VISION & BALANCE
THE OPTOMETRIST'S ROLE IN MANAGING PATIENTS WITH DIZZINESS & VESTIBULAR DYSFUNCTION

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Abstract

Balance is maintained by coordinating sensory input from the vestibular, somatosensory, and visual systems. A deficit in any of these systems may cause a patient to experience symptoms of vertigo, dizziness, or disequilibrium. In order to properly evaluate and manage patients presenting with such symptoms, it is important for optometrists to appreciate and clinically apply the important role that vision plays in maintaining balance. Specifically, optometrists are uniquely positioned to evaluate a patient's refractive condition, binocular vision, extra-ocular motility, and vestibular ocular reflexes, all of which play a role in vestibular system function. Optometric diagnostic and management protocols for individuals with vestibular dysfunctions are provided along with two case histories.

Key Words

balance, dizziness, disequilibrium, oscillopsia, vertigo, vestibular ocular reflex, vestibular rehabilitation, vestibular system, vision therapy

INTRODUCTION

Over 90 million Americans age 17 or older have experienced dizziness or a balance disorder.[1] All too often, though, balance disorders go untreated. One study reported that in an outpatient primary care medical clinic 80% of patients presented with symptoms of dizziness, but only 44% received treatment.[2] Balance problems can present with a myriad of symptoms and can be the result of an even more varied assortment of etiologies**.** Many of these patients see their optometrist as a result of the visual component of their symptoms, such as the illusion of a room spinning or diplopia. The intent of this article is to provide an overview of the vestibular system and the most common etiologies of dizziness. This will serve as the basis for the author's recommendation of what constitutes optimal optometric management of the dizzy patient. Two case studies will demonstrate the application of these recommendations.

The Vestibular System

The vestibular system serves to stabilize eye position and movements during changes in head position and is an essential mechanism for clear vision.[3] Vestibular, somatosensory, and visual input are integrated to maintain posture and balance, distinguish environmental movement from self motion, and to coordinate head and body movement. Problems arise when the three inputs are not in agreement or when one of these sources is failing. For example, if the visual system detects motion, but the somatosensory system senses that one is standing still, then a sensory conflict may arise resulting in dizziness.[3]

The vestibular apparatus is located in the membranous labyrinth of the inner ear and makes up the peripheral component of the vestibular system.[3] The semicircular canals are three ring-shaped structures that monitor rotational and/or angular head movements along the x, y, and z axes. The orientation of the semicircular canals mirrors the functional actions of the extra-ocular muscles. In addition, two otoliths process information about linear acceleration and gravity, as when moving forward while riding in a car or moving up or down in an elevator respectively. Vestibular information from the left and right inner ear is integrated to determine the direction and velocity of head movement. The receptor organ of the vestibular apparatus is the hair cell. Deformation of the hair cells by the movement of fluid in the inner ear, the endolymph, results in a change in the tonic neuronal firing rate.[3]

The vestibulocochlear nerve, pathways, and nuclei make up the central vestibular system.[4] Vestibular and auditory information are transmitted to the brain via the vestibulocochlear nerve (CN VIII) and are processed in multiple vestibular nuclei in the brainstem and cerebellum.[3] The vestibular nuclei are important centers of integration, receiving input from the vestibular nuclei of the opposite side, as well as from the cerebellum and the visual and somatosensory systems.[4]

The vestibular ocular reflex (VOR) is an important component of the vestibular system.[5] It maintains bifoveal fixation by orchestrating compensatory eye movements to head and body movements. Each semicircular canal has connections to one ipsilateral and one contralateral extra-ocular muscle. For horizontal eye

movements, the signal generated in the horizontal semicircular canal travels to multiple vestibular nuclei in the brain stem. From there it goes to the contralateral abducens nerve (CN VI) nucleus and the ipsilateral oculomotor nerve (CN III) nucleus via the medial longitudinal fasciculus (MLF).[5] The physical response of a head turn to the left is an eye turn to the right, which is generated by an excitatory signal to the right lateral rectus and left medial rectus. A second-order inhibitory signal is also sent to the right medial rectus and the left lateral rectus.

Etiologies of Dizziness

Symptoms of dizziness can result from any disturbance in the balance control systems. This can include the peripheral vestibular system, the cardiovascular system, the central nervous system, or the visual pathways.

Peripheral vestibular problems result from dysfunction of, or injury to, the vestibular apparatus in the inner ear. Examples include benign paroxysmal positional vertigo (BPPV),[6] acute labrynithitis[7], vestibular neuritis,[2] ototoxicity from prescription medications,[7] a perilymphatic fistula,[8] and Meniere's disease.[9] BPPV is the most commonly diagnosed vestibular disorder in the elderly, occurring in about 40% to 50% of patients experiencing dizziness.[2,6] It is the result of the displacement of small calcium carbonate crystals, known as otoconia, into the semicircular canal, which causes inappropriate neural signaling when the head stops moving. Benign paroxysmal positional vertigo may be the result of a head injury, but is most often idiopathic.

Central nervous system disorders that may contribute to dizziness include seizures, traumatic brain injury, and cerebral vascular accidents.[7] Tumors may also directly impinge on the central vestibular pathway. Of particular note are acoustic neuromas which can affect both auditory and vestibular functioning. An acoustic neuroma, or vestibular schwannoma, is a benign tumor of the myelin sheath that follows the course of CN VIII and is considered to be the third largest group of all intracranial tumors.[7]

Vascular problems can also cause dizziness, or more commonly, symptoms of light-headedness.[7] Orthostatic hypotension is a decrease in systolic blood pressure by approximately 20 mmHg when the patient goes from a seated position to a standing position. Symptoms are often transient and are proportional to the disruption in blood flow to the brain. Transient ischemic attacks (TIA) result from vertebrobasilar artery insufficiency and cause acute symptoms of dizziness associated with transient vision loss, limb weakness, and confusion. Patients who suffer from migraines may also experience dizziness that can last from approximately four to forty minutes and may be associated with a headache or with the headache prodrome.

Visual pathway problems that contribute to dizziness can include spectacle adaptation, anisometropia and aniseikonia, diplopia, and visual field loss.[10] A change in a patient's refractive correction may lead to temporary discomfort as a result of a sensory mismatch. Patients must adapt their perception of objects in response to a change in image size and clarity. Similarly, patients with anisometropia or aniseikonia may have sensory confusion as a result of different image sizes. Horizontal and vertical misalignments can also result in faulty visual input to the vestibular system. Patients with small intermittent deviations are often the most symptomatic as it is difficult to adapt to such deviations.[10] Finally, it is also important to remember that the brain uses information from the peripheral visual field along with vestibular information to determine sense of position in the environment. If the visual field is constricted, the patient's sense of position may be distorted, causing the patient to be disoriented or unsteady.

Table 1. Differential Diagnosis of Dizziness

Term	Symptoms	Mechanism
Vertigo	Illusion of movement - rotation, linear movement or tilt	Sudden imbalance in tonic firing rates to vestibular nucleus
Disequalibrium	Imbalance or unsteadyness while standing or walking	Loss of proprioception, visual and/or motor function; joint pain or instability; psychological factors
Light-headedness	Presyncope	Decreased blood flow to the brain
Psychological	Floating, spinning, rocking and spinning inside head	Anxiety and depression
Oscillopsia	Illusion of visual motion	Spontaneous: acquired nystagmus Head induced: severe, bilateral loss of VOR

Optometric Assessment of the Dizzy Patient

Case History

The case history is one of the most important parts of the clinical evaluation. Patients' complaints are often vague and can be confounded by other anxiety-provoked symptoms. A careful history will help isolate the cause of the dizziness and aid in the appropriate management. The key components of the history are a careful differentiation of the symptoms, delineation of the timing of attacks and associated factors, and a review of the patient's medications.

Symptoms

Patients will define almost any state involving loss of normal mental processing as dizziness.[7] For diagnostic and treatment purposes, it is important to obtain an accurate description of what the patient is experiencing. Dizziness can be broken down into five more specific sensory experiences: vertigo, disequilibrium, presyncope, psychologically-induced, and oscillopsia.[7] See Table 1.

Vertigo is an illusion of motion.[2] Patients will often report a rocking sensation or that the room is spinning. Vertigo usually results from a sensory mismatch between visual, vestibular, and somatosensory information and can be caused by rapid head movement or damage to the inner ear, cranial nerve VIII, the vestibular nucleus, or the cerebellum. Vertigo is often accompanied by excessive autonomic nervous system activity in the form of sweating, nausea, vomiting, and pallor.[2]

Patients who complain of a loss of balance without the illusion of movement suffer from disequilibrium. These patients are often very unsteady, especially when walking on uneven surfaces or on stairs. Disequilibrium results from a disruption in sensory-motor integration and is most common in the elderly secondary to widespread degeneration of the musculoskeletal, neuromuscular, and sensory systems. It can also be the result of double vision, blurred vision, impaired

Table 2. Chair-side Tests of the Vestibular Ocular Reflex		
Dynamic VA	1. Measure static distance VA 2. Measure distance VA while manually oscillating the patient's head horizontally at ~ 2Hz	Patient should be able to read the same line or one line larger than static VA If patient loses 3 lines of VA or more, then there is a likely a vestibular defect
Head Thrust	1. Patient fixates on a distant target 2. Observe the position of the eyes immediately after a small thrust of the head to the left and the right	A refixation saccade after the head thrust indicates decreased VOR
Head Shaking Nystagmus	1. Position the patient with his head down ~ 30º 2. Oscillate the head horizontally 20 times and then observe head movement	Resultant jerk nystagmus indicates a vestibular imbalance Works best when fixation is blocked

vestibular function, joint pain, or peripheral neuropathy. These patients will often support themselves on walls or tables and may require a cane or walker.[10]

Presyncope is a feeling of lightheadedness. The patient feels faint, but does not experience a loss of consciousness. These are often acute attacks, lasting seconds, and can be accompanied by a buzzing in the head, rubbery leg feeling, the sensation of tunnel vision, as well as nausea, pallor, and sweating. This is most often a manifestation of cardiac or vascular disease and results from restriction of blood flow to the brain.[10]

The most challenging type of dizziness to differentiate is psychologically-induced dizziness.[10] In a study done at a clinic specializing in dizziness, between one in three and one in five patients with dizziness was diagnosed with a psychogenic etiology.[10] Patients will describe a sensation of floating or swimming in their head often associated with acute bouts of anxiety lasting seconds to minutes, also known as panic attacks. Patients may also experience chest pain, lightheadedness, extreme irrational fear, or an intense need to escape. These symptoms are often dismissed by physicians as "being all in the patient's head." However, since they may be the presenting sign of a psychological disorder, they warrant further evaluation.

Finally, oscillopsia is the illusion of movement of stationary objects.[5] Patients may say that objects have a shimmery appearance or simply that images are blurred. This usually occurs in patients with an acquired, large amplitude nystagmus or a severely impaired vestibular ocular reflex. In contrast, oscillopsia will not be present in patients with congenital nystagmus.[5]

The timing of a patient's symptoms can also be very diagnostic.[7] Acute symptoms are those that last less than three days and are associated with sudden changes that may include a brainstem infarct or infection such as labrynithitis. Chronic symptoms are persistent over more than three days and are indicative of a central nervous system problem, a unilateral vestibular defect, or bilateral hypofunction of the vestibular system. Chronic symptoms are also common in psychological disorders. Episodic attacks can last seconds, minutes, or hours. Those lasting seconds are usually associated with BPPV or orthostatic hypotension. If the attack lasts for minutes at a time, then TIA or migraine is the most likely diagnosis. Episodes lasting hours are most frequently associated with Meniere's disease, a disorder characterized by dysregulation of the fluid pressure in the inner ear.[9]

It is also important to determine under what circumstances the patient experiences dizziness. Symptoms may be spontaneous or associated with movement.[7] For example, a patient who experiences dizziness upon standing may have orthostatic hypotension. If symptoms are brought on by moving the head backwards or looking up at something, then BPPV should be ruled out. Some patients are hypersensitive to visual information and may note symptoms in crowded or visually-busy environments. Finally, patients with compromised vestibular systems are often visually-dependent, meaning they rely predominantly on visual cues for balance and proprioception, and may report that symptoms are worse in the dark or when the eyes are closed.

Although a review of current medications is a routine part of a clinical exam, it is especially critical when examining the dizzy patient. Dizziness and vertigo are potential adverse effects of many prescription medications. One study suggested that of the more than 4000 drugs listed on a computerized version of the *Physician's Desk Reference*, over 25% had dizziness listed as a possible side effect.[8] Most notable are antihypertensive medications, such as hydrochlorothiazide, atenolol, and verapamil, and antidepressants, such as amitryptyline and sertraline.[7] Other medications may either permanently or reversibly damage the inner ear. Most well-known in this category is gentamicin. However, streptomycin, tobramycin, amiodarone, quinine, and furosemide are all potentially ototoxic.[7]

Clinical Testing

The standard optometric evaluation should be conducted.[11] In this regard, a dilated fundus exam should be performed unless is it contraindicated. A thorough refractive analysis is extremely important, since even small, uncorrected refractive errors may be potentially troublesome in patients with a compromised vestibular system.[12] This is due to their increased reliance on visual cues.

A detailed oculo-motor evaluation should also be performed. This includes the assessment of the accuracy and stability of monocular and binocular fixation. A 20D lens can be used to magnify the eye while the patient fixates on a small near target. The magnification allows the optometrist to watch for small refixation movements or intrusion saccades. Careful **c**over testing in all positions of gaze and phorometic testing to assess the patient's ocular alignment should also be performed. It should be kept in mind that even small vertical and horizontal misalignments, as low as one or two prism diopters, may need to be compensated for because of the reliance on intact visual function in the dizzy patient.

If a patient presents with unsteady monocular or binocular fixation, nystagmus, or poor smooth pursuit and/or saccadic function, three tests of VOR function should be performed.[7] See Table 2. These tests require no special equipment and only minimal chair-time. The first test is dynamic visual acuity. To perform this test, the patient's head is manually oscillated horizontally at a rate of about two hertz, or two cycles per second. While the head is rotated, the patient is asked to read the

distance visual acuity chart. He or she should be able to read the same line or one larger than their static visual acuity. If the patient loses three or more lines of visual acuity and there is no other ocular motility defect, then there is likely a lesion in the inner ear, vestibular pathway, or brainstem. This test is contraindicated in patients with neck or spinal problems that impair their range of motion.

The next test is the head thrust. The patient is asked to fixate on a distant target. The patient's eyes are then carefully observed immediately following a small thrust of the head to the left and to the right. A refixation saccade means the VOR failed to hold eyes steady in place and the eyes drifted along with the head during the thrust.

The final test of the VOR is the head-shaking nystagmus test. The patient's head is initially positioned downward approximately thirty degrees. Then, the patient's head is oscillated horizontally approximately twenty times. When the patient's head is stabilized, the eyes are observed for any nystagmus. In this case, a resultant jerk nystagmus, or a nystagmus with a slow drift in one direction and a fast recovery in the opposite direction, indicates a vestibular imbalance or a weakness in the VOR. This test is best performed when fixation is blocked. This can be achieved by using high-plus lenses on the patient. The lenses will also magnify the eyes making any nystagmus easier to observe.

Additional testing can include determination of the patient's perception of his/her body midline. When this is determined to be misplaced, the use of yoked prisms has been recommended to improve symptomotology.[13] Further, a protocol for determining the efficacy of tinted lenses to reduce symptoms that include dizziness should be considered.[14] Assessment of the patient's visual perceptual processing has also been recommended.[12]

The optometrist should consider referral for other vestibular diagnostic testing in the event these evaluations have not been performed. These tests are conducted by audiologists, vestibular rehabilitation therapists, physical therapists, or neurologists. Bithermal caloric testing is considered to be the most specific test of vestibular function.[7] The ear canal is irrigated with warm and cold air or water which creates a temperature gradient and a resultant nystagmus. Caloric testing is used to differentiate peripheral vestibular lesions from central vestibular lesions.

Videonystagmography utilizes special video goggles to record and quantify eye movements during vestibular and visual tracking exercises. Computerized dynamic posturography is used to measure changes in posture when visual and somatosensory cues are altered or absent. Neuroimaging is warranted in cases where a central nervous system lesion is suspected.[3] Finally, because of the close relationship between the auditory and vestibular system, a hearing test is also often indicated.

Optometric Management of the Dizzy Patient

Optimizing the visual system input is essential in the presence of a vestibular weakness. Any visual system disorder, from ocular health problems such as dry eye to visual impairment or visual field loss, needs to be addressed. It is particularly important to provide the appropriate optical correction. I have found that patients with vestibular problems frequently perform better with separate distance and near spectacles. As a general rule, progressive lenses, bifocals, and multifocal or monovision contact lenses are contraindicated. Additionally, the judicious use of prisms to correct vertical misalignments and to achieve binocular fusional stability is critical.

Cohen cites four main treatment goals for vision therapy of vestibular dysfunction.[14] The first goal is to enhance the stability of the visual system. This is achieved by stabilizing fixation, improving motor control and motor planning, and enhancing peripheral awareness. Secondly, it is important to develop a stable binocular vision system which includes accommodative and vergence flexibility, rapid recovery of fusion, and central fusional vergence stability. The binocular vision system should be challenged in the context of a dynamic visual environment by incorporating head and body movement and decreasing somatosensory cues. Third, the speed and facility of visual perceptual processing should be optimized. This includes training in the areas of speed and span of perception, figure-ground analysis, visual closure, and visual sequencing. Finally, visual field deficits must be addressed by means of yoked prism systems, visual rehabilitation, and/or low vision rehabilitation.

Standard visual therapy techniques are used with the dizzy patient. However, I have found that the addition of dynamic tasks that involve balance and head and body movements is desirable. For example, patients should do near point of convergence techniques while walking, or perform smooth pursuit techniques while balancing a sponge on their heads or standing on an uneven surface. Prisms can also be used to create a sensory mismatch and further challenge the integration of sensory information for balance control. It should be kept in mind that the dizzy patient may experience significant discomfort with these techniques and will need to be coached extensively at least through the initial stages of therapy.

In at least some instances, the optometrist should make a referral based on the patient's symptoms and clinical findings and what has been determined to be the most likely etiology of the dizziness. For example, if orthostatic hypotension is suspected, the patient should be referred to his/her cardiologist. If a central nervous system etiology is suspected, the patient should be referred for an MRI and follow-up with a neurologist. Primary care physicians should be consulted in cases of possible adverse reactions to prescription medications.

The optometrist might also deem it beneficial to refer the patient to be evaluated for a vestibular rehabilitation program. These are designed and supervised by specially trained physical or occupational therapists.[15] These are exercise-based programs that seek to promote central nervous system compensation for vestibular dysfunction. The first goal of vestibular rehabilitation is to improve gaze stability by means of VOR adaptation and fixation exercises. This can include focusing on a distance target while moving the head and walking. Habituation exercises involve persistent exposure to provoking stimuli with the goal of long-term sensory adaption. This can result in reduced sensitivity to symptoms. Postural and gait stability can be addressed by incorporating exercises that improve coordination of muscle responses as well as the organization of sensory information. Furthermore, cardiovascular conditioning is an important part of vestibular rehabilitation. Patients should be on a supervised program of brisk walking, stair climbing, or swimming for improved vascular health.

There is clinical evidence that demonstrates the effectiveness of vestibular rehabilitation. In a study by Venosa and Bittar, 87 patients with a clinical history

of acute vertigo were given VOR adaptation exercises to do three days a week for three weeks.[16] The first exercise was a head rotation to the left and to the right while fixating on a stationary target. The second exercise involved fixating on a hand-held target that was moved in the opposite direction of head rotation. Each of these exercises was to be performed for one minute in the horizontal and vertical directions. It was reported that the patients' symptoms improved faster with therapy, their need for medications was less, and their neuro-otologic testing normalized faster than control patients who were given stationary fixation exercises.

CASE REVIEWS

Case 1

Patient RG is a 52-year-old white male who presented to the clinic with a chief complaint of dizziness and unsteady gait for the last five years. He stated that his symptoms had worsened over the last six months and that he was afraid to leave his house as a result of his dizziness. He described his dizziness as if he was spinning while the room remained still. The patient walked into the clinic with a wide, swaying gait and had to hold onto the walls for balance.

Patient RG was treated for hypertension, hyperlipidemia, and peripheral neuropathy and was reportedly taking lisinopril, felodipine, and vardenafil. He denied any history of head trauma, tinnitus, or migraines. He also had no specific visual complaints of diplopia, eye strain, or blurred vision.

Optometric examination revealed best corrected visual acuity of 20/20 in each eye with a myopic refractive correction. The patient had end-gaze horizontal nystagmus which was greater in left gaze, however his monocular and binocular fixation were stable. On cover testing, the patient was found to have a slight exophoria at distance and a moderate exophoria at near. His near point of convergence was five inches and was reduced to ten inches when looking through red-green lenses. Phorometric and free space testing revealed low fusional vergence ranges at distance and near. On dynamic visual acuity testing, which was performed to assess the VOR, the patient was unable to read any line on the acuity chart while his head was oscillated horizontally. Midline-shift testing[12] revealed an egocentric shift to the left. The patient's performance improved significantly with four prism diopters yoked base left prism. Slit lamp and dilated fundus exam were both unremarkable. The patient was diagnosed with midline shift, convergence insufficiency, and oculo-motor dysfunction. He reported increased visual comfort with blue tinted lenses.[13] He was enrolled in vision therapy program to optimize all visual functioning, and was also evaluated by a vestibular rehabilitation specialist and an audiologist.

Patient RG underwent videonystagmography testing and bithermal caloric testing which were both suggestive of a central nervous system etiology of this dizziness. Loss of sensation in his lower extremities was also thought to be contributing to his disequilibrium. He was enrolled in a physical therapy program and advised to change his footwear from cowboy boots to sneakers. After five weeks of physical therapy, the patient demonstrated improved balance and gait and was started on a home-based exercise program.

After 27 sessions of vision therapy the patient was re-evaluated and dismissed on a home vision therapy program. He had been prescribed single vision distance spectacles with a light blue tint and four prism diopters yoked base left prism. This dramatically improved his gait and postural stability. At the time of dismissal he still had a convergence insufficiency but had greater fusional vergence stability and flexibility. Most notably, he could walk down the hall slowly without bracing himself on the walls.

Case 2

Patient JG is a 58-year-old white male that presented to the clinic with a chief complaint of gradually progressive dizziness and unsteadiness over the last year. He reported that his symptoms were worse when he turned his head, especially when driving and trying to look at signs. He complained of feeling like he was moving while the world was still. He also complained of being unable to read comfortably because he felt that everything was moving and he could not maintain his focus. He denied any hearing loss, tinnitus, or head trauma.

Patient JG was being treated for hypertension, hyperlipidemia, peripheral vascular disease, depression, and benign prostatic hypertrophy. He also had a history of lung cancer and had undergone a left lower lobectomy. He was taking eleven different medications for these conditions, the vast majority of which had a potential side effect of dizziness or vertigo. Most notable were amlodipine (*Norvasc*) for hypertension; diazepam (*Valium*) and fluoxetine (*Prozac*) for depression and anxiety; and doxazosin (*Cardura*), finasteride, and oxybutynin (*Ditropan XL*) for benign prostatic hypertrophy.

On clinical evaluation he was found to have normal, symmetric acuity with a low hyperopic refractive error. He was mildly exophoric at near but had a normal near point of convergence. He exhibited end-gaze horizontal nystagmus in both left and right gazes and undershot his saccades by about 30% in all gazes.

The results of vestibular ocular reflex testing were consistent with vestibular dysfunction. He lost three lines of visual acuity on dynamic visual acuity testing and had difficulty maintaining focus during the head thrust test. Orthostatic hypotension was ruled out since there was no change in seated and standing blood pressures. Bithermal caloric testing was also performed and suggested a central nervous system etiology for his dizziness. It was determined that patient JG's vertigo and disequilibrium were likely related to a central nervous system disorder. He was also diagnosed with vestibular ocular reflex dysfunction, binocular instability, and oculo-motor dysfunction. He was referred to neurology for evaluation and imaging to rule out a central nervous system lesion, especially metastatic lesions given his history of lung cancer. He was started on a vision therapy program with emphasis on oculo-motor skills and VOR adaptation. Initially, separate distance and near spectacles were ordered, but the patient complained of more significant dizziness in switching between the glasses than he experienced with his bifocals. Thus, the patient continued to wear his bifocal prescription. Patient JG's biggest obstacle in vision therapy was overcoming his nausea in order to consistently perform the recommended activities. Daily activities were started with the patient seated and then progressed to standing and then to the use of yoked prism as tolerated by the patient. Subjectively, his progress was slow. Nonetheless, he continued to improve both in his time at task and in the number of exercises tolerated each week.

CONCLUSION

Dizziness is an umbrella term used to describe variable sensory experiences such as vertigo, disequilibrium, presyncope, anxiety, and oscillopsia. A detailed case

history is critical in the diagnosis and management of the dizzy patient. Based on clinical observations, the patient should be referred to neurology, cardiology, primary care, or psychology for further evaluation and diagnosis. Furthermore, many patients can benefit from vestibular rehabilitation in conjunction with vision therapy.

Visual input makes up one-third of the sensory information used for balance and equilibrium. Therefore, optometrists play a key role in the management of balance disorders. Optimal correction of refractive errors, binocular vision problems, and visual field loss with optical lenses and prisms is the first step in optimizing visual input into the vestibular system. Optometrists trained in functional vision rehabilitation can assist patients by enhancing the stability of the visual system, improving the stability and flexibility of binocular vision, optimizing visual perceptual processing and motor planning skills, and managing visual field loss with prisms and low vision devices. Finally, communication with neurologists, audiologists, physical therapists, and occupational therapists is essential to optimize the patient's rehabilitation program.

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