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Self-Induced Motion Sickness and Body Movement During Passive Restraint

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While standing on a force platform, participants were subjected to passive restraint by being strapped to a vertical surface at the head, shoulders, hips, and knees. Despite the restraint, small movements of the body were possible. During restraint, there was no imposed motion of any kind. Twenty-two percent of participants became motion sick, suggesting that passive restraint during stance may be inherently nauseogenic. Motion sickness was preceded by changes in displacements

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of the center of pressure. During passive restraint, the amplitude of center of pressure displacements tended to increase over time for the participants who later reported motion sickness, whereas for participants who did not report motion sickness, center of pressure displacements tended to be stable over time. The results are consistent with predictions made by the postural instability theory of motion sickness (Riccio & Stoffregen, 1991).

Motion sickness is commonly associated with imposed motion, such as occurs when we are passengers in real or simulated vehicles. Indeed, it is this association with imposed motion that accounts for the name, *motion* sickness. For example, Money (1990) argued that motion sickness did not occur before the invention of vehicles such as ships, automobiles, and aircraft. Yet there have been occasional reports of persons stating that they were motion sick in the absence of imposed motion (i.e., either inertial or visual motion). Perhaps the most prominent example occurs in spaceflight. In orbital flight above the atmosphere, people are weightless and there is no imposed motion of the body relative to the spacecraft. Despite the absence of imposed motion, astronauts and cosmonauts often experience what appears to be motion sickness. During spaceflight, sickness is associated with self-generated movements, such as head turns (e.g., Oman, Lichtenberg, Money, & McCoy, 1986). Under terrestrial conditions, motion sickness is possible without imposed motion and can be associated with voluntary movement. For example, motion sickness sometimes occurs when standing up and wearing inverted prism spectacles (e.g., Dolezal, 1982) or with vigorous rotation of the torso in stance (Bouyer & Watt, 1996).

Motion sickness can also occur in the absence of imposed motion, voluntary movement, or any perceptual manipulation. Perhaps the most carefully documented instance was reported by Smart, Pagulayan, and Stoffregen (1998). They conducted experiments in which standing participants were asked to look at stationary targets (objects) presented at different distances. Participants were instructed to stand comfortably, with the feet side by side. Each trial lasted 70 s, and there were up to 57 trials. The purpose of the study was to investigate possible relations between the distance of fixation and the amplitude of postural sway. The study was not about motion sickness, that is, the experimenters did not intend or expect participants to become motion sick and did not inform participants that motion sickness might occur. Despite the seemingly innocuous experimental situation, 21% of participants (9 of 42) spontaneously reported to the experimenters that they felt motion sick. Among participants who stated that they were motion sick, symptoms were similar to the ones found in the motion sickness literature. Some of the participants who reported motion sickness were asked to fill out a standard instrument used to assess symptom severity (the Simulator Sickness Questionnaire; Kennedy, Lane, Berbaum, & Lilienthal, 1993). Scores on this instrument closely resembled the scores of military personnel who became motion sick in flight simulators.

Some symptoms of motion sickness are associated with other, unrelated conditions, including various illnesses, eyestrain, headache, and food poisoning. This reflects the fact that there is no precise, exclusionary definition of motion sickness (e.g., see the debate over whether the symptoms experienced in weightlessness constitute motion sickness; Oman et al., 1986). Similarly, there is no measurement that gives a definitive indication of whether any given individual is motion sick rather than experiencing any other disorder with similar symptoms. In the majority of motion sickness research, the judgment that a person is motion sick is made primarily on the basis of their self-report, either in their own words or in responses to questionnaires (e.g., Bouyer & Watt, 1996; Dichgans & Brandt, 1973; Guignard & McCauley, 1990; Kennedy et al., 1993; Lawther & Griffin, 1986; Lee & Lishman, 1975; Morrissey & Bitner, 1986; Oman et al., 1986; Stoffregen, 1985; Wiker, Kennedy, McCauley, & Pepper, 1979). In the present study, we accepted participants' direct, explicit statements that they were motion sick.

MOTION SICKNESS AND PASSIVE RESTRAINT

In classical theories of motion sickness etiology, nauseogenic situations are characterized in terms of sensory conflict (e.g., Duh, Parker, Philips, & Furness, 2004; Oman, 1982; Reason, 1978). According to this view, patterns of perceptual stimulation that differ from patterns expected on the basis of past experience constitute sensory conflict. When the magnitude of these differences exceeds some threshold, motion sickness occurs. Many forms of sensory conflict have been proposed in the literature (e.g., input conflict or output conflict), but Riccio and Stoffregen (1991) argued that all forms of sensory conflict necessarily entail the idea that current patterns of multisensory stimulation are discrepant relative to patterns expected on the basis of previous experience. In presentations of the sensory conflict theory, postural instability is sometimes seen as a consequence— a symptom—of motion sickness but has no role in causing motion sickness (e.g., Cobb, 1999). In their theory of motion sickness, Riccio and Stoffregen argued that postural instability is a necessary and sufficient precursor of motion sickness.

In the experiment of Smart et al. (1998), it is unclear how sensory conflict could explain the occurrence of sickness because participants were not exposed to any imposed motion or unusual (i.e., unexpected) perceptual patterns. By contrast, the postural instability theory of motion sickness (Riccio & Stoffregen, 1991) offers an explanation of the results of Smart et al. (1998). The postural instability theory predicts that motion sickness will be preceded by instabilities in the control of posture. Smart et al. (1998) collected data on postural sway and observed that sway amplitude was significantly greater among participants who reported motion sickness. However, participants were not told to discontinue participation at symptom onset, and so it was not possible to know whether

patterns of postural motion preceded the onset of symptoms. The results of Smart et al. (1998) suggest that, in some situations, terrestrial motion sickness may be induced by factors other than imposed motion. The same suggestion arises from a recent study by Faugloire, Bonnet, Riley, Bardy, and Stoffregen (2007), who sought to evaluate a central prediction of the postural instability theory of motion sickness. 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), people 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 & Mayne, 1953; Johnson & Taylor, 1961; Lackner, Graybiel, & DiZio, 1991; Mills & Griffin, 2000). However, in other studies, motion sickness has been observed among persons subjected to passive restraint (e.g., Graybiel & Miller, 1970; Warwick-Evans & Beaumont, 1995; Warwick-Evans, Symons, Fitch, & Burrows, 1998). Thus, previous studies are ambiguous with respect to the claim that passive restraint prevents motion sickness. However, 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 & Stoffregen, 1991, p. 223). This possibility provides a strong motivation to measure any movements that might occur during passive restraint. Unlike previous studies relating motion sickness to passive restraint, Faugloire et al. (2007) included measurements of body movement. In their study, passively restrained participants were exposed to visual motion that is known to induce motion sickness in unrestrained persons (Bonnet, Faugloire, Riley, Bardy, & Stoffregen, 2006; Smart, Stoffregen, & Bardy, 2002; Stoffregen & Smart, 1998). They found that motion sickness occurred during passive restraint (incidence = 39%) and that motion sickness was preceded by significant changes in displacements of the center of pressure (relative to participants who did not become motion sick). During exposure to an optic flow stimulus, the variability, velocity, and range of movement tended to increase over time among participants who later reported motion sickness, whereas these same variables tended to be stable over time among participants who did not become sick. Faugloire et al. concluded that motion sickness was possible during passive restraint, that passive restraint did not preclude all movement, and that motion sickness during passive restraint was preceded by changes in body movement.

Faugloire et al. (2007) showed that motion sickness is preceded by changes in displacement of the center of pressure even during passive restraint. However,

they did not definitively establish whether postural instability and/or motion sickness during passive restraint were induced by visual motion. Postural instability and/or motion sickness might have been induced by the imposed optic flow. Alternately, instability and/or motion sickness might have been induced by passive restraint per se or by consequences of passive restraint, such as claustrophobia. In the present study, we did not attempt to distinguish between direct and secondary effects of passive restraint.

Faugloire et al. (2007) recognized that passive restraint might give rise to claustrophobia and, accordingly, they assessed the incidence and severity of claustrophobia. There was a significant increase in the severity of subjective symptoms of claustrophobia following passive restraint. However, this increase was not general; it occurred only among participants who reported motion sickness. This finding raised the possibility that passive restraint might have played a causal role in the etiology of motion sickness, independent of any role played by imposed visual motion.[1]

In the present study, we sought to determine whether motion sickness could be induced by passive restraint of standing persons in the absence of any imposed motion. We also evaluated the hypothesis that motion sickness would be preceded by changes in displacements of the center of pressure. Finally, we assessed relations between motion sickness and claustrophobia in the context of passive restraint. The apparatus, experimental design, and procedure were identical to those used by Faugloire et al. (2007) with one exception. In the present experiment, participants were not subjected to imposed motion of any kind during passive restraint.

METHOD

Participants

Eighteen students from the University of Minneapolis (graduate and undergraduate) participated in this experiment. There were 8 males and 10 females, ranging in age from 19 to 29 years with a mean age of 22.39 years. Participants ranged in weight from 55.34 kg to 111.12 kg with a mean weight of 68.44 kg. Participants ranged in height from 1.58 m to 2.01 m with a mean height of 1.74 m. No participants reported any history of recurrent falls or vestibular (inner ear) dysfunction, and all had normal or corrected to normal vision. They

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[1]We take as given the idea that claustrophobia is not related to motion sickness etiology in the absence of passive restraint (e.g., on ships, in automobiles). In a study of motion sickness during unrestrained stance, Bonnet et al. (2006) measured claustrophobia symptoms before and after exposure to potentially nauseogenic visual motion. There were no pre-post differences in claustrophobia scores for participants who became motion sick or for those who did not.

were all able to stand on one foot for 30 s with eyes open. Participants stated that they were in good health and were not pregnant. Participants had not participated in any other studies of motion sickness.

Apparatus

The experiment was conducted in a moving room (Lee & Lishman, 1975; Smart et al., 2002) consisting of a cubical frame (2.44 m on a side) mounted on wheels that could be moved in one axis along rails by an electric motor under computer control (Figure 1A). 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 a large, detailed map of the continental United States (53 × 80 cm; $19° \times 28°$). Illumination was provided by four incandescent floodlights mounted on the interior walls of the room and oriented so that shadows were minimized.
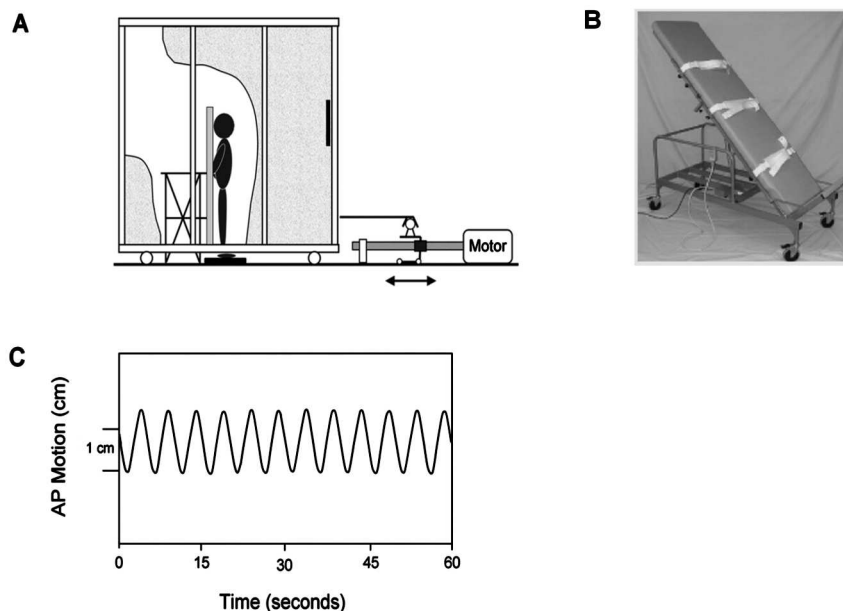

FIGURE 1   A: The moving room and the tilt table. B: Manufacturer's photograph of the tilt table. The straps in the photograph are not the ones used in our experiment. C: Motion pattern of the moving room during Trials 3, 4, and 9: Motion at 0.2 Hz with amplitude 1.5 cm and duration 60 s. AP motion: anteroposterior motion.

Participants stood on a force platform (AccuSwayPlus, AMTI, Chicago) that was used to measure displacements of the center of pressure. The force platform rested on the carpeted concrete laboratory floor (the carpet was very short-napped, and there was no padding under the carpet), such that there was no imposed inertial motion. Center of pressure (COP) data were collected in the antero-posterior (AP) and medio-lateral (ML) axes at 50 Hz.

Restraint was achieved by strapping participants to a tilt table (801 Electronic Tilt Table; ActiveAid, Inc., Redwood Falls, MN) that was rotated so that the table surface was locked in its vertical position (Figures 1A and 1B).

Procedure

We separately assessed the incidence of motion sickness, using participants' direct, yes/no statements about whether they were motion sick and the severity of motion sickness symptoms (e.g., Bonnet et al., 2006; Stoffregen & Smart, 1998). We assessed motion sickness symptoms using the Simulator Sickness Questionnaire (SSQ; Kennedy et al., 1993). We separately assessed the incidence of claustrophobia, using participants' direct, yes/no statements about whether they were claustrophobic and the severity of claustrophobia symptoms. We assessed claustrophobia symptoms using the Claustrophobia Questionnaire (CLQ; Radomsky, Rachman, Thordarson, McIsaac, & Teachman, 2001). To assess participants' initial level of symptoms, and to ensure that they were familiar with motion sickness and claustrophobia symptoms, participants were asked to complete the SSQ and CLQ at the beginning of the experiment. We used the SSQ and CLQ pre-exposure scores as a baseline against which post-exposure data could be compared (Regan & Price, 1994; Smart et al., 2002). The SSQ and CLQ are standard instruments that have been extensively validated.

Each participant successfully completed a pre-test in which they were asked to stand on one foot for 30 s with their eyes open. They then entered the moving room and 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. Participants were warned that they might become ill or claustrophobic 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. For the duration of each trial with eyes open, participants were asked to keep their gaze on the map on the front wall (there was no fixation point). 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 with a sinusoidal pattern (Figure 1C) 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,

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, unrestrained
4	1 min, eyes closed, 0.2 Hz, 1.5 cm amplitude, unrestrained
5–8	10 min, eyes open, no imposed motion, 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

each of which was 10 min (600 s) in duration, during which the room was stationary. During these stationary trials, standing participants were restrained by being strapped to the vertical tilt table. Participants were strapped using elastic bands at the forehead, shoulders, hips, and knees, which were adjusted by the experimenter so that participants felt them to be tight but not uncomfortable. Participants' feet remained in contact with the force platform, and their body weight was not supported by the tilt table. Participants were briefly released from restraint at the end of each restrained trial. Before Trial 1, after Trials 6 and 8, we repeated the instruction for participants to discontinue the experiment immediately if they began to experience any noticeable symptoms of motion sickness or claustrophobia. For participants who completed all four restrained trials without discontinuing, the conditions of Trials 1, 2, and 3 were repeated (Trials 9–11). This permitted us to evaluate pre-post differences in spontaneous sway and in responses to the simple, 0.2 Hz imposed optic flow. Participants were monitored continuously by an experimenter stationed outside the moving room. The monitoring was for their safety and to ensure compliance with instructions.

For the duration of Trials 1–4 and 9–11 (if completed), participants were asked to keep their hands in their pockets or clasped behind or in front of them. They were free to change hand position between 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. During the long trials (5–8), participants' arms and hands were restrained along their body. Participants were instructed to stand comfortable, in a relaxed manner, and to avoid voluntary movements.

If participants discontinued their participation, they were asked whether they felt motion sick and/or claustrophobic and were asked to describe any symptoms, after which they were asked to fill out the SSQ and CLQ. Participants who stated that they were not motion sick were asked to report on their motion sickness and claustrophobia status over the next 24 hr. They were asked to indicate on a yes/no basis whether they developed motion sickness and/or claustrophobia, to

indicate when symptoms developed, 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 hr if no symptoms developed. Symptom onset is sometimes delayed up to an hour following termination of exposure to imposed visual motion (e.g., Stoffregen, 1985).

Analysis of COP Displacements

We conducted several analyses of postural motion before, during, and after exposure to passive restraint. We quantified displacements of the COP along the AP and ML axes by computing the variability (standard deviation of COP position), the range (difference between maximum and minimum COP positions), and the mean velocity (or mean speed) for each trial. These variables were selected, in part, to permit direct comparison with the results of Faugloire et al. (2007), who used the same variables. An additional motivation arises from the fact that there are not widely accepted definitions of stability and instability in the literature on human movement. Our use of these variables is exploratory and can help to inform more formal efforts at formalizing these concepts. Analyses of movements during the 10-min trials were based on criteria derived from the data and, therefore, are described in the following section. Prior to statistical analyses, we filtered the COP data, using a low-pass filter (FFT filter) with a cutoff frequency of 7 Hz. For each significant effect in our Analyses of Variance (ANOVAs), we estimated the effect size using partial eta squared (partial η^2).

RESULTS

Subjective Reports

Incidence and discontinuation.   Four participants stated that they were motion sick (22%) and were placed in the Sick group. One of the sick participants discontinued during Trial 6 and one during Trial 7. The two other participants completed the experiment. One became sick within 30 min after leaving the laboratory. The other participant felt sick the next morning and attributed the illness to her participation in our study. The incidence of motion sickness did not differ from the 39% observed by Faugloire et al. (2007), who exposed standing participants to nauseogenic optic flow during passive restraint, $\chi^2(1) = 2.10$, *ns*.

One participant reported being claustrophobic. This participant, who discontinued at the end of Trial 7, was one of the four who reported motion sickness.

Motion sickness and claustrophobia history.   Of the 4 Sick participants, 3 reported having been motion sick in the past (75%), whereas 8 of the 14 Well participants (57%) had been sick in the past. Two members of the Sick group reported having been claustrophobic in the past (50%), whereas only 2 of the Well participants (17%) had previously experienced claustrophobia.

Simulator Sickness Questionnaire (SSQ).   For each participant, we computed the total severity score in the recommended manner (Kennedy et al., 1993). Mean scores are presented in Figure 2A. Because the distribution of post-exposure SSQ scores across participants was positively skewed, we used nonparametric tests to compare the rank of SSQ scores between the Sick and
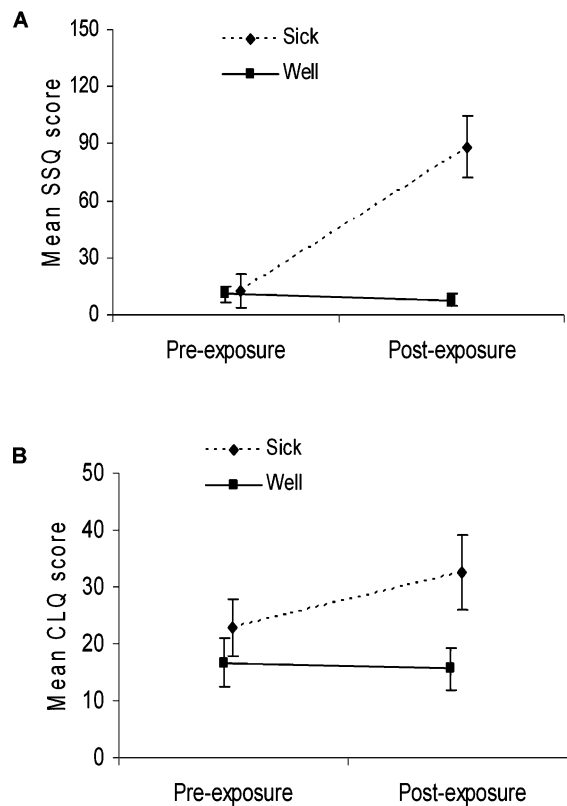

FIGURE 2   Pre-exposure and post-exposure (A) Simulator Sickness Questionnaire score (SSQ) and (B) Claustrophobia Questionnaire score (CLQ) for both Sick and Well participants. The error bars represent standard error.

Well groups (Mann-Whitney U test) and to compare the pre-post differences within each group (Wilcoxon Signed Rank Test). We adjusted the alpha criterion to .025 because each data set was used twice.

The Mann–Whitney U test revealed that there was no significant difference in rank between the Sick and Well groups for the pre-ranks, $U = 28.00$, $p = 1.00$. However, there was a significant difference in rank between the two groups for the post-ranks, $U = 0.00$, $p = .001$. After the experiment, the Sick group exhibited a higher rank (Mean Rank $= 16.50$) than the Well group (Mean Rank $= 7.50$). Concerning the Wilcoxon Signed Rank Test, no significant difference between pre-rank (Mean Rank $= 5.50$) and post-rank (Mean Rank $= 5.50$) was found for the Well group ($z = -.57$, $p = .31$). Similarly, no significant difference existed between the mean pre-rank (Mean Rank $= 0.00$) and post-rank (Mean Rank $= 2.50$) for the Sick group ($z = -1.83$, $p = .063$).

Claustrophobia Questionnaire (CLQ).   CLQ scores for each participant were calculated in the recommended manner (Radomsky et al., 2001). The data distributions were not skewed and the variances were homogeneous. Thus, we conducted a two-factor Time (pre-exposure vs. post-exposure) × Group (Sick vs. Well) ANOVA with repeated measures on the first factor.

The data are summarized in Figure 2B. There was no main effect of Time (pre-exposure vs. post-exposure) or of Group (Sick vs. Well), each $F(1, 16) < 3.05$, $p > .10$. However, Time × Group interaction was significant, $F(1, 16) = 4.88$, $p = .042$, partial $\eta^2 = .23$.

Relations between claustrophobia and motion sickness.   Nonparametric Spearman rho correlations between SSQ ranks and CLQ ranks were conducted independently on the pre-scores and on the post-scores. The correlation coefficient was not significant for the pre-exposure score, $rs(16) = .36$, $p > .14$, but was significant for the post-exposure score, $rs(16) = .57$, $p = .014$ (Figure 3).

Movement Data

Spontaneous sway, unrestrained (Trials 1 and 2).   The data are summarized in Table 2. We conducted two-factor, Vision (eyes open vs. eyes closed) × Group (Sick vs. Well) ANOVAs with repeated measures on the first factor for six dependent variables: variability, velocity, and range, each in the AP and ML axes. The analyses revealed significant main effect of Vision on variability, velocity, and range in the AP axis, each $F(1, 16) > 12.38$, $p < .003$, partial $\eta^2 = .46$, .44, and .44, respectively. For each significant effect, motion was greater when the eyes were closed. There were no other significant main effects or interactions effects for any dependent variable in any axis, each $F(1, 16) < 2.63$, *ns*.
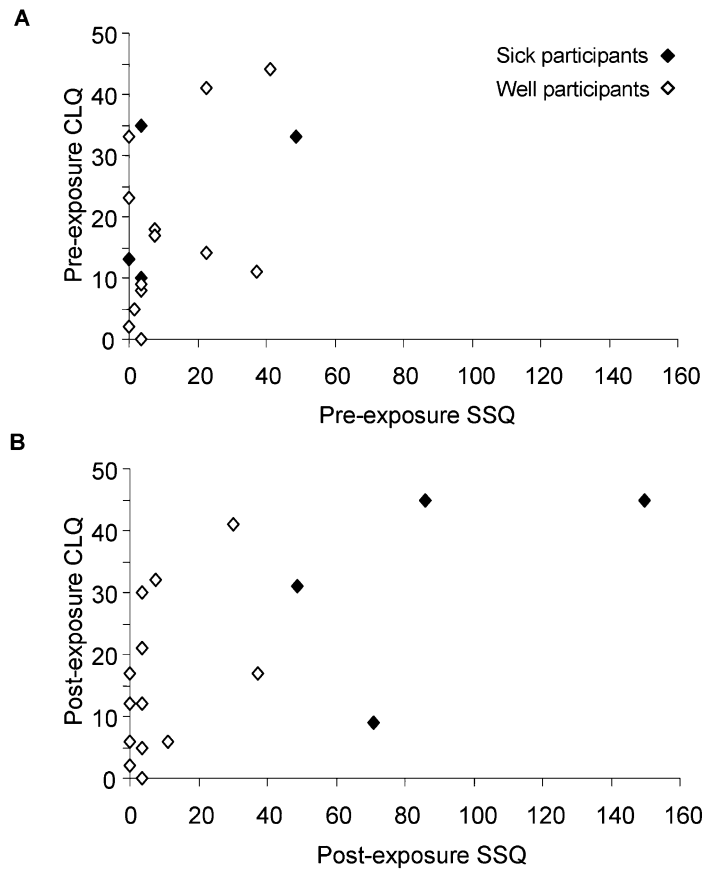
FIGURE 3   Correlation between motion sickness and claustrophobia scores for (A) the pre-exposure, $rs(16) = .36$, *ns*, and (B) the post-exposure, $rs(16) = .57$, $p < .05$. Black: Sick participants. White: Well participants. SSQ: Simulator Sickness Questionnaire score; CLQ: Claustrophobia Questionnaire score.

0.2 Hz stimulus, unrestrained (Trials 3 and 4).   The same variables and the same analysis as for Trials 1 and 2 were conducted for Trials 3 and 4. There were no significant effects of Vision or Group or any Group × Vision interactions.

Passive restraint, no imposed motion (Trials 5–8).   Each participant in the Well group completed all of the 10-min trials and, therefore, was restrained for a total of 40 min. Two participants in the Sick group completed the experiment, but the other two discontinued without completing the four restrained

TABLE 2
Unrestrained Trials: Descriptive Statistics for the Significant Main Effects of Vision on
Spontaneous Sway. Trial 1: Eyes Open. Trial 2: Eyes Closed. AP: Antero-Posterior.

	Variability AP (cm)		Velocity AP (cm.s^{-1})		Range AP (cm)	
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	Trial 1	Trial 2	Trial 1	Trial 2	Trial 1	Trial 2
M	0.24	0.38	0.52	0.85	1.17	1.86
SD	0.08	0.11	0.09	0.33	0.36	0.53

trials. 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 the data for Trials 5 and 6 but not for Trial 7. This procedure ensured that our analysis included only movement that occurred prior to the onset of motion sickness symptoms.

Overall movement.   We began by evaluating Sick/Well differences in overall movement, that is, computing the means across trials for each participant. We conducted independent *t* tests on these means for each dependent variable. As in Faugloire et al. (2007), there were no significant effects, indicating that the restrained condition did not produce any overall differences in movement between the Sick and Well participants, each $t(16) < 2.09$, $p > .05$.

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 passive restraint. 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 s to 360 s of Trial 6, and the final window was from 481 s to 600 s of

Trial 7. The windows selected for the Well participants were based on the fact that, on average, participants in the Sick group completed three restrained trials. Accordingly, for the Well group, we took the first 2 min of Trial 5, the middle 2 min of Trial 6 (from 241 s to 360 s), and the final 2 min of Trial 7. For one Sick participant, we found an outlier in one window of one trial. The participant exhibited a single very large movement (during restraint) during the last few seconds of the final window. To be conservative, we elected to shift this window forward in time (i.e., toward the beginning of the trial) so as to exclude this single movement. 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. Raw data from representative Well and Sick participants are illustrated in Figure 4.

ANOVAs revealed main effects of Window on variability, velocity, and range in the ML axis, each $F(2, 32) > 3.31$, $p < .05$, partial $\eta^2 = .17$, .36, and .20, respectively (Table 3). In addition, we found a main effect of Group on variability in the ML axis, $F(1, 16) = 7.19$, $p = .016$, partial $\eta^2 = .31$. The Sick group exhibited higher variability ($M = 0.13$ cm, $SD = 0.08$) than the
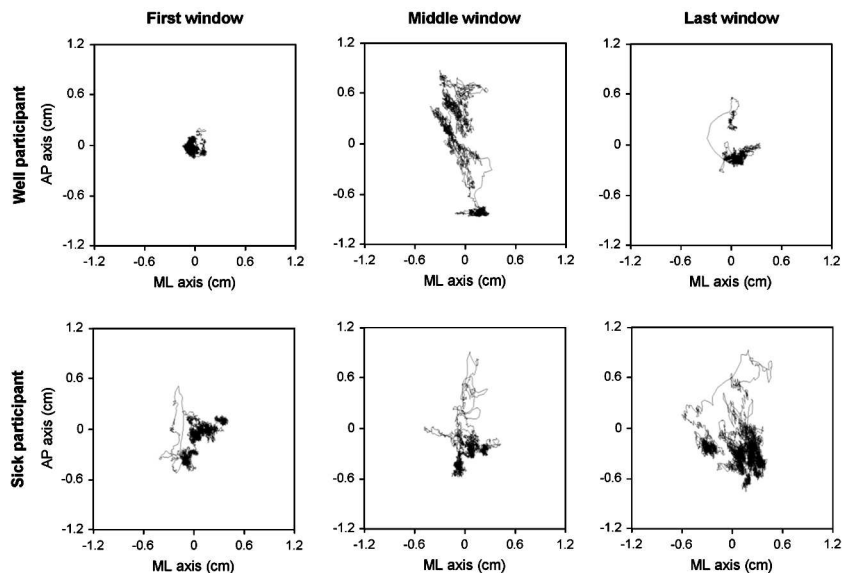

FIGURE 4   Center of pressure data for representative participants at the beginning (left), middle (center), and end (right) of restraint trials (Trials 5–8). Top: A participant who did not report motion sickness. Bottom: A participant who reported motion sickness. AP axis: antero-posterior axis; ML axis: medio-lateral axis. The data were collected before sickness onset.

TABLE 3
Restrained Trials: Means and Standard Deviations (in Parentheses) for the
Dependent Variables That Had Significant Main Effects of Window.
ML: medio-lateral.

Dependent variables	First window	Middle window	Last window
Variability ML (cm)	0.23 (0.14)	0.23 (0.21)	0.26 (0.20)
Velocity ML (cm.s^{-1})	0.35 (0.08)	0.37 (0.12)	0.44 (0.15)
Range ML (cm)	1.52 (0.99)	1.40 (1.28)	1.80 (1.43)

Well group ($M = 0.09$ cm, $SD = 0.05$). We found significant Group × Window
interactions for variability and range in the ML axis and for velocity in the AP
axis, each $F(2, 32) > 3.91$, $p < .03$, partial $\eta^2 = .30$, .20, and .26, respectively
(Figure 5).

Comparison of sway before and after passive restraint. Due to dis-
continuation, only two participants in the Sick group completed Trials 9–11.
Therefore, the comparison between pre-exposure and post-exposure sway was
performed only for the Well group. For each dependent variable, we conducted
three *t* tests comparing spontaneous sway with the eyes open (Trial 1 vs. 10),
spontaneous sway with the eyes closed (Trial 2 vs. 11), and sway during exposure
to the 0.2 Hz stimulus with the eyes open (Trials 3 vs. 9). Due to technical
problems, movement data were not recorded for one participant during Trial 9
and for another participant during Trial 11. Consequently, the following analyses
include 13 participants for comparisons between Trials 2–11 and Trials 3–9 and
14 participants for comparisons between Trials 1–10.

The data are summarized in Table 4. For spontaneous sway with the eyes
open, the velocity and range of movement in the AP axis were greater during
Trial 10 than during Trial 1, each $t(13) > 2.76$, $p < .017$. For spontaneous
sway with the eyes closed, sway was greater during Trial 11 than during Trial 2
for variability and range in both AP and ML axes, each $t(12) > 2.31$, $p < .041$.
No other differences were significant.

DISCUSSION

Standing participants were subjected to passive restraint in the absence of any
imposed motion. Twenty-two percent of participants stated that they were motion
sick. Passive restraint was associated with subjective symptoms of claustrophobia
but only among participants in the Sick group. Displacements of the center of
pressure were different for the Sick and Well groups. Participants were asked to
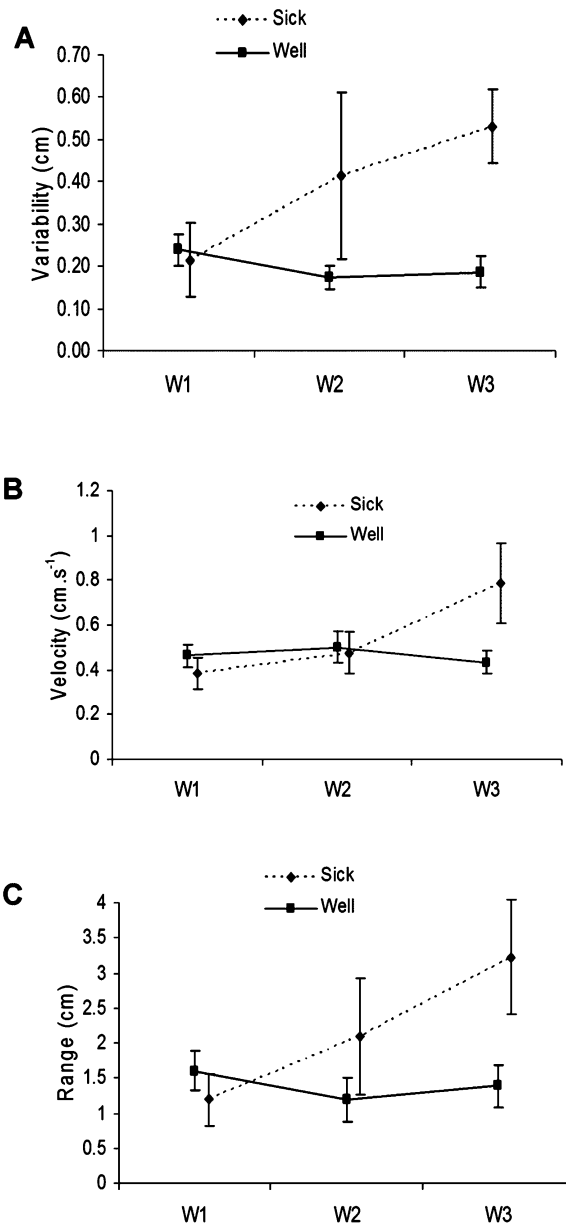
FIGURE 5   Significant Group × Window interactions for center of pressure data during passive restraint (Trials 5–8). A: Variability of position in the medio-lateral (ML) axis. B: Velocity of movement in the antero-posterior (AP) axis. C: Range of movement in the ML axis. W1, W2, and W3 refer to the first, middle, and last windows, respectively. The error bars represent standard error.

TABLE 4
Unrestrained Trials: Means and Standard Deviations (in Parentheses) for the Changes in
Sway Before and After Passive Restraint (Data From the Well Group Only).
AP: antero-posterior: ML: medio-lateral.

Dependent variables	Trial 1	Trial 10	Trial 2	Trial 11
Velocity AP ($cm.s^{-1}$)	0.52 (0.10)*	0.65 (0.16)*	0.92 (0.36)	1.11 (0.37)
Variability AP (cm)	0.24 (0.09)	0.27 (0.07)	0.38 (0.12)*	0.57 (0.21)*
Range AP (cm)	1.15 (0.38)*	1.46 (0.38)*	1.84 (0.52)*	2.94 (1.32)*
Variability ML (cm)	0.15 (0.07)	0.15 (0.06)	0.20 (0.15)*	0.24 (0.18)*
Range ML (cm)	0.79 (0.41)	0.80 (0.29)	1.02 (0.83)*	1.32 (1.02)*

*$p < .05$.

discontinue immediately at the appearance of any symptom of motion sickness.
Also, trials that were not completed (due to discontinuation) were not included
in our analysis of the movement data. For these reasons, we can conclude that
differences in movement between the Sick and Well groups preceded the onset
of motion sickness. The differences in movement between Sick and Well groups
resembled findings from previous studies in which unrestrained participants were
exposed to nauseogenic visual motion stimuli. In interpreting the present results,
we focus on this resemblance.

Motion Sickness, or Something Else?

The analyses of the displacement of the center of pressure revealed statistically
significant differences between the Sick and Well groups. These results constitute
objective evidence that body movements were different in the two groups during
the restraint conditions. Previous studies suggest that our participants' statements
that they were motion sick should be taken at face value (Faugloire et al., 2007;
Smart et al., 1998) and, therefore, that the differences in movement are related
to motion sickness. However, given the fact that participants were restrained
and were not subjected to any imposed motion, one may wonder whether
participants' subjective symptoms might have been caused by something other
than motion sickness.

   One possible post-hoc interpretation of our results is that participants in the
present study may have suffered from orthostatic hypotension rather than motion
sickness. The U.S. National Institute of Neurological Disorders and Stroke
(2007) defines orthostatic hypotension as "a sudden fall in blood pressure that
occurs when a person assumes a standing position." Symptoms generally occur
after sudden standing and include dizziness, light-headedness, blurred vision,
and syncope (temporary loss of consciousness). The subjective symptoms of
orthostatic hypotension are similar to some of the subjective symptoms of motion

sickness. However, we have our participants' direct, specific statements that they were motion sick. For methodological purposes, participants were excluded from our study if they reported any history of dizziness, seizures, balance disorders, or vestibular dysfunction. Thus, it is very unlikely that orthostatic hypotension occurred in connection with any medical condition. Of the four participants who reported motion sickness, two experienced the malady only after leaving our laboratory. We know of no reports of orthostatic hypotension occurring after such delays.

It is possible that participants in our study experienced both orthostatic hypotension and motion sickness. That is, orthostatic hypotension might be linked to motion sickness, as is known to be the case following parabolic flight (Schlegel et al., 2001) and after spaceflight (Buckey et al., 1996). Similarly, some studies (e.g., Stewart et al., 1999) have observed nausea and retching among healthy participants who experienced orthostatic hypotension. Stewart et al. did not consider the possibility that participants may have experienced motion sickness: Participants were not warned to expect motion sickness, were not asked whether they were motion sick, and were not informed that many of the symptoms they reported were also symptoms of motion sickness. In addition, studies of orthostatic hypotension do not include quantitative data on body movement. Thus, in the extant literature on terrestrial orthostatic hypotension, it is not possible to evaluate the hypothesis that either postural instability or motion sickness may have occurred. Physiological changes associated with orthostatic hypotension, such as a reduction in blood pressure, could influence the perception and/or control of bodily orientation. In fact, reduction in blood pressure yields, within several seconds, a subsequent reduction in the force produced by muscle contraction (Fitzpatrick, Taylor, & McCloskey, 1996). It is possible, then, that orthostatic hypotension leads to unstable control of the body that, in turn, could lead to motion sickness. It would be interesting, in studies of orthostatic hypotension, to collect data on body movement, and to ask participants whether they feel motion sick. Affirmative answers to this question would support they hypothesis that orthostatic hypotension may sometimes lead to motion sickness.

Self-Induced Motion Sickness?

Self-induced motion sickness has been reported in several widely different situations, including orbital spaceflight (Oman et al., 1986), during wearing of prism spectacles (Dolezal, 1982), or with vigorous rotation of the torso in stance (Bouyer & Watt, 1996). Smart et al. (1998), in a study of body sway in the absence of any imposed motion, were surprised when participants began spontaneously to report being motion sick. In the present study, participants reported motion sickness despite the fact that they were not exposed to any

experimental motion. This finding resembles the study of Smart et al. (1998). The incidence of motion sickness in our restrained condition was very similar (22%) to that reported by Smart et al. (1998; 21%). Smart et al. suggested that motion sickness in their study was self-induced; that characterization seems credible in the present study as well. We interpret the present study as adding to the small but diverse literature on motion sickness that occurs in the absence of imposed motion.

Motion Sickness Severity

The SSQ scores of the Sick group did not increase significantly from pre-exposure to post-exposure. It might appear, then, that individuals in the Sick group were not truly motion sick or that symptoms were mild. However, post-exposure SSQ scores were significantly higher for the Sick group than for the Well group, confirming that symptoms differed between participants who became motion sick and those who did not. Moreover, each participant in the Sick group stated that they were motion sick. Participants had no reason to lie because they knew that they could discontinue participation at any time for any reason.

Divergence between the incidence of motion sickness and the severity of motion sickness symptoms has been observed elsewhere (e.g., Lawson, Graeber, Mead, & Muth, 2002). As one recent example, Merhi, Faugloire, Flanagan, and Stoffregen (2007) asked participants to play a console video game that was presented through a head mounted display. The Sick group exhibited a significant increase in SSQ scores following game play. However, there was also a significant post-exposure increase in SSQ scores among the Well group. The same result was also found in Faugloire et al. (2007), in which the Well group was found to have a significant increase in SSQ post-exposure scores (each participant in the Well group stated that they were not motion sick). It must be remembered that some symptoms included in the SSQ are associated with a variety of conditions and not solely with motion sickness. Examples include eyestrain, fatigue, and headache. The results of the present study underscore the relevance of the distinction between the incidence of motion sickness and the severity of symptoms that are associated with motion sickness.

Unrestrained Stance

During spontaneous, unrestrained stance, displacements of the center of pressure were greater when the eyes were closed (Trial 2) than when they were open (Trial 1). This finding replicates classical effects. Some previous studies have also found that spontaneous sway and/or postural responses to the 0.2 Hz stimulus differed between the Sick and Well groups (Bonnet et al., 2006; Faugloire et al., 2007; Smart et al., 2002; Stoffregen, Hettinger, Haas, Roe, & Smart, 2000;

Stoffregen & Smart, 1998). In the present study, we found no effects in Trials 1–4 that were related to whether participants later became motion sick. The absence of such effects in the present study compared with former studies may suggest that self-induced motion sickness during passive restraint is unrelated to postural control in the absence of restraint.

Movement During Passive Restraint

In interpreting the movement data it is important to recall that our analysis included only movement prior to the onset of motion sickness symptoms. The center of pressure data showed that both Well and Sick participants moved during passive restraint, that is, passive restraint was not complete. The same was true for Faugloire et al. (2007), who observed displacements of the center of pressure during passive restraint. These findings underscore the practical difficulty of achieving complete passive restraint, that is, a state in which body movement relative to the restraint system is not possible.

We observed several differences in center of pressure displacements between the Well and Sick groups. Our window analysis revealed that changes in movement over time during passive restraint differed for the Well and Sick groups. The Sick group exhibited higher variability in the mediolateral axis than the Well group. In addition, the Sick group was characterized by increasing variability over time, whereas movements of the Well group tended to be stable over time. These results are compatible with the hypothesis that motion sickness should be preceded by unstable control of posture.

The data suggest that the movements of Sick participants changed despite the fact that participants did not need to control their stance during passive restraint. Indeed, the definition of postural control used by Riccio and Stoffregen (1991) was more general than the control of stance. Their definition referred to states of the body with regard to perception and action, and it is thus appropriate for any kind of posture, in general. The existence of differences in movement of Sick and Well groups, prior to the onset of subjective symptoms, replicates similar effects from previous studies. In Faugloire et al. (2007) passively restrained participants were exposed to visual oscillations in the range 0.1–0.4 Hz. Some participants reported motion sickness, and center of pressure data collected during restraint revealed differences in movement of the Sick and Well groups before the onset of subjective symptoms. Similar effects have been observed in several studies of both standing and sitting participants who were not subjected to any restraint but who were exposed to optical motion stimuli (e.g., Bonnet et al., 2006; Merhi et al., 2007; Stoffregen et al., 2000; Stoffregen & Smart, 1998). In each of these studies, as in the present study, reports of motion sickness were reliably preceded by changes in movement of the head and torso and/or changes in displacement of the center of mass. In each case, the movement effects

were predicted by the postural instability theory of motion sickness (Riccio & Stoffregen).

As noted in the introduction, several studies have reported the occurrence of motion sickness among participants who were passively restrained (e.g., Graybiel & Miller, 1970; Warwick-Evans & Beaumont, 1995; Warwick-Evans et al., 1998). Quantitative data about movement during passive restraint were not collected in any of these studies. Thus, it is possible that the movement effects observed in the present study may also have occurred in these earlier studies.

The Role of Claustrophobia

Faugloire et al. (2007) observed postural instability and (subsequent) motion sickness among participants who were passively restrained during exposure to imposed optic flow. Faugloire et al. could not differentiate whether postural instability was caused by the imposed optic flow or by factors relating to passive restraint. In the present experiment, there was no imposed optic flow. Our results indicate that motion sickness can occur in situations of passive restraint.

In the present study, only one participant reported being claustrophobic. However, we found a significant Time × Group interaction, indicating that claustrophobia symptoms increased (following passive restraint) only among Sick participants. Also, after exposure to passive restraint, there was a significant correlation between the severity of claustrophobia and the severity of motion sickness symptoms (cf. Faugloire et al., 2007, who found similar results). Of course, claustrophobia symptoms are not an inevitable accompaniment of motion sickness (e.g., Bonnet et al., 2006). Similarly, claustrophobic situations are not necessary for the occurrence of self-induced motion sickness (e.g., Smart et al., 1998). But in the present study and in Faugloire et al. (2007), the fact that symptoms of claustrophobia developed only among Sick participants suggests that claustrophobia and motion sickness can be related in claustrophobic situations.

Faugloire et al. (2007) suggested that claustrophobia could be an indirect cause of motion sickness. During passive restraint, feelings of claustrophobia might be related to functional characteristics of perception and action. There could be individual differences in the tendency to use postural movements as a means to generate information about the qualitative dynamics of the animal-environment system. For these people, passive restraint might lead to a reduction in the available perceptual information that, in turn, might be related to the subjective experience of claustrophobia. Such feelings could lead to unusual movements that, if poorly controlled, could produce motion sickness. An advantage of this interpretation is that it can explain relations between all three types of data (claustrophobia, motion sickness, and movement). Inversely, it would be possible to claim that claustrophobia was caused by motion sickness,

but such a claim would not be able to account for the movement data (i.e., the fact that motion sickness was preceded by changes in center of pressure displacements).

An advantage of the previous interpretation is that Riccio and Stoffregen (1991) wrote their theory in the context of both perception and action. They defined instability in terms of the consequences of any given movement for other actions. Participants who became motion sick during restraint may have perceived instability relative to their movement goals. Of relevance here are claims that perception of nonmotion is possible (Riccio, 1995) and that it may be possible to perceive consequences of instability for perceiving and acting (Riccio & Stoffregen, 1991).

Theories of Motion Sickness Etiology

As noted earlier, the incidence of motion sickness in the present study was similar to that observed by Smart et al. (1998). Smart et al. observed self-induced motion sickness in the absence of imposed motion. In their study, standing participants were unrestrained and were obliged to control their stance. Smart et al. (1998) noted that no version of the sensory conflict theory already predicted the occurrence of motion sickness during quiet stance, in the absence of any imposed motion. In the present study, there was no imposed motion during passive restraint. In the present experiment, it is not clear what might have given rise to sensory conflict. It would seem that the present form of the sensory conflict theory would not predict sickness. The incidence of motion sickness in the present study was not significantly different from the incidence reported by Faugloire et al. (2007), in which standing participants were exposed to nauseogenic optic flow during passive restraint. The finding that the incidence of motion sickness did not differ as a function of whether participants were exposed to experimental motion does not appear to be readily compatible with the sensory conflict theory of motion sickness. If sensory conflict existed, its magnitude should have been greater during exposure to imposed optic flow than in the absence of imposed optic flow. The present study together with those of Faugloire et al. (2007) and Smart et al. (1998) raise new questions about the generality and explanatory power of the sensory conflict theory of motion sickness.

The postural instability theory of motion sickness (Riccio & Stoffregen, 1991) can account for the occurrence of motion sickness during unperturbed stance in the study of Smart et al. (1998) and during passive restraint, as in the present study and the study of Faugloire et al. (2007). The theory predicts that participants entering and maintaining states of postural instability will become motion sick (Riccio & Stoffregen, 1991, p. 206). This prediction is valid with or without imposed motion and regardless of the cause of the postural instability (e.g., unsteadiness, claustrophobia, voluntary movement).

Our study demonstrated that Sick and Well participants moved differently prior to the onset of motion sickness. Because we assessed only the kinematics of the center of pressure, we do not know the source of these differences in terms of rotations around different joints or the contractions of different muscles. Riccio and Stoffregen (1991) proposed that stability and instability could be defined in terms of kinematics, rather than in terms of kinetics, or muscle activity. However, muscular activity (and the resulting somatosensory stimulation) could be used with respect to Riccio and Stoffregen's theory to detect some changes in body motion that would not be detected by the force platform in a restrained condition, such as co-contraction or skeletal strain. Measurements of muscular activity might provide a new means to define instability related to motion sickness. For these reasons, causal relations between center of pressure kinematics and the kinematics and dynamics of muscles and joints are subjects for future research.

CONCLUSION

During passive restraint, participants reported motion sickness in the absence of any imposed motion. Prior to the onset of motion sickness, movement differed between the Well and Sick groups. The results support the postural instability theory of motion sickness. The results also confirm previous findings of the existence of self-induced motion sickness (e.g., Oman et al., 1986; Smart et al., 1998) and extend this finding to the situation of passive restraint.

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