

Chronic Dizziness

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Opinion statement

The mainstay of treatment is a specialized form of physical therapy that intentionally induces symptoms of dizziness with head movements, moving visual targets, and changes in position. Patients must be motivated to experience discomfort during the early stages of rehabilitation, doing the types of activities that they likely have been avoiding. Time must be spent convincing the patient of the necessity to deliberately provoke dizziness in order for the exercises to work. It is important to not overmedicate with vestibular suppressants, and to treat any co-existing mood or anxiety disorder, as these interfere with the adaptive plasticity that is the basis of successful rehabilitation.

Introduction

This article addresses symptoms of dizziness that are present chronically or are provoked by certain environments or activities on a regular basis. These types of complaints frequently co-exist with true spontaneous bouts of vertigo. In this case, a specific etiology for the paroxysmal episodes of vertigo must be sought and treated appropriately. Benign paroxysmal positional vertigo (BPPV), which is provoked by pitch-plane head movements, was dealt with previously [1•, Class III]. Other common conditions causing recurrent spontaneous vertigo include Ménière's syndrome, vertebrobasilar transient ischemic attacks, and migraine headaches. Vestibular neuronitis (sudden onset vertigo without any associated hearing loss) and true labyrinthitis (sudden onset vertigo associated with hearing loss) are most always a monophasic illness, but frequently lead to a chronic impairment as patients fail to fully compensate for a partial or full residual unilateral peripheral loss, and continue to complain of dizziness long after the initial onset of symptoms.

A commonality in chronic disorders of "dizziness" originating from an initial insult to the peripheral or central vestibular system is the lack of a completed central vestibular compensation process. The reason for such a lack of full compensation in a patient most often takes one or more of the following forms: 1) the patient is suffering from an unstable central or peripheral vestibular system process causing repeated changes in the

functional status of the system, *eg*, Ménière's disease, recurrent BPPV, or degenerative condition of the peripheral or central system such as hereditary or autoimmune. 2) The patient develops maladaptive behaviors of avoidance in movements following a pathologic insult that creates a stable locus of lesion and, hence stalls the compensation process in a partial state, *eg*, the intermittent symptoms with head movements following a vestibular neuronitis event. 3) The patient for whom a second disease process causes repeated interference with the compensation process following an initial stable insult, *eg*, development of a secondary anxiety disorder with or without panic, or the patient with migraine-associated dizziness. 4) The patient in whom the compensation process is stalled secondary to the chronic use of medications that were initiated at the onset of the insult for symptom control, but have not been appropriately withdrawn. These are typically medications thought of as vestibular suppressants, *eg*, meclizine or any of the benzodiazepines.

The second major cause for chronic disorders of dizziness involve other disorders of a chronic nature that can manifest their symptoms in the form of vertigo or lightheadedness or imbalance. The two most common of these would be migraine-associated vertigo and a primary anxiety disorder with or without panic.

In consideration of difficulties with anxiety in the primary or secondary form, the symptoms of vertigo,

imbalance, and lightheadedness are most commonly provoked by naturally occurring stimuli of visual motion activity. Several diagnostic entities or descriptors have been offered—space motion phobia, phobic postural vertigo, and somatoform disorder—all dealing with the anxiety and defensive reactions to vestibular sensations encountered in normal activities that, in the secondary developed anxiety disorder, perhaps trigger conditioned responses to a previous vestibular crisis. Frequently, the description of a patient's symptoms evolves from an initial crisis involving vertigo, ataxia, and nausea and vomiting to more mild lightheadedness and imbalance, though they may still use the terms *spinning* or *dizziness*. Rather than having a direct vestibular cause, these chronic complaints are more likely due to a number of other mechanisms—anxiety, avoidance, hyperventilation, orthostasis, and medication side effects being among the most common.

Mal de débarquement is the persistent sensation of rocking, swaying, and imbalance usually following a sea voyage, but may occur following any boat travel or even air travel. It almost always occurs in women, with onset during the age of 40s and 50s. Although symptoms generally decline over time, the duration of the condition can last for years. Treatment with vestibular suppressants (meclizine, scopolamine) is ineffective. Benzodiazepines appear to work best, with a modest benefit from vestibular rehabilitation noted.

PREDISPOSING FACTORS

People are normally subjected to illusions of motion on a regular basis—a typical example is sitting stationary car when a large vehicle to the side begins to move forward. The perception of backward self-motion initiates a rapid increase in pressure on the brake pedal, and usually some

amount of anxiety or momentary panic. In the patient who has had a vestibular crisis during which they may have had severe vertigo, nausea, vomiting, and ataxia in the midst of a public space with no one nearby to help, any reminder of such an experience (such as the benign visual illusion described) can elicit a defensive reaction or even a full-blown panic attack in a susceptible individual. This is frequently associated with hyperventilation, which ensures a vicious cycle of increasing symptoms as they try to control symptoms with deep sighs or deliberate breathing. The resulting hypocarbic state causes cerebrovascular constriction and hypoperfusion, increasing lightheadedness and presyncopal sensations. These are all considered *dizziness* by the patient, and not necessarily distinguished from their initial vestibular event. They, therefore, report recurrent spontaneous events of dizziness, though, in reality, they had a single vertiginous event that triggered an anxiety or panic disorder manifesting in environments likely to present a challenge to the spatial orientation system. Treatment of an underlying anxiety disorder is often necessary in managing patients with chronic dizziness. Behavioral and cognitive therapies may be effective, as well as pharmacologic treatment.

Migraine-associated vertigo is becoming increasingly recognized. The vestibular symptoms can take many forms, from vague dizziness to spinning vertigo. The duration of symptoms can also range from brief spells to episodes lasting for days. Headache often does not accompany the dizziness, although a clear history of vascular headache should be present in order to entertain this etiology. Motion sickness susceptibility, food triggers for headache, caffeine-withdrawal headache, relief with sleep, and family history of migraine all suggest the diagnosis of migraine-associated vertigo, though it should remain a diagnosis of exclusion.

Treatment

- Management options available for the chronic dizziness patient presented in this paper presuppose that the evaluative process has identified any likely diagnoses that could be responsible for spontaneous fluctuations in vestibular function. The treatments described here are useful for symptoms that are present on a daily basis or are reliably reproduced by certain activities or environments.

Interventional procedures

- Actually, what is discussed under this heading is the patient interview, because this is frequently a crucial intervention. Because patients often present with voluminous records documenting repeatedly normal neurologic examinations by colleagues, imaging studies, laboratory tests, audiograms, and so forth, it may be clear to the experienced provider that the patient is just dizzy. Many patients have been told previously by other providers that “We don't know what's wrong with you but it is nothing

serious," "It will go away on its own," or "It is all 'in your head'" or "Nothing to worry about." In the case of previous patients, these have proven either false or were not accepted. The history as obtained from the patient, though often time consuming, is in fact therapeutic. The willingness of the patient to accept the treatment plan necessary for recovery is generally proportional to the time spent in the encounter.

- Although little specific diagnostic information may be gained, it is often helpful to hear an account of the patient's perception of his or her illness prior to pursuing more specific questions. One can often gain a sense of the degree of functional disability the vestibular symptoms have produced. The psychosocial impact of the illness may also become clear in the patient's initial comments assisting in suspicion of psychologic disorders playing a role in ongoing symptoms. The patient may become tearful when describing their frustration at the duration of their condition, or they may be observed to take frequent sighs when describing provocative stimuli. Sometimes patients will volunteer that their symptoms are trivial or have resolved completely, but they simply want to make sure that they have not suffered a stroke or developed a brain tumor. If the patient is not permitted to share this information freely, important aspects of the individual's care may be overlooked.

Pharmacologic treatment

- There is no role for chronic treatment of dizziness or vertigo with meclizine, scopolamine, or other antihistamine or anticholinergic medications. These are appropriately used acutely in the first days to a week after a vestibular crisis, and on a daily basis when significant nausea or spontaneous spells of vertigo are expected. Patients may wish to keep some on hand as "security," but habitual use of these agents generally is not helpful and may be counterproductive to the central compensation process.
- Additional history regarding current or prior use of medications should be elicited. Many patients are under the mistaken impression that vestibular suppressants will prevent spells of vertigo and take them habitually. Because oversedation from these centrally acting drugs may retard central nervous system compensation for vestibular lesions, one should consider tapering or discontinuing these medications whenever possible. Medications that must be continued should be directed toward particular symptoms that specifically interfere with the patient's recovery process.
- Use of specific medications for disorders related to migraine headache [2, Class III; 3, Class I] is often successful in treating chronic dizziness in patients with vascular headache. The treatment is similar to headache patients in general; a migraine diet should be given to identify triggers, triptans may be effective abortants, and prophylactic agents (beta-blockers, tricyclic antidepressants, and acetazolamide) are usually effective.
- Recently, selective serotonin reuptake inhibitors (SSRIs) have been found beneficial in an open-label series of patients with chronic dizziness, regardless of whether a co-existing psychiatric diagnosis is present. Patients with complaint of exercise-induced dizziness, often young, athletic individuals, may be particularly helped with this treatment [4, Class II]. Use of SSRIs has been shown to be effective in a special group of patients presenting with exertion induced vague complaints of dizziness and symptoms of autonomic dysregulation [5, Class II].
- Because otolaryngologists, who are most accustomed to thinking about Ménière's syndrome, see many patients, treatment is frequently initiated with a diuretic medication or a sodium-restricted diet. Both of these

interventions (often effective when the diagnosis is correct) contribute to orthostatic intolerance, and again patients interpret their presyncopal sensations as another attack of dizziness or vertigo. If the diagnosis of Ménière's is not confirmed over time (there are no test specific to this disorder) with the development of the full profile involving a progressive, yet fluctuant sensorineural hearing loss, appropriate withdrawal of the diuretic in a diagnostic trial format maybe effective in reducing orthostatic intolerance complaints.

Surgery

- There is very little role for surgical intervention in the patient with chronic dizziness alone. When there are ongoing, superimposed true spontaneous events of tinnitus, aural fullness, hearing loss, and vertigo, transtympanic gentamicin injections or vestibular nerve section may be appropriate treatments for Ménière's syndrome.
- An uncommon cause of chronic dizziness is superior semicircular canal dehiscence, in which patients may experience a continuous sense of unsteadiness due to a defect in the temporal bone overlying this canal. On specific questioning, the patient usually notes exacerbation of symptoms with Valsalva maneuver or with sound (Tullio phenomenon). On examination, a mixed torsional vertical nystagmus may be elicited during attempted forced exhalation against pinched nostrils. Thin section CT scan of the temporal bone is diagnostic. A craniotomy can be performed to repair this defect with good result.
- Perilymphatic fistula should be entertained only with a good history of antecedent barotrauma or head injury, with associated hearing loss. Microvascular decompression of the eighth nerve should be considered only if there is a clear response to carbamazepine. Before either of these procedures is contemplated, an adequate course of physical therapy as outlined, and evaluation for any possible anxiety component, should be undertaken.

Diet and lifestyle

- Avoidance of migraine triggers (habitual caffeine or acetaminophen, chocolate, red wine, and so forth).
- Lower sodium and increase fresh water if Ménière's is suspected.
- Increased sodium and water if orthostatic symptoms are described.
- Root of ginger for nausea.

Physical therapy and exercise

- The primary treatment modality for the majority of chronic dizzy patients involves the use of vestibular and balance rehabilitation therapy (VBRT). This is a specific form of physical therapy exercises customized to the patient's specific complaints and the objective functional findings during the therapy evaluation. Maladaptive postural adjustments such as en bloc turning and avoidance of head motion with resulting neck discomfort and even headache are handled with the use of VBRT. Patients must be motivated to experience discomfort during the early stages of rehabilitation, with the expectation that they will benefit from markedly reduced (or absent) symptoms later. Patients must be convinced of the necessity to deliberately provoke dizziness in order for the exercises to work. The overall goal of the therapy, which should be performed at least once per day, is to

induce to completion the central compensation process. In many patients, this will involve activities that provoke symptoms of vertigo, often with head movements, moving visual targets or both, progressing to performing these while upright and walking. Exercises serve a number of specific goals—habituation to symptoms associated with unilateral peripheral vestibular loss, prophylaxis against return of BPPV and early warning if BPPV should return, confidence building, and maintenance of compensation. Patients are told “the brain only solves problems with which it is confronted” in order to justify their being asked to perform the very activities they have been assiduously avoiding because of symptom generation.

- Vestibular and balance rehabilitation therapy is a symptom-driven program for determination of who is an appropriate candidate. Therefore, it is during the history that the first possible indications for the use of this management technique would be developed. For the most part, other than the evaluations of posturography, the laboratory tests of electronystagmography (ENG) and rotational chair do not provide indications for the use of this treatment option. There are situations when the symptom complaints are questionable for use of the program, yet findings on postural control studies suggest functional deficits that can be addressed with the VBRT techniques and its use would be appropriate.
- In the case of bilateral vestibular paresis, the rotary chair is the only test that can provide information about the extent of the bilateral labyrinthine involvement. With those chair results, VBRT programs can be altered for more realistic goals. Findings from ENG of a unilateral reduced vestibular response to caloric irrigation, or abnormal timing relationship (phase) between eye and head movement from a rotary chair study support the use of adaptation exercises to improve vestibulo-ocular reflex gain during the VBRT. It is unlikely that repeat use of ENG or rotary chair following use of VBRT would be called for, because the use of these tools in determining compensation status is very limited and the primary focus in a VBRT program is to enhance the compensation process in many dimensions. However, repeated use of various postural control [6,7, Class II] and dynamic visual acuity assessments would be useful as a monitor or final outcome measure of the effectiveness of a VBRT program, because these studies are primarily functional in nature. The patients that are the most appropriate for VBRT on first review are suggested from any one of the following: patients with symptoms provoked by head or visual motion; symptoms that are continuous with motion exacerbation; evaluations revealing balance or gait dysfunction with or without either of the traits listed.
- The patients that are most probably not appropriate for use of VBRT as the initial management technique have the following characteristics to their symptoms: symptoms of only spontaneous events that are more frequent than once every 6 to 8 weeks and last longer than 15 minutes at a time; no provocative activity or balance dysfunction can be realized during the therapy evaluation, therefore, nothing is found on which to base exercise activities; progressive central lesions involving gait and balance have not been shown to respond to therapy for balance and gait. These patients may benefit from exercises to reduce eye and head movement sensitivity and safety with ambulation-oriented goals.
- The general principles of designing vestibular rehabilitation programs involve exposing the patient to the stimuli that provoke vertigo, cause slippage of the visual signal on the retina and challenging areas of deficiency in postural control and ambulation. First, the therapist must identify those activities or environmental situations that provoke symptoms. Second, the patient’s functional deficits regarding balance and gait must be identified. These may be caused by the vestibular symptoms,

lesions in nonvestibular regions or by maladaptive behavior that has developed in response to the symptoms. Lastly, it is desirable to challenge the sedentary lifestyle that the vestibular disorder patient often adopts. An active lifestyle including regular exercise that accounts for age and other health constraints will serve as a maintenance program once active therapy is completed.

- An additional tool, Tai Chi has demonstrated efficacy in treating complaints of imbalance when standing still in patients of any age [8,9, Class I].

Other treatments

- If available, the various resources of a balance center may be of assistance in sorting out complicated patient presentations. Such centers optimally include providers from neurology, otorhinolaryngology, physical therapy or physiatry, audiology, and psychiatry. These centers provide for extensive vestibular and balance testing laboratories. Patients are told that their diagnosis and treatment may be challenging, and that help from many quarters may be needed for best care. At the same time, however, the fact that many other patients with the same types of complaints have been effectively treated is reinforced.
- It is important that both the clinician and patient understand the place that laboratory testing has in the management of the chronic dizzy patient. The evaluation of the dizzy patient should proceed guided by what information is needed to make initial and subsequent management decisions. When various tests are reviewed, and correlated with high level activities of daily living, virtually no significant relationships exist for the chronic dizzy patient. Tests considered extent and site-of-lesion studies, ENG [10, Class III], rotational chair [11, Class III], and specific protocols in postural control assessment [12, Class III], give results that are unable to be used to predict symptom type, magnitude, or the level of disability of an individual patient. Conversely, patient complaints can not be used to predict the outcomes of these tests.
- In a limited manner, more functionally oriented evaluation tools—computerized dynamic posturography (CDP) and dynamic visual acuity testing [13, Class II] provide for some correlation between results, patient symptoms, and functional limitations. Add to the testing specific or general health inventories like the Dizziness Handicap Inventory and predictive assessment of disability is improved but remains significantly limited. It is hypothesized that the reason for this dichotomy in test results versus functional disability and symptom complaints is the inability of the tests to adequately characterize the status of the central vestibular compensation process [15,16, Class II]. The limited laboratory tools available can assess compensation to a unilateral peripheral vestibular loss by measuring any spontaneous and positional nystagmus in darkness certain parameters of the caloric irrigation test, the rotational chair test, and tests of postural control [10–12, Class III]. At the bedside, intolerance of rapid head movement to the point of trepidation with the vestibular examination indicates inadequate compensation or the development of a phobic or defensive, maladaptive strategy.
- It is the exception, not the rule, that vestibular and balance laboratory tests provide results that would drive management of the dizzy patient. It would be extremely rare that these studies return a diagnosis. Therefore, the routine use of these tools to determine how to proceed with the management of a dizzy patient is a false line of reasoning and not productive in the majority of the patients. What, then, is the roll of the laboratory studies of ENG, rotary chair, and CDP? Determination of extent and site-of-lesion within the peripheral and central vestibular system and the functional

limitations in static and dynamic postural control (this may or may not be related directly to gait abnormalities). The use of this information is in the confirmation of the suspected site-of-lesion and diagnosis derived from the patient history and direct physical examination including aspects of the direct office vestibular evaluation. This does not imply a prioritized order to the testing versus the office visit, as with chronic dizzy patients it can be very useful to triage them to at least core laboratory evaluations (full history, ENG, and screening postural control testing) prior to the office visit.

- To summarize the discussion, the following are required elements to make management decisions for the chronic dizzy patient; detailed neurologic history, office vestibular and physical examination, and formal audiometric testing given the inescapable anatomic relationship between the auditory and vestibular peripheral systems. The following are considered important, however, less likely to directly drive the management in the typical case: laboratory vestibular and balance function studies, neuroradiographic evaluations, and serologic tests. It is important to realize that there will be patients for whom unexpected findings on any one of these latter studies will either alter the complete course of the management or add dimensions to the management not originally considered. But for the majority of chronic patients the vestibular and balance tests will be confirmatory in nature.

Other readings

- Multiple sources are available for the reader interested in further detailed discussions of the use of the neurologic history for the dizzy patient [15,17,18–23].
- A full discussion of the office vestibular examination is beyond the scope of this article and the reader is referred to other resources that provide for this discussion [13, Class II; 20,24, Class III].
- The reader is referred to two recent summary articles that discuss the area of psychologic impact and the management techniques available to deal with this important area [25,26, Class III].
- Baloh and Halmagyi [19] and Cass and Furman [6, Class II] contain detailed descriptions of specific disorders by history and test findings and the office vestibular examination.
- Extensive literature is available to discuss protocols, techniques, and the significant efficacy of vestibular rehabilitation therapy to which the interested reader is referred [27,28,29, Class III; 30, Class I; 31, Class III].

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