GUEST EDITORIAL

What is Visual Motion Hypersensitivity?

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Symptoms of visual motion hypersensitivity (VMH), also known as visual vertigo and/or motion sickness, can be provoked by motion of the individual through the environment (riding in a car, boat, or plane), or motion of the environment (visual surroundings) while a patient stands still (standing in an active shopping center or driving on a busy road).[1,2] Symptoms of VMH include dizziness, nausea, vomiting, vertigo, imbalance, diplopia, headache, and disorientation.[3,4] There are several theories regarding the origin of VMH. It is thought that a combination of a vestibular disorder and a strong visual reliance on the magnocellular system and peripheral visual field are contributory.[1] A link between VMH and visual abnormalities such as unsteady fixation in a moving visual background and binocular visual dysfunction has also been found.[2] Diagnosis such as traumatic head injury and concussion increase the incidence of motion sickness.[5] The leading theory, however, is that there is a mismatch in the information received by the brain between the visual, vestibular, and somatosensory systems.[1-6]

The vestibular sensory system is responsible for providing us with information about motion,head acceleration, deceleration and position, and spatial orientation, as well as allowing us to balance, maintain posture, and stabilize the head and body during movement.[1,2] The vestibular system uses several organs to fulfil its function. The complex set of integrated sensorimotor-control systems which regulate balance have interlacing feedback mechanisms from the inner ear, eyes, muscles, and joints. Any disconnect or conflict between the vestibular, visual and/or musculoskeletal proprioceptive systems will cause symptoms of visual motion hypersensitivity (motion sickness).[6-7]

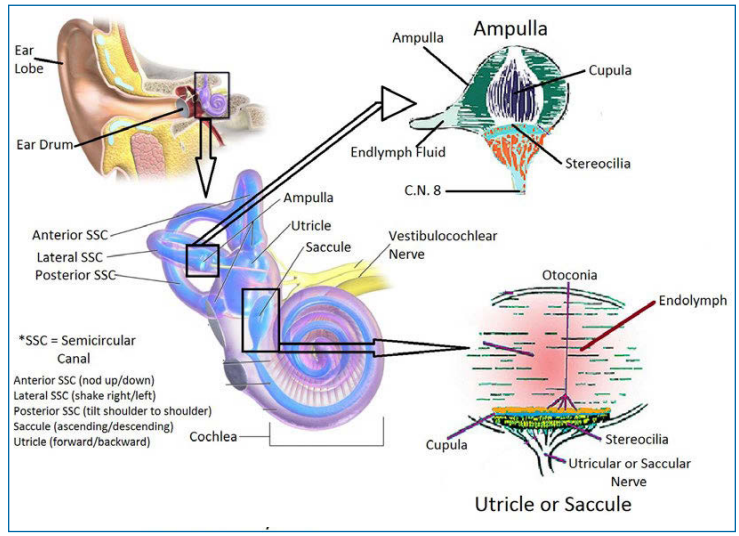

Figure 1. Representation of the vestibular labyrinth

The vestibular labyrinth, which is continuous with the cochlea, is a system of compartments found in each inner ear. The brainstem gains information from the vestibular system about the head's position and movement as well as what the body needs to maintain or regain balance.[8-10]

- **Angular acceleration** – Inside the labyrinth are three interconnected bony fluid filled tubes, called **semicircular canals,** which are each situated in a plane corresponding with head movement (Figure 1). The *anterior* (a.k.a. superior) semicircular canal is sensitive to movement and rotation in the lateral plane (nodding up and down/pitch), the posterior semicircular canal is sensitive to movement or rotation in the anterior-posterior plane (tilting shoulder to shoulder/roll), and the lateral (a.k.a.horizontal) semicircular canal is sensitive to movement or rotation in the transverse plane (shaking right and left/yaw). The **endolymph fluid** inside each semicircular canal moves through

the canal corresponding to the plane of movement when the head is either moved up and down, side to side, or right and left. As the fluid moves with the corresponding head movement, it flows into an outpouching at the end of the canal, called the **ampulla**. The ampulla houses a thick gel, called the **cupula**. The sensory hair receptor cells (**stereocilia**) of the vestibular system sit within the cupula gel. As endolymph fluid moves into the ampulla and causes movement of the gelatinous cupula, the sensory stereocilia hair cells nestled inside the gel deflect and move. As the stereocilia move, they convert mechanical energy into electrical energy and neurotransmitters are released with information about that specific plane of movement to the brain via the vestibulocochlear nerve (C.N. 8). The stimulation of one canal in the right ear results in the inhibition of the same canal in the left ear (and vice versa) allowing sensation of all directions of rotation. If the movement becomes continuous without any acceleration or deceleration, the endolymph fluid stabilizes and stops applying pressure on the cupula and stereocilia - stopping the signal. If the continuous movement is then suddenly stopped, you would feel as though you were turning in the other direction as the endolymph fluid rushes out of the ampulla. This same 'stimulation' and 'inhibition' process is true for all coupled canals.

- **Linear acceleration** – The **otolith organs**, which lay inside the vestibule just between the semicircular canals and cochlea, are two fluid-filled pouches that help detect forward and backward movement, up and down movement, as well as gravitational force (where the head is with respect to gravity – sitting up, laying down, leaning back...). The vestibular labyrinth has two otolith organs: the **utricle** (responsible for detection in the horizontal plane) and the **saccule** (responsible for detection in the vertical plane). Calcium carbonate crystals called **otoconia** move and shift in response to horizontal movement in the utricle or vertical movement in the saccule. The shift of the otoconia crystals is detected by specialized sensory hair cells (stereocilia and kinocilium) inside the utricle and saccule.

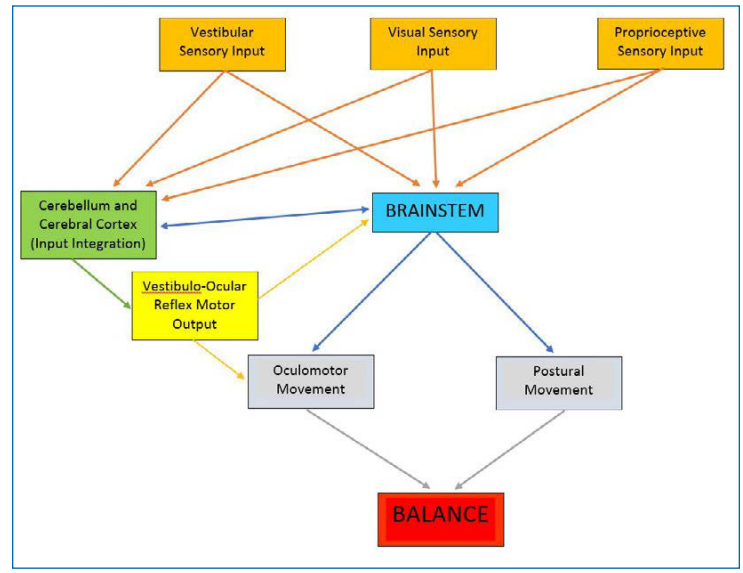

Figure 2. Sensory and Motor Integration for Balance

Information provided by the eyes, muscles, and joints is sent along with information from the vestibular system and is summated in the vestibular nuclei of the brainstem (Figure 2). The information is then analyzed and integrated with information sent to the brainstem from the cerebellum and cerebral cortex. The cerebellum sends information about learned movements which have become automatic through repetition (walking up stairs) while the cerebral cortex sends information about learned movements due to past experience (walking carefully when it is snowing because we know the ground is slippery and we may fall).[8-10]

The vestibulo-ocular reflex (VOR) keeps an image stable on the retina while the head is in motion (Figure 3). The VOR initiates eye movements in the opposite direction of

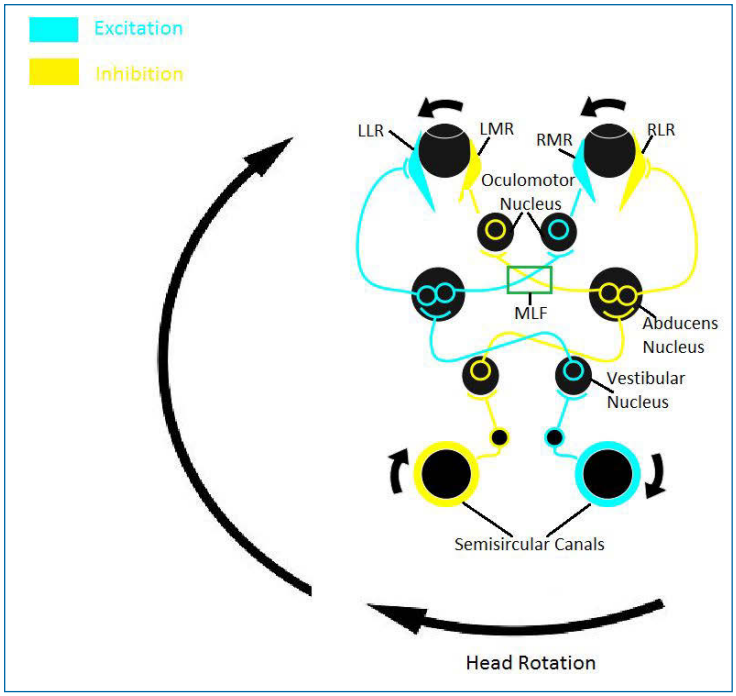

Figure 3: VOR to the LEFT during RIGHT head movement

Person turns head to the right –> excitatory signals from the right semicircular canal are sent through the right vestibular nerve to the right vestibular nuclei in the brainstem.

From the right vestibular nuclei, excitatory fibers leave the brainstem and cross to innervate the contralateral left abducens nucleus and go on to stimulate the left lateral rectus via the left abducens nerve (abducting the left eye). The excitatory fibers will also travel through the MLF and innervate the right oculomotor nerve which will then innervate the right medial rectus via the right oculomotor nerve (adducting the right eye).

Meahwhile, the inhibitory signals from the left semicircular canal are sent through to the left vestibular nuclei in the brainstem. Through the same pathway as above, they inhibit the right lateral rectus and left medial rectus.

As a result, both eyes will turn to the right while the head turns to the left.

head movement and prevents "retinal slip" of an image. There are horizontal, vertical and torsional components to the VOR. In the horizontal rotational VOR, the semicircular canals and otoliths become activated by head movement and send efferent information through the vestibular nerve (C.N 8) to the vestibular nuclei in the brainstem. After synapsing in the brainstem, fibers cross to the contralateral side and synapse in the contralateral abducens nucleus (C.N. 6). From the abducens nucleus, the fibers travel to (1) the contralateral lateral rectus via the abducens nerve and (2) project to the Medial Longitudinal Fasciculus (MLF) to synapse at the ipsilateral oculomotor nucleus (C.N. 3). From the oculomotor nucleus, the fibers go on to innervate the ipsilateral medial rectus muscle.[8-10]

The vestibular system continuously sends oculomotor control signals through the VOR. When the head is stationary, the number of impulses from the left and right vestibular system are equal. When the head is rotated, the number of impulses on that same side increase while there is a decrease of impulses or inhibition on the opposite side. For example, if a person rotates their head to the left, excitatory signals from the left semicircular canal are sent through the left vestibular nerve to the left vestibular nuclei in the brainstem. The fibers leave the brainstem and cross to innervate the contralateral right abducens nucleus and go on to stimulate the right lateral rectus via the right abducens nerve (abducting the right eye). They will also travel through the MLF and innervate the left oculomotor nerve which will then innervate the left medial rectus via the left oculomotor nerve (adducting the left eye). As a result, both eyes will turn to the right while the head turns to the left. The difference in the impulses sent from each side control eye movement to stabilize gaze and allow for stable viewing during head movement. Very similar pathways exist for the vertical and torsional VOR and involve the superior and inferior obliques and superior and inferior recti muscles.

If the head movement to the side is short lived, the direct VOR path is sufficient to keep the eyes pointing in the opposite direction of head movement. If the head stays turned, however, the eyes will drift back to the center since the direct VOR does not maintain the signal. In this case, a tonic input (from the prepositus hypoglossi (PPH) via the indirect pathway) is required (Figure 4). The PPH nucleus converts the short lasting phasic vestibular input from the VOR into a long lasting tonic signal for the extraocular muscles.

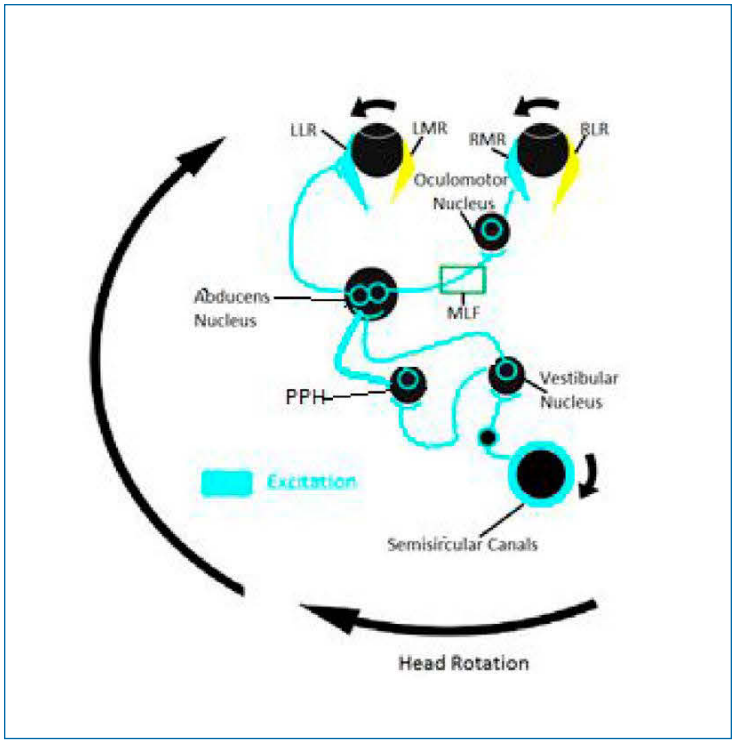

Figure 4. Tonic PPH pathway

As long as the head is turned and stays in the same location and the patient is fixating on a target, the pathway 'remembers' how far and at what angle the head is turned and stabilizes the eyes pointing in the opposite direction for stable fixation.

If a person starts to spin with their eyes closed, the cupula will initially move causing the stereocilia to send impulses to the brainstem. After prolonged rotation with stable velocity, the cupula gradually restores to its upright, stationary position and the stereocilia stop firing, and the VOR pathways stops as well. If they open their eyes at that instance, they will see that they are in fact moving (producing a disconnect between the vestibular and visual systems) and causing the sense of dizziness and nausea. The visual system can also drive the VOR and produce a sense of motion without any movement or vestibular drive via the optokinetic nystagmus (OKN). The OKN movements stabilize the eyes during tracking of a moving visual target which will cause an illusion and sensation of circular vection (feeling of motion when the body is stationary) in the opposite direction. If a person is stationary but something in their visual field moves, this will produce an optokinetic response and a false sense of motion.[8-10]

Any mismatch of information or conflict between the vestibular, visual and/or musculoskeletal proprioceptive systems will cause symptoms of dizziness, nausea, vertigo, motion sickness, double vision, and headaches. Visual motion hypersensitivity or motion sickness during transportation is a common symptom and occurs when the visual and vestibular systems are in conflict. If a person is in the cabin of a boat or reading in the car, the head/vestibular system feels the motion but the visual system does not see the world moving since it is concentrating on a stationary object. This disconnect is the cause of motion sickness during transportation and why looking at the horizon and seeing the world moving helps resolve them.[8-14]

Charles Darwin once wrote, "If it was not for sea-sickness, the whole world would be sailors." Depending on the type of vehicle, the prevalence of motion sickness ranges from 3-60% and the prevalence has increased since the creation of smartphones and tablets.[10-14] The car manufacturer, Citroën, is claiming to have developed the first glasses against motion sickness called LUNETTES SEETROËN (Figure 5).[15,16] We were unable to find any independent studies relating to the product and have not had any personal or anecdotal experience with the product, but it is important that the optometric community be aware of the products available to our patients.

The manufacturer claims that the new glasses are 95% effective in relieving motion sickness and online reviews have, so far, been positive. The "boarding ring technology" glasses consist of a central and peripheral round transparent frame that is half filled with a colored liquid. As a person looks at an immobile object in a moving car, the fluid moves around with the motion of the vehicle, providing a moving, virtual horizon in the

Figure 5. LUNETTES SEETROËN glasses[18]

peripheral visual field (in synch with the motion felt by the inner ear) in both the frontal (right / left) and sagittal (front / back) directions. As the colored liquid moves in the periphery, it re-creates an artificial moving horizon, re-synchronizing the senses and removing the disconnect between the auditory and visual systems. The glasses do not disrupt central vision and can be worn over prescription glasses.[15-17]

Citroën directs that the glasses should be put on at the first sign of motion sickness. They are to be worn for 10-12 minutes (until the auditory and visual systems resynchronize) and can be removed after symptoms have resolved to enjoy the rest of the trip without them. The glasses cannot be used on children under 10 years old, since the inner ear has not yet finished developing and should not be used in conjunction with anti-motion sickness drugs as they may negate the effects of the glasses. The glasses cost around $115 and may be ordered online at lifestyle.citroen.com. The company's product video can be viewed at https://youtu.be/CBqpTqc8Kpc.[19]

Given that motion sickness affects so many of our patients via the inability to ready or look at a smartphone while traveling, this product, though awkward in appearance and not yet independently verified, may prove to be quite useful in people's day to day commute. One can understand the mechanism when using this device on a smooth road with constant acceleration but it would be necessary to evaluate the effect on a vehicle with constantly changing direction and acceleration. Clinical testing of the device with people that experience visual motion hypersensitivity both during locomotion and during motion of the visual surroundings is warranted. On another note, vertigo and dizziness are a prominent complaint in many traumatic head injury and concussion patients. The pathogenesis for this differs vastly, but it would be interesting to evaluate the effect of this boarding ring technology in patients with traumatic brain injury who suffer from VMH.

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