

Could OKAN be an objective indicator of the susceptibility to visually induced motion sickness?

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ABSTRACT

International Workshop Agreement 3 organized by the International Standard Organization calls for more research to determine simple objective ways to assess susceptibility to visually induce motion sickness (VIMS) without making viewers sick (So and Ujike, 2010). This study examines the use of measurable optokinetic afternystagmus (OKAN) parameters to predict susceptibility to VIMS. Eighteen participants were recruited. They were exposed to a sickness provoking virtual rotating drum (210 degrees field-of-view) with striped patterns rotating at 60 degrees per second for 30 minutes (Phase 1). Sickness data were collected before, during, and after the exposure. These participants were invited back for OKAN measurements at least two weeks after Phase 1 was completed to minimize any adaption effect (Phase 2). Out of the 18 participants, 10 participants (i.e., 55%) exhibited consistent patterns of OKAN. Correlations between the time constants of OKAN and levels of VIMS experienced by the same viewers were found. The possibility of using OKAN as an objective indicator of the susceptibility to visually induced motion sickness is discussed.

KEYWORDS: OKAN, Visually induced motion sickness, Velocity storage mechanism, Motion sickness.

INDEX TERMS: simulation and behavior; psychology; immersion; cybersickness

1 INTRODUCTION

Watching striped patterns rotating in the yaw axis can trigger optokinetic nystagmus (OKN) and visually induced motion sickness (VIMS) (Ebenholtz, 1992; Ji *et al.*, 2009; Lo and So, 2001). About 30% of the British and Chinese population are susceptible to motion sickness (Griffin, 1992; So *et al.*, 1999) and about 30% of viewers of virtual reality (VR) simulation are susceptible to VIMS (Lo and So, 2001; Kiryu and So, 2008; So *et al.*, 2001). A typical OKN cycle consists of a slow drift of the eyes in the direction of the stimuli followed by a rapid saccadic return. While viewers are experiencing OKN, a sudden immersion into total darkness will stop OKN in some viewers and allow slow decays of OKN in others. The latter is called optokinetic afternystagmus (OKAN). It is a continuation of OKN but with a slowly decaying drifting velocity (Ohm, 1927). OKAN occurs in the same direction as OKN. Sometimes it is followed by a

secondary afternystagmus (OKAN II) beating in the opposite direction to OKN (Aschan and Bergstedt, 1955). If OKN response can be divided into the visual perception part and the vestibular response part, OKAN should reflect the part involving the activation of the vestibular nucleus (Tijssen *et al.*, 1989). It has been known that patients with bilateral labyrinthectomy will not experience OKAN (Cohen *et al.*, 1973) and OKAN can be suppressed after surgical removal of the flocculi and portions of the paraflocculi within the vestibular organ (Waespe, 1983). Dellepiane *et al.* (2006) reported that the prevalence rate of OKAN increased with increasing vestibular reflex in individuals. This suggests that the vestibular system plays an important role in the genesis of OKAN.

On the other hand, the vestibular nuclei have also been reported to play a central role in the generation of visually induced motion sickness (VIMS). Cheung *et al.* (1991) reported that bilateral labyrinthine-defective subjects were immune from VIMS but can experiencevection sensation (illusion of self-motion). A review of literature indicate that self-report questionnaires have been the most common way to measure levels of VIMS (e.g., Kennedy *et al.*, 1993; Ji *et al.*, 2009; Lo and So, 2001; So and Lo, 1999; So *et al.*, 2002). Although gastric myoelectric activities have been shown to correlate with rated levels of visually induced motion sickness, its measurement requires making the participants sick (Hu *et al.*, 1989). In 2005, ISO International Workshop Agreement 3 (IWA3) called for the development of simple objective tests to assess susceptibility to VIMS. Consequently, a study has been conducted to investigate whether OKAN can be an objective measure for quantifying susceptibility to VIMS (So and Ujike, 2010).

OKAN is known to be a reflection of the velocity storage mechanism (VSM). There are two pathways related to the generation of OKN and OKAN. A direct pathway linked to the smooth pursuit system is responsible for the rapid rise of OKN and rapid release of OKAN (Muratore and Zee, 1979). The secondary slow rise and steady-state of OKN slow phase velocity and the slow decay of OKAN slow phase velocity is mediated by an indirect vestibular pathway. The direct pathway is also known as the fast pathway and the indirect pathway is known as the slow pathway. During optokinetic stimulation, the indirect component would engage eye velocity signals to contribute to the build-up of OKN velocity. When in total darkness, the discharge of the eye velocity storage would maintain the eye response, resulting in OKAN. This has been referred to as the velocity storage mechanism (VSM). VSM can be triggered either by visual or vestibular stimulus, which can be reflected by the time course of OKAN or the vestibulo-ocular reflex (VOR).

The authors of this paper are not the first to link OKAN with motion sickness. When testing patients with labyrinthine lesions, Dai *et al.* (2003) reported that susceptibility to physical motion sickness is related to the indirect vestibular pathway (also known as the slow pathway) known to control the steady-state of OKN slow phase velocity and its decline during OKAN. Perhaps the strongest evidence reported so far is the ability of 'Baclofen' (a

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drug) to increase the activities of GABA receptors resulting in shortening of the time constant of velocity decay in OKAN as well as preventing symptoms of physical motion sickness in specific forms of vertigo (Dai *et al.*, 2005). We, therefore, hypothesized in this study that, the slow phase velocity decay time constants of OKAN correlates with individual susceptibility to VIMS (H1). This hypothesis is also consistent with past studies on vestibular ocular reflex (VOR). Significant correlations between motion sickness susceptibility and the angular VOR time constants have been reported and it has been suggested that motion sickness is related to the indirect pathway (Dai *et al.*, 2007; Takahashi *et al.*, 1997).

Further literature reviews indicate that studies to relate OKAN and VIMS on normal human beings are not yet available.

2 METHOD

Nine male and nine female subjects were recruited for the experiment. They were exposed to a virtual rotating drum which has previously been used to provoke VIMS and postural instability among viewers (Ji *et al.*, 2009; Chow *et al.*, 2007). Figure 1 illustrates the virtual rotating drum. During this phase (Phase 1), participants were required to watch alternate black-and-white vertical stripes rotating clockwise at 60 degrees per second for 30 minutes while standing. A chin-rest was used to restrain head movements. In addition, the head positions of participants were monitored using a Polhemus FASTRAK sensor. The display was updated at 60 frames per second. A previous study indicates that additional display lag of 40ms may affect subject ratings (So and Griffin, 1991a,b, 1992). In this experiment, images were projected on a curved screen and their projection onto viewers' retinas response instantly to eye movements. Symptoms of motion sickness were measured before, during, and after the exposure using a pre-exposure simulator sickness questionnaire (SSQ), a 7-point nausea rating (Golding, 2006), and a post-exposure SSQ. The slow phase velocities of OKNs were measured using an EOG system (EOG 100C from BIAPAC®).

Two weeks after the exposure in Phase 1, participants were invited back for Phase 2 of the study. They were exposed to the same virtual rotating drum for six repeated trials. The six repetitions were separated into two consecutive days to minimize eye fatigue. Phases 1 and 2 shared the same laboratory environment and apparatus.

Each trial consisted of three stages: (i) participants were instructed to stare at a stationary fixation dot projected on a blank screen for 15 seconds; (ii) after the 15-second period, the virtual drum was started and rotated at 60 degrees per second in the clockwise direction for two minutes. During this period, participants were instructed to look straight ahead; (iii) on the 120th second, the stimulus as well as all light in the laboratory were switched off for one minute and participants were instructed to keep looking straight ahead during this period.

Eye movement data was recorded by the EOG system (EOG100C, BIAPAC®). Two commonly used parameters of

OKAN were analyzed (Dai *et al.*, 2003): the initial velocity of OKAN and the time constant of OKAN. Data corresponding to the first second (i.e., the 120th second) after the light was switched off was discarded following the literature (Tijssen *et al.*, 1989).

The initial velocity of OKAN was calculated from the EOG data collected during the 2nd second after the light was switched off (i.e., the 121st second). Most participants had more than one OKAN cycle during the 121st second. For those who had only one cycle, the first OKAN cycle was used to calculate the initial velocity of OKAN. The decline of the slow phase velocity was modelled with an exponential function (Cohen *et al.*, 1977, Muratore and Zee, 1979, Maioli, 1988, and Tijssen *et al.*, 1989). The time constant was determined by the reciprocal of the slope of the fitted line to plots of the natural log (ln) of the slow phase velocity versus the time of exposure to total darkness.

3 RESULTS

3.1 Head Movements

The average standard deviations of the lateral head movements of the eighteen participants ranged from 0.02 cm to 0.32 cm. The data indicate that participants' heads were well restrained by the chin rest.

3.2 Prevalence Rates of OKAN

The prevalence rate of OKAN among the 18 participants was 55%. 10 out of 18 participants exhibited patterns of OKAN. There was no significant correlation between the occurrence of OKAN and the rated level of nausea reported in Phase 1 of the study. Figures 2 and 3 are examples of consistent occurrence and

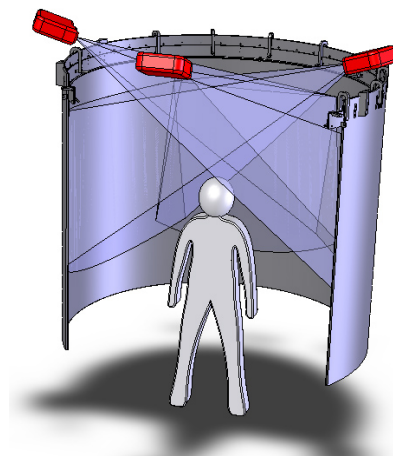


Figure 1. An illustration of a viewer facing the virtual rotating drum with field-of-view of 210° (horizontal) by 50° (vertical).

Table 1. Tally of OKAN occurrence among the 18 participants

Consistently present	Inconsistently/Occasionally present		Consistently absent
All 6 trials	5 out of 6 trials	1 to 4 trials	0% (as in "None of the trials"?)
5	2	3	8

consistent absence of OKAN respectively. As shown in Table 1, only 5 out of the 18 participants showed consistent occurrence of OKAN in all six trials.

3.3 Initial Slow Phase Velocity of OKAN

Among the eighteen participants, eight participants did not show patterns of OKAN. The average 7-point nausea rating of these eight participants was 3.82, ranging from 1.5 to 5.8, while the average 7-point nausea rating for the ten participants who exhibited OKAN was 3.95, ranging from 1 to 5.8. The average 7-point nausea rating for all 18 participants was 3.85. No observable difference in susceptibility to VIMS was found between the two groups of participants.

The average initial velocity of all ten subjects who exhibited OKAN was 7.9 dps, ranging from 3.8 dps to 11.5 dps. This represented a reduction of 80% in slow phase velocity from OKN to OKAN. There was no observable difference in initial velocities between male and female participants.

Correlation tests were conducted between average nausea ratings and the initial velocities of OKAN. There was no significant correlation between the susceptibility to motion sickness and the initial velocity of OKAN (Pearson correlation: $\rho = -0.365$, $p = 0.05$). The discharge rates (initial SPV / SPV of OKN) were calculated for each trial and there was no significant correlation between the rated levels of nausea in Phase 1 and the discharge rates ($\rho = 0.121$, $p > 0.05$).



Figure 2. An example of a viewer showing consistent occurrence of OKAN. The horizontal axis is time and the vertical axis is the voltage of EOG signals. OKN patterns can be observed before the light was turned off (indicated by the vertical black line). OKAN patterns can be observed after the light was turned off (adapted from Guo *et al.*, 2011).

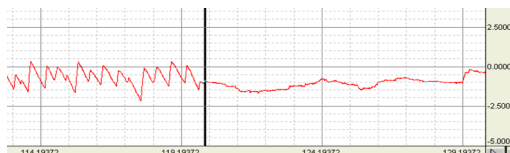


Figure 3. An example of a viewer showing consistent absence of OKAN. The horizontal axis is time and the vertical axis is the voltage of EOG signals. OKN patterns can be observed before the light was turned off (indicated by the vertical black line). No OKAN patterns can be observed after the light was turned off (adapted from Guo *et al.*, 2011).

3.4 Time Constants of the Decaying OKAN velocity

The decaying curve of OKAN velocity was modelled by an exponential function (Tijssen *et al.*, 1989). Linearity correlation between the logarithm of the slow phase velocity of OKAN and duration in each trial was tested before fitting a linear regression line. Those showing no significant linearity were discarded in the calculation of time constants which were the slopes of the fitted

lines. All except one trial reported significant linear correlation ($p < 0.05$). The one that did not show a significant correlation was due to a very short duration of OKAN.

The average time constant of the ten participants who exhibited OKAN patterns was $17.9s \pm 5s$, ranging from 6.7s to 30s; for male subjects it was 16.8s and for female subjects it was 18.81s. There was no significant difference between the time constants of male and female participants. Pearson correlation tests showed that there was marginally significant correlation between the time constants and the rated levels of nausea reported in Phase 1 of the study ($p = 0.05$, correlation coefficient: $\rho = 0.484$). This is an important and interesting finding which extends Dai *et al.* (2003)'s result to visually induced motion sickness. The authors of this paper are delighted to have found significant results even with simple forms of data extraction. An inspection of Figures 1, 2 and 3 indicates that OKAN cycles are noisy. More sophisticated data smoothing techniques may result in better signal to noise ratio. The authors are currently consulting signal processing experts about the best way to detect and extract OKAN data.

The authors 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 significantly correlated with those calculated using just the 121st second data ($\rho = 0.8$, $p < 0.001$). This supports the validity of the fitted regression lines in modelling the slow phase velocity of OKAN. .

4 DISCUSSION AND CONCLUSION

This study examined the correlation of OKAN parameters with rated levels of VIMS on normal human participants. Eighteen participants were studied and ten of them exhibited patterns of OKAN. The initial slow phase velocity and time constant of OKAN were characterized. Based on the statistical results, there are two major findings. First, motion sickness susceptibility, as indicated by the rated levels of nausea in Phase 1 of this study, significantly correlated with the time constants of decaying OKAN slow phase velocity. Since the decaying process is related to the indirect velocity storage pathway, the significant correlation suggests that the susceptibility of VIMS may also be related to the indirect velocity storage pathway. Second, motion sickness susceptibility is independent of the direct pathway. This was indicated by the non-significant correlation between the initial slow phase velocity of OKAN and the rated levels of nausea and the non-significant correlation between the discharge rate of OKAN and the rated level of nausea. These two findings are consistent with past studies concerning the correlation relationships between vestibulo-ocular reflex (VOR) and susceptibility to motion sickness. Susceptibility to physical motion sickness have been shown to be related to the indirect pathway (significantly correlated with angular VOR time constants: Dai *et al.*, 2007) but not to the direct pathway (non-significant correlation with angular VOR gains: Cohen *et al.*, 2003; Dai *et al.*, 2003; 2007). The authors would like to stress that most/all previous studies have been focused on physical motion sickness and no study relating OKAN time constants to levels of VIMS could be found.

The current findings indicate that OKAN measurement is/can be a quick and simple test to measure individuals' susceptibility to visually induced motion sickness without making them sick. However, this study only involved 18 participants. Future studies with more participants are desirable to confirm the findings. Also, the requirement of a complete dark surround should also be addressed. In this study, all windows in the experimental room needed to be sealed. Future work exploring the use of head-

mounted display with tight seal should be conducted to improve the usability of this method.

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