Postural Instability and Simulator Seasickness

Kyle A. Pettijohn; Daniel Geyer; Jacqueline Gomez; William J. Becker; Adam T. Biggs

BACKGROUND: Motion sickness is a serious issue for many individuals, but the problem is particularly important among military personnel who may regularly experience unusual or extreme motion profiles as a part of their duties. As such, it is important to understand the underlying mechanisms that contribute to motion sickness, which in turn can lead to new and more effective countermeasures. The current study investigated causal etiology by examining the predictions of postural instability theory. Subjects experienced multiple motion profiles while reporting their sickness symptoms.

METHODS: Postural instability was directly manipulated by including both an active and passive condition. In the active condition, subjects could actively adapt their posture to the motion profile. In the passive condition, subjects had their feet affixed in place and could not effectively adapt their posture to the motion profiles. Subjects completed both conditions to control for individual differences in motion sickness susceptibility.

RESULTS: Active condition subjects had greater postural stability as measured by sample entropy ($M = 0.179$ Active, $M = 0.136$ Passive), and sickness symptoms increased with time. Both results provide a methodological check against our manipulation. However, there were no differences in symptoms between active or passive conditions as measured by the simulator sickness questionnaire ($M = 16.56$ and $M = 18.25$, respectively), and no relationship between our measure of postural instability and symptomology.

DISCUSSION: These results do not support postural instability as the primary causal factor in motion sickness; however, more research is needed to elucidate the mechanisms of motion sickness etiology.

KEYWORDS: Simulator sickness, motion sickness, sensory conflict, postural instability, virtual reality.

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Motion sickness (MS) is a serious problem for the armed forces where optimal performance is critical for mission and training success. Unfortunately, the exact etiology of MS is unknown and MS susceptibility differs across individuals. Therefore, we can only treat MS symptoms rather than combating MS a priori. As a result, millions of dollars are spent annually on pharmaceutical countermeasures designed to treat MS symptoms. Although many of these medications are effective, they have undesirable side effects (e.g., cognitive impairments and drowsiness), and thus their use is problematic for military personnel who need to be able to perform optimally. If we can gain a clearer understanding of MS etiology, we might develop better, more targeted MS mitigation techniques.

Multiple theories have already been proposed to explain MS, although Sensory Conflict Theory (SCT) is easily the leading perspective. SCT suggests that MS originates from conflicting sensory input (vestibular, visual, and proprioceptive) that is generated as a result of unusual or provocative motion.[15,16] Common examples include airsickness, sea sickness, or car sickness, where vestibular-detected motion differs from perceived visual motion. This conflicting sensory input "travels" from the peripheral vestibular and visual centers through the vestibular nuclei to a centrally located "comparator." In turn, incoming sensory input is compared with previously stored information from a "neural store"—a theoretical repository of past motion information. If incoming sensory input conflicts with previously stored information, the comparator generates a mismatch signal response[15,16,24] and the autonomic response of MS is induced. After repeated exposures to an offending motion, the neural store is gradually updated with a novel motion profile. This graduated adaptation attenuates the autonomic response to MS, until complete adaptation occurs. An

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example of this effect would be student pilots who experience severe airsickness during their first flight, but each subsequent flight results in diminished airsickness until the negative response is entirely eliminated. A related hypothesis to SCT is that the MS is caused by the difference between what one senses as the vertical versus what one expects the vertical to be based on previous experience or expectation.[3]

Additional hypotheses include the poison hypothesis[25] that proposes the response to MS is similar to that caused by ingesting a toxin, and the same processes used to rid oneself of the toxin may be employed during MS-causing motions. This has been expanded on by Lawson[12] to suggest that there may be an evolutionary basis for avoiding potentially unsafe environments, rather than poison alone being the primary cause. Finally, there is the idea that MS arises from combinations of reflexive and voluntary eye movements during motion.[4] These hypotheses have been the driving forces behind how researchers examine, think about, and explain MS while also tackling the need to develop effective MS countermeasures.

An additional hypothesis is Postural Instability Theory (PIT), which is rooted in ecological perception theory. Riccio and Stoffregen[18] defined postural stability as "the state in which uncontrolled movements of the perception action systems are minimized" (p. 202). PIT postulates that when an individual is placed in a novel motion environment, the individual must adopt a new posture compatible with the new environment, where "uncontrolled movements are minimized."[18] When this adaptation does not occur, the subsequent postural instability leads to MS. Therefore, differences in postural degradation during exposure to a provocative motion environment may be predictive of the MS response. This degraded stability is most likely to occur during the process of adaptation. Several studies have found that postural stability is degraded during periods of provocative stimulation, and those with worse stability are more likely to become motion sick.[10,22,23]

Adaptation refers to one's ability to modulate one's actions in response to environmental changes or changes in particular task constraints.[6,17] In order for successful adaptation, one must adopt behaviors that are appropriate for proper exploration and utilize the appropriate information in order to regain a functional correspondence (a perception-action relationship that affords successful attainment of a goal state) with the environment.[8,19] Ultimately, the end goal of adaptation is a return to one's ability to exhibit prospective control—the ability to utilize perceptual information to regulate and guide future-oriented, or goal-directed, behaviors or actions.[1,5,26] In other words, this refers to the ability to exhibit active anticipatory (prospective) behaviors rather than passive compensatory (reactionary) behaviors, which, by definition, do not allow for prospectivity.[13]

PIT can be assessed empirically by directly comparing postural behavior to MS symptoms within a given scenario. For example, participants who became motion sick after exposure to various types of optical motion exhibited greater sway as measured by elliptical area and path length.[21] Another proxy for postural behavior is center of displacement, which has been found to increase over time for those who eventually become motion sick compared to those who do not.[2] The current investigation thus compared the type of postural behavior (extent of self-similarity) for individuals along a continuum of MS susceptibility. Many MS studies only utilize individuals susceptible to MS. Although that may be advantageous for examining the efficacy of an anti-MS drug, this drastically limits these studies from examining MS susceptibility differences from different theoretical perspectives. The current study examines how passive behavior (fixed stance width) impacts MS incidence for these same individuals. While it is important to note that successful adaptation requires a person to be able to anticipate what future movements will be needed to maintain stability, this may not always be possible. The aim of the current experiment is to examine PIT in the context of a more ecologically valid motion environment—riding as a passenger on a small boat. Through comparisons of active and passive behavior, the current study contributes to a more comprehensive understanding of the etiology of MS. In addition, it may help illuminate issues that specifically relate to seasickness. For example, the provocative motion that causes seasickness is unique in that it is characterized by large, somewhat predictable, motions caused by swells as well as smaller, and less predictable, perturbations. Movement on other forms of transport is generally more restricted, but the working environment of a ship may require its occupants to travel around the craft, even when motion is severe.

We hypothesize that: 1) individuals who do not experience MS will demonstrate more functionally adaptive behavior (more self-similar) than those who do experience MS; and 2) most individuals will experience MS during passive motion with a fixed stance width. For the first hypothesis, functionally adaptive behavior represents better anticipatory control, and this adaptation corresponds to greater success in mitigating MS symptoms. For the second hypothesis, a fixed stance width (9 inches on center) reduces one's base of support and forces an individual into an unusual posture, resulting in increased and prolonged compensatory behavior simply to remain upright. This increased compensatory action can create postural instability and contribute to the increase of MS symptoms. We expect forcing someone into an unusual posture will lead to increased postural instability and MS symptoms. The narrow width stance that will be imposed on people will limit their ability to make compensatory motions with their hips and knees. In the active condition, they will have full freedom to make any necessary adjustments to maintain balance; however, the lack of unusual posture does not constitute a true control condition because subjects will not be able to perfectly anticipate the needed compensatory movements to maintain stability. The focus of this experiment is on how well PIT is able to account for seasickness.

METHODS

Subjects

An a priori power analysis with a medium effect size ($F = 0.3$) revealed that 12 subjects are required to ensure an observed

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power of 0.86 with alpha set at the 0.05 level. We do not report "observed" power because, as pointed out by O'Keefe,[14] the power of the test is the same no matter when it is computed. That is, if the population effect size is what we assume based on previous studies, the power to detect that effect does not change after the data are collected. If we are to calculate power using the effect size observed in our data rather than the expected population effect size, we already know that the power will be low given that we have found a nonsignificant result. This is a function of the relationship between *P* value and power. Hoenig and Heisey[7] demonstrate that significance level determines observed power. Because of this relationship, "nonsignificant p [sic] values always correspond to low observed power" (p. 2). The study protocol was approved by the Naval Medical Research Unit Dayton Institutional Review Board in compliance with all applicable Federal regulations governing the protection of human subjects. There were 17 subjects (4 women) who were recruited from active duty military members and those covered by Department of Defense insurance at Wright-Patterson AFB, OH. Subjects were between the ages of 21 and 63 yr ($M = 37.4$, $SE = 3.5$). During an initial screening, they answered a preliminary questionnaire to ensure that they did not have any conditions (inner ear disorder, temporary illness, etc.) that could be exacerbated by motion sickness. Subjects were also informed that in order to maintain eligibility, they must refrain from drinking alcohol for 24 h before an experimental day and must avoid taking any medication that could affect balance, inner-ear fluid levels, or cause dizziness or lightheadedness. Female subjects were administered a pregnancy test prior to the experimental sessions to ensure that pregnancy-related nausea would not affect the results. Three subjects discontinued the study and were not included in the analyses: the first stopped approximately 1 min into the passive condition and approximately 2.5 min into the active condition; the second stopped approximately 9 min into the passive condition; and the third did not return for the passive condition session. Thus, there were 14 subjects included in the final analyses.

Equipment and Materials

The experiment utilized a within-subjects design with each subject participating in both the active control (active) and passive restraint (passive) conditions. The order of the conditions was counterbalanced to account for any confounding effects of order. For the passive condition, subjects' feet were strapped into modified snowboard bindings to reduce their ability to make postural adjustments. The centers of the bindings were placed 9 in. (22.86 cm) apart. The modifications consisted of removing the straps, buckles, and hibacks as these could provide additional support. Thus, the modified bindings contained the baseplate, sideplates, and heelcups (**Fig. 1**). Subjects completed a series of three 10-min simulated sea state profiles in either the active condition or the control condition. The two additional profiles were created by removing the first 1 or 2 min of collected sea state data and appending it to the end of the profile. The order of the motion profiles was counterbalanced across subjects.

During the experimental procedure, subjects completed the Motion Sickness Susceptibility Questionnaire Short-form (MSSQ[16]), the Simulator Sickness Questionnaire (SSQ[9]), a demographics and compliance questionnaire, and a preliminary screening. The MSSQ provides an assessment of how susceptible a person is to motion sickness and what types of motion are likely to elicit it. The SSQ is a measure of the severity of motion sickness symptoms a person is currently experiencing and can be further divided into three subscales: Nausea, Oculomotor Discomfort, and Disorientation. The preliminary screening asked potential subjects about any conditions, medication, or activities (e.g., blood or plasma donation within the last 30 d, alcohol consumption) that might prevent them from taking part in the study, and the compliance questionnaire ensured that subjects met the eligibility requirements for each session.

Motion was conveyed through a 6 degree of freedom Stewart platform that moves in the x, y, z, yaw, pitch, and roll axes; however, for this experiment only the yaw, pitch, and roll axes were used. Thus, the motion of the platform mainly consisted of roll and pitch perturbations, with occasional more pronounced movements caused by encountering a large wave. Mean roll frequency was 4.37×10^{-5} Hz (SE = 0.004), and mean pitch frequency was 1.69×10^{-5} Hz (SE = 0.006). The platform was covered with antislip tape to ensure subject safety. Platform motion was controlled/driven by real world "sea state data" captured by accelerometers on a small boat traveling across a bay.

The head mounted display (HMD) was an nVisor SX60 (NVIS, Inc., Reston, VA) that displayed a virtual environment programmed in the Godot Engine (Version 2.1; Fig. 1). The HMD displayed a virtual sky, ocean, and rigid hull inflatable boat (similar to what was used to collect the sea state data) via two 1280×1024 displays with a 60° diagonal field of vision. An InterSense IntertiaCube 2 (Thales Visionix, Inc., Aurora, IL) was mounted to the HMD to track head motion. When the participant moved his or her head around, the display would update in real time, providing an immersive experience of being aboard a small boat on the ocean.

Procedure

After signing the consent form subjects filled out the demographic, compliance, and preliminary simulator sickness questionnaires and performed two balance checks. The first consisted of walking in a straight line, heel to toe, approximately 10 ft (3 m) one way and 10 ft back the other way. The second required they stand on one leg with their eyes closed for 30 s. Following this, subjects climbed onto the motion platform (and their feet were restrained if they were in the passive condition). The HMD was placed on their heads and adjusted to minimize its movement during the motion profiles. Participants were informed that they should notify the researcher and discontinue if symptoms of motion sickness emerged. They experienced a motion profile and verbally completed an SSQ after each until all three profiles had been administered. The time between profiles was long enough to administer the SSQ and load the next profile—approximately 2 min. A final SSQ was filled out no more than 5 min after the final profile for a total of five SSQs. At the 4-, 5-, or

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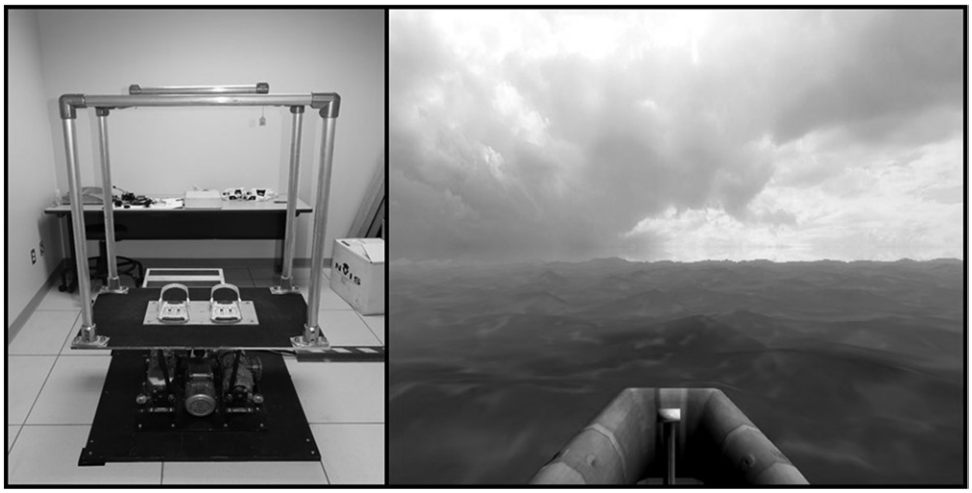

Fig. 1. Sample image of the motion platform (left) with the bindings for the passive condition (active condition used the same setup except the bindings were removed from the platform). Right image represents the participant view within the simulated environment.

6-min mark of each profile, subjects were asked to count backward from a three digit number by threes for 60 s (e.g., 319, 316, 313, etc.). This was done to "break up" the profiles around their midpoints. Participants were not given any other instructions as to what to do during the profile. They were told not to hold onto the safety railing but only grab it if they felt they were about to fall.

The experimental session was repeated on a different day for the active and passive conditions. Because the effects of motion sickness can persist for 24 h, each session was conducted at least 24 h apart [M = 306.9 h (12.79 d), SE = 55.8 (2.33)]. One subject's second session took place the day after the first; all other sessions were at least 5 d apart. The time of day the subject participated varied according to that person's availability. Condition and profile order were counterbalanced to ensure that a particularly provocative condition-profile combination did not skew the results.

Statistical Analysis

Results for SSQ data are presented in **Fig. 2** and **Table I**. The Total SSQ scores were submitted to a 2 (Condition) × 5 (Time) repeated-measures ANOVA. There was no main effect of Condition [$F(1,13) = 0.16$, MSE = 161.15, $P = 0.697$, $\eta_p^2 = 0.01$], but there was a main effect of Time [$F(4,52) = 12.29$, MSE = 126.18, $P < 0.001$, $\eta_p^2 = 0.49$]. Importantly, the interaction was not significant [$F(4,52) = 0.59$, MSE = 39.47, $P = 0.671$, $\eta_p^2 = 0.04$]. The main effect of time demonstrates that the effects of motion sickness accumulate over time, thus the symptoms become more severe. An ANCOVA using MSSQ score as the covariate revealed the same pattern of results; no main effect of Condition, main effect of Time, and no interaction. The data were also analyzed to determine if there were differences on the subscales of the SSQ. The same pattern of results was found for all subscales, again indicating that the symptoms of motion sickness increased over time for people in both conditions.

Often, SSQ data are not normally distributed. To account for this, the data were also analyzed using Wilcoxon signed rank tests. No group differences were found on the Total SSQ score or any of the subscales ($P > 0.10$). Pairwise comparisons were conducted on Total SSQ score for each pair of consecutive time points with a Bonferroni correction for multiple comparisons. There was a significant increase from Pretest to Time 1 ($M_{diff} = 14.69$, $P < 0.001$), and a significant decrease from Time 3 to Posttest ($M_{diff} = -12.42$, $P = 0.009$). None of the other comparisons was significant ($P > 12$). Postural stability was assessed in two ways using the head tracking data. First, the ellipses that contained 98% of a participant's head movements for each trial were analyzed (**Fig. 3**). Ellipses represent a measure of spatial complexity or magnitude of postural sway. Euclidean norms were calculated for each movement vector. The distribution of norms was fit to a Rayleigh distribution, and data points that fell outside of the 98th quantile were removed to prevent extreme excursions from exerting undue influence on the area calculation. The areas of the ellipses were submitted to a 2 (Condition) × 3 (Trial) ANOVA. Head movement data was not collected along with the pretest or posttest SSQ scores, and so the postural stability assessments are limited only to the three motion profiles. The main effect of Condition was significant [$F(1,13) = 5.08$, MSE = 0.004, $P = 0.042$, $\eta_p^2 = 0.28$], with subjects having larger ellipse areas in the Active condition than the Passive condition (M = 0.091, SE = 0.025 and M = 0.062, SE = 0.013, respectively). The main effect of Trial was also significant [$F(2,26) = 4.12$, MSE = 0.001, $P = 0.028$ (when the Greenhouse-Geisser correction is applied, $P = 0.052$), $\eta_p^2 = 0.24$], reflecting the fact that ellipses grew larger over time. Again, the interaction was not significant [$F(2,26) = 1.93$, MSE = 0.001, $P = 0.166$, $\eta_p^2 = 0.13$]. These differences indicate that individuals' postural sway in the passive condition had lower spatial complexity and exhibited a more rigid coordinative structure. In other words, the group difference in ellipse area suggests that the restraint imposed in the passive condition made it more difficult for people to make postural adjustments, and their ellipse areas were smaller as a result.

The second measure of postural stability was sample entropy. Sample entropy is a measure of the amount of complexity of time series data. Higher sample entropy indicates greater data complexity, and a wider array of movements (including smaller adjustments to increase stability) indicate increased ability and attempts to maintain postural stability. Thus, higher scores can be interpreted as a quantitative description of greater postural stability (with lower scores indicating greater postural instability). The MATLAB code used to calculate sample entropy is

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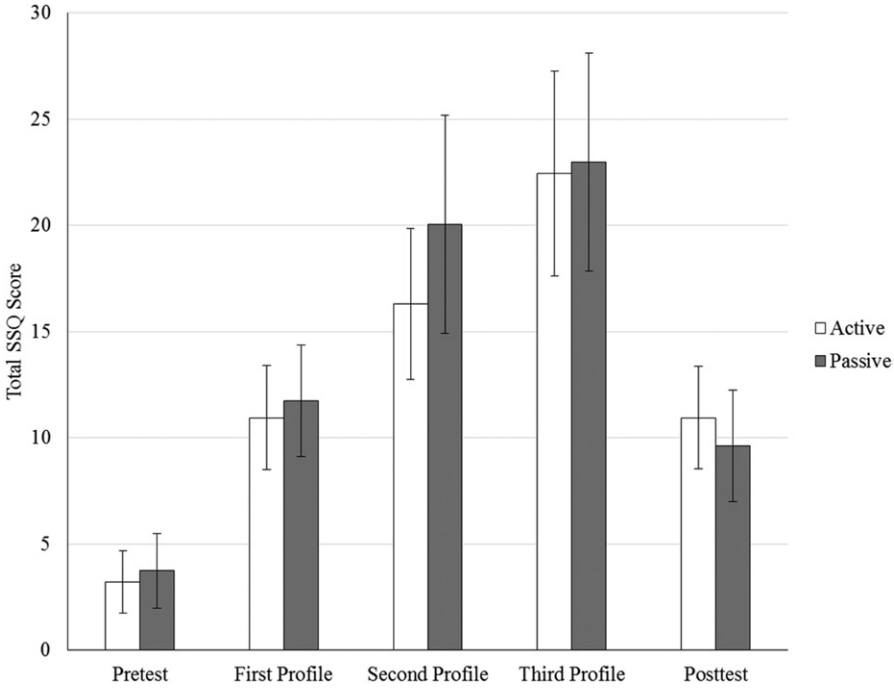

Fig. 2. Total SSQ score over time as divided by the five assessments. First, second, and third participant profile assessments were all given while standing on the platform, whereas the pretest and posttest assessments were not made while subjects were on the platform. Note: active refers to the ability to actively adapt one's posture.

provided in **Appendix A** [online only supplemental data: https://doi.org/10.3357/amhp.4998sd.2018]. The data were trimmed as described above, and entropy was calculated in both the anterior-posterior and lateral axes (Fig. 3). Both sets of data were submitted to 2 (Condition) × 3 (Trial) ANOVAs. There were no significant effects in the anterior-posterior axis. In the lateral axis there was a significant main effect of Condition [$F(1,13) = 37.66$, MSE = 0.04, $P < 0.001$, $\eta_p^2 = 0.74$], reflecting the fact that entropy was higher in the Active condition. The main effect of Time was not significant [$F(2,26) = 0.06$, MSE = 0.001, $P = 0.943$, $\eta_p^2 = 0.01$], and the interaction also failed to reach significance [$F(2,26) = 1.78$, MSE = 0.001, $P = 0.189$, $\eta_p^2 = 0.12$]. Higher sample entropy in the Active condition suggests that when people were free to adjust their posture, they were able to make more, smaller adjustments to maintain stability. Because of this, there is more complexity in Active group head movements. Movements in the Passive condition were characterized by larger, more regular movements, which resulted in lower entropy scores (Fig. 3, bottom panels). This evidence indicates that individuals exhibited more rigid postural behavior during the passive condition compared to the more functionally adaptive behavior during the active condition.

To ensure that the motion profiles did not differ in the amount of motion sickness they induced, Total SSQ scores were submitted to a 2 (Condition) × 3 (Profile) ANOVA. Neither the main effect of Condition nor Profile was significant [$F(1,13) = 0.74$, MSE = 135.10, $P = 0.405$, $\eta_p^2 = 0.05$] and [$F(2,26) = 2.32$, MSE = 204.39, $P = 0.119$, $\eta_p^2 = 0.15$], respectively. The interaction also failed to reach significance [$F(2,26) = 0.86$, MSE = 59.50, $P = 0.434$, $\eta_p^2 = 0.06$]. Thus, no differences were detected with respect to one profile being more or less provocative than the others. This finding is not surprising because the motion profiles are all started from different points within the same sea state data profile, but the analysis was a necessary methodological check.

Finally, MSSQ scores were analyzed to determine whether there were any effects based on one's propensity for motion sickness. The MSSQ data are presented in **Table II**. One outlier with a particularly high MSSQ score (more than 3 SD above the mean) was removed, and partial correlations accounting for MSSQ were calculated between SSQ and entropy in the lateral axis. There were no significant correlations following any profile in either the Active or Passive condition ($P > 0.16$). Additionally, correlations between average entropy and MSSQ were calculated. For the Active condition, the correlation was not significant ($P = 0.99$, $r < 0.01$); this was also true for the Passive condition ($P = 0.24$, $r = 0.42$). The same analyses were run with the outlier included, and the same pattern of results was found.

Because the motion may not have been enough to elicit motion sickness, as evidenced by the low dropout rate and relatively low SSQ scores, additional analyses were conducted only on those who exhibited more extreme scores. The results are presented in **Appendix B** [online only supplemental data: https://doi.org/10.3357/amhp.4998sd.2018]. Briefly, the pattern of results displayed by those classified as motion sick did not differ from the sample as a whole. This suggests that those who found this stimulus more provocative showed similar behaviors as those who did not. That is, they exhibited the same postural instability with respect to ellipsis area and sample entropy, but did not differ in MS symptomatology even under conditions of reduced stability.

Finally, it is possible that the data may show a temporal relationship between postural stability and MS symptoms. Previous evidence suggests that instability appears before subjects report feeling motion sick.[23] To examine this, measures of instability (sample entropy and ellipsis area) were correlated with the SSQ administered after the measure was taken (e.g., entropy after the first profile was correlated with SSQ after the second profile). None of the correlations was significant.

DISCUSSION

Although the exact etiology of MS is unknown, PIT suggests that MS symptoms arise from an inability to adapt postural balance to the demands of the current environment. The current

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Table I. Total and Subscale SSQ Scores.

	NAUSEA		OCULOMOTOR		DISORIENTATION		TOTAL	
TIME	ACTIVE	PASSIVE	ACTIVE	PASSIVE	ACTIVE	PASSIVE	ACTIVE	PASSIVE
Pretest	3.41 (2.15)	4.09 (1.65)	3.79 (1.54)	3.79 (2.21)	0.00 (0.00)	0.99 (0.99)	3.21 (1.46)	3.74 (1.75)
Profile 1	14.31 (4.33)	11.58 (2.86)	8.12 (1.68)	10.29 (2.59)	4.97 (2.77)	7.95 (4.05)	10.95 (2.46)	11.75 (2.63)
Profile 2	20.44 (4.99)	19.76 (4.63)	13.54 (3.39)	19.49 (5.37)	5.97 (4.05)	9.94 (4.71)	16.30 (3.56)	20.04 (5.14)
Profile 3	25.89 (5.14)	23.85 (5.08)	20.03 (5.43)	22.20 (5.42)	8.95 (3.13)	9.94 (5.54)	22.44 (4.83)	22.97 (5.14)
Posttest	12.95 (3.41)	8.18 (3.30)	9.75 (2.69)	10.83 (3.05)	3.98 (1.74)	3.98 (2.27)	10.95 (2.40)	9.62 (2.62)

investigation assessed PIT by comparing simulator seasickness symptoms within a virtual reality environment that incorporated actual motion taken from a rigid-hulled inflatable boat, a type of boat commonly used by the Navy. This is a more ecologically valid motion than that typically used in a lab setting; however, there are still some limitations that should be noted. People generally do not stand up while on the type of ship simulated, although there may be operational reasons this is necessary. As mentioned before, the motion is relatively unpredictable, but it is possible that the overall sea state allows for some adaptation. Thus, the nature of the task was reactive, but subjects may have been able to interpret some characteristics of the motion.

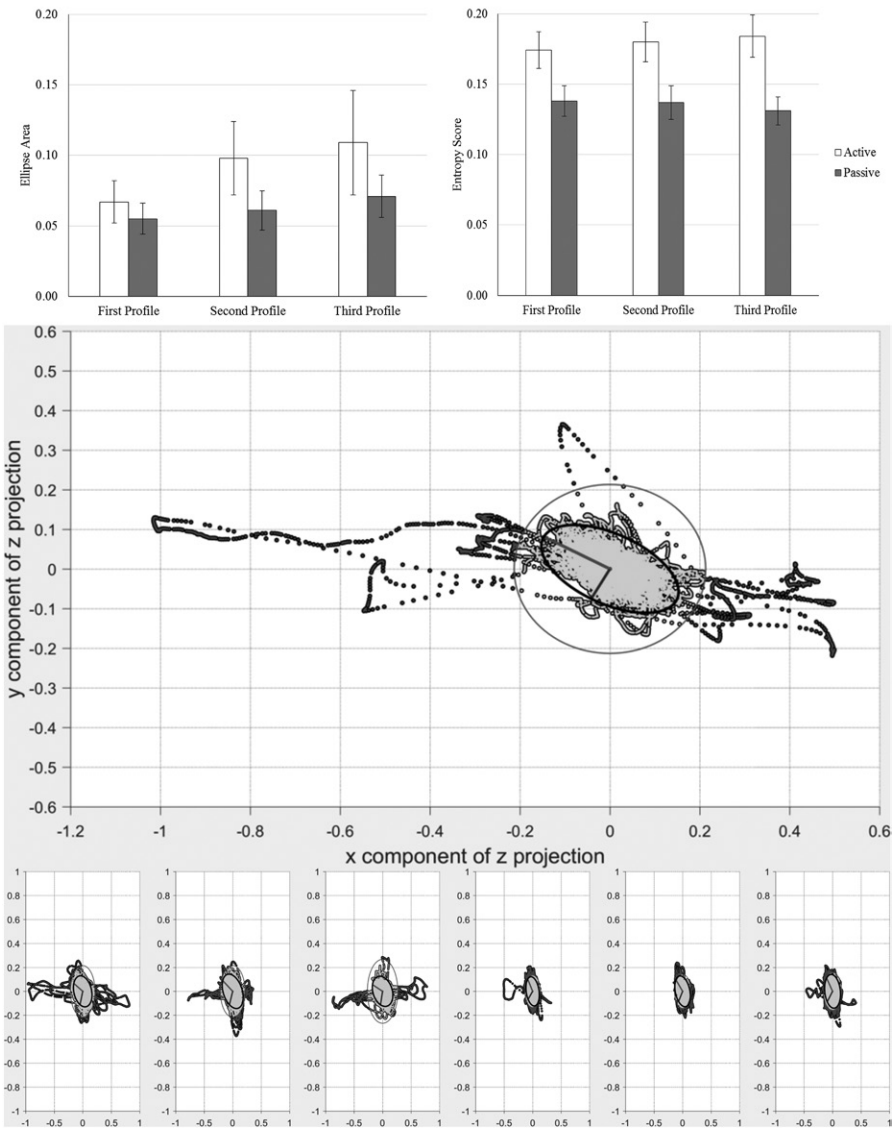

Fig. 3. Ellipse area (top left panel) and Entropy Score (top right panel) following each profile. Example head tracking data from a single subject showing an oval surrounding the ellipse that encompasses 98% of the data and individual head positions during motion exposure (middle panel). Head tracking data from the same subject showing the difference between the passive (first three graphs) and active (second three graphs) conditions (bottom panel).

Postural instability was imposed on subjects by having them participate in both an active and passive version of the experiment. Both experimental conditions involved a virtual seafaring environment presented through an HMD while a moving platform created actual motion in sync with the simulated motion. In the active condition, subjects were free to control their foot width while participating in the three simulated sea state profiles and the accompanying counting tasks. In the passive condition, subjects were constrained to a fixed foot width and were not able to move their feet during the simulated sea state profiles. It is possible that subjects in the Passive condition could use the foot bindings for additional support. We do not believe this is the case for two primary reasons. First, the nature of the motion profile was such that subjects had to react to the motion rather than anticipate it. Even though the bindings could be used to exert additional pressure, the reactive nature of the task makes this difficult, and the bindings prevented the subjects from fully making the microadjustments necessary to control posture. Second, the bindings were placed relatively close together, which would also reduce their utility for maintaining postural control. While it is possible that subjects may have been able to use the bindings to aid posture, the overall effect was to reduce postural control as evidenced by the ellipsis and entropy measures.

Our methodological checks confirm that the passive condition and overall simulation were effective for three reasons: 1) restricting the feet likewise restricted head movement; 2) subjects

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Table II. MSSQ Data.

	SUBJECT	MSSQ
	1	6.00
	2	2.25
	3	0.00
	4	2.13
	5	6.00
	6	3.00
	7	4.50
	8	10.00
	9	5.00
	10	1.29
	11	32.14
	12	0.00
	13	7.00
	14	1.00
Mean		5.74
Standard Error		2.17

did exhibit greater postural instability in the passive condition; and 3) simulator sickness increased throughout the experiment, indicating that the experimental setup was capable of inducing simulator sickness. Thus, the experimental setup did effectively create differences in postural instability between active and passive conditions while simultaneously simulating an experience capable of inducing significant symptomology.

Although the experimental method provided strong empirical support for the setup itself, there was no evidence that postural instability contributed directly to sickness symptoms. There were no significant or even marginally significant differences between active or passive conditions within reported symptoms. Moreover, there is no evident relationship between reported symptoms and individual differences in postural instability. Taken together, these results do not support PIT as a causal explanation of MS symptoms.

These findings then raise questions about why our results differ from previous evidence supporting PIT. For example, Scuderi[20], Smart et al.[22], and Stoffregen et al.[23] found that differences in postural motion prior to MS could be used to predict who will get sick. Active exploration is necessary for successful adaptation according to PIT, so the theory would predict that the individual would be more likely to experience MS independent of past MS susceptibility if that person were forced into a passive situation (fixed stance width). Our results suggest that concurrent differences in postural instability do not have a direct impact on symptomology. It remains possible that some individual differences prior to the motion experience are contributing to symptom severity, and that the current dataset lacks sufficient statistical power to detect such individual differences. Still, the current results suggest that the postural behaviors do not have an immediate impact on symptomology. This is not a novel result. For example, Warwick-Evans et al.[27] found that people had greater MS when in a restrained position (lying down or by rigid bars). Presumably, the restraint should increase postural stability and reduce MS. Other studies have failed to find any relationship between posture and MS.[11]

A primary limitation of the current study involved our methodological approach in the "active" condition. Although subjects could freely adapt their posture by regulating their stance width, subjects could not anticipate and adapt their posture in advance of the motion. This hypothetical condition would represent the purest active condition possible such as when pilots can anticipate the change in motion if they are in full control of the aircraft and there was no turbulence. The current study thus used an active condition that allowed for functionally adaptive behavior without truly allowing the participant to anticipate the changes. In a sea state environment, there is a question of whether such a true active condition is even possible as fully predicting the wave motion would be near impossible even if the individual were steering the boat. It is possible for people to adapt to the predominant wave motions for a given sea state (i.e., getting one's "sea legs"); however, individual perturbations will remain and conditions will change. The lack of a "true" control condition limits the conclusions that can be made regarding postural stability. However, the present experimental conditions were conducted in a simulated naval operating environment and provided subjects the opportunity to adapt their postural stability to the limitations of that environment. It is possible that a true active condition is necessary for PIT to better describe behavior, albeit a true active condition remains more of a theoretical possibility in a sea state environment and would not readily apply to seasickness.

In summary, people experienced motion equivalent to being on a small boat in a bay under two conditions. In one (Passive), their feet were restrained, making it more difficult to maintain a stable posture. In the other (Active), they could adjust their stance freely. Posture was less stable in the Passive condition as shown by the smaller ellipse area and lower sample entropy exhibited in head motion data. However, there were no differences in Simulator Sickness Questionnaire scores. The main result was that motion sickness scores increased over time, which is to be expected. Postural Instability Theory would suggest that as instability increases, motion sickness should show a corresponding increase. However, as **Fig. 4** shows, there does not appear to be any relationship between stability as measured by sample entropy or ellipsis area and the severity of motion sickness symptoms one experiences, nor were there any significant symptom differences between the active and passive conditions. Taken together, the results of this experiment suggest that postural instability is not a good correlate of motion sickness.

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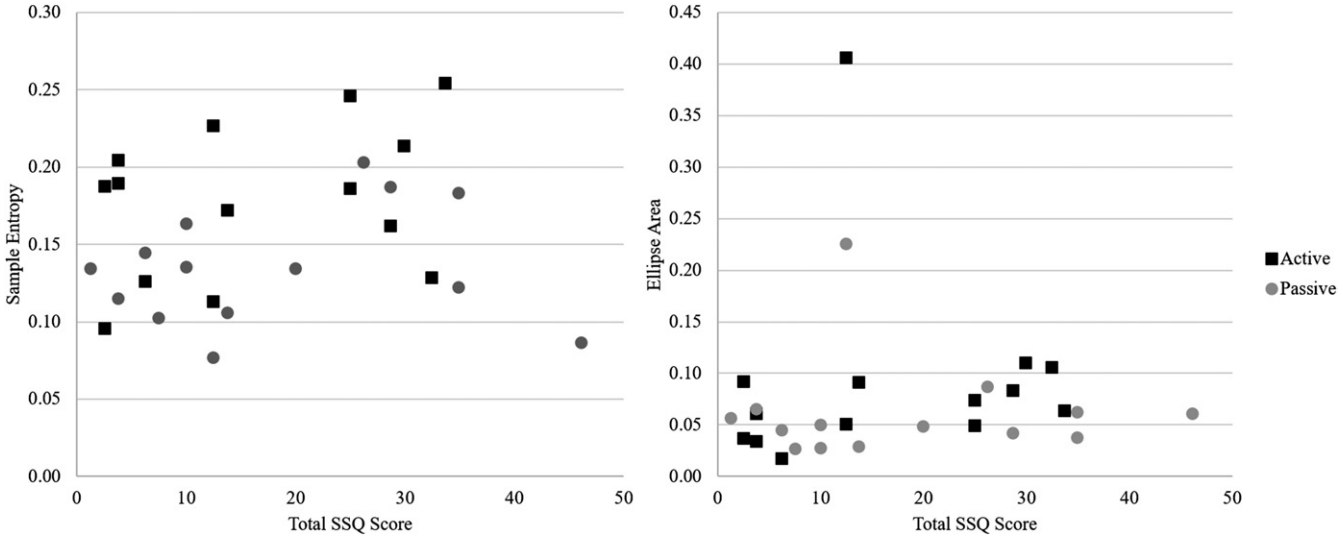

Fig. 4. Scatterplots of sample entropy and total SSQ score (left) and ellipsis area and total SSQ score (right) averaged over the three motion profiles.

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