Isolating the Effects of Vection and Optokinetic Nystagmus on Optokinetic Rotation-Induced Motion Sickness

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Objective: This study investigates isolated effects of vection and optokinetic nystagmus (OKN) on visually induced motion sickness (VIMS) provoked by rotating optokinetic drum patterns. **Background:** VIMS was the subject of recent standardization activities, but the effects of OKN have not been studied in the absence of vection. **Method:** Experiment 1 suppressed OKN by eye fixation and examined VIMS severity (both ordinal and ratio scale) and time spent in saturated vection at four pattern rotating velocities of 0, 2, 14, and 34 degrees per second (dps). Experiment 2 suppressed vection by adding a peripheral visual field rotating in the opposite direction to the rotating patterns. VIMS severity and OKN slow-phase velocity were studied at four rotating velocities of 0, 30, 60, and 90 dps. **Results:** Results from Experiment 1 indicated that VIMS severity increased as the pattern velocity increased from 0 dps to 34 dps. Results from Experiment 2 indicated that as the velocity of the rotating pattern increased, the slow-phase velocity of OKN and the severity of VIMS increased and peaked in the 60-dps condition. In both experiments, ratio-scaled nausea data significantly correlated with ordinal-scaled nausea ratings. **Conclusion:** VIMS can still occur in the absence of either vection or OKN. Interestingly, the profile of the summed results of the two experiments matches nicely with the profile reported by Hu et al. in which neither OKN nor vection were controlled. **Application:** Potential applications include modeling and reduction of VIMS in computer gaming environments.

INTRODUCTION

Visually induced motion sickness (VIMS) is a major ergonomics concern with the use of virtual reality (VR) technology (Stanney et al., 1998). It was reported that after 1 hr of exposure to a VR environment, 88% of the users experienced various VIMS symptoms, 71% experienced nausea, and nearly 50% had to terminate their exposure prematurely (Stanney, Kingdon, & Kennedy, 2002). In 2005, concerns about VIMS among viewers of computer games and entertainment prompted the publication of ISO International Workshop Agreement 3 on image safety (ISO, 2005), and in 2006, the Commission Internationale de l'Eclairage commissioned a technical committee, TC1-67, to study the effects of dynamic and stereo visual images on human health.

Viewers exposed to optokinetic stimuli can exhibit vection, optokinetic nystagmus (OKN; Buttner and Buttner-Ennever, 1988), and VIMS (Flanagan, May, & Dobie, 2002; Webb & Griffin, 2002). VIMS is a variant of motion sickness (MS; Griffin, 1992; Hettinger & Ricco, 1992; Reason, 1978), and sensory rearrangement theory is the most frequently cited theory to explain VIMS occurrence (Reason, 1978). This theory predicts that vection and not OKN is critical for generating VIMS. Hettinger, Berbaum, Kennedy, Dunlap, and Nolan (1990) reported that vection, the illusion of self-motion, was a necessary condition for VIMS elicitation. Consequently, VIMS provoked by rotating optokinetic stimuli has been referred to as "vection-induced" MS (e.g., Hu et al., 1997; Hu & Stern, 1998; Stern, Hu, LeBlanc, & Koch, 1990). A review of the literature indicates that participants in these studies also exhibited OKN.

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In 1994, Ebenholtz and his colleagues proposed the extraocular afferent hypothesis to predict that VIMS is caused by abnormal OKN responses. This prediction was supported by Webb and Griffin (2002), who observed that OKN suppression significantly reduces VIMS severity without significant changes in vection perception. As of today, the effects of vection and OKN on VIMS are still the subject of debate.

Although the effect of vection and OKN on VIMS has been studied by several empirical studies (Flanagan, May, & Dobie, 2004; Stern, Hu, Anderson, Leibowitz, & Koch, 1990; Webb & Griffin, 2002, 2003), a study examining both the isolated effects of vection and the isolated effects of OKN on VIMS under the same experimental setup could not be found. Hu, Stern, Vasey, and Koch (1989) reported an inverted *U*-shaped relationship between rotation velocity of optokinetic stimuli and VIMS severity. In their study, both OKN and vection were not suppressed, and VIMS severity peaked when the stimuli rotated at 60 degrees per second (dps) in the yaw axis.

In this study, we sought to repeat Hu et al.'s (1989) study but to isolate the effects of vection and OKN. The specific objectives were to (a) examine the isolated role of vection perception and the isolated role of OKN in VIMS severity under a similar experimental setup, (b) determine the VIMS response characteristics as functions of the rotating velocity of stimuli in the absence of OKN or vection, and (c) compare the use of ratio-scaled and ordinal-scaled VIMS severity data. The third objective was prompted by the lack of ratio-scaled VIMS data. A review of the literature indicates that nearly all the previous VIMS studies measured VIMS severity using ordinal scales (e.g., the Simulator Sickness Questionnaire [SSQ] by Kennedy, Lane, Berbaum, & Lilienthal, 1993, and the 7-point nausea rating scale by Golding & Kerguelen, 1992; Webb & Griffin, 2002, 2003). One important application of ratio-scaled data is in the development of computational models (Oman, 1982).

In summary, this article is a report of the results of two experiments. In Experiment 1, we examined the effect of the rotating velocity of an optokinetic pattern (pattern velocity) on VIMS severity in the absence of OKN and in the presence of vection. In Experiment 2, we examined the effect of pattern velocity on VIMS severity in the absence of vection and in the presence of OKN. Participants in both experiments were exposed to moving image patterns. However, instead of controlling the rotating velocities of the patterns to be the same in the two experiments, we controlled the retinal slip velocities of the pattern stimuli. Retinal slip velocities are the velocities of image projections on the retinas.

There were two reasons for controlling the retinal slip velocities in our study: (a) The two-visual system theory suggests that motion perception is initiated when motion-sensitive receptors on the retinas detect the retinal slip of moving images (Leibowitz & Post, 1982), and (b) as the eyes follow a moving image pattern, the retinal slip is reduced. Consequently, we sought to remove the confounding relationship between the presence of OKN and retinal slip velocities.

EXPERIMENT 1: ISOLATED EFFECTS OF VECTION ON VIMSWITH OKN SUPPRESSION

Objective and Hypothesis

In Experiment 1, we considered the relationship between vection and the severity of VIMS in the absence of OKN in viewers watching a wide-field-of-view optokinetic striped pattern rotating in the yaw direction at different velocities (pattern velocity). We hypothesized that (a) pattern velocity, in the absence of OKN, will have a significant main effect on the time spent in saturated vection (Hypothesis 1) and on reported VIMS severity (Hypothesis 2) (on the basis of Hu et al., 1989), and (b) as the pattern velocity increases, VIMS will increase, peak, and reduce (Hypothesis 3) (on the basis of Hu et al., 1989).

Method and Design

Method to suppress and monitor OKN. Eye fixation has been shown to completely suppress OKN (e.g., Brandt, Dichgans, & Koenig, 1973; Flanagan et al., 2002; Stern et al., 1990; Webb & Griffin, 2002). Brandt et al. (1973) used an eye fixation point subtending 1° to suppress OKN. To verify whether an eye fixation point of 1° in diameter could completely suppress OKN in this study, a pilot test was conducted under a similar experimental setup as Experiment 1. The visual stimulus took the form of an alternating black-and-white-striped pattern (cf. Hu et al., 1997) rotating at a speed of 34 dps. Eight participants watched the stimulus without eye fixation for 1 min, followed by a 2nd min of exposure with eye fixation.

Electrooculogram (EOG) recordings were made using the BIOPAC® EOG amplifier system (EOG100C, BIOPAC Systems Inc., CA, USA) with the EOG data acquisition and analysis software (AcqKnowledge, BIOPAC). The EOG data sampling rate was set at 200 samples per second with a low-pass filter set at 35 Hz. Both horizontal and vertical eye movements were monitored and recorded by separate channels.

All EOG recordings had clear slow-phase and fast-phase OKN cycles when the participants did not fixate their eyes. When the participants fixated their eyes, the EOG recordings showed no OKN cycles. These results confirmed that OKN could be completely suppressed by an eye fixation of 1° in diameter in Experiment 1. This is consistent with the previous findings that OKN can be fully suppressed by eye fixation point at a drum rotation velocity of 60 dps or faster (Brandt et al., 1973; Flanagan et al., 2002; Webb & Griffin, 2002).

Participants. Past studies have shown that gender can influence symptoms of motion sickness (Griffin, 1992), and female Chinese have been shown to be hypersusceptible to VIMS (Stern et al., 1993). In this study, female Chinese participants were recruited to examine the effects of vection and OKN on VIMS. Participants in Experiment 1 were 16 Chinese female university students ages 22 to 28. All were consenting volunteers who were healthy and free of medication or illness. This experiment was approved by the Human Subject and Research Ethics Committee at the Hong Kong University of Science and Technology.

Two participants vomited during their first exposure and decided to quit the experiment before completing all four conditions. The other 14 participants completed all four conditions, and their data were used in the data analysis. Their mean Motion Sickness Susceptibility Questionnaire (MSSQ)–Short raw score (Golding, 2006) was 19.5 ($SD = 12.6$), which was significantly higher than the average score of 12.9 ($SD = 9.9$) reported by Golding (2006). This was consistent with the finding by Stern et al. (1993) that Chinese females are hypersusceptible to motion sickness.

All participants had uncorrected or corrected visual acuity greater than or equal to 20/20 in both eyes, and they were all able to complete a series of line length estimation tests using free modulus estimation methods (Stevens, 1971). Participants were compensated for their time at an hourly rate of HK$50 (about US$7).

Visual stimulus and apparatus. The optokinetic drum used by Hu et al. (1989) was reconstructed virtually by a computer. Images of this computer-generated virtual drum were then projected on a circular wide-angle screen (Da-Lite Inc., USA: Matte White) via three projectors. Similar to the optokinetic drum used by Hu et al. (1989), the virtual drum had 24 pairs of black and white stripes with uneven widths (a white stripe had an angular width of 9.3° and a black stripe had an angular width of 5.7°). Views from the center of the virtual drum were projected on a 200° (horizontal) × 50° (vertical) circular screen. The black-and-white-striped pattern was presented at a refresh rate of 60 Hz and a resolution of 1,920 × 480 pixels by a NVIDIA GeForce 7600GT graphics card running on an Intel Core 2TM computer (2.4 GHz CPU, 2 GB memory).

An external hardware, Matrox Triple Head2Go, was used to split the view into three images for the three projectors. Each of the three images had about 6° horizontal overlap with the adjacent images to create a seamless, wide-angle, rotating image pattern. Gradient image blending programmed by Visual Basic was used to fade the edges formed by image overlapping.

Figure 1 shows a participant standing in front of the circular screen. A chin rest was used to fix the head position, and participants were required to hold on to a rigid frame with both hands. Full restraint was not used as it might increase levels of claustrophobia and sickness symptoms (Faugloire, Bonnet, Riley, Bardy, & Stoffregen, 2007; Smart, Stoffregen, & Bardy, 2002). A video camera was used to monitor the participants during the entire exposure period. An OPTEC® 2000 vision tester was used to measure the visual acuity of the participants.

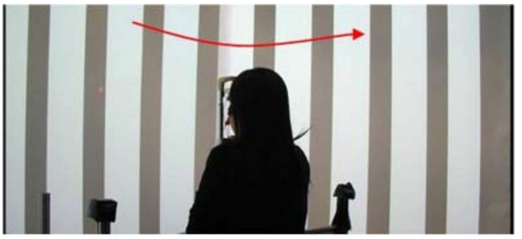

Figure 1. A participant standing in front of the rotating striped pattern used in Experiment 1. The arrow is added to indicate the direction of pattern rotation. The head of the participant was fixed by a chin rest. Participants were required to hold on to self-made rigid brackets with both hands during the entire exposure period.

Experimental design. The only independent variable in Experiment 1 was the pattern velocity, and it had four levels: 0, 2, 14, and 34 dps. We used a within-subject design and a balanced 4 × 4 Latin square to counterbalance the order of presenting the four conditions to 4 participants. This design was repeated four times for the 16 participants. To reduce effects of habituation, the time interval between two successive conditions was at least 7 days (Regan, 1995).

In Hu et al. (1989), velocities of 30, 60, and 90 dps were used. Because Hu did not suppress OKNs in his participants, the resulting image velocities on the retinas were much reduced. From the OKN gain data reported by Koenig, Allumm, and Dichgans (1978; 0.91, 0.72, and 0.63 for pattern velocities of 30, 60, and 90 dps), the retinal image velocities were estimated to be 2.7, 16.8, and 33.4 dps. Because of the technical limitations of the display system, the closest pattern velocities that could be displayed were 2, 14, and 34 dps. These were the velocity levels used in this experiment. These four conditions of pattern velocity were designed to map the four conditions of retinal slip velocity in Experiment 2. The reason for controlling the retinal slip was explained in the Introduction.

Procedure. Before the experiment, all participants completed a series of visual acuity tests, a motion sickness susceptibility survey questionnaire (So, Finney, & Goonetilleke, 1999), and the MSSQ–Short questionnaire (Golding, 2006). Before the commencement of each condition, participants were requested to complete a line length estimation test to familiarize them with the free modulus magnitude estimation method. After that, participants completed a pre-exposure SSQ. If the participants reported any "moderate" symptoms, they would be asked to rest in an air-conditioned room until the symptom had subsided. If the symptom continued after resting for 15 min, participants were asked to come back on another day.

The participants were assigned to one of the four conditions according to the counterbalanced Latin square, and the duration of exposure of each condition was 30 min. At every 2 min during the exposure, participants reported their subjective nausea severity level using both the free modulus magnitude estimation method and the 7-point nausea rating scale (Golding & Kerguelen, 1992; Webb & Griffin, 2003). In conditions other than the 0-dps condition, participants also needed to report any change in the perceived vection velocity using a fixed-modulus magnitude estimation method (Stevens, 1971). EOG recordings were taken throughout the entire exposure period. At the end of each 30-min condition, participants completed a postexposure SSQ.

Fixed-modulus magnitude estimation of vection velocity. To identify an appropriate anchor point for the fixed-modulus magnitude estimation of the vection velocity (i.e., the reference 100), participants were exposed to a precondition session lasting up to 90 s. A break of 5 to 10 min was given between each precondition session and the main condition.

In the precondition session, participants were exposed to rotating patterns at the same speed as the upcoming condition. Out of the 14 participants, eight reported brief moments of experiencing full vection saturation in all four precondition sessions (i.e., a visual illusion that they were rotating while the patterns appeared to be stationary). The participants were asked to remember that experience as the reference point of 100 for the subsequent estimation of vection velocity using the fixed-modulus magnitude estimation method. For the 6 participants who did not experience full vection saturation in one or more precondition sessions, they were educated about the meaning of the 100 reference rating. The reference rating of 100 refers to the experience of self-motion with a similar magnitude to the velocity of the rotating pattern but in the opposite direction. In other words, a rating of 100 was referenced to the sensation of full vection saturation.

Data analysis. The time spent in saturated vection and vection velocity (relative to pattern velocity) was measured for each participant in each condition. The nausea ratio scale data collected by the free-modulus magnitude estimation method was transformed by the modulus equalization method (Stevens, 1971). Because the data were not normally distributed, nonparametric statistical tests were used. Friedman two-way ANOVAs and Wilcoxon signed rank tests were used to test the main effects of pattern velocity. Spearman's rank correlation tests were also used. All the individual time-series recordings of EOG along the horizontal axis were analyzed to identify valid OKN cycles with the use of Matlab™ codes and were confirmed by visual inspection. For all participants in all four conditions, no repetitive OKN cycle was identified. The OKN slow-phase velocity (SPV) for all four conditions was therefore zero. This means that OKN was fully suppressed by eye fixation in Experiment 1.

Results

Two participants dropped out because they got too sick. As a result, one of the 4 × 4 Latin squares used to balance the order of presentation could not be completed. Results of the Spearman's correlation tests indicated that the order of presentation did not significantly correlate with VIMS severity (Spearman, $N = 56$; nausea ratio scale, rho = 0.037, $p = .789$; 7-point nausea rating, rho = 0.015, $p = .914$; postexposure SSQ Nausea, rho = −0.103, $p = .449$) and time spent in saturated vection (Spearman's rho = −0.087, $N = 56$, $p = 0.524$).

Consistent with the observations reported in Hu et al. (1989), as the retinal slip velocity increased from 14 dps (corresponded *with* the 60-dps drum velocity condition in Hu et al., 1989) to approximately 34 dps (corresponded with the 90-dps drum velocity condition in Hu et al., 1989), participants spent less time in saturated vection, although the change was not significant (Wilcoxon, $N = 14$, $z = -1.15$, $p = .249$). This result did not support Hypothesis 1.

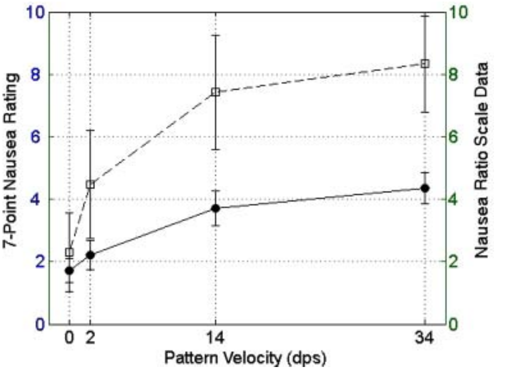

Figure 2. Ratio-scaled visually induced motion sickness severity (□) and ordinal-scaled nausea ratings (●) recorded at the end of 30-min exposures to rotating black-and-white patterns at 0°, 2°, 4°, and 14° per second. Participants fixed their eyes to eliminate OKN. Means and standard errors are shown.

Figure 2 shows that as the pattern velocity increased, VIMS severity as measured by the 7-point nausea rating and the nausea ratio scale increased. The postexposure SSQ Nausea subscores data also followed a similar trend (Table 1). Results of the Friedman ANOVAs indicated that increases in pattern velocity resulted in significant increases in VIMS severity as measured by the nausea ratio scale (Friedman, $s = 6.88$, $df = 3$, $p = .014$), 7-point nausea ratings (Friedman, $s = 20.12$, $df = 3$, $p = .000$) and postexposure SSQ Nausea subscores (Friedman, $s = 11.16$, $df = 3$, $p < .011$). These results support Hypothesis 2.

Results of the Wilcoxon tests indicated that when the pattern velocity increased from 2 dps to 14 dps, significant increases in both average nausea ratings (Wilcoxon, $N = 14$, $z = -2.199$, $p = .028$) and postexposure SSQ Nausea subscores were found (Wilcoxon, $N = 14$, $z = -2.135$, $p = .033$). The nausea ratio scale data in the 14-dps and 34-dps pattern velocity conditions were significantly larger than the nausea ratio data collected in the 0-dps pattern velocity condition (Wilcoxon, $N = 14$; 0 dps vs. 14 dps, $z = -2.521$, $p = .012$; 0 dps vs. 34 dps, $z = -2.497$, $p = 0.013$). The increases of VIMS severity as pattern velocity increased from 14 dps to 34 dps did not support Hypothesis 3.

Correlation analyses indicated significant positive relationships among the ratio-scaled

TABLE 1: Rotating Velocity of the Stimuli, Retinal Slip Velocities, and Postexposure SSQ Nausea Subscores (Means And Standard Errors) Collected in Experiments 1 and 2

Experiment 1: The Presence of Vection Without OKN			Experiment 2: The Presence of OKN Without Vection		
Rotating Speed of the Stimuli in dps	Retinal Slip Velocity in dps	Postexposure SSQ Nausea Subscore *M* (*SE*)	Rotating Speed of the Stimuli in dps	Retinal Slip Velocity in dps	Postexposure SSQ Nausea Subscore *M* (*SE*)
0	0	29.3 (6.5)	0	0	14.3 (5.1)
2	2	32.0 (8.0)	30	2.1	35.4 (9.2)
14	14	58.6 (9.4)	60	12.2	42.2 (9.3)
34	34	60.0 (12.7)	90	48.6	34.1 (9.4)

Note. SSQ = Simulator Sickness Questionnaire (Kennedy, Lane, Berbaum, & Lilienthal, 1993); OKN = optokinetic nystagmus; dps = degrees per second.

nausea severity ratings (using free magnitude estimation; Stevens, 1971), nausea ratings (7-point nausea scale; Golding & Kerguelen, 1992) and the postexposure SSQ Nausea scores (Kennedy et al., 1993) (Spearman, $N = 56$; nausea ratio scale vs. 7-point nausea rating, rho = 0.883, $p = .000$; nausea ratio scale vs. postexposure SSQ Nausea, rho = 0.794, $p = .000$; 7-point nausea rating vs. postexposure SSQ Nausea, rho = 0.851, $p = .000$). These results indicated that ordinal-scaled and ratio-scaled levels of VIMS were consistent.

EXPERIMENT TWO: ISOLATED EFFECTS OF OKN ON VIMS WITH VECTION SUPPRESSION

Objective and Hypotheses

Experiment 2 studied the relationship between OKN and VIMS severity in the absence of vection while the participants watched patterns rotating at different velocities in the yaw direction. We hypothesized that (a) pattern velocity, in the absence of vection, will significantly influence OKN SPV (Hypothesis 4a, based on Koenig et al., 1978;) and VIMS severity (Hypothesis 4b, based on Hu et al., 1989); (b) as pattern velocity increases from 0 dps to 90 dps, VIMS severity will peak, increase, and reduce (Hypothesis 5); and (c) OKN SPV will be significantly correlated with VIMS severity (Hypothesis 6, based on Webb & Griffin, 2002, 2003). Hypothesis 5 was based on Hu et al. (1989), who reported that VIMS severity peaked when participants were exposed to patterns rotating at 60 dps in the presence of both OKN and vection.

Method and Design

Method to suppress and monitor vection. Brandt, Dichgans, and Koenig (1973) reported that when viewing a central and peripheral striped pattern rotating in the yaw axis but in opposite directions, viewers experienced OKN without vection. The rationale was that while OKN is stimulated by the central rotating patterns, vection is stimulated by both the central and peripheral rotating patterns. Brandt and his colleagues reported that if the central and peripheral patterns are rotating in opposite directions, OKN will follow the central rotating patterns, whereas vection can be completely suppressed when the field of view of the central rotating pattern is approximately 100° in diameter.

Inspired by the finding of Brandt et al. (1973), we designed a central and peripheral striped pattern rotating in opposite directions at the same speed (Figure 3b). A pilot test was conducted to determine the size of the central visual field needed to suppress vection in each pattern velocity condition. Participants were the same as those in Experiment 2. The horizontal field of view of the central patterns was varied from 10° to 180° in 10° steps. The maximum vertical size of the central stimulus was 50°, limited by the screen size.

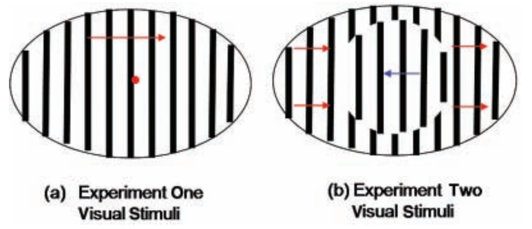

Figure 3. Illustrations of visual stimuli used in Experiments 1 and 2. (a) Alternate black-and-white-striped patterns with an eye fixation point to eliminate optokinetic nystagmus in viewers. (b) Central and peripheral striped patterns rotating in opposite directions to reduce perceived vection in viewers. The size of the central pattern was personalized.

Each participant was exposed to patterns of each size rotating at four different velocities: 15 dps, 30 dps, 60 dps, and 90 dps. In total, each participant was exposed to 64 different trials (18 sizes × 4 velocities). During each trial, the participants were requested to report if vection was experienced and the direction of the perceived self-rotation. If no vection was perceived continuously for 90 s, the trial was terminated. There was a 3-min break between each trial. The ranges of stimuli sizes at which each participant reported no vection were recorded, and the lower limits of the ranges were used as their personalized sizes of the central visual field in Experiment 2.

Participants. We recruited 16 Chinese female university students, ages between 21 and 30, to participate in Experiment 2. Also two of them participated in Experiment 1, with at least 7 days between the two experiments. One participant, not the same person as in Experiment 1, vomited after exposure to one condition and decided to quit. Another participant reported a very high level of nausea even for the condition of 0 dps (i.e., stationary stimuli). After seeking medical advice and consulting Professor Graham Harding (an expert in photosensitive epileptic seizure [PES]; Harding & Harding, 2007), we concluded that the person was not likely to have experienced PES-related symptoms but that it was prudent to relieve her from participating in the experiment.

The remaining 14 participants completed all four sessions, and their data were used in the data analysis. Their mean MSSQ–Short raw score was 14.6 (*SD* = 7.6), statistically no different from 12.9 (*SD* = 9.9) reported by Golding (2006). All participants had corrected or uncorrected visual acuity of 20/20 or better for both eyes and were able to complete a series of line length estimation tests using the free-modulus magnitude estimation method (Stevens, 1971).

Visual stimulus and apparatus. The visual stimulus consisted of a central rotating striped pattern with personalized fields of view and a peripheral striped pattern rotating in the opposite direction at the same constant speed along the earth-vertical axis (Brandt et al., 1973). The horizontal size of the central visual field was personalized to each participant (ranging from 100° to 180°). Illustrations of visual stimuli used in Experiments 1 and 2 are shown in Figure 3. A chin rest was used to constrain head movements. Experiment 2 involved the use of the same apparatus as in Experiment 1.

Experimental design. Experiment 2 had four conditions to study the effects of the four rotating velocities: 0, 30, 60, and 90 dps. The three nonzero velocities were the same as those used in Hu et al. (1989) and were expected to produce image velocities on the participants' retinas similar to the four conditions used in Experiment 1 (see Experiment 1). Each condition lasted 30 min. Experiment 2 also used a within-subject design and a balanced 4 × 4 Latin square to balance the order of presentation. To minimize effects of habituation, the time interval between two conditions for the same participant was at least 7 days (Regan, 1995).

Procedure. Before the experiment, participants completed a series of line length estimation tests, a visual acuity test, the motion sickness susceptibility survey, and the MSSQ–Short questionnaire. As in Experiment 1, participants completed a pre-exposure SSQ; if anyone reported any "moderate" symptoms, the participant was asked to rest in an air-conditioned room until her symptoms had subsided. Participants whose symptoms persisted after 15 min of resting were asked to come back on another day. EOG calibration runs were conducted before the start of the exposure.

During the 30-min exposure, participants were requested to report subjective nausea severity using free-modulus magnitude estimation and the 7-point nausea rating scale. Unlike in Experiment 1, they were not asked to estimate the vection velocity. Instead, they were asked to rate the vection intensity on a 4-point vection scale (Webb & Griffin, 2002) every 2 min. Participants then completed a postexposure SSQ.

Data analysis. EOG recordings were used to calculate SPVs of the OKNs. To transform the unit of raw EOG data from volts (V) into degrees, an individualized mapping constant was calculated from EOG records of six calibrated eye movements. Because the distributions of SPV data do not follow normal distributions, nonparametric tests are used to analyze the data. Similar to Experiment 1, recordings of EOG were analyzed by Matlab codes to identify cycles of OKNs and to calculate the SPVs.

Results

None of the 14 participants reported any nonzero vection intensity rating during the entire exposure period of the 60-dps and 90-dps conditions. This means that vection perception was fully suppressed by Brandt's method (Brandt et al., 1973). In conditions in which the patterns were rotating at 30 dps, vection perception was fully suppressed in all 14 participants for up to 18 min of exposure. Two participants reported a rating of 1 (intermittent vection) at 20 min and 22 min after the start of the stimulus. Results of the Spearman's correlation test indicate that the order of presentation did not have significant correlation with VIMS severity (Spearman, $N = 56$; nausea ratio scale, rho = –0.076, $p = .579$; 7-point nausea rating, rho = –0.187, $p = .168$; postexposure SSQ Nausea, rho = –0.168, $p = .216$) and OKN SPV (Spearman's rho = –0.023, $N = 56$, $p = .864$).

Results of the Friedman ANOVAs indicate that pattern velocity had a significant main effect on OKN SPV (Friedman, $s = 26.23$, $df = 3$, $p = .000$). This supported Hypothesis 4a. Consistent with Koenig et al. (1978), the results of the Wilcoxon tests indicated that when the pattern velocity increased from 0 dps to 30 dps and from 30 dps to 60 dps, significant increases in OKN SPV were found (Wilcoxon, $N = 14$; 0 dps vs. 30 dps, $z = -3.301$, $p = .001$; 30 dps vs. 60 dps, $z = -2.229$, $p = .026$). When pattern velocity was at 90 dps, although the median

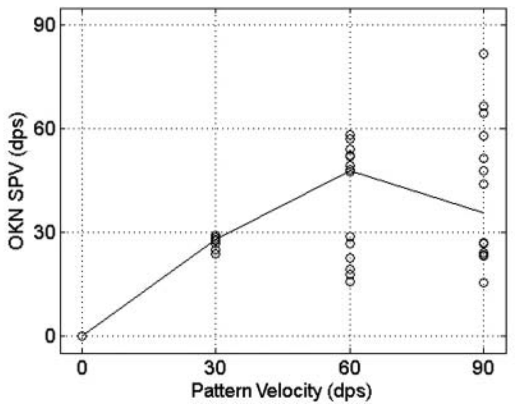

Figure 4. A scatter plot of optokinetic nystagmus slow-phase velocities (OKN SPV; averaged across 30 min) of 14 participants when viewing black-and-white-striped patterns rotating at 0°, 30°, 60°, and 90° per second in the yaw axis. A peripheral striped pattern rotating in the opposite direction of the central striped pattern at the same speed was used to suppress vection. The solid line connects the medians of four pattern velocity conditions.

OKN SPV reduced, the reduced SPV was not significantly different from that reported at 30 dps and at 60 dps (Wilcoxon, $N = 14$; 60 dps vs. 90 dps, $z = -0.91$, $p = .363$; 30 dps vs. 90 dps, $z = -1.726$, $p = .084$, Figure 4).

Figure 5 illustrates the effects of pattern velocity on the ratio-scaled VIMS severity and ordinal-scaled nausea ratings. The figure indicates that VIMS severity increases, peaks, and reduces as the pattern velocity increases. Friedman ANOVAs indicate that these changes were significant in the ratio-scaled VIMS severity ratings (Friedman, $s = 8.35$, $df = 3$, $p = .039$), the ordinal-scaled nausea ratings (Friedman, $s = 20.89$, $df = 3$, $p = .000$), and the postexposure SSQ Nausea scores (Friedman, $s = 8.07$, $df = 3$, $p = .045$; Table 1). These results supported Hypothesis 4b.

When the pattern velocity was at 60 dps, both the ratio-scaled VIMS severity and the ordinal-scaled nausea ratings had the highest values (Figure 5), and these values are significantly greater than their corresponding values when the velocity was at 0 dps (Wilcoxon, $N = 14$; nausea ratio scale, $z = -2.524$, $p = .012$; 7-point nausea rating, $z = -2.994$, $p = .003$). When the pattern velocity was at 90 dps, both ratio-scaled VIMS severity and postexposure SSQ Nausea

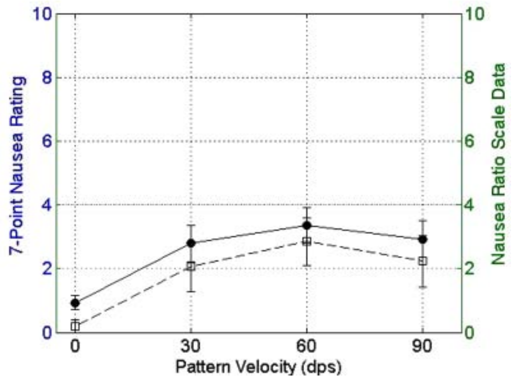

Figure 5. Ratio-scaled visually induced motion sickness (VIMS) severity (□) and ordinal-scaled nausea ratings (●) recorded at the end of 30-min exposures to rotating black-and-white patterns at 0°, 30°, 60°, and 90° per second. Perceived vection in participants was suppressed. Means and standard errors are shown.

scores were not significantly different from those reported in the 0 dps condition (Wilcoxon, $N = 14$; nausea ratio scale, $z = -1.992$, $p = .05$; postexposure SSQ Nausea score, $z = -1.933$, $p = .053$). Results from the Wilcoxon tests supported Hypothesis 5.

Consistent with the findings of Webb and Griffin (2002, 2003), average OKN velocities were significantly correlated with average VIMS severity measured by the 7-point nausea rating (Spearman's rho = 0.4, $N = 56$, $p = .000$) and the nausea ratio scale data (Spearman's rho = 0.31, $N = 56$, $p = .02$). These results supported Hypothesis 6.

Results of the Spearman's correlation test indicated that the ratio-scaled VIMS severity data, the ordinal-scaled nausea ratings, and the postexposure SSQ Nausea subscores were significantly correlated with each other (Spearman, $N = 56$; nausea ratio scale vs. 7-point nausea rating, rho = 0.858, $p = 0.000$; nausea ratio scale vs. postexposure SSQ Nausea, rho = 0.759, $p = .000$; 7-point nausea rating, rho = 0.898, $p = .000$). This result suggested that using ratio-scaled and ordinal-scaled methods to assess symptoms of VIMS can result in correlated findings.

In this experiment, the median OKN gains calculated from the EOG recordings were 0.93, 0.8, and 0.4 for the three conditions with pattern velocities of 30, 60, and 90 dps, respectively. The trend of reducing OKN gains with increasing pattern velocity was consistent with Koenig et al. (1978), who reported OKN gains of 0.91, 0.72, and 0.63 when viewers were watching stimuli rotating at 30, 60, and 90 dps, respectively.

DISCUSSION

The Isolated Effect of Vection on VIMS

Modern VIMS theories suggest that there are three factors that are potentially the primary causes of VIMS: (a) vection (sensory rearrangement theory; Reason, 1978), (b) OKN (extraocular afferent hypothesis; Ebenholtz, Cohen, & Linder, 1994), and (c) postural instability (postural instability theory; Riccio & Stoffregen, 1991). Among these three factors, vection was the only factor that was not purposely controlled or suppressed in Experiment 1. In fact, vection was purposely stimulated in Experiment 1. OKNs were completely suppressed by eye fixation as confirmed by the EOG recordings. Head movements were constrained by a chin rest. Unfortunately, head position was not measured.

A post hoc test indicated that a head sway movement that is 2° in amplitude can cause an observable and consistent pattern in EOG recordings. Examination of the EOG recordings indicated no evidence of such a pattern among the participants. Consequently, we are confident that the participants did not move their heads by more than 2° in the first experiment. We do acknowledge that body movement was not measured, however.

Results of Experiment 1 indicated that in the presence of vection and in the absence of OKN, VIMS severity increased as the pattern velocity increased from 0 dps to 2 dps, 14 dps, and 34 dps. However, the averaged times spent in saturated vection were not significantly affected by the changes of pattern velocity from 2 dps to 14 dps and 34 dps.

The observed decoupling between the increases in VIMS severity and the changes in time spent in saturated vection is consistent with Flanagan et al. (2002) but appeared to be contradictive to the findings in Kennedy, Hettinger, Harm, Ordy, and Dunlap (1996) and Hettinger et al. (1990). Further investigations revealed that different vection measurements were used in the latter two studies. Hettinger et al. separated participants into a vection group and a no-vection group and reported a higher VIMS severity in the vection group. Kennedy et al. measured vection velocity and reported that vection velocity monotonically increased along with the increase of pattern velocity. In Experiment 1, times spent in saturated vection were measured.

Since the absence of both vection and OKN produced near-zero levels of VIMS severity among the participants, the authors would suggest that while the presence vection alone can significantly increase VIMS severity, the level of sickness severity will also depend on the retinal slip velocity of the moving patterns.

Because body posture was not measured in this study, we cannot rule out possible interactions between postural instability and reported sickness. However, all things equal, the main treatments in the two experiments were still the moving stimuli.

The Isolated Effect of OKN on VIMS

In Experiment 2, vection was suppressed by a visual stimulus with central and peripheral visual fields rotating at the same speed but in opposite directions. Head movements were constrained by a chin rest. Because head sways were not measured in this study, we cannot rule out possible interactions between postural instability and reported sickness. However, all things equal, the main treatments in Experiment 2 were the speed of the moving stimuli and associated OKNs.

Results of Experiment 2 indicate that as the pattern velocities increased from 0 dps to 30, 60, and 90 dps, VIMS severity increased, peaked, and reduced. These effects of pattern velocity on VIMS severity reported in Experiment 2 are different from the monotonic increasing patterns reported in Experiment 1. The OKN SPV calculated from the EOG recordings in Experiment 2 had median values of 27.9, 47.8, and 35.5 dps for the conditions with pattern velocities of 30, 60, and 90 dps, respectively. Given that both the VIMS severity and OKN SPV peaked under the same condition, this result suggests that OKN SPV may influence levels of VIMS. Indeed, the correlations between OKN SPV and VIMS severity were significant (Spearman, $p < .05$). We argue that OKN can be a covariant of VIMS severity.

The Role of Vection and OKN in Generating Symptoms of VIMS

In this study, both the presence of retinal slip without OKN and the presence of retinal slip without vection were found to influence both ratio-scaled and ordinal-scaled ratings of nausea significantly. Interestingly, the profile of the changes in VIMS severity with increasing retinal slip velocity in the presence of both OKN and vection matches nicely with the profile of the sum of the changes in VIMS severity reported in Experiments 1 and 2 (Figure 6c). This suggests that both OKN and vection are related to the generation of VIMS.

It can be observed in Figures 6a and 6b that when images were moving at low retinal slip velocities (approximately 2 dps), higher nausea levels were generated in the presence of OKN than in the presence of vection. When images were moving at higher retinal slip velocities (approximately 34 dps), lower nausea levels were generated in the presence of OKN than in the presence of vection. Further studies to investigate the reasons are desirable.

Findings from the two experiments demonstrate that VIMS can occur in the presence of vection without OKN and in the presence of OKN without vection. This implies that neither OKN nor vection is a necessary condition for VIMS to occur. This finding is in disagreement with Hettinger and Ricco's (1990) claim that vection is necessary for VIMS.

Figure 6 indicates that as the retinal velocity increased from 0 dps to approximately 12.2 or 14 dps, symptoms of VIMS increased significantly regardless of the absence of OKN or vection. However, when the retinal velocity increased from 12.2 or 14 dps to approximately 34 or 48.6 dps, respectively, VIMS symptoms continued to increase in the absence of OKN, but they reduced in the absence of vection, although both changes were not statistically significant. Under conditions when the retinal velocities were approximately 34 or 48.6 dps, the presence of vection without OKN led to significantly higher VIMS severity measured on the ratio scale than did the presence of OKN without vection (Mann-Whitney test, $N = 14$,

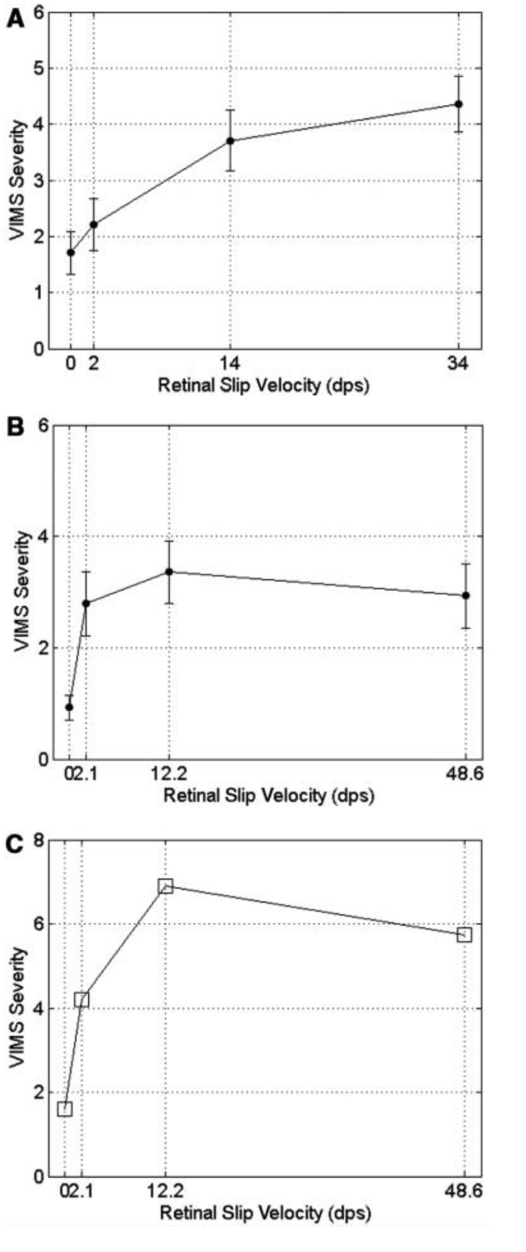

Figure 6. A diagram illustrating that visually induced motion sickness (VIMS) severity caused by image movements on the retina in the presence of both vection and OKN (as reported by Hu et al., 1989) (c) can be observed as a combination of similar effects reported in Experiment 1 without optokinetic nystagmus (OKN) (a) and Experiment 2 without vection (b). The *y*-axis of Figure 6c used VIMS severity data published in Hu et al. (1989), and the *x*-axis used retinal slip velocity calculated using OKN gain that was not published in Hu et al.

$W = 261$, $p = .0059$). This suggests that the presence of vection was associated with more severe VIMS than was the presence of OKN when participants watched stimuli that were moving at approximately 40 dps across the retina.

Nausea Ratio Scale and Ordinal Scale

In this study, a nausea ratio scale and a 7-point nausea rating scale were used to measure VIMS simultaneously. The significant correlation between VIMS severity measured by the nausea ratio scale and the 7-point nausea rating scale is an important finding. The data collected on the 7-point nausea rating scale gave a meaning to the ratio-scaled nausea number. We acknowledge that one possible reason for the correlation between the ordinal and ratio VIMS ratings may be the deliberate cognitive action of the participants.

Potential Applications of the Findings

Studying the isolated effects of OKN and vection on VIMS can help to identify effective ways to reduce VIMS. For example, if either the absence of OKN or the absence of vection can prevent the occurrence of VIMS, then solutions to reduce VIMS can focus on the reduction of the appropriate factors. Unfortunately, the findings of the two experiments indicate that neither OKN nor vection is a necessary condition for VIMS to occur.

In this study, OKN SPV, VIMS severity, and vection velocity (relative to pattern velocity) were measured using ratio scale methods. This enabled the development of mathematical models to simulate VIMS generation (e.g., Oman, 1982). Currently, the empirical ratio scale data reported in this article are being used to verify a computational model simulating VIMS reported from viewers of an optokinetic drum rotating along the earth-vertical axis (Ji et al., 2007). We hope that as computational models to predict VIMS become more and more accurate, they will lead to the development of a system that can predict the probability of VIMS in users playing newly developed computer games. This model could also be included in future standards of image safety (e.g., ISO, 2005).

CONCLUSION, LIMITATIONS, AND FUTURE WORK

This study successfully applied Brandt et al.'s (1973) method to suppress vection perception in 14 participants watching rotating scenes at 30, 60, and 90 dps.

OKN gains as functions of the rotating scene velocity reported by Koenig et al. (1978) are confirmed.

Image motion projected on the retina in the presence of vection and the absence of OKN can significantly increase the severity of VIMS. As the velocity of the images projected on the retina increased from 0 to 2, 14, and 34 dps, the severity of VIMS significantly increased.

Image motion projected on the retina in the presence of OKN and the absence of vection can also significantly increase the severity of VIMS. As the velocity of the images projected on the retina increased from 0, to 2.1, 12.2, and 48.6 dps, both the OKN SPV and the severity of VIMS significantly increased, peaked, and reduced.

Ratio-scaled nausea data have been found to be significantly correlated with ordinal-scaled nausea data. The collection of both ratio- and ordinal-scaled data enable the future development of mathematical models of VIMS generation.

Future studies to examine the effects of retinal slip on VIMS in the presence of both OKN and vection are desirable. We recommend the measure of absolute perceived vection velocities in future studies because it comprises the presence of vection and the effects of retinal slips. Kennedy et al. (1996) reported that human participants can reliably report perceived vection velocity.

We acknowledge that only female Chinese participants were used in this study and that they have been reported to be hypersusceptible to motion sickness (Stern et al., 1993). We would also like to point out that 2 out of 16 participants dropped out from the study because they got too sick to participate. Their data have been excluded in the analyses because they did not participate in all the conditions.

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REFERENCES

Brandt, T., Dichgans, J., & Koenig, E. (1973). Differential effects of central versus peripheral vision on egocentric and exocentric motion perception. *Experimental Brain Research*, *16*, 476–491.

Buttner, U., & Buttner-Ennever, J. A. (1988). Present concepts of oculomotor organization. In J. A. Buttner-Ennever (Ed.), *Neuroanatomy of the oculomotor system* (pp. 1–42). Amsterdam: Elsevier BV.

Ebenholtz, S. M., Cohen, M. M., & Linder, B. J. (1994). The possible role of nystagmus in motion sickness: A hypothesis. *Aviation, Space and Environmental Medicine*, *65*, 1032–1035.

Faugloire, E., Bonnet, C. T., Riley, M. A., Bardy, B. G., & Stoffregen, T. A. (2007). Motion sickness, body movement, and claustrophobia during passive restraint. *Experimental Brain Research*, *177*, 520–532.

Flanagan, M. B., May, J. G., & Dobie, T. G. (2002). Optokinetic nystagmus, vection, and motion sickness. *Aviation, Space and Environmental Medicine*, *73*, 1067–1073.

Flanagan M. B., May, J. G., & Dobie, T. G. (2004). The role of vection, eye movements and postural instability in the etiology of motion sickness. *Journal of Vestibular Research*, *14*, 335–46.

Golding, J. (2006). Predicting individual differences in motion sickness susceptibility by questionnaire. *Personality and Individual Differences*, *41*, 237–248.

Golding, J., & Kerguelen, M. (1992). A comparison of the nauseogenic potential of low-frequency vertical versus horizontal linear oscillation. *Aviation, Space, and Environmental Medicine*, *63*, 491–497.

Griffin, M. J. (1992). *Handbook of human vibration.* London: Academic Press.

Harding, G. F. A., & Harding, P. F. (2007). Photosensitive epilepsy and image safety. In So, R. H. Y., Cheung, R., Chow, E., Ji, J. T. T., and Lam, A. (Eds.), *Proceedings of the First International Symposium on Visually Induced Motion Sickness, Fatigue, and Photosensitive Epileptic Seizures (VIMS2007)* (pp. 43–48). Clear Water Bay, Kowloon, Hong Kong: Secretariat of VIMS2007, Hong Kong University of Science and Technology, Department of Industrial Engineering and Logistics Management.

Hettinger, L. J., Berbaum, K. S., Kennedy, R. S., Dunlap, W. P., & Nolan, M. D. (1990). Vection and simulator sickness. *Military Psychology*, *2*, 171–181.

Hettinger, L. J., & Ricco, G. E. (1992). Visually induced motion sickness in virtual environments. *Presence*, *1*, 306–310.

Hu, S., Davis, M. S., Klose, A. H., Zabinsky, E. M., Meux, S. P., & Jacobsen, H. A. (1997). Effects of spatial frequency of a vertically striped rotating drum on vection-induced motion sickness. *Aviation, Space and Environmental Medicine*, *68*, 306–311.

Hu, S., & Stern, R. M. (1998). Optokinetic nystagmus correlates with severity of vection-induced motion sickness and gastric tachyarrhythmia. *Aviation, Space and Environmental Medicine*, *69*, 1162–1165.

Hu, S., Stern, R. M., Vasey, M. W., & Koch, K. L. (1989). Motion sickness and gastric myoelectric activity as a function of speed of rotation of a circular vection drum. *Aerospace Medical Association*, *60*, 411–414.

ISO. (2005). *ISO International Workshop Agreement-IWA3: Image safety. Reducing the incidence of undesirable biomedical effects caused by visual image sequences* (IWA 3:2005[E]).

Available from ISO copyright office, Case postale 56, CH-1211 Geneva 20, Switzerland.

Ji, J. T. T., Chow, E., Lor, F., So, R. H. Y., Cheung, R., Stanney, K., & Howarth, P. A. (2007). A biologically inspired computational model relating vection and visually induced motion sickness: Individual differences and sensitivity analysis. In So, R. H. Y., Cheung, R., Chow, E., Ji, J. T. T., and Lam, A. (Eds.), *Proceedings of the First International Symposium on Visually Induced Motion Sickness, Fatigue, and Photosensitive Epileptic Seizures (VIMS2007)* (pp. 33–40). Clear Water Bay, Kowloon, Hong Kong: Secretariat of VIMS2007, Hong Kong University of Science and Technology, Department of Industrial Engineering and Logistics Management.

Kennedy R. S., Hettinger, L. J., Harm, D. L., Ordy, J. M., & Dunlap, W. P. (1996). Psychophysical scaling of circular vection (CV) produced by optokinetic (OKN) motion: Individual differences and effects of practice. *Journal of Vestibular Research*, *6*, 331–341.

Kennedy, R. S., Lane, N. E., Berbaum, K. S., & Lilienthal, M. J. (1993). Simulator sickness questionnaire: An enhanced method for quantifying simulator sickness. *International Journal of Aviation Psychology*, *3*, 203–220.

Koenig, E. J., Allumm, J. H. K., & Dichgans, J. (1978). Visual-vestibular interaction upon nystagmus slow phase velocity in man. *Acta Oto-Laryngologica*, *85*, 397–410.

Leibowitz, H. W., & Post, R. B. (1982). The two modes of processing concept and some implications. In J. Beck (Ed.), *Organization and representation in perception* (pp. 343–363). Hillsdale, NJ: Lawrence Erlbaum.

Oman, C. M. (1982). A heuristic mathematical model for the dynamic of sensory conflict and motion sickness. *Acta Oto-Laryngologica Supplementum*, *392*, 5–44.

Reason, J. T. (1978). Motion sickness adaptation: A neural mismatch model. *Journal of the Royal Society of Medicine*, *71*, 819–829.

Regan, C. E. (1995). An investigation into nausea and other side-effects of head-coupled immersive virtual reality. *Virtual Reality*, *1*(1), 17–31.

Riccio, G. E., & Stoffregen, T. A. (1991). An ecological theory of motion sickness and postural instability. *Ecological Psychology*, *3*, 195–240.

Smart, L. J., Stoffregen, T. A., & Bardy, B. G.(2002). Visually induced motion sickness predicted by postural instability. *Human Factors*, *44*, 451–465.

So, R. H. Y., Finney, C. M., & Goonetilleke, R. S. (1999). Motion sickness susceptibility and occurrence in Hong Kong Chinese. In M. A. Hanson, E. J. Lovesey, & S. A. Robertson (Eds.), *Contemporary ergonomics 1999* (pp. 88–92). London: Taylor and Francis.

Stanney, K. M., Kingdon, K. S., & Kennedy, R. S. (2002). Dropouts and aftereffects: Examining general accessibility to virtual environment technology. In *Proceedings of the Human Factors and Ergonomics Society 46th Annual Meeting* (pp. 2114–2118). Santa Monica, CA: Human Factors and Ergonomics Society.

Stanney, K. M., Salvendy, G., Desisinger, J., Dizio, P., Ellis, S., Ellison, J., Fogleman, G., Gallimore, J., et al. (1998). Aftereffects and sense of presence in virtual environments: Formulation of a research and development agenda. *International Journal of Human-Computer Interaction*, *10*, 135–187.

Stern, R. M., Hu, S., Anderson, R. B., Leibowitz, H. W., & Koch, K. L. (1990). The effects of fixation and restricted visual field on vection-induced motion sickness. *Aviation, Space and Environmental Medicine*, *61*, 712–715.

Stern, R. M., Hu, S., LeBlanc, R., & Koch, K. L. (1993). Chinese hyper-susceptibility to vection-induced motion sickness. *Aviation, Space and Environmental Medicine*, *64*, 827–830.

Stevens, S. S. (1957). On the psychophysical law. *Psychological Review*, *64*, 153–181.

Stevens, S. S. (1971). Issues in psychophysical measurement. *Psychological Review, 78*, 426-450.

Webb, N. A., & Griffin, M. J. (2002). Optokinetic stimuli: Motion sickness, visual acuity, and eye movements. *Aviation, Space and Environmental Medicine*, *73*, 351–358.

Webb, N. A., & Griffin, M. J. (2003). Eye movements, vection and motion sickness with foveal and peripheral vision. *Aviation, Space and Environmental Medicine*, *74*, 622–625.

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