Visual-Vestibular Interaction and Treatment of Dizziness: A Case Report

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Abstract

Background: *The visual and vestibular systems are closely related both physiologically and functionally. Dysfunction in one or both systems can lead to subjective feelings of dizziness and/or vertigo. A common cause of vestibular dysfunction is head trauma leading to traumatic brain injury. Throughout the rehabilitation process, optometrists providing rehabilitative vision therapy services play a vital role in the recovery of visual and vestibular function and quality of life.*

Case Report: *The patient in this case suffered a closed head trauma resulting in visual and vestibular dysfunction. Following vestibular rehabilitation and occupational therapy, she demonstrated residual symptoms of disequilibrium and eye strain that were exacerbated by computer use and large, crowded spaces. She was enrolled in a course of in-office vision therapy for treatment. Following rehabilitative vision therapy, the patient reported significant improvements in her everyday activities and quality of life. She noted that her vision was more stable, balance had improved, and reading was easier with improved comprehension.*

Conclusion: *A thorough visual assessment coupled with appropriate optometric intervention is essential for patients suffering traumatic brain injury, especially when vestibular dysfunction is present. Through the coordinated use of lenses, prisms, and rehabilitative vision therapy, relief or even resolution of symptoms can occur.*

Key Words

traumatic brain injury, vertigo, vestibular rehabilitation, vision therapy

The Vestibular System

Postural control and coordination of movement, including eye and head movement, is mediated primarily through the vestibular system and its input to the central nervous system (CNS).[1] An in-depth discussion of the anatomy of the system is beyond the scope of this article; however, a basic understanding of its structure and how it relates to the visual system is essential.

The vestibular system consists of three main components: a peripheral sensory apparatus, a central processor, and a motor output system.[2] The peripheral sensory mechanism resides within the inner ear and is where the sensory cells of the system reside.[2] Within the peripheral vestibular system are the semicircular canals, which contribute to sensation of velocity and communicate via the vestibular nuclei with motor neurons of the extraocular muscles (EOMs).[1,2] The orientation of the three semicircular canals within each ear parallels the actions of each of the EOMs, and each canal has major neural connections to one ipsilateral and one contralateral EOM.[2-4] This relationship is the basis of the vestibulo-ocular reflex (VOR), which is responsible for maintaining stable retinal images and thus maintaining gaze stability during head movement. Because of their anatomical positioning, the semicircular canals are able to sense all rotational movements in space and therefore maintain stable vision for every direction of head movement.[3]

Central processing of vestibular information takes place in two main locations: the vestibular nuclear complex and the cerebellum.[5] Sensory input from the peripheral vestibular system travels to the vestibular nuclei where it is combined with visual, somatosensory, and cerebellar information for the maintenance of balance and equilibrium.[4,5] From the vestibular nuclei, numerous fibers communicate with the cerebellum, which serves an adaptive role: it modifies the VOR as needed and adjusts the gain for saccadic and vergence eye movements.[2,5,6] Vestibular information is also transmitted to various association cortex areas for further interpretation; however, there is no primary vestibular cortex.[3] Once interpreted, vestibular data is integrated with visual and somatosensory inputs and the final step in the process, vestibular motor output, occurs. This is accomplished via various motor neurons that produce both eye and postural movements resulting in maintenance of visual stability as well as overall bodily balance and coordination.[2,5]

Vestibular Dysfunction

Dysfunction along any pathway in the vestibular system can lead to the sensation of dizziness and/or vertigo. Patients often use these terms interchangeably; however, due to differences in underlying etiology, it is important to distinguish between them. Dizziness can refer to any subjective feeling of floating, light-headedness, or disorientation and is a commonly experienced sensation following traumatic brain injury (TBI). A feeling of dizziness often, but does not always, involve vestibular dysfunction.[1] The underlying cause of dizziness

may be vestibular, somatosensory, or visual in origin or may arise from any combination of the three.[6] Non-vestibular etiologies of dizziness include presyncope, disequilibrium, and psychologically induced dizziness. Presyncope is a feeling of light-headedness caused by restriction of blood flow to the brain[4] and may be accompanied by sweating, nausea, and constriction of the visual field.[7] Disequilibrium is the loss of balance without any illusion of movement and results from disturbances in sensory-motor integration.[4,7] It is often exacerbated by unfamiliar or dimly lit places.[7] Psychologically induced dizziness is frequently related to high anxiety and often accompanies panic attacks.[4,7]

Vertigo is the illusion of movement - either of one's self or one's surroundings - when there is none. The underlying cause is a lack of concurrence between visual, vestibular, and/or somatosensory inputs.[4,7] Such an incongruity could be caused by damage to the inner ear, the vestibulocochlear nerve, the vestibular nucleus, and/or the cerebellum.[4] Vertigo results when the brain is unable to resolve the inconsistency between various sensory inputs.[7] Unlike dizziness, true vertigo *implies* vestibular involvement.[1] In patients recovering from TBI, dizziness, vertigo, and imbalance are all common.[8]

It is imperative to keep in mind that a major portion of the sensory input contributing to balance and coordination comes from the visual system. Therefore, it should not be overlooked that unreliable visual input can also create sensory conflicts resulting in dizziness or vertigo. There are multiple routes through which visual input can be distorted: abnormal eye movements (such as nystagmus), an unclear image, image size differences between the two eyes (aniseikonia), binocular dysfunction, and/or visual field deficits.[7] Each of these present a potential problem that should be addressed as part of a thorough visual assessment with patients complaining of dizziness or vertigo. If there is concurrent vestibular dysfunction, be cautious when prescribing spectacles. These patients may be less able to adapt to new lens prescriptions, and changes can exacerbate any symptoms they may be experiencing.[7] The greater the change in lens power, the more the system is required to recalibrate to a new gain in the VOR system when gaze is directed away from the optical center of the lenses. In addition, there may be hypersensitivity to other changes such as alteration in base curve or frame shape.

The presence of ocular misalignment (heterophoria, fixation disparity, or strabismus), can require continuous effort by the patient to overcome. Those with intermittent deviations are often the most symptomatic,[4] as maintaining alignment is a constant struggle. In addition, the brain must decipher disparate information from the two eyes before incorporating it with incoming vestibular and proprioceptive data.[7] This extra energy and concentration toward what should be a subconscious process can lead to symptoms of dizziness.[7] Yang et al.[9] found that adults with normally functioning vestibular systems who were experiencing symptoms of vestibular dysfunction actually had binocular problems, demonstrating deficiencies of both saccadic and vergence eye movements. Similar results have been found in children experiencing visually-induced vertigo, for whom treatment with orthoptic training and/or therapeutic lenses alleviated symptoms.[10,11]

Because of the intimate relationship in function between the visual and vestibular systems, dysfunction in the vestibular system can affect visual function. One such effect is a decrease in ability to maintain steady fixation.[3,6] As a result, patients may experience their world as being jerky or bouncy. Such gaze instability may lead to complaints of difficulty with reading and/or comprehension, as text that appears to be moving requires much more effort to process, thus leaving little cognitive function for interpretation and understanding. Reading on a computer presents an additional challenge due to actual movement of text on the screen or a heightened sensitivity to the screen's flicker, which is often seen in vestibular dysfunction.[12] Additional visual difficulties that may accompany vestibular dysfunction include deficits of accommodation, vergence, versions, visual perception, and visual field.[6] Convergence insufficiency and uncompensated fixation disparity or phoria are common findings.[3] Vestibular dysfunction does not necessarily produce a fixation disparity or heterophoria, but can decrease the patient's ability to compensate for pre-existing binocular conditions. When binocularity breaks down, it further increases the disparity between visual and vestibular inputs, affecting the VOR and sense of balance.[3] With VOR impairment, nystagmus, oscillopsia, photosensitivity, and/or discomfort with flickering lights can occur, with fluorescent lights being especially aversive.[12] Situations in which both fluorescent lighting and moving objects are present can be particularly strenuous, making shopping in large spaces such as a grocery store or shopping mall very challenging.[12] Peripheral awareness may also be affected, as the ambient visual process–which provides information regarding contrast and movement and supports posture, balance, and spatial orientation–is integrated via the vestibular nuclei.[3,8,12]

Optometric Intervention in Vestibular Dysfunction

Because multiple sensory systems are affected when vestibular dysfunction is present, multi-disciplinary therapy is essential for recovery. The first step is to address the vestibular dysfunction directly. Aside from surgical or pharmacological intervention, vestibular therapy is often accomplished through a guided exercise program designed specifically for the patient and administered by occupational and/or physical therapists.[4] Such programs are individually designed for each patient to address their specific symptoms while taking into account any limitations they may have.[13] The purpose of the exercises is to decrease dizziness by improving the ability to compensate for vestibular deficits. Success is highly reliant on CNS plasticity, as improved compensation is accomplished through reorganization of sensory and motor aspects of postural control within the system.[4,13,14] In addition to prescribed rehabilitative activities, a general exercise routine should be encouraged, as it will facilitate the recovery process.[13]

Although often overlooked, the visual system contributes heavily when vestibular dysfunction is present. When patients are unable to compensate for deficits in vestibular function with vestibular rehabilitation alone, one of the most common reasons is impairment in one or both of the other sensory inputs necessary for balance, one of which is vision.[13] In a normally functioning system, visual input makes up one third of the sensory information used for coordination and balance.[4] In the context of vestibular dysfunction, vision may take over as the primary sensory input for posture and balance, even if the visual system itself is impaired.[6,8] Erroneous visual input can cause these patients to lose their balance or even fall,[15]

potentially leading to further complications. It is therefore essential that visual function be assessed and optimized as part of the rehabilitation process.

The first step to optimizing visual function is finding the proper spectacle prescription. Because visual input is relied upon so heavily in vestibular dysfunction, a quality refraction is of utmost importance. When prescribing, it is best to have separate lenses for distance and near viewing, as the optical effects of progressive addition lenses, multifocal contact lenses, or monovision viewing could exacerbate vestibular symptoms, especially if there are induced peripheral distortions.[4] In addition, spectacles with smaller lenses serve to reduce the amount of optical aberrations, thus helping to decrease symptoms of dizziness and/or vertigo.[12] Prescription of correcting prism should be considered, even for small deviations, to maximize quality and stability of visual input. When prescribing, keep in mind the aforementioned difficulty that vestibular patients may have adapting to major changes in lens prescription.

After successful ophthalmic correction has maximized optical quality of visual input, a course of rehabilitative vision therapy (VT) should be undertaken to decrease symptoms and maximize visual performance. Because of the close physiological and functional relationships between the visual and vestibular systems, it is not uncommon for patients to show significant improvements with a decrease in symptoms following a course of rehabilitative VT.[5,6] According to Mejia,[4] there are four main treatment goals in VT for vestibular rehabilitation:

1. Enhance the stability of the visual system;
2. Stabilize fixation, improve motor control and planning, and enhance peripheral awareness;
3. Develop a stable binocular system, including accommodation and vergence flexibility, rapid recovery of fusion, and central fusional vergence stability;
4. Challenge the binocular system in the context of a dynamic visual environment and incorporate head and body movement while decreasing somatosensory cues.

In addition, visual perceptual processing——including cognitive speed and flexibility—should be addressed, along with any visual field deficits that are present.[4] If the patient is able, activities involving head and body movement should be incorporated. Yoked prisms are a useful tool and can be used to generate a sensory disruption to which the patient must adapt in order to maintain balance,[4] thereby increasing their ability to integrate changes in visual stimuli. Throughout the course of therapy, activities should be performed at a level that challenges the patient and that may even provoke mild symptoms. It is this level of stress to the system that will take advantage of CNS plasticity and drive the changes necessary for improvement.[6] However, proceed with caution with patients engaged in any concurrent therapies; patients involved in more than one mode of treatment are easily over-stimulated, and may become sick for hours or even days if it occurs.[6] Ultimately, through the coordinated use of lenses, prisms, and rehabilitative VT, relief from or even resolution of symptoms can occur.

Case Report

MC, a 52-year-old Asian female, presented on 2 June 2010 for a vision therapy evaluation with complaints of asthenopia, blur, and pain/discomfort with near work. She had sustained a TBI when she was hit by a car in January 2009, and after completing vestibular rehabilitation, was experiencing residual vestibular symptoms. She reported that maintaining attention during driving and computer work was difficult, and that she felt off-balance after driving, especially long distances. She was also uncomfortable and felt nauseated and off-balance in large spaces with many people, such as markets and shopping malls. In addition, she felt that her peripheral awareness was not as good as it had been before the accident. Her visual and vestibular symptoms were exacerbated by computer work and stress. At the time of her initial evaluation, she was receiving occupational therapy and had recently undergone the Sensory Organization Test (SOT) at Kessler Institute for Rehabilitation. The SOT serves to identify abnormalities in ability to balance using the three contributory sensory systems – visual, vestibular, and somatosensory.[16] MC's SOT revealed abnormalities in her overall balance control as well as in her ability to use visual and vestibular cues to maintain balance. She had completed a course of physical therapy for vestibular dysfunction and was also being seen regularly by an ear, nose, and throat (ENT) specialist, who referred her to the office for optometric evaluation and was also recommending exploratory inner ear surgery.

MC's last comprehensive eye exam was in March 2010, and the records obtained from that exam indicated that ocular health was normal. She was diagnosed at that time with myopia, presbyopia, and astigmatism and was given a spectacle prescription for full-time wear. She presented on the day of the VT evaluation with a habitual spectacle prescription of -3.25-0.75x059 OD, -0.25-0.50x093 OS with a +1.50 add OU in a progressive addition lens which she used almost exclusively for working at the computer. When not wearing her spectacles, MC's refractive error provided natural monovision viewing conditions, of which she chose to take advantage.

Entering unaided distance visual acuities were 20/400 OD, 20/20- OS. Autorefraction was performed with a Grand Seiko Open-View Autorefractor, and distance refractive error was revealed to be -2.50-0.25x066 OD, +1.50-1.25x081 OS. This assessment was repeated at each vision therapy progress evaluation and remained relatively stable throughout the course of treatment. Given her habitual prescription at the time of evaluation, it appeared that she was over-minused in both eyes. This likely contributed to her symptoms and may have been the reason she preferred monovision viewing. The prospect of separate prescriptions for distance and reading was discussed, but MC was not receptive to the idea as she only used her glasses for computer work and did not feel separate prescriptions were necessary. Reading eye movements were assessed using a Visagraph II, which revealed an overall grade level efficiency of 1.7, substantiating her subjective complaint of difficulty with near work. All parameters measured–fixations, regressions, average span of recognition and average duration of fixation–were inadequate. Overall reading rate was 100 words per minute. Binocular testing with the Keystone Telebinocular revealed a convergence insufficiency profile with diplopia at near; no vertical heterophoria was present. The Keystone Telebinocular is useful in determining several basic visual skills. For MC, it was used to evaluate distance and near heterophorias, vertical heterophoria, and fusion abilities at distance and near. Stereopsis was evaluated with the Randot Stereotest; stereoacuity was 20 seconds of arc on

Wirt Circles, and no random dot stereopsis was present. On performance of the VO Star, MC demonstrated a slight central suppression of the left eye with an overall downward shift of the tracing. A central 40 visual field screening was performed; no abnormalities were present. Based on the results of the evaluation, 48 sessions of weekly in-office VT were recommended, with progress evaluations to be performed after every eight sessions.

MC commenced in-office VT on 5 August 2010. Therapy was initiated with a monocular phase, the purpose of which was to equalize basic visual skills—pursuits, saccades, and accommodation—between the two eyes. All activities were initially performed seated with a blank, uncluttered background in order to decrease the effects of balance and optic flow on symptoms and performance. As therapy progressed, activities were performed standing and background clutter was gradually increased. Peripheral awareness activities were incorporated to engage the ambient visual system and increase MC's ability to integrate central and peripheral awareness.

Once monocular visual skills were equalized, MC transitioned through a phase of monocular fixation in a binocular field (MFBF) into binocular therapy. As in the monocular phase, activities were introduced with MC seated, and as skills were acquired, standing, movement, and background clutter were incorporated. Performance of binocular skills, both subjective and objective, was often compared with and without MC's habitual prescription. In instances where the lenses improved performance, they were worn. If no difference was noted, the activity was performed without correction. Peripheral awareness training continued throughout all phases of therapy, and all peripheral awareness activities of this type were performed without correction to facilitate integration of central and peripheral vision without having the spectacle frame as a potential source of visual field disruption.

In the final phase of therapy, high powered yoked prisms were incorporated to create visual disruption from which MC had to recover and to which she had to habituate. Given the interaction of the visual and vestibular systems in maintaining fixation with head and background movement, optic flow was simulated during binocular activities in order to help MC with the difficulties she experienced in crowded spaces. This was achieved using movement of head, eyes, and body and through the use of an optokinetic drum in the background.

After her first month in therapy, MC's ENT was no longer recommending inner ear surgery, as her results on various balance tests—with eyes open and closed—had improved significantly. MC's visual skills improved throughout the course of therapy, and her vestibular symptoms gradually decreased. By the time of her final progress evaluation on 11 January 2011, MC reported significant improvements in her daily activities and resolution of most of her vestibular symptoms. Although reading eye movements were not specifically addressed in therapy, her performance on Visagraph recordings improved throughout, reaching a peak grade level efficiency of 13.0 with overall reading rate of 211 words per minute. Improvement was seen in every parameter measured except average duration of fixation. When asked, MC reported that she had noticed improvements in both reading comfort and comprehension.

At the final progress evaluation, MC's performance on all Keystone visual skills had normalized and diplopia was no longer present. Her binocular fusion ranges were normal in both base in and base out directions, and SILO responses were obtained on vergence testing. Worth 4-dot revealed fusion at distance and near with a lustre response at near. Phoria testing with the modified Thorington card revealed orthophoria at near. Her performance on VO Star tracing still suggested a slight central suppression of the left eye; however, the tracing was centered vertically, with no downward shift as was noted at her initial evaluation. Stereopsis was assessed again using the Randot Stereotest, and MC now demonstrated random dot stereopsis, accurately identifying all random dot targets. Stereoacuity was retained at 20 seconds of arc on Wirt Circles. It should be noted that stereopsis testing was performed with her habitual prescription, whereas initially she was tested uncorrected. Throughout the course of therapy, stereopsis was tested both with and without her spectacles; random dot stereopsis was never attained without correction. Based on results of her final progress exam, MC was dismissed from vision therapy for one month with instruction to continue maintenance therapy consisting of eccentric circles, infinity walking, and Brock string two to three days per week at home.

MC returned to the office for follow-up on 8 February 2011. Her binocular fusion ranges remained consistent, and her Keystone Visual Skills profile revealed slight exophoria at near with no diplopia. She reported that walking through a crowd still made her feel off-balance, and that she would be seeing her ENT in March for routine follow-up. She was advised to continue her home therapy with head and body movement to stimulate vestibular components. It was also recommended that she wear her habitual prescription more often, to correct her anisometropia and enhance binocularity. She returned to the office on 3 May 2011 for her final post-VT follow-up exam. She had not performed any VT-related activities since her previous visit, but reported no dizziness and a decrease in symptoms while in a crowd. She had undergone six weeks of twice weekly physical therapy, which helped with her vestibular symptoms. Keystone Visual Skills testing revealed stability from her prior exam with slight exophoria at near and no diplopia. Binocular fusion ranges were normal and remained stable from the prior exam. She was released from VT follow-up and will return to the office yearly for annual exams.

Discussion

The number of reported TBIs seen in emergency departments throughout the United States yearly is staggering—up to 1.4 million.[1] TBI, even if mild, has the potential to produce vestibular dysfunction resulting from damage to any part of the pathway. Up to 65% of patients suffering TBI will experience signs and symptoms of vestibular dysfunction during the course of their recovery.[14] Oculomotor dysfunction is also common following TBI, and may include deficits of version, vergence, and/or accommodation.[17] In addition, patients suffering closed head trauma often experience headaches, visual field loss, and/or reading difficulties.[18] Due to the close anatomical and functional relationships between the visual and vestibular systems, dysfunction in either can affect the other; dysfunction in both has the potential to be incapacitating. Studies[5,6,8,17] have indicated that both visual and vestibular symptoms secondary to TBI are amenable to improvement through rehabilitative

Table 1. Why Rehabilitative VT Leads to Alleviation of Visual-Vestibular Symptoms

Increased accommodative function and convergence through lenses or prisms because brain injured patients may lack phoria adaptation
Improvement in balance between focal and ambient processing systems
Improvement in VOR gain with improved refractive status and/or accommodation
Integration of erroneous visual information with veridical vestibular and somatosensory information creates conflicting sensory information so the patient must "choose" a single system for balance
Habituation or desensitization to visual surround movement

vision therapy. There are multiple theories in the literature as to why rehabilitative VT leads to alleviation of visual-vestibular symptoms. Summarized by Hellerstein and Winkler,[6] these theories are presented in Table 1.

When treating patients with vestibular dysfunction following TBI, keep in mind that recovery can take up to twice as long than for those with vestibular dysfunction resulting from other causes.[14] The most effective rehabilitation for these patients should not only address vestibular function but should work to maximize quality and stability of visual input as well, taking into account refractive status, binocular function, visual perceptual processing, and visual field defects (if present).[18] Therapy should be tailored to the patient's specific concerns and abilities with the ultimate goal being the optimization of visual-vestibular integration to improve quality of life.

Summary

The patient in this case suffered a closed head trauma resulting in both visual and vestibular dysfunction. The case demonstrates that a thorough visual assessment coupled with appropriate optometric intervention is essential for patients suffering TBI, especially when vestibular dysfunction is present. It also highlights the necessity for collaboration between various professionals involved in the rehabilitation process to bring about the best possible outcome for the patient.

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