was also significant for velocity in the ML axis, $F(1, 13) = 9.91$, $P < 0.05$, partial $\eta^2 = 0.43$, with a higher velocity in the eyes closed than in the eyes open condition. The significant main effects of Vision are summarized in Table 3. There was a significant Group × Vision interaction for velocity in the AP axis, $F(1, 13) = 5.17$, $P < 0.05$, partial $\eta^2 = 0.28$. Sick participants moved more rapidly when their eyes were closed ($M = 1.04$ cm s^{-1}, $SD = 0.39$ for Trial 3 and $M = 1.11$ cm s^{-1}, $SD = 0.33$ for Trial 4), while Well participants moved more rapidly when their eyes were open ($M = 1.05$ cm s^{-1}, $SD = 0.23$ for Trial 3 and $M = 0.97$ cm s^{-1}, $SD = 0.23$ for Trial 4).

Sum-of-sines stimulus, restrained (Trials 5–8)

Due to discontinuation, we did not have the same amount of postural data for each participant in Trials

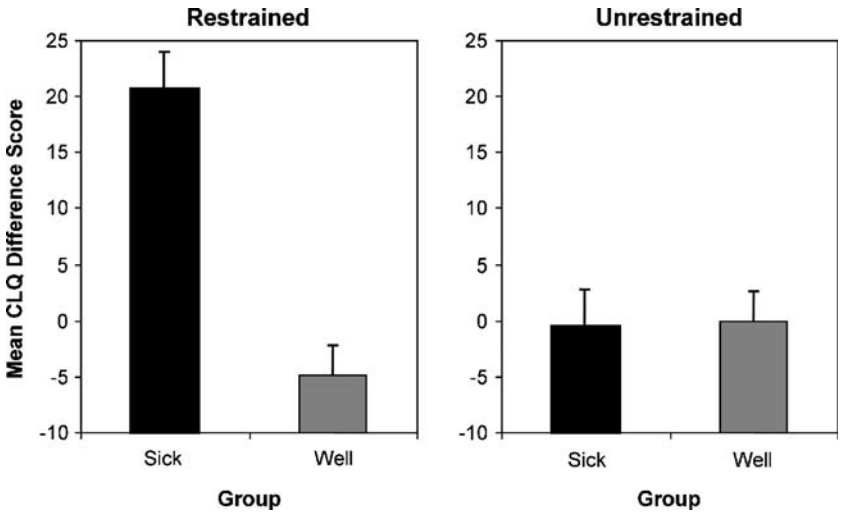

Fig. 3 The significant interaction Group (Sick vs. Well) × Experiment (Restrained vs. Unrestrained) for mean difference scores (post-test–pre-test) for claustrophobia. Data for the unrestrained experiment are from Bonnet et al. (2006). The error bars represent standard error

Table 2 Means and standard deviations for the significant main effects of vision on spontaneous sway

	Variability AP (cm)		Velocity AP (cm s^{-1})		Range AP (cm)		Variability ML (cm)		Range ML (cm)	
	Trial 1	Trial 2	Trial 1	Trial 2	Trial 1	Trial 2	Trial 1	Trial 2	Trial 1	Trial 2
Mean	0.33	0.48	0.84	1.17	1.52	2.41	0.18	0.23	0.97	1.30
Standard deviation	0.18	0.18	0.20	0.36	0.61	0.89	0.08	0.08	0.43	0.38

Trial 1: eyes open, Trial 2: eyes closed

Table 3 Means and standard deviations for the significant main effects of vision on postural motion during exposure to the sinusoidal stimulus (0.2 Hz)

	Variability AP (cm)		Range AP (cm)		Velocity ML (cm s^{-1})	
	Trial 3	Trial 4	Trial 3	Trial 4	Trial 3	Trial 4
Mean	0.61	0.43	3.00	2.51	0.56	0.58
Standard deviation	0.25	0.15	0.84	0.69	0.15	0.13

Trial 3: eyes open, Trial 4: eyes closed

5–8. 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. One participant discontinued during Trial 5, and so, was excluded from the following analyses.

Overall movement When the sum-of-sines was presented, we observed displacement of the center of pressure despite the fact that participants were restrained. We began by evaluating Sick/Well differences in overall movement, that is, computing the means across trials for each participant. Independent *t* tests conducted on these means for each dependent variable showed no significant differences in overall motion between the Sick and Well groups, each $t(15) > -1.25$, *ns*.

Evolution of movement during exposure We next evaluated the hypothesis that there might be differences between the Sick and Well groups in the evolution of COP displacements over time, using the procedure developed by Bonnet et al. (2006). We selected three windows from the data, each of which was 2 min in duration. Due to discontinuation, participants in the Sick and Well groups did not have the same duration of exposure to the sum-of-sines stimulus. We judged it to be important to ensure that the windows for the Sick and Well groups represented similar exposure durations. To ensure this we tied the selection of windows for the Well group to the mean exposure duration of the Sick group. For the Sick group, we chose the first, the middle, and the final 2 min 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 to 360 s of Trial 6, and the final window was from 481 to 600 s of Trial 7. The windows selected for the Well participants were based on the 22 min mean exposure of participants in the Sick group. Accordingly, for the Well group, we took the first 2 min of Trial 5, the final 2 min of Trial 5, and the final 2 min of Trial 6. For each of the dependent variables, we conducted separate 2-factor Group (Sick vs. Well) × Window (first, middle, last) ANOVAs with repeated measures on the second factor.

The windowing analysis revealed significant main effects of Window on the variability and range of COP displacements in both the AP and ML axes, and on velocity in the ML axis, each $F(2, 30) > 3.35$, $P < 0.05$, partial $\eta^2 = 0.23$, 0.25, 0.18, 0.21, and 0.31, respectively (Fig. 4). Each of these effects indicates that movement increased over time during exposure to the sum-of-sines. There were no significant main effects of Group, each $F(1, 15) < 1$, *ns*. We found significant Group × Window interactions for variability in the AP axis, and for velocity and range in the ML axis, each $F(2, 30) > 3.45$, $P < 0.05$, partial $\eta^2 = 0.19$, 0.30, and 0.20, respectively (Fig. 5). Each of these interactions revealed that in the Sick group movement increased over time, while in the Well group movement tended to be stable over time. Representative data from the three windows of the sum-of sines trials are presented in Fig. 6 for Sick and Well participants.

Comparison of sway before and after the sum-of-sines stimulus

Due to discontinuation of sick participants, the comparison between pre-exposure and post-exposure sway was conducted only for the Well group ($N = 11$). For each dependent variable, we conducted three *t*-tests comparing trials having similar conditions before and after the sum-of-sines trials (Trial 1 vs. 10, Trial 2 vs. 11 and Trials 3 vs. 9). Due to technical problems we were unable to record postural data during Trials 10 and 11 for one participant of the Well group. Consequently, the following analyses include 10 participants for the comparisons between Trials 1 and 10, and Trials 2 and 11.

For spontaneous sway with the eyes open (Trial 1 vs. 10), the analyses revealed significant differences in the ML axis for variability and range, each $t(9) < -2.72$, $P < 0.05$. In both cases, values were higher for Trial 10 (mean variability = 0.23 cm; mean range = 1.32 cm) than for Trial 1 (mean variability = 0.18 cm; mean range = 0.99 cm). For responses to the 0.2 Hz stimulus motion with the eyes open (Trial 3 vs. Trial 9), we found a significant effect for velocity in the ML axis, $t(10) = -2.68$, $P < 0.05$. Again, values were higher for the post-exposure trial (mean velocity = 0.61 cm s^{-1}) than for the pre-exposure trial (mean velocity = 0.53 cm s^{-1}). All other comparisons were not significant, $-1.91 < ts < 0.32$, *ns*.

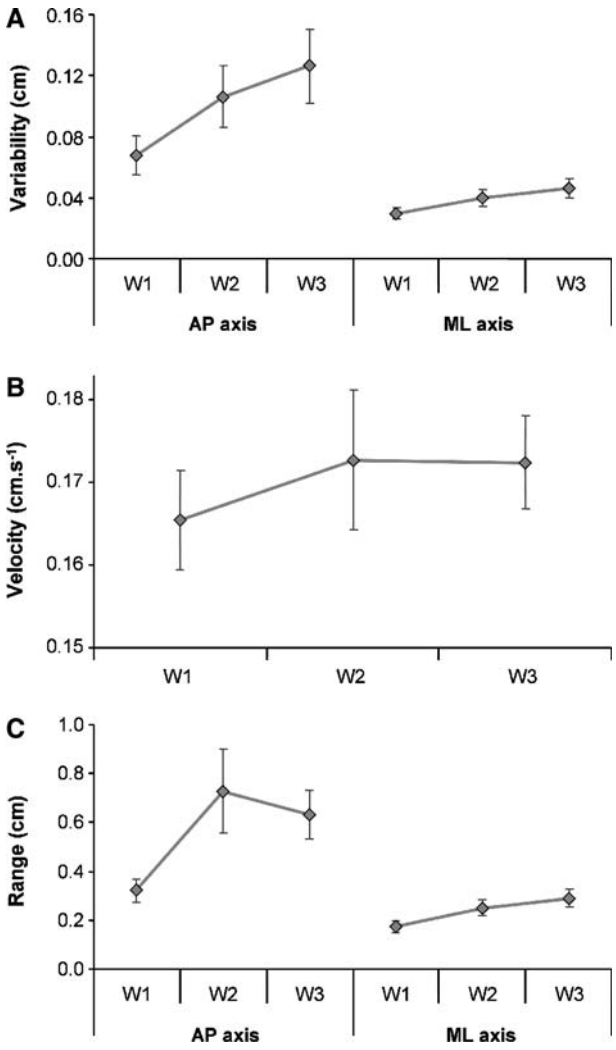

Fig. 4 Main effects of windows on kinematics of the center of pressure when restrained participants were exposed to the sum-of-sines stimulus. **a** Variability in the anterior-posterior (AP) and medio-lateral (ML) axes. **b** Velocity in the ML axis. **c** Range in the AP and ML axes. W1, W2, and W3 refer to the first, middle, and last windows, respectively. The error bars represent standard error

Correlations between variables

Because our dependent variables on postural motion were computed from a single data set (displacements

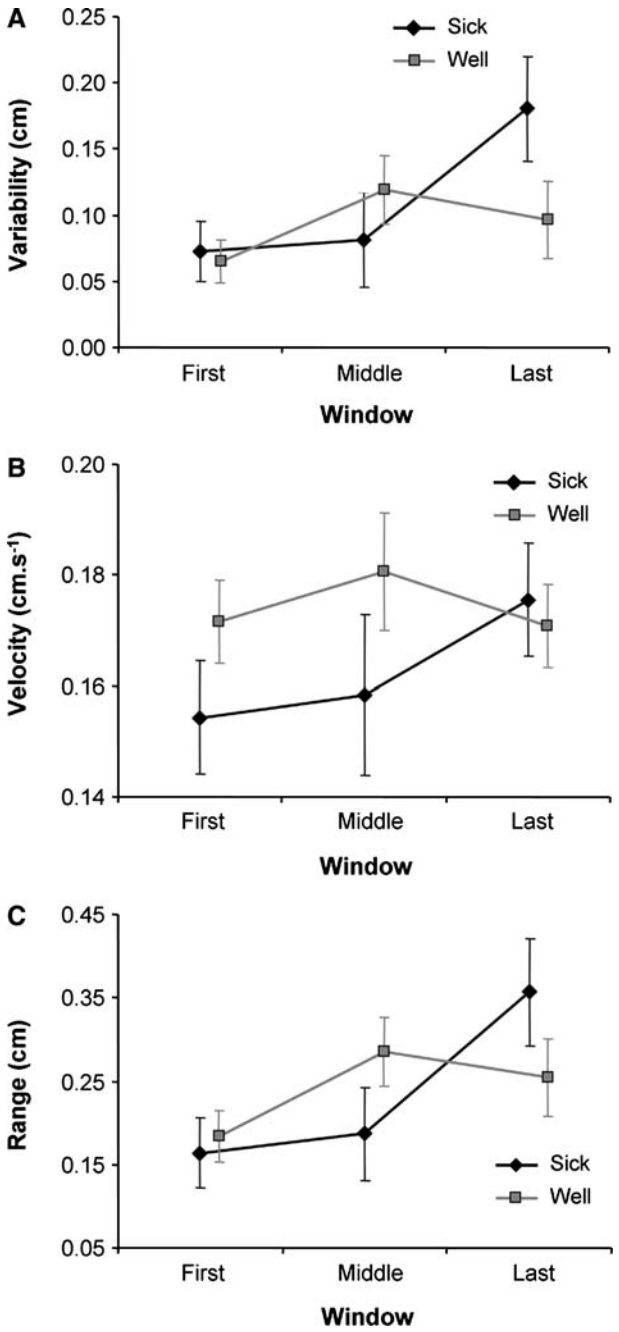

Fig. 5 Significant Group × Window interactions for center of pressure data when restrained participants were exposed to the sum-of-sines stimulus. **a** Variability of position in the AP axis. **b** Velocity of movement in the ML axis. **c** Range of movement in the ML axis. W1, W2, and W3 refer to the first, middle, and last windows, respectively. The error bars represent standard error

of the center of pressure), we expected them to be at least moderately correlated with each other. We conducted correlation analyses over the means of each participant for every trial. The highest correlation coefficients were between range and variability in the AP (0.79) and ML (0.78) axes, and between velocity in the AP axis and velocity in the ML axis (0.67). All other correlation coefficients ranged between 0.05 and 0.52. The magnitude of the correlations mandates caution in interpreting the number of significant effects in our analyses. As one example, the three effects illustrated in Fig. 5 cannot be assumed to be independent (Smart et al. 2002)

Discussion

Standing participants were passively restrained and exposed to visual motion that simulated the amplitude and frequency of ordinary (i.e., unrestrained) body sway. Motion sickness occurred despite the fact of passive restraint. Prior to the onset of motion sickness, displacements of the center of pressure evolved differently among participants who eventually became sick, relative to those who did not. The severity of motion sickness symptoms was similar to studies in which participants have not been restrained. Claustrophobia symptoms increased more in the present experiment than in an earlier study in which participants were not restrained (Bonnet et al. 2006). However, the severity of claustrophobia appeared to be related more closely to motion sickness than to restraint, per se.

Subjective reports and the influence of restraint

Seven of eighteen participants (39%) reported motion sickness during or after exposure to the sum-of-sines optical stimulus. Both the incidence and severity of motion sickness during passive restraint did not differ from previous studies in which participants were not restrained (Bonnet et al. 2006). We can conclude, following several previous studies (e.g., Graybiel and Miller 1970; O'Hanlon and McCauley 1974; Warwick-Evans et al. 1998), that attempts to passively restrain a person do not guarantee immunity from motion sickness. By contrast, the severity of claustrophobia was significantly greater during passive restraint (in the present study) than in the absence of passive restraint (Bonnet et al. 2006). The increase in claustrophobia when participants were restrained is not surprising. However, a significant interaction revealed that claustrophobia was not a general consequence of passive restraint. Rather, claustrophobia symptoms increased only for restrained participants who became motion sick. This finding raises questions about causal relations between motion sickness and claustrophobia, which are discussed below (Claustrophobia, motion sickness, and postural instability).

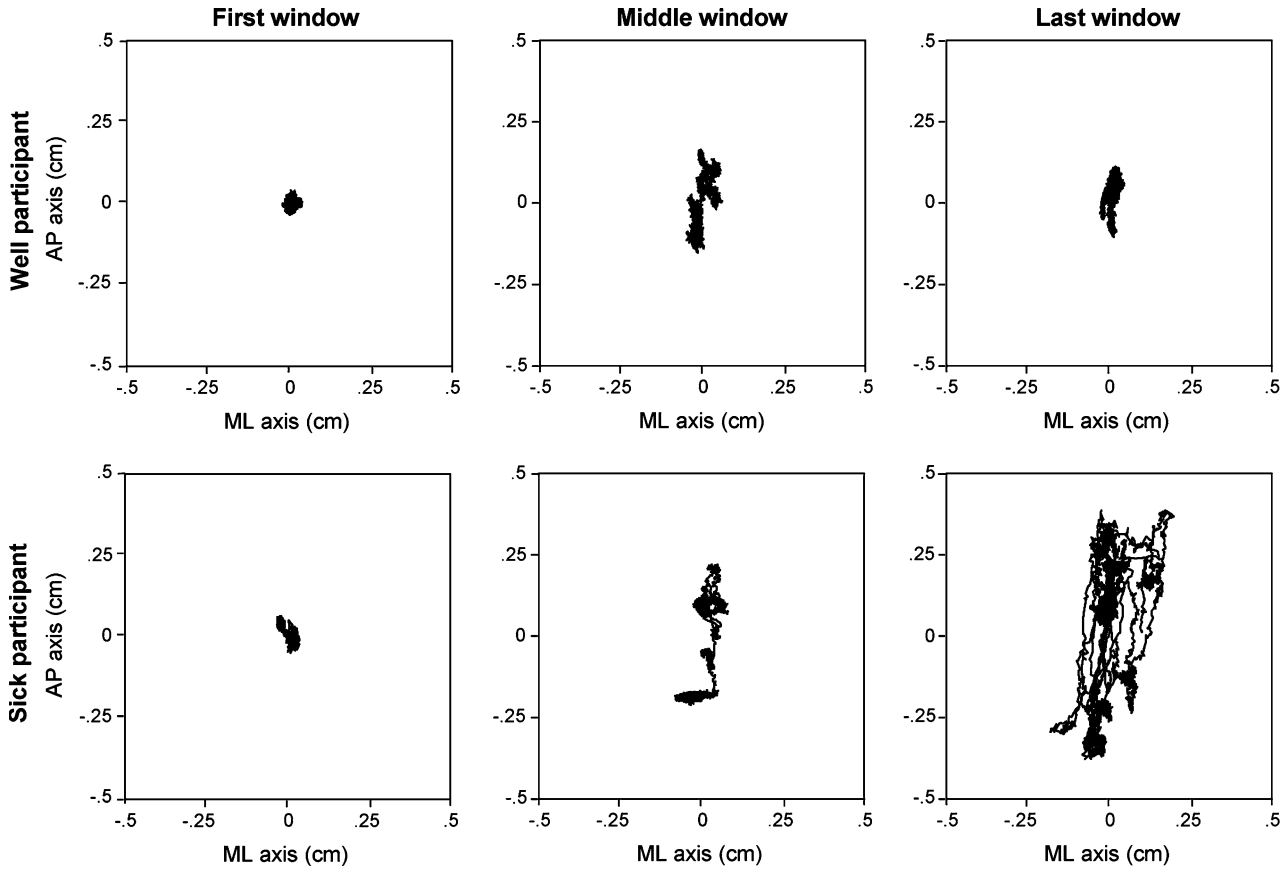

Fig. 6 Center of pressure data for representative participants at the beginning (*left*), middle (*center*) and end (*right*) of exposure to the sum-of-sines stimulus, during restraint. *Top*: A participant who did not report motion sickness. *Bottom*: A participant who reported motion sickness. The data were collected before sickness onsets

Movement during the sum-of-sines trials

It must be stressed that all movement data were collected before participants reported any subjective symptoms of motion sickness. During passive restraint and before participants reported any subjective symptoms of motion sickness, there were reliable differences in the evolution of body movement between participants who reported motion sickness and those who did not. Participants in the Sick group exhibited steady increases in the variability, velocity, and range of body movement during exposure to the sum-of-sines stimulus. By contrast, in the Well group, these variables tended to be stable over time. The fact that movements evolved differently among Sick and Well participants prior to the onset of motion sickness is consistent with the postural instability theory of motion sickness (Riccio and Stoffregen 1991).

While there are essential similarities in results across studies, some of the results of the present study also differ from previous studies in important ways. First, previous studies have found significant differences between Sick and Well groups for overall sway during exposure to the sum-of-sines (Bonnet et al. 2006; Smart et al. 2002; Stoffregen et al. 2000; Stoffregen and Smart 1998). In the present study, there were no significant main effects of Group on movement of restrained participants during exposure to the sum-of-sines. Second, in the only previous study that included an analysis of changes in sway during exposure to the sum-of-sines, Bonnet et al. (2006) found no significant interactions between Group and Window. This contrasts with the present study, in which we found significant Group × Window interactions (Fig. 5). For Bonnet et al., the presence of main effects for Group coupled with the absence of interactions between Group and Window suggested that differences between Sick and Well participants in postural responses to the sum-of-sines were present at the onset of stimulus motion, and did not develop during exposure. In the present study, we must reach the opposite conclusion. During passive restraint, the initial response to sum-of-sines optic flow was the same for the Sick and Well groups, and Group-related differences in movement developed during

exposure. Differences in the pattern of results between the present study and previous studies may be related to our use of passive restraint. One possible interpretation, suggested by our claustrophobia data, is presented below (Claustrophobia, motion sickness, and postural instability).

Sway before exposure to the sum-of-sines

In the present study, Trials 1–4 were identical to those used in our previous research (Bonnet et al. 2006; Smart et al. 2002; Stoffregen et al. 2000, Stoffregen and Smart 1998) in the sense that participants were not restrained during these trials. Each of these studies found significant main effects of Group (Sick vs. Well) for spontaneous sway (Trials 1 and 2) and/or for sway during exposure to the 0.2 Hz optic flow stimulus (Trials 3 and 4). No such main effects of Group were found in the present study.

We found one significant interaction between Group and Vision. During exposure to the 0.2 Hz optic flow stimulus (Trials 3 and 4), the velocity of sway in the AP axis was higher for the Sick group than for the Well group, when participants' eyes were closed. The effect indicates that there was at least one difference in postural motion between Sick and Well participants before they were exposed to the nauseogenic stimulus (the sum-of-sines). In this sense, this significant interaction is consistent with previous studies, each of which has found at least one significant effect involving Group for postural motion before exposure to the sum-of-sines. However, we do not have a clear interpretation of this effect: We do not know why the presence versus absence of vision should have a different effect on sway for sick and well participants when the room was moving.

Claustrophobia, motion sickness, and postural instability

As might have been expected, claustrophobia symptoms increased during passive restraint. However, this effect was not observed for each participant: the severity of claustrophobia interacted with the incidence of motion sickness, such that claustrophobia increased (from pre- to post-test) only among participants who reported motion sickness. We can conclude that passive restraint did not guarantee claustrophobia. A more interesting issue raised by this result relates to causal relations between claustrophobia and motion sickness. In the context of the overall motion sickness literature, it is clear that motion sickness can occur in the absence of claustrophobia (e.g., Bonnet et al. 2006). However, the possibility remains that claustrophobia may have been causally related to motion sickness in the present study. Our interpretation of this possibility is based on the severity of motion sickness and claustrophobia symptoms and on relations between subjective reports and movement data. During passive restraint, feelings of claustrophobia may be related to functional characteristics of perception and action. Berlyne (1960) suggested that complete stasis of the body may be inherently aversive. From a perception-action perspective, complete stasis is undesirable because it eliminates information about system dynamics that is made available through motion (Riccio et al. 1997). The reduction of information about the dynamics of the animal-environment system might explain the aversion that many people feel for situations in which their movements are restricted. Thus, the correlation between claustrophobia and the incidence of motion sickness (together with the changes in body motion observed among Sick participants) may indicate that some individuals are more reliant on these subtle exploratory motions, such that they attempted (unsuccessfully) to generate them even when passively restrained. In this way, claustrophobia might have been an indirect cause of motion sickness, if it induced claustrophobic participants to move differently than participants who were not claustrophobic. One way to test this interpretation would be to bring these same participants back to the laboratory and assess their susceptibility to motion sickness (and postural instability) in the absence of passive restraint. The hypothesis that claustrophobia could be an indirect cause of motion sickness would be supported if participants who had become sick in the restrained condition did not become sick in the unrestrained condition. A problem with this design is that the incidence of sickness might be higher among participants who had been sick in the earlier experiment (e.g., Johnson 2005). Another approach would be to subject standing participants to passive restraint in the absence of any imposed motion (e.g., without the sum-of-sines optic flow stimulus). Passively restrained participants who became claustrophobic might experience motion sickness and exhibit unstable movement in the absence of any imposed motion. Such effects would be consistent with the idea that claustrophobia and motion sickness are related to individual differences in reliance on exploratory movement.

Conclusion

We used passive restraint in an attempt to eliminate actively controlled movement. While under passive

restraint, our participants moved, and so this attempt failed, that is, we were not able to estimate the incidence of motion sickness in the absence of actively controlled body movement. To test this key prediction of the postural instability theory of motion sickness, alternative methods may be needed. For example, people might be exposed to 0.1–0.4 Hz optic flow during water immersion at neutral buoyancy. At neutral buoyancy, the body is passively stable; uncontrolled movements tend to be damped by the viscosity of the water. The absence of motion sickness in such conditions would be compatible with the fact that motion sickness is rare among swimmers and scuba divers. While we were not able to eliminate actively controlled movement, we succeeded in showing that motion sickness was preceded by changes in movement during restraint. This finding is compatible with our predictions, and it may explain previous findings that passive restraint sometimes does not prevent motion sickness (e.g., Graybiel and Miller 1970; Warwick-Evans et al. 1998).

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References

Bensel CK, Dzendolet E (1968) Power spectral density analysis of the standing sway of males. Percept Psychophys 4:285–288

Berlyne DE (1960) Conflict, arousal, and curiosity. McGraw Hill, New York

Bonnet CT, Faugloire E, Riley MA, Bardy BG, Stoffregen TA (2006) Motion sickness preceded by unstable displacements of the center of pressure. Hum Mov Sci (in press)

Draper MH, Viirre ES, Gawron VJ, Furness TA (2001) The effects of virtual image scale and system delay on simulator sickness within head-coupled virtual environments. Hum Factors 43:129–146

Duh HB, Parker D, Philips JO, Furness TA (2004) "Conflicting" motion cues to the visual and vestibular self-motion systems around 0.06 Hz evoke simulator sickness. Hum Factors 46:142–153

Ebenholtz SM, Cohen MM, Linder BJ (1994) The possible role of nystagmus in motion sickness: a hypothesis. Aviat Space Environ Med 65(11):1032–1035

Fox S, Arnon I (1988) Motion sickness and anxiety. Aviat Space Environ Med 59(8):728–733

Fox RA, Daunton NG, Coleman J (1982) Susceptibility of the squirrel monkey to several different motion conditions. Neurosci Abstr 8:698

Golding JF, Finch MI, Stott JR (1997) Frequency effect of 0.35–1.0 Hz horizontal translational oscillation on motion sickness and the somatogavic illusion. Aviat Space Environ Med 68:396–402

Graybiel A, Miller EF (1970) Off-vertical rotation: a convenient precise means of exposing the passive human subject to a rotating linear acceleration vector. Aerosp Med 41(4):407–410

Guignard JC, McCauley ME (1990) The accelerative stimulus for motion sickness. In: Crampton GH (ed) Motion and space sickness. CRC Press, Boca Raton, pp 123–152

Johnson DM (2005) Introduction to and review of simulator sickness research (ARI Res. Rep. 1832). Arlington, VA: U.S. Army Research Institute for the Behavioral and Social Sciences

Johnson WH, Mayne JW (1953) Stimulus required to produce motion sickness; restriction of head movement as a preventive of airsickness; field studies on airborne troops. J Aviat Med 24(5):400–411

Johnson WH, Taylor NBG (1961) Some experiments on the relative effectiveness of various types of accelerations on motion sickness. Aerospace Med 32(3):205–208

Kennedy RS, Lilienthal MG (1994) Measurement and control of motion sickness aftereffects from immersion in virtual reality. In: Proceedings of "Virtual reality and medicine: The cutting edge", Inc SIG-Advanced Applications, Inc., New York, pp 111–119

Kennedy RS, Lane NE, Berbaum KS, Lilienthal MG (1993) Simulator sickness questionnaire: an enhanced method for quantifying simulator sickness. Int J of Aviat Psychol 3:203–220

Lackner JR, Graybiel A, DiZio PA (1991) Altered sensorimotor control of the body as an etiological factor in space motion sickness. Aviat Space Environ Med 62(8):765–771

Lawther A, Griffin MJ (1988) Motion sickness and motion characteristics of vessels at sea. Ergonomics 31(10):1373–1394

Lee DN, Lishman JR (1975) Visual proprioceptive control of stance. J Hum Movement Stud 1:87–95

Mills KL, Griffin MJ (2000) Effect of seating, vision and direction of horizontal oscillation on motion sickness. Aviat Space Environ Med 71(10):996–1002

Money K (1970) Motion sickness. Physiol Rev 50(1):1–39

O'Hanlon JF, McCauley ME (1974) Motion sickness incidence as a function of the frequency and acceleration of vertical sinusoidal motion. Aerosp Med 45(4):366–369

Oman CM (1982) A heuristic mathematical model for the dynamics of sensory conflict and motion sickness. Acta Otolaryngol Suppl 392:1–44

Owen N, Leadbetter AG, Yardley L (1998) Relationship between postural control and motion sickness in healthy subjects. Brain Res Bull 47(5):471–474

Radomsky AS, Rachman S, Thordarson DS, McIsaac HK, Teachman BA (2001) The claustrophobia questionnaire. J Anxiety Disord 15(4):287–297

Reason JT (1978) Motion sickness adaptation: a neural mismatch model. J R Soc Med 71(11):819–829

Riccio GE, Stoffregen TA (1991) An ecological theory of motion sickness and postural instability. Ecol Psycho 3:195–240

Riccio GE, McDonald V, Peters BT, Layne CS, Bloomberg JJ (1997) Understanding Skill in EVA Mass Handling. vol I: Theoretical and operational foundations. NASA Technical Paper #3684, National Aeronautics and Space Administration, Washington D.C

Smart LJ, Stoffregen TA, Bardy BG (2002) Visually-induced motion sickness predicted by postural instability. Hum Factors 44(3):451–465

Stoffregen TA (1985) Flow structure versus retinal location in the optical control of stance. J Exp Psychol Hum Percept Perform. 11(5):554–565

Stoffregen TA, Riccio GE (1991) An ecological critique of the sensory conflict theory of motion sickness. Ecol Psychol 3:159–194

Stoffregen TA, Smart LJ (1998) Postural instability precedes motion sickness. Brain Res Bull 47(5):437–448
Stoffregen TA, Hettinger LJ, Haas MW, Roe M, Smart LJ (2000) Postural instability and motion sickness in a fixed-base flight simulator. Hum Factors 42(3):458–469
Strogatz SH (1993) Nonlinear dynamics and chaos. Addison-Wesley, Reading
Tucker GJ, Reinhardt RF (1967) Airsickness and anxiety. Aerosp Med 38(8):855–858
Warwick-Evans LA, Symons N, Fitch T, Burrows L (1998) Evaluating sensory conflict and postural instability theories of motion sickness. Brain Res Bull 47(5):465–469