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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.

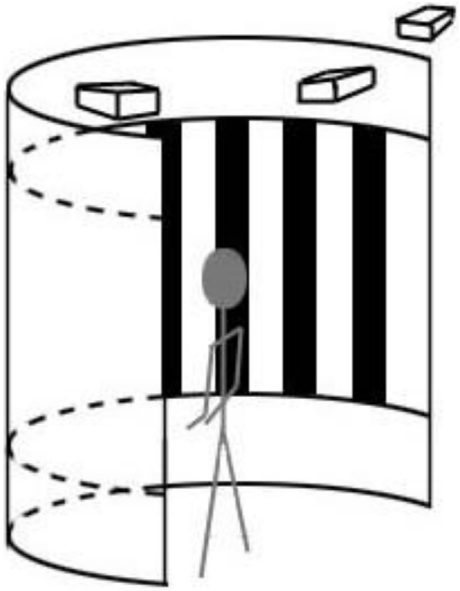

Fig. 1. An illustration of the wide field-of-view virtual rotating drum used to provoke symptoms of VIMS. Visual patterns with field of view of 200° (horizontally) by 65° (vertically) were projected on a curved screen with a radius of 115 cm via three projectors. Proprietary software was used to combine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