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A Search for Possible Neural Pathways Leading to Visually Induced Motion Sickness

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1. Viewing wide field-of-view image movement can cause nausea.

Numerous studies have proved that, either when seated or standing, and watching continuously rotating, oscillating, or translating scene movements, about 30% of humans will suffer from mild to moderate sickness symptoms ranging from headache to nausea and about 10% will prefer to stop watching the visual stimulation. There is also evidence to link this type of visually-induced motion sickness to postural instability. Recently, the ISO Technical Management Board approved a call for an ISO International Workshop Agreement (IWA3) on image safety that addresses this issue of visually induced motion sickness (VIMS).

2. While empirical studies are important to discovering the effects of various factors, understanding the neural mechanism responsible for visually induced motion sickness should be the ultimate goal.

With advances in brain imaging techniques, great advances in neurosciences have been achieved in the last decade. The authors believe that the timing is right for scientists who have been studying visually induced motion sickness to search for neural mechanisms that are consistent with their empirical findings. In this lecture, a possible neural pathway for visually motion sickness is presented and summarized. Please note that the possible neural pathways proposed in this lecture are by no means the final proven ones. There is still too many unknown to make the final decision and the purpose of this lecture is to stimulate interest among vision scientists to study the neural mechanism behind VIMS.

3. Possible neural pathways for generating visually induced nausea are illustrated in Fig. 1.

Light traveling through a pupil is detected by rod and cone cells inside the photoreceptor layer of the retina. Once a photoreceptor cell receives enough light quanta, it emits a signal to corresponding ganglion cells with different combinations of horizontal, bipolar and marine cells. Each ganglion cell has its own receptive field on the retina that is organized in a circular, concentric, antagonistic center-surround manner. There are 3 types of ganglion cells:

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alpha, beta and gamma. Alpha (i.e., parasol or m-type) ganglion cells have large cell bodies and receptive fields and are sensitive to visual stimuli of low spatial frequency and high temporal frequency. They are responsible for detecting motion. Beta (i.e., midget or p-type) ganglion cells have medium-sized cell bodies and small receptive fields and are sensitive to the spectral frequency of visual stimuli of high spatial frequency and low temporal frequency. Their main function is to cope with the details of vision. The remaining type is called a gamma (k-type) ganglion cell, which have been found to be useful in color vision. A review of the literature indicates that viewing static images is not the cause while viewing moving images is causative of VIMS. Consequently, we propose that the motion pathway plays an important role in explaining the effects of VIMS. Continuing through the M-stream (magnocellular-stream), the output signals of alpha ganglion cells are routed via the optical nerve and tract to the magnocellular layer of the dorsal lateral geniculate nucleus (dLGN) situated in the thalamus. dLGN is a relay station with afferents from the retina and efferents to the primary visual cortex (V1). The signals from dLGN via optical radiation terminate at the cells of layers 4Cα and 4B in the V1 cortical hypercolumns. Up to this point, the optical information sensed by the retina is still organized according to the spatial structure of the retina (referred to as the retinotopic map). In the V1 region, each hypercolumn contains an aggregation of simple, complex, and hyper-complex cells that act as filters responding to optical information originating from a selected area of the retina (referred to as the receptive field) according to

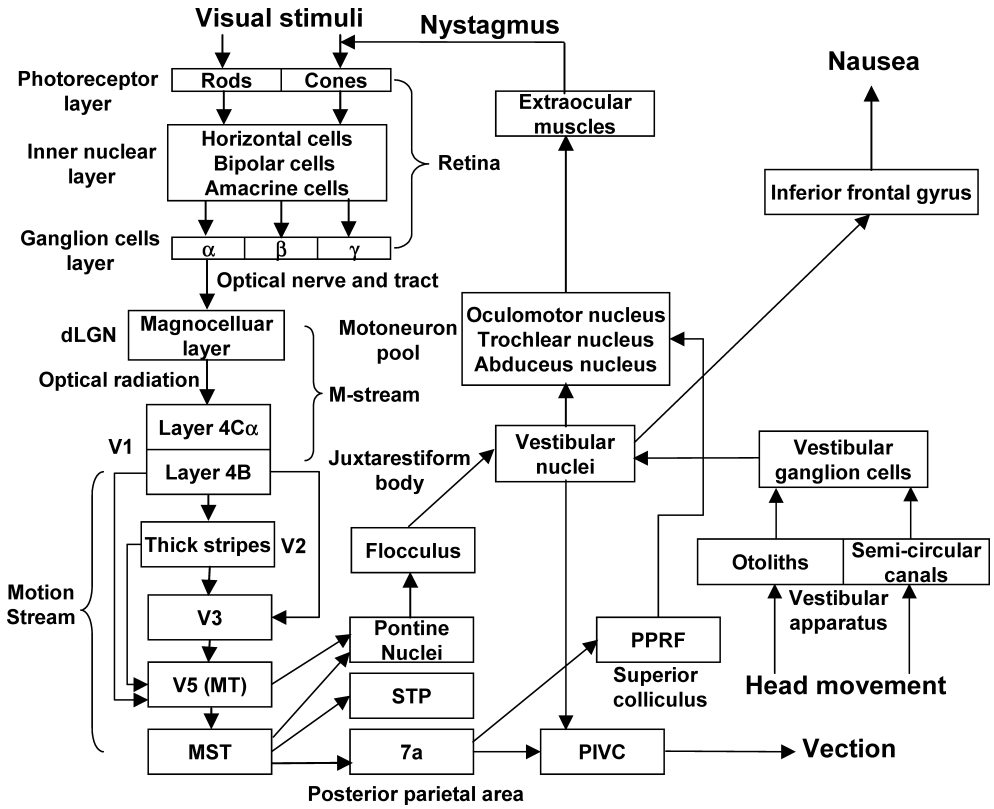

Fig. 1. An illustration of the preliminary biological pathways for the generation of vection and nausea.

the spatial frequencies and orientation of the spatial patterns, as well as movements of these patterns. The output signals from these V1 cells are then fed to the middle temporal area (MT or V5) directly or indirectly through the regions' thick stripes of V2 and V3. Those regions are responsible for the detection of changes in depth and spatial patterns, respectively. As we are focusing on the M-stream pathway, we will bypass the functions of V2 and V3 for the time being.

The main function of V5 (MT) is to evaluate the optic flow of local receptive fields. After that, V5 projects processed signals to nearby extrastriate areas, such as the medial superior temporal area (MST). The receptive field of MST almost covers the entire area of the retina. It integrates signals from local fields and constructs the global optic flow field for the entire visual field. This, we believe, is relevant to the generation of VIMS. The output of the m-stream from MST is sent to different parts of the deeper cortex, such as the posterior parietal area (7a) and the superior temporal polysensory area (STP), for complex motion interpretation (Pomplun et al. 2002, Vaina et al. 2001). Brandt (1999) reported that the parietal-insular vestibular cortex (PIVC) has a strong correlation with the generation of vection while the inferior frontal gyrus is correlated with the generation of nausea (Yates et al. 1998). In many empirical studies of VIMS, reported levels of vection have been found to correlate with levels of nausea. Since the occurrence of nystagmus has been found to correlate with levels of vection, the initial pathway of nystagmus generation is also determined and shown in Fig. 1. To generate the fast corrective phase of a nystagma in the horizontal direction, the posterior parietal area projects motion signals to the paramedian pontine reticular formation (PPRF) of the superior colliculus (SC) (Dietrich et al. 1998). Then, PPRF processes the information and sends commands to the extra-ocular muscles via the oculomotor nucleus, the trochlear nucleus and the abducens nucleus. With a slow motion arc of a nystagma along the horizontal direction, motion signals are transmitted directly from MT and MST to the pontine nuclei in the brainstem. Pontine nuclei then trigger the flocculus in the cerebellum to generate signals that feed into vestibular nuclei through the juxtarestiform body. The vestibular nuclei then project command signals to the oculomotor, trochlear, and abducent nuclei to drive the extra-ocular muscles appropriately. Webb and Griffin (2002) reported that levels of VIMS are correlated with the amount of nystagma but not vection. As a consequence, the nystagma pathway may play an important role in VIMS.

As illustrated in Fig. 1, vestibular nuclei also receive primary vestibular afferents from vestibular ganglion cells. The input to the vestibular ganglion cells is generated by the vestibular apparatus consisting of two parts: semi-circular canals and otoliths, which are responsible to measure, respectively, the rotation and translational movement of the head. Vestibular stimuli not only influence nystagmus, but also contribute to the generation of nausea and vection. The convergence of visual and vestibular signals at the vestibular nuclei has been empirically proved by Dichgans and Brandt (1972). Given the central role of the vestibular nuclei, the authors acknowledge that the current version of the pathways as illustrated in Fig. 1 does not give enough attentions on the vestibular nuclei. The proposed pathway is still under refinement and future work includes (i) the improvement of the pathway model and (ii) the development of a computational simulation algorithm for the proposed model. The ultimate

goal is to develop a biological computational algorithm that can simulate the generation of VIMS.

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