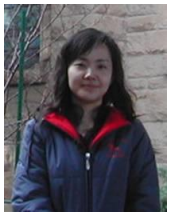
VISUALLY INDUCED MOTION SICKNESS: AN INSIGHT FROM NEUROSCIENCE



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ABSTRACT

Most empirical studies on visually induced motion sickness (VIMS) have cited the sensory rearrangement theory to explain the generation of VIMS. However, since 1990, there has been a surge of competing theories for VIMS (e.g., postural stability theory; nystagmus theory; and subjective vertical conflict theory). This has sparked off much intellectual debate on the possible mechanisms and etiologies of VIMS. On the other hands, neuroscience experiments have advanced our understanding of the neural mechanisms behind vection perception and vertigo sensation. This paper seeks to identify the possible neural pathways concerning the etiologies of VIMS.

Keywords

Motion sickness, cybersickness, neuro-ergonomics, computational ergonomics

INTRODUCTION

Visually induced motion sickness

Lo and So (2001) reviewed 37 empirical studies on VIMS caused by viewing virtual reality displays published in or before 1999. Those 37 studies examined effects of 17 independent variables including variables related to the content of VR simulation (e.g., types of scene background and scene movement), variables related to how the VR simulation is being presented (e.g., exposure duration, types of VR display, display's field-of-view, image delays, use of stereoscopic, inter-pupillary-distance mismatch, method of navigation), variables related to the physical movement of participants (e.g., postures during simulation, amounts of head movement during simulation), and variables related to individual characteristics (e.g., age, gender, pre-exposure posture stability, habituation, menstrual phase, drug treatment). A further review of the literature indicates that between 1999 and 2005, another 36 empirical studies were

published. Given the ample amount of published empirical data, the authors are convinced that the timing is appropriate to determine the fundamental mechanism responsible for generating VIMS in human.

Visually induced motion sickness (VIMS) has been the subject of many empirical studies in the past 100 years. As the display technology evolved over the years, the apparatus used to produce the visual stimuli also changed (telescope: Stratton, 1896, 1897; rotating drums: Stern *et al.*, 1985, 1989, 1993; simulators: Kennedy and Frank, 1995, Kennedy and Massey, 1995, Kennedy *et al.*, 1989, 1995; virtual reality displays: Ji *et al.*, 2004; Lo and So, 2001, McCauley and Sharkey, 1992, Stanney *et al.,* 1998, Stoffregen and Smart, 1998, and Wilson, 1996). Although it has been reported that the profiles of symptoms can change as the display technology changes (e.g., Kennedy *et al.*, 1997), most empirical studies on VIMS have cited the sensory rearrangement theory (Reason, 1967; Reason and Brand, 1975) to explain the generation of VIMS. Since 1990, there has been a surge of competing theories for VIMS (e.g., reflex-response theory: Griffin, 1990; postural stability theory: Riccio and Stoffregen, 1992; nystagmus theory: Ebenhotz *et al.*, 1994; and subjective vertical conflict theory: Bles *et al.*, 1998). This has sparked off much intellectual debate on the possible mechanisms and etiologies of VIMS. A review of literature indicates that there are conflicting empirical findings. For examples, Stern *et al.* (1990) and Flanagan and May *et al.* (2002) reported an association between significant reductions of sickness symptoms with significant reductions of circular vection ratings. However, both Webb and Griffin (2002, 2003) reported that circular vection ratings were not correlated with sickness ratings. Instead, they were correlated with eye-movements. The former finding is more consistent with the sensory rearrangement theory while the latter is more consistent with the nystagmus theory. One obvious way to resolve these conflicts is to conduct more empirical studies. This paper, however, proposes an additional approach that can go hand-in-hand with the empirical study approach. With the recent advances in our understanding of human brain functions, the authors propose to utilize the knowledge accumulated in the field of neuroscience to hypothesize a set of possible neural pathways leading to the generation of VIMS. These hypothetical pathways can then be tested with future empirical studies.

Advances in neuroscience

A review of literature indicates that there has been much development in (i) signal processing models to account for sensory rearrangement and visual-vestibular interactions (e.g., Oman, 1982, 1990, 1991, 1998; Zupan et al., 2002; and Telban et al., 2001, 2002) as well as (ii) understanding of the neuro-mechanism of self-motion perceptions, vertigo, and motion sickness (e.g., Brandt, 1999; Lappe, 2000; Van Essen et al., 1992; Takedo et al., 2001). The understanding of human brain functions provides an opportunity to identify hypothetical neural pathways to explain the generation of VIMS (Ji *et al.*, 2005).

The benefits of determining hypothetical neural pathways of VIMS

Once the hypothetical neural pathways of VIMS are established, software or hardware simulation can be developed to simulate the functions of those pathways. The simulated results can then be compared to empirical results so that the pathways themselves can be tested and verified. This process is consistent with the concept of 'studying an ergonomics process by building an ergonomics process' proposed in So and Lor (2004) and referred to as 'computational ergonomics' approach. Under this approach, empirical studies are tools to refine a biological inspired algorithm or hardware that simulates an ergonomics process until the simulation systems can accurately simulate the corresponding ergonomics process.

To further illustrate the benefits of studying an ergonomics process by building an ergonomics process. Let us use an imaginative example. If scientists who had been studying simulator sickness in the 80's also developed a biological inspired computational algorithm that could simulate the generation of simulator sickness, when VIMS caused by viewing virtual reality displays (i.e., cybersickness) became a cause for concerns, scientists can refine or modify the algorithm developed for simulator sickness to simulate the generation of cybersickness. As this algorithm is biologically inspired, it should have a functional structure similar to the real biological process of cybersickness generation. In other words, the algorithm should reflect the true neural mechanism behind the generation of cybersickness. Furthermore, scientists from all over the world can work on the same algorithm and shared their refined codes via the Internet.

In 2005, the authors were funded by the Hong Kong Research Grants Council to develop a biological inspired algorithm to simulate the VIMS produced after viewing virtual reality displays. This paper presents selected preliminary findings of this study.

THE ROLE OF VECTION IN VIMS: AN INSIGHT FROM NEUROSCIENCE

As mentioned in the introduction section, there have been conflicting reports on the relationships between vection sensation and VIMS (e.g., Stern *et al.,* 1990; Flanagan and May *et al.,* 2002; Webb and Griffin, 2002, 2003). Establishing an hypothetical neural pathway for vection perception and nausea symptoms can be an important guide to future studies in this area.

A review of literature indicates that the magnocellular pathways within the cells in the retina are responsible for the perception of visual motion (Kandel, 1991). The m-type ganglion cells along the pathways are less sensitive to color information and visual patterns of high spatial frequencies. The insensitive to visual color information is consistent with the empirical findings that change of color of a virtual reality simulation did not significantly change the rated levels of nausea ratings and scores of simulator sickness questionnaire (Yuen, 2001). The magnocellular pathway projects onto the primary visual cortex (V1 area) via the thalamus. Cells in V1 area process the visual information within their respective field-of-views (known as receptive fields) according to the spatial frequency characteristics, orientation of spatial patterns, as well as the temporal movements of these patterns. The processed signals are then fed to the middle temporal area (MT area or V5 area) (Rolls and Deco, 2002) and the parieto-occipatal area PO(V6) (Brandt, 1999). Both MT area and the area PO(V6) project signals to the medial superior temporal area (MST area) (Rolls and Deco, 2002). Longstaff (2000) reported that patients with lesions in area V5 could lose their ability to perceive motion and, hence, vection. At the MST area, signal representing the global optic flow are formed and passed to the parietal-insular vestibular cortex (PIVC) area via the inferior parietal area 7a. It has been known that the area PO(V6) and the PIVC are related to the visually induced circular vection and the vestibular-induced circular vection respectively and reciprocally (Brandt, 1999).

A review of literature shows that nausea sensation can be triggered by signal projects from the inferior frontal gyrus (IFG) and vomiting actions are associated with nucleus tractus solitarius (NTS) and lateral medullary reticular formation (LMRF) in the brain stem (Miller *et al.*, 1996; Yates *et al.*, 1998). However, no direct neural pathway was found between area PO(V6) and these three areas (IFG, NTS, and LMRF). Indirect

pathways were found via area V6A, inferior parietal area 7a, PIVC, vestibular nuclei, and thalamus. Although the PIVC is not oxygenated during visually induced circular vection (Brandt, 1999), it is not clear whether the reciprocal actions between PIVC and PO(V6) can play a 'push-pull' role in linking the visually-induced circular vection associated with area PO(V6) and the nausea sensation associated with the inferior frontal gyrus.

FINAL REMARKS

This paper presents selected preliminary findings of a study to identify the possible neural pathways for VIMS. So far, the identified neural pathways support that vection is generated in association with sickness symptoms of VIMS but does not have a clear evidence for a direct cause-and-effect link between the vection signals associated with the parieto-occipital area PO(V6) and nausea and vomiting signals associated with the inferior frontal gyrus, the nucleus tractus solitarius, and the lateral medullary reticular formation, respectively. At the conference, the pathways concerning the generation of optokinetic nystagmus and their relationships with vomiting and other sickness symptoms of VIMS will also be presented. In addition, the interior structure and the role of vestibular nuclei on VIMS will also be discussed.

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