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Correlations between individual susceptibility to visually induced motion sickness and decaying time constant of after-nystagmus

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ABSTRACT

This study examines the correlations between optokinetic after-nystagmus (OKAN) parameters and individual susceptibility to visually induced motion sickness (VIMS). Twenty-seven participants were exposed to vertical black-and-white stripes drifting along the yaw axis at 60° per second for 30 min to collect individual VIMS data (Phase 1). Two weeks after the exposure, OKANs were measured (Phase 2). 19 out of 27 participants (i.e., 70%) exhibited consistent OKAN patterns. Significant correlations between the time constants of OKAN and levels of VIMS experienced by the same viewers were found. Four months later, these 27 participants were invited back for a second OKAN measurement (Phase 3). Twenty-one participants came back. Their two OKAN measurements were significantly correlated ($r = 0.69$, $p = 0.001$). Rated levels of VIMS in phase 1 significantly correlated with the time constant of OKAN in both Phase 2 ($r = 0.51$, $p = 0.044$) and Phase 3 ($r = 0.74$, $p = 0.006$). The implications of the correlation results are discussed.

1. Introduction

Viewing moving visual scene can cause visually induced motion sickness (VIMS). The prevalence rate of VIMS is about 30% (So and Lo, 2001; Kiryu and So, 2008; So and Ujike, 2010) which is similar to that of motion sickness provoked by physical motion (Griffin, 1990; So et al., 1999). However, the prevalence of VIMS can also vary from 1% to 70% depending on the apparatus and stimuli (Kennedy et al., 1996a,b; 2010). The occurrence of VIMS among computer gamers has been frequently reported (Stoffregen et al., 2008; Qualls, 2014; Davis, 2016). In 2005, VIMS scientists and representatives from the gaming industry met in Tokyo and published an ISO International Workshop Agreement 3 (IWA 3, 2005) declaring the need for more research on the assessment and prevention on VIMS (So and Ujike, 2010). In particular, the IWA3 called for the development of a simple objective test to predict individual susceptibility to VIMS without making the person sick. A review of literature indicates that the most common way to measure levels of VIMS has been through subjective questionnaires (e.g., the simulator sickness questionnaire (SSQ): Kennedy et al., 1993; nausea ratings: Golding and Kerguelen, 1992; motion sickness susceptibility questionnaire (MSSQ): Golding, 1998; studies using questionnaires: Keshavarz and Hecht, 2011; Lo and So, 2001; So and Lo, 1999; So et al., 2002). Although researchers have successfully used electrogastrography (EGG) activities to quantify levels of VIMS (Hu et al., 1989), it required making the participants sick before such objective measurements could be obtained. In summary, an objective measure that can predict individual susceptibility to VIMS could not be found. This study examines the possible correlations between after-nystagmus (OKAN) parameter and the individual susceptibility to VIMS.

Watching visual patterns drifting horizontally can trigger both optokinetic nystagmus (OKN) and VIMS (Ebenholtz et al., 1994; Ji et al., 2009; Lo and So, 2001). Suppression of OKN using an eye fixation point has been shown to significantly reduce rated levels of VIMS (Ji et al., 2009; Webb and Griffin, 2002). This suggests that OKNs may play a key role in the generation of VIMS. Indeed, hypotheses have been proposed to suggest that abnormal ocular-motor afferent activities are part of the causes of VIMS (e.g., Ebenholtz et al., 1994; Gupta, 2005). A typical OKN cycle consists of

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a slow drift of the eyes, at the slow-phase velocity, in the direction of the stimuli followed by a rapid saccadic return. When viewers are experiencing OKN, a sudden immersion into total darkness will stop OKN abruptly in some viewers but allow OKN to decay slowly in most viewers. The latter is called optokinetic after-nystagmus (OKAN). It is a continuation of OKN but with a slowly decaying slow-phase velocity (Aschan and Bergstedt, 1955; Cohen et al., 1973; Ventre-Dominey and Luyat, 2009). OKAN has been shown to have a close association with the vestibular nucleus (Dellepiane et al., 2006; Tijssen et al., 1989). Patients who have undergone bilateral labyrinthectomy will not experience OKAN (Cohen et al., 1973) and OKAN can be suppressed after surgical removal of part of the vestibular organ (Waespe et al., 1983). This suggests that the vestibular system plays an important role in the genesis of OKAN. Since vestibular nuclei are known to be associated with the generation of VIMS (bilateral labyrinthine-defective subjects were immune to VIMS: Cheung et al., 1991), the association between OKAN and vestibular nuclei suggests a hypothetical relationship between OKAN and VIMS. In this study, we hypothesize that the individual susceptibility of VIMS will correlate with the decaying time constant of slow-phase velocity of OKAN. Indeed, the OKAN time constant has been found lower in patients with bilateral vestibular disorders than in normal people (Dellepiane et al., 2006) and Cheung et al. (1991) has shown that patients with bilateral vestibular disorders do not report symptoms of VIMS.

Our OKAN hypothesis is also consistent with the reflex theory of motion sickness proposed in Griffin (1990) because OKAN is a reflex associated with velocity storage mechanism (VSM) theory (Muratore and Zee, 1979; Bertolini et al., 2011). According the VSM theory, there are two pathways related to the generation of OKN and OKAN: a direct (fast) and an indirect (slow) pathways.

The VSM theory predicts that during optokinetic stimulation, the indirect pathway would “store” information related to the slow-phase velocity in OKN within their neural circuitry and in total darkness, these “stored” information would be discharged to maintain the eye response, resulting in OKAN. In other words, the indirect pathway mediates the gradual decay of the slow-phase velocity of OKAN (Bertolini et al., 2011).

A review of literature indicates that the current study is the first attempt to correlate VIMS with the time constant of OKAN experimentally. The closest studies are Takahashi et al. (1997) and Dai et al. (2003). They reported correlations between OKAN's decaying time constants and severity of motion sickness provoked by physical motion. Using patients with labyrinthine lesions, their studies found that susceptibility to physical motion sickness is related to the function of indirect VSM pathway which is known to affect OKAN decaying time constants. In 2006, Dai and his colleagues successfully shortened the decay time constant of OKAN and suppressed symptoms of physically provoked motion sickness with muscle relaxing medicine (Dai et al., 2006). Furthermore, our hypothesis is also consistent with past studies on vestibular ocular reflex (VOR). Significant correlations between motion sickness susceptibility and the time constants of angular VOR have been reported and it has been suggested that motion sickness is related to the indirect pathway of VSM known to be related to OKAN (Dai et al., 2007).

2. Method

2.1. Apparatus and stimulus

Fig. 1 illustrates the virtual rotating drum used to provoke symptoms of VIMS. Visual patterns with field-of-view of 200° (horizontally) by 65° (vertically) were projected on a curved screen with a radius of 115 cm. Alternate black-and-white vertical stripes drifting clockwise at 60° per second served as the stimulus. This wide field-of-view projection system has been used in previous studies related to VIMS (Chen et al., 2016; Guo and So, 2012; Ji et al., 2009). The black and white stripes formed angles of 5.7° and 9.3° with the horizontal, respectively (c.f., Hu et al., 1989). Participants stood at the center of the virtual drum. Foot rests were used to align the participants' eye levels with the screen and a chin rest was used to restrain head movements. Head positions and orientations were monitored using a Polhemus FASTRAK system at 100 samples per second. Electrooculography (EOG) recordings were made using a BIAPAC® EOG 100C system at 200 samples per second. The EOG recordings were used to estimate the relative OKN and OKAN movements. The experimental setting was similar to that used in Ji et al. (2009) during which significant increases in rated levels of VIMS were reported.

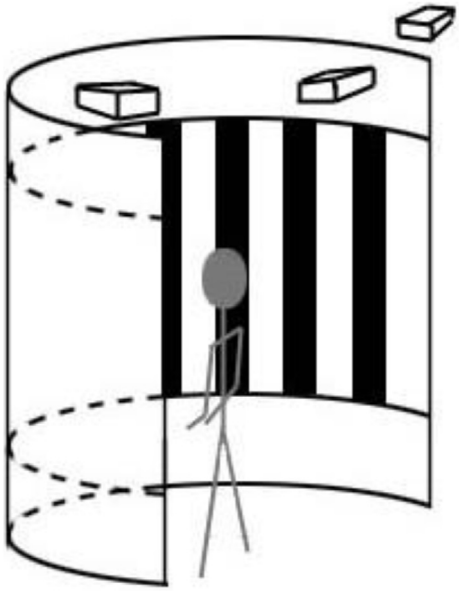

Fig. 1. An illustration of the wide field-of-view virtual rotating drum used to provoke symptoms of VIMS. Visual patterns with field of view of 200° (horizontally) by 65° (vertically) were projected on a curved screen with a radius of 115 cm via three projectors. Proprietary software was used to combine the three images seamlessly.

2.2. Design of experiment

Twenty-seven postgraduate students (13 males) aged 22 to 30 were recruited from the Hong Kong University of Science and Technology to participate in the study. All of them were exposed to the drifting patterns for 30 min in Phase 1. Symptoms of motion sickness were measured before, during, and after the exposure using a pre-exposure simulator sickness questionnaire (SSQ, Kennedy et al., 1993), a seven-point nausea rating (Golding and Kerguelen, 1992), and a post-exposure SSQ. Nausea ratings (1: no symptoms; 4: mild nausea; 7: moderate nausea, want to stop) were taken at two-minute intervals. When a ‘7’ was reported, the presentation of the drifting pattern would be stopped and a ‘7’ would be assigned to the remaining time. The measurement of VIMS was conducted first in order to minimize the effects of habituation on VIMS. Since OKAN is part of an involuntary reflex, it is relatively less affected by habituation.

In Phase 2, which took place two weeks after the exposure in Phase 1, participants were invited back for measurements of OKAN with six repeated trials. Each trial consisted of three stages. Participants were instructed to stare at a stationary eye-fixation dot

projected on a stationary stripe pattern for 15 s (first stage). After the 15-second period, the dot disappeared and the stripes started to drift at 60° per second from left to right for two minutes and participants were instructed to look straight ahead the whole time (second stage, Fig. 1). Immediately after this two-minute period, i.e. at the start of the 120th second, the stimulus (i.e., the virtual drum) and all the light in the laboratory were switched off for one minute and participants were instructed to keep looking straight ahead the whole time (third stage). The six repeated trials were performed over two consecutive days to minimize eye fatigue.

Two commonly used parameters of OKAN were analyzed in Phase 2 (Dai et al., 2003): (i) the initial velocity of OKAN and (ii) the time constant of OKAN. The moment the light was turned off was recorded through a light sensing circuit and the initial velocity of OKAN was calculated from the EOG data collected during the first second immediately after the light was switched off. Most participants experienced more than one OKAN cycle during the first second in total darkness and the initial velocity of the first OKAN cycle was extracted and calculated. The decay of the slow-phase velocity was modeled with an exponential function (Cohen et al., 1977; Muratore and Zee, 1979; Maioli, 1988; Tijssen et al., 1989). Following previously published methodology, the time constant was determined from the reciprocal of the slope (K) of the regression line fitted to a plot of the natural log (ln) of the slow-phase velocity versus the time of exposure (t) to total darkness (Tijssen et al., 1989). In other words, the time constant (TC) is determined as:

$$TC = 1/K$$

where

$$SPV\ (\text{slow-phase velocity}) = V_0\ (\text{initial velocity}) \times e^{-Kt}$$
$$\text{or } \ln(SPV) = \ln(V_0) - Kt$$

In Phase 3, which took place four months after Phase 2 of the study, 21 participants agreed to come back for another six repeated trials to measure their OKAN responses. Phases 1, 2 and 3 used the same laboratory and apparatus.

3. Results

Partial initial results collected in Phase 2 were presented at an International conference (Guo et al., 2011).

3.1. Head movements

The lateral head movements of the 27 participants had average standard deviations ranging from 0.02 cm to 0.32 cm. These results indicate that the chin rests succeeded in keeping head movements to a minimum and no motion artefact was observed in the EOG data.

3.2. The occurrence of OKAN and its repeatability

As the light was turned off at the 120th second, any eye movements with OKN patterns measured after that second would be OKAN. In order to separate the occurrence of OKAN from the residual of a decaying OKN, rules were adopted from Tijssen et al. (1989). OKAN had occurred only if one of the following conditions was satisfied: (i) at least two consecutive cycles of the eye movements in complete darkness were of the OKN type, each displaying an asymmetric saw-tooth-shaped EOG signal whose amplitude is reduced compared to that of the previous two cycles of OKN eye movements; or (ii) at least four cycles of the eye movements in complete darkness were of the OKN type with at most one cycle of non-OKN-type eye movements in between. The prevalence rate of OKAN among the 27 participants in Phase 2 was 70%. In other words, 19 out of 27 participants exhibited patterns of OKAN. Among these 19 participants, seven reported 100% (i.e., six out of six trials during Phase 2) occurrence of OKAN, four reported 83% (i.e., five out of six trials), and eight reported 17%–67% occurrence (Table 1). During Phase 3, the prevalence rate of OKAN was 81% among the 21 participants who agreed to a second set of measurements. For these 21 participants, the prevalence rate was 71% during Phase 2 (i.e., 17 out of 21 participants). Among the 17 participants who reported experiencing OKAN, five reported 100% occurrence, four reported 83%, and eight reported 17%–67% occurrence (Table 1). An inspection of Table 1 reveals that the prevalence rate increased because two of the participants reported zero occurrence of OKAN in Phase 2 but 17% occurrence in Phase 3 (i.e., one out of six trials) and one participant reported zero occurrence of OKAN in Phase 2 but 83% occurrence (i.e., five out of six trials) in Phase 3. Examples of EOG patterns measured from a participant exhibiting 100% OKAN occurrence in Phase 2 and 0% OKAN occurrence in Phase 3 are shown in Fig. 2 and Fig. 3, respectively.

The occurrence of OKAN among the 21 participants who took part in both phases 2 and 3 of the study is shown in Table 1 for comparison. The number of repeated trials (maximum is 6) that exhibited OKAN in Phase 2 and that in Phase 3 per each participant correlated significantly with each other (correlation coefficient = 0.69, $p = 0.001$, Pearson). In other words, those who exhibited more OKAN patterns in Phase 2 continued to do so in Phase 3 and so were those who exhibited less. Results of the Wilcoxon signed-rank test showed no significant difference between the percent occurrence of OKAN during phases 2 and 3 ($Z = -0.78$, $p > 0.1$). This suggests that the measures of OKAN occurrence were

Table 1
Prevalence rate of OKAN during Phase 2 and Phase 3: "—" means the individual did not participate in Phase 3.

Subject no.	No. of trials exhibiting OKAN out of six repeated trials and prevalence rate				Change from Phase 2 to Phase 3
	Phase 2		Phase 3		
1	0	0.00%	1	16.67%	+1/6
2	4	66.67%	4	66.67%	0/6
3	6	100.00%	4	66.67%	−2/6
4	1	16.67%	0	0.00%	−1/6
5	3	50.00%	–	–	–
6	0	0.00%	0	0.00%	0/6
7	5	83.33%	5	83.33%	0/6
8	0	0.00%	1	16.67%	+1/6
9	4	66.67%	–	–	–
10	0	0.00%	0	0.00%	0/6
11	5	83.33%	–	–	–
12	6	100.00%	3	50.00%	−3/6
13	6	100.00%	6	100.00%	0/6
14	6	100.00%	4	66.67%	−2/6
15	5	83.33%	6	100.00%	+1/6
16	0	0.00%	–	–	–
17	0	0.00%	0	0.00%	0/6
18	2	33.33%	4	66.67%	+2/6
19	0	0.00%	5	83.33%	+5/6
20	5	83.33%	6	100.00%	+1/6
21	6	100.00%	4	66.67%	−2/6
22	3	50.00%	–	–	–
23	2	33.33%	5	83.33%	+3/6
24	6	100.00%	6	100.00%	0/6
25	0	0.00%	–	–	–
26	6	100.00%	6	100.00%	0/6
27	1	16.67%	5	83.33%	+4/6

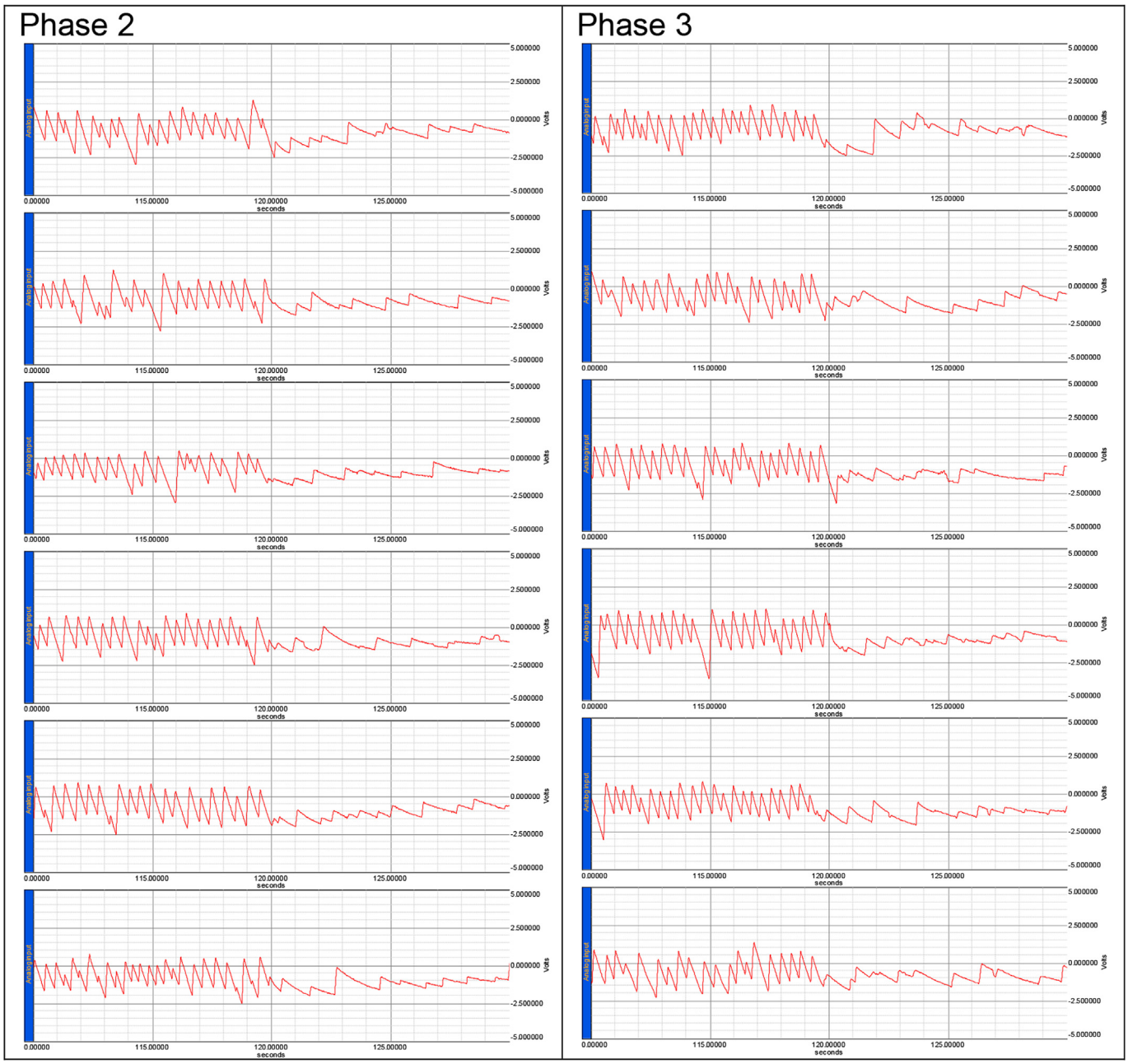

Fig. 2. Examples of OKAN occurring in all six repeated trials for one participant. The six EOG plots on the left were collected from Phase 2 and the six plots on the right were from Phase 3. The x-axis measures the time in seconds and the y-axis measures the EOG signals in volts. Light was turned off at the 120th second. Data before that time represent OKN and data after that time represent OKAN.

stable. Inspections of Table 1 indicated that among the 21 participants who took part in both Phases 2 and 3, 90% of those who reported high occurrence of OKAN (5 or more out of the 6 trials) continued to exhibit high occurrence of OKAN after a gap of four months. Since the later analyses of OKAN parameters focused on those who reported OKAN, the 90% consistence provide support on the repeatability of the later findings.

3.3. Relationship between the occurrence of OKAN and susceptibility to VIMS

The average seven-point nausea ratings measured in Phase 1 from the 8 participants with 0% occurrence of OKAN ranged from 1.5 to 6.06 with an average of 3.92. The seven-point nausea ratings for the 19 participants who exhibited OKAN ranged from 1 to 6.06 with an average of 3.98. The average seven-point nausea rating for all 27 participants was 3.96 ('4' is mild nausea) with an average SSQ total score, nausea sub-score, oculomotor sub-score and disorientation sub-score of 66, 65, 48 and 62, respectively. There was no observable difference in susceptibility to VIMS between those who exhibited OKAN and those who did not (Table 2).

3.4. Initial slow-phase velocity of OKAN

The initial slow-phase velocity of OKAN was calculated using the statistical software Minitab™. In Phase 2, the initial velocities of the

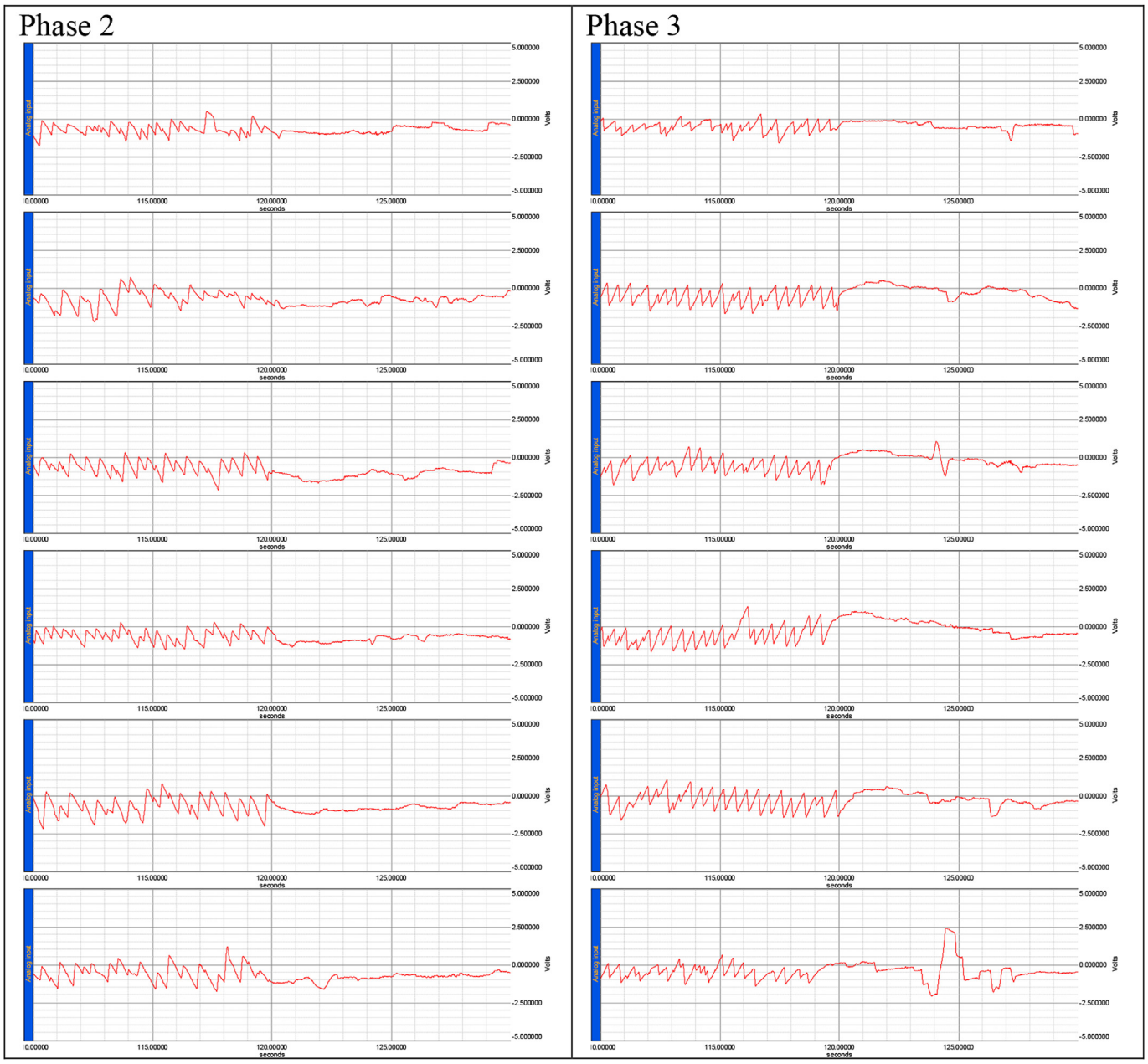

Fig. 3. Example of OKAN not occurring in any of the six repeated trials for one participant. The six EOG plots on the left were collected from Phase 2 and the six plots on the right were from Phase 3. The x-axis measures the time in seconds and the y-axis measures the EOG signals in volts. Light was turned off at the 120th second. Data before that time represent OKN and data after that time represent OKAN.

Table 2
Nausea ratings and SSQ scores groups of participants with or without exhibiting OKAN.

	Mean Nausea ratings (SD)	Mean SSQ scores (SD)			
		Total score	Nausea sub-scores	Disorientation sub-scores	Oculomotor sub-scores
exhibited OKAN	3.98 (1.5)	66.5 (28)	69.3 (30.14)	48.7 (22.5)	59.3 (35.9)
without OKAN	3.92 (1.6)	63.6 (57.2)	54.9 (45.3)	47.3 (41.7)	59.6 (78.4)

19 participants ranged from 5.9° per second (dps) to 18.1 dps with an average of 9.6 dps ± 3.8 dps. The average ratio of the initial velocity of OKAN to the average slow-phase velocity of OKN during the two-minute exposure was 79.0% ± 7.0%. This is referred to as the discharge rate, which suggests that once the light was turned off, the amplitude of the first OKAN pattern reduced by 80% on average. In Phase 3, the initial velocities of the 17 participants ranged from 5.5 dps to 19.3 dps with an average of 11.3 dps ± 3.7 dps. The average ratio of the initial velocity of OKAN to the average slow-phase velocity of OKN during the two-minute exposure was

75.0% ± 7.1%. The initial velocity in Phase 2 was correlated significantly with that in Phase 3 (Pearson correlation: $r = 0.833$, $p < 0.001$), and no significant difference was found between the average initial velocities in the two phases ($p > 0.05$). Again, this suggests that the OKAN patterns measured in phases 2 and 3 from the same participant were similar.

3.5. Time constant

The decay of OKAN velocity was modeled by an exponential function (Tijssen et al., 1989). The weighted least squares linear regression model was used. Data with R-squared values below 0.6 were discarded in the calculation. In Phase 2, the time constant was calculated for 16 participants (out of 19 participants). In Phase 3, among the 17 participants who exhibited OKAN, the time constant was calculated for 12 of them.

During Phase 2, the time constant ranged from 6.4s to 30.6s with an average of 17.6s ± 6.7s for the 16 participants; in Phase 3, the time constant ranged from 1.7s to 17.7s with an average of 10.6s ± 5.6s for the 12 participants. The Pearson correlation test showed that there was significant correlation in both Phase 2 ($r = 0.51$, $p = 0.044$) and Phase 3 ($r = 0.74$, $p = 0.006$) between motion sickness susceptibility and time constant at the 0.05 significance level (Figs. 4 and 5). In this study, we adopted the criteria of calculating the decaying time constants of OKAN from past studies (Tijssen et al., 1989) and had to exclude 16% and 30% of those who reported inconsistent OKAN in Phases 2 and 3, respectively. We acknowledge that this may be a potential drawback for using OKAN decaying time constant as a predictor variable for VIMS. However, the repeated significant correlation results between OKAN decaying time constant and VIMS in both Phases 2 and 3 indicate that the relationship is not by chances. Future efforts to develop better ways to trigger OKAN and more reliable measures may be useful.

We also used the intercepts of the fitted regression lines as an alternative method to determine the initial slow-phase velocity of OKAN. The initial velocities calculated using this method

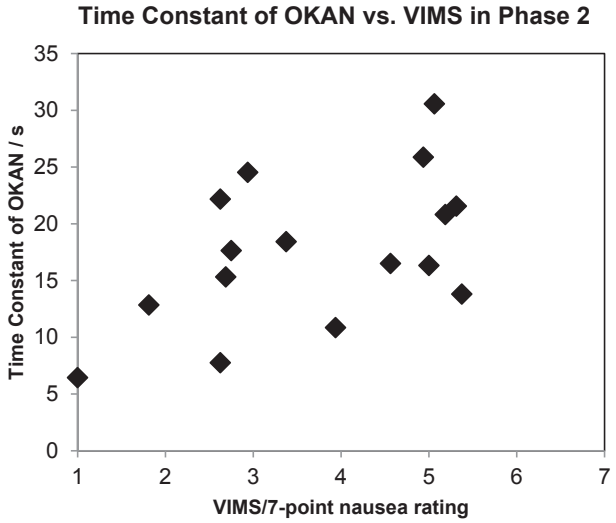

Fig. 4. Scatter plot of decaying time constants of OKAN versus the mean seven-point nausea ratings. The time constants are correlated significantly with the nausea ratings (r = 0.51, p = 0.044). The time constants were estimated from data collected in Phase 2 from 16 participants who exhibited enough OKAN cycles for the estimation to be made. Each filled square represents data from one subject. The x-axis measures the level of VIMS as measured by the average seven-point nausea rating during the 30-minute exposure in Phase 1 and the y-axis measures the mean of time constants from six repeated trials collected in Phase 2.

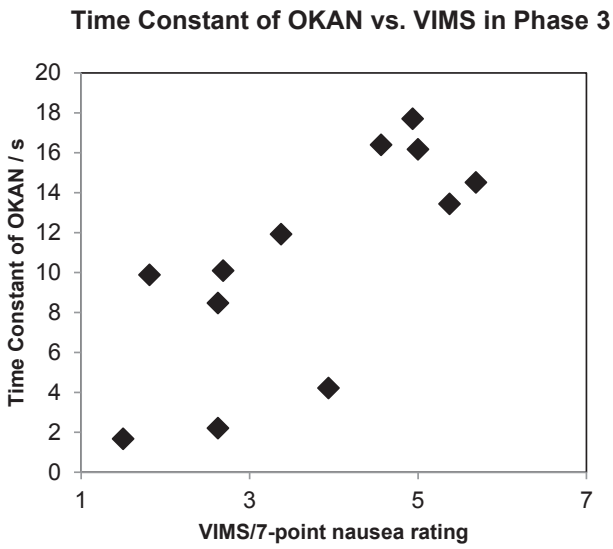

Fig. 5. Scatter plot of decaying time constants of OKAN versus the mean seven-point nausea ratings. The time constants are correlated significantly with the nausea ratings (r = 0.74, p = 0.006). The time constants were estimated from data collected in Phase 3 from 12 participants who exhibited enough OKAN cycles for the estimation to be made. Each filled square represents data from one participant. The x-axis measures the level of VIMS as measured by the average seven-point nausea rating during the 30-minute exposure in Phase 1 and the y-axis measures the mean of time constants from six repeated trials collected in Phase 3.

significantly correlated with those calculated using data on just the first OKAN cycle collected at the 121st second ($r = 0.82$, $p < 0.001$). This supports the validity of the fitted regression lines for modeling the slow-phase velocity of OKAN.

4. Discussion

The significant correlations between the time constants of OKAN and the rated levels of nausea are consistent with past studies concerning VOR and susceptibility to motion sickness (MS) due to physical motion. It was indicated that susceptibility to physically provoked motion sickness is significantly correlated with the time constants of angular VOR (Dai et al., 2007). As Dai et al.'s study only concerned physically provoked motion sickness, the current study is the first to link OKAN time constants to levels of VIMS. Nonetheless, due to the convergence of visual and vestibular neural signals, neural mechanisms for VIMS and motion sickness triggered by physical motion may have much in common. According to the theory of velocity storage mechanism (VSM: Bertolini et al., 2011), both OKAN addressed in this study, and angular VOR(Dai et al., 2007), are known to be controlled by VSM. In particular, for OKAN, the indirect (slow) pathway of VSM would "store" information during OKN and discharge them in total darkness to facilitate OKAN. Results of this study may indicate that participants with higher susceptibility to VIMS exhibited larger decaying time constants and slower decaying rates. Based on VSM theory, larger time constants (i.e., taking longer time for the velocities of OKAN to decay to 0.37 (1/e) of their initial values) suggest that "more" information was being stored during the OKN phase. In other words, the "more" the stored information in the indirect pathway of VSM, the higher the susceptibility to VIMS. The presence of OKN has been shown to increase levels of VIMS significantly (Webb and Griffin, 2002), one could infer that the "stored" information within VSM may related to the symptoms of VIMS. The current results suggest that more works should be conducted to uncover the function of the VSM which may hold the key to the

etiology of VIMS and its prevention. Also, the parallel correlations between the time constants of VOR and MS severity along with the time constants of OKAN and VIMS severity suggest that future studies on the etiology of VIMS can benefit from the more established research on MS.

Indeed, Dai and his colleagues successfully reduced the severity of MS symptoms using a drug called Baclofen (Dai et al., 2006; Cohen et al., 2008). This drug was shown to reduce OKN velocity and decaying time constant of OKAN in monkeys (Cohen et al., 1988) and we hope that the current findings can spur on the possible examination of using Baclofen to reduce VIMS as well as the search for an objective predictive measurement within the VSM parameters for VIMS susceptibility.

5. Conclusions, implications and limitations of the findings

This study examined the correlation of OKAN parameters with rated levels of VIMS in healthy human participants. Of the 27 participants, 19 exhibited patterns of OKAN. Twenty-one participants repeated the OKAN measurement study after four months. The occurrence of OKAN in Phases 2 and 3 were significantly correlated ($r = 0.69$, $p = 0.001$) with 90% of those who reported high occurrence of OKAN continued to report high occurrence after a period of four months. There were individuals who reported less occurrence of OKAN in Phase 2 exhibited more OKAN in Phase 3, further studies to explore a better method to measure OKAN are desirable.

The initial slow-phase velocity and time constant of OKAN were characterized, leading to two major findings. First, the occurrence of OKAN are correlated even with a gap of four months ($p < 0.001$, correlations). Second, visually induced motion sickness (VIMS) susceptibility, as indicated by the rated levels of nausea, correlated significantly with the time constants of decay of the slow-phase velocity of OKAN among those who exhibit OKAN. We acknowledge that the prevalence rate of OKAN is about 70% (19 out of 27). Nonetheless, the reported significant correlation suggests a possible relationship between ocular motor system and VIMS. Since the decay process of OKAN is related to the indirect velocity storage pathway, the significant correlation may suggest that susceptibility to VIMS could also be related to the indirect velocity storage pathway (Dellepiane et al., 2006). This is consistent with the proposed role of vestibular velocity storage in handling conflicting sensory cues (Cohen et al., 2003).

This is the first attempt to explore the possible correlation relationship between the decaying time constant of OKAN and the measured susceptibility of VIMS. Given the central role of OKN in the etiology of visually induced motion sickness (VIMS), further studies to examine how and why the decaying time of OKAN are related to the susceptibility of VIMS may increase our understanding of VIMS and lead to the development of objective predictors for VIMS.

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