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**Introduction**

There are three basic inputs – vision, proprioception, and the vestibular semicircular canals and otolith organs – that are used by the brain to provide ocular stability, gait control, and balance during active and passive body movements. A disorder of the vestibular system is a major disruptor of these critical functions and a significant source of spatial disorientation symptoms. A patient's complaint of dizziness can be caused by a variety of factors including pre-syncopal lightheadedness, dysequilibrium, visual distortion and multi-sensory dysfunction and must be differentiated clinically from vertigo or dizziness of vestibular origin. The differential diagnosis of vertigo has remained stable over the past several decades, but management strategies continue to improve. The goal of this discussion is to review the most common types of vestibular disorders encountered by the otolaryngologist and discuss the medical management and rehabilitation therapy strategies currently at use.

**Pathophysiology**

The vestibular labyrinth is responsible for detecting both linear and angular head movements. Each is composed of three semicircular canals (SCC) and two otolithic organs. The SCC detects rotational movement or angular accelerations and the otolith organs detect linear acceleration. The sensory hair cells within the membranous labyrinth detect specific movements during hyper or depolarization of the stereocilia and kinocilia extending from the cell body. Within the SCC, the hair cells are organized under a gelatin film called the cupula. In the utricle and saccule, the hair cells are attached to a layer of otoconia on the macula. The otoconia remain stationary relative to linear head accelerations, which causes deflection and stimulation of the underlying hair cells. The hair cells project one or more afferent nerve endings onto the body of the cell. In addition there can be a direct and indirect efferent innervation of the hair cell body. The fiber bundles from each of the five sensory epithelia join to make two branches of the vestibular nerve: the superior and inferior branches. The afferent nerve fibers are bipolar and have one synapse at the hair cell and the other in the vestibular nucleus. The cell bodies are located in between within Scarpa's ganglion. Peripheral vestibular disorders are presumed to be restricted anatomically to the aforementioned structures.

In addition to a normally functioning vestibular system, balance requires input from the visual (vestibulo-ocular) and proprioceptive (vestibulospinal) systems. Vestibular input is balanced and compared to these inputs. Central causes of vestibular dysfunction compromise the central vestibular circuits that mediate vestibular influences on posture, control of gaze, and autonomic functions. Any insult that disrupts the calibration or balance between the two peripheral vestibular systems or between

the vestibular system and its visual and proprioceptive input leads to the sensation of vertigo or loss of balance. If this process is acute, vertigo usually results. If it is more chronic, dysequilibrium may be the presenting symptom. The goal of treatment then becomes to restore balance between the input systems.

Also, the vestibular system influences or contributes to brainstem autonomic circuits. There is an intimate linkage in components of brainstem pathways that process vestibular and visceral inputs. This explains the autonomic dysfunction associated with alterations of vestibular input, including nausea, vomiting, and pallor, as well as changes in respiration and circulation.

## **Medical Treatment**

The treatment of vertigo can be divided into two components: symptomatic and specific. Symptomatic treatment should focus on relieving the acute symptoms and autonomic complaints of vertigo. Specific treatments are geared towards targeting the underlying cause of the vertigo.

### **Symptomatic pharmacotherapy**

There are several transmitters in the vestibular nuclei, including cholinergic, H1-histaminergic receptors, and GABA. The vomiting center is stimulated by serotonergic, dopaminergic (D2) and histaminergic (H1) systems. Thus, many pathways and neurotransmitters are responsible for vestibular symptoms and associated autonomic complaints. This explains why so many classes of drugs are used to manage acute vertigo attacks. The main classes of drugs used for symptoms of acute vertigo include antihistamines, anticholinergic agents, anti-dopaminergic agents, and (gamma)-aminobutyric acid-enhancing (GABA-ergic) agents. These drugs will not eliminate but rather reduce the severity of vertiginous symptoms.

Although the exact mechanism of action of these drugs is unclear, they act at the level of the neurotransmitters involved in the propagation of impulses from primary to secondary vestibular neurons and in the maintenance of tone in the vestibular nuclei. They also act on the areas of the nervous system that control vomiting, including the central components, or “emetic center” and the peripheral components of the gastrointestinal tract. Two recent clinical trials comparing IV dimenhydrinate (50mg) with lorazepam (2 mg) and IM dimenhydrinate (50mg) with droperidol (2.5mg) for the treatment of peripheral vertigo in patients in the emergency department, found that dimenhydrinate was more effective than lorazepam and that dimenhydrinate and droperidol were equally effective. The response is clearly dose-dependent, so higher doses can be tried if the initial dose is not effective. It is important to note that all the medications used can be sedating, so they should not be used in patients attempting to perform activities, which necessitate a high level of alertness (i.e., driving, operating machinery or athletics). Less sedating drugs, such as meclizine and transdermal scopolamine are useful for milder vertigo and prevention of motion sickness. The newer nonsedating antihistamines do not enter the CNS and have little value in the treatment of acute vertiginous attacks.

### **Specific Pharmacotherapy**

#### ***Vestibular Neuritis***

Characterized by the sudden onset of prolonged vertigo of peripheral origin usually without hearing loss, vestibular neuritis (a.k.a. vestibular neuronitis) typically exhibits severe vertigo over a period of hours, lasting a few days, and then subsiding over the course of a few weeks. It is thought to result from a selective inflammation of the vestibular nerve, presumably of viral origin. The onset may be preceded by a viral infection of the respiratory or gastrointestinal systems. This finding and the occurrence of epidemics, several family members afflicted, and more common spring and early summer

disease all support a potential viral etiology. Therefore, treatment aimed at stopping the inflammation has been proposed. Ariyasu et al treated 20 patients in a double-blinded, crossover study with methylprednisolone versus placebo for acute vestibular vertigo. They found that 90% experienced a decrease in vertigo within 24 hours, compared with only 30% of the placebo group. The placebo groups were switched to methylprednisolone after 24 hours and subsequently had a decrease in vertigo within 24 hours of starting the steroid. The electro-nystagmogram in the 16 patients who took methylprednisolone with resolution of vertigo returned to normal within 1 month.

Most patients will have spontaneous, complete symptomatic recovery even only with supportive treatment. Patients who have persistent unsteadiness or motion provoked symptoms may have incomplete central compensation and should benefit from a customized vestibular rehabilitation program.

### ***Meniere's Disease***

The treatment for Meniere's disease or endolymphatic hydrops continues to evade clinicians, because the precise etiology of the disease is unknown. The histiologic finding of hydrops by Hallpike and Cairns in 1938 implicated a disturbance of salt and water balance as the pathology in patients suffering from Meniere's. The widely accepted medical treatment has, therefore, been dietary salt restriction and diuretics. It is thought that diuretics can alter the fluid balance in the inner ear leading to a decrease in endolymph and resolution of the hydrops. Thiazide diuretics are traditionally used nowadays in combination with a potassium sparing diuretic such as triamterene (Maxzide or Dyazide). It is important to stay on the diuretic therapy for at least 3 months before a decision is made to discontinue therapy. If allergies to sulfa drugs contradict the use of thiazides, loop diuretics or other therapies may be considered.

Carbonic anhydrase inhibitors (e.g., acetazolamide), which have been used by ophthalmologists to decrease intraocular pressure in treating glaucoma, have been tried for Meniere's. The analogy drawn between the two disease states (Meniere's has been called "inner ear glaucoma") led to the trial of these agents. These agents decrease sodium-hydrogen exchange in the renal tubule leading to diuresis, however the diuretic effects are not as long lasting as thiazides and loop-diuretics. In addition, acetazolamide is capable of decreasing CSF production. Possible adverse effects include nephrocalcinosis, mild metabolic acidosis and GI disturbances with chronic therapy.

Vasodilators have also been used for the treatment of Meniere's disease, based on the hypothesis that the pathogenesis of endolymphatic hydrops results from ischemia of the stria vascularis. The rationale is to improve the metabolic function of a diseased ear. IV histamine, isosorbide dinitrate, cinnarizine (calcium antagonist) and betahistine (oral histamine analogue) have all been used with anecdotal success, but no studies have demonstrated definitive beneficial effects of vasodilator therapy in reversing hydrops.

It has recently become apparent that Meniere's disease may be a disease of multifactorial inheritance. In some patient's there is thought to be an association of immune-mediated phenomena. There have been numerous reports of association of allergies and Meniere's disease. A study by Gottschlich et al., found that fifty percent of subjects meeting the criteria for Meniere's were found to have antibodies to a 70-kD heat-shock protein, which has been implicated in autoimmune sensorineural hearing loss. Therefore, treatment with immunosuppressive agents has gained favor, especially for patients presenting with bilateral disease. Systemic and intratympanic glucocorticoids, cyclophosphamide, and methotrexate have all been used by clinicians. Shea reported 48 patients following dexamethasone transtympanic perfusion for intractable Meniere's disease with a 66.7% success rate for the elimination of vertigo and a 35.4% improvement in hearing.

For intractable disease with disabling vertigo despite medical treatment, vestibular surgery should be considered. The chemical labyrinthectomy, or transtympanic gentamicin (intratympanic aminoglycoside [ITAG]), allows treatment of unilateral disease without producing systemic toxicity or affecting the opposite ear. It also carries the advantage of being a nonsurgical office procedure. Gentamicin is primarily vestibulotoxic, and may impair the function of vestibular dark cells, which are thought to play a role in the production of endolymph. However, there is an inherent risk of hearing loss that may be sudden, severe, and irreversible, which averages around 30%.

A stock solution of 40mg/mL of gentamicin is used and 10mg to 20mg is injected over the round window after anesthetizing the tympanic membrane. The patient remains in the supine position with the injected ear up for 30 minutes and is encouraged not to swallow. Bolus injections are typically repeated either weekly or biweekly, although the end point of therapy is variable between different authors, some advocating only one treatment and a possible follow-up. Clinical signs of vestibular hypofunction such as spontaneous nystagmus or headshake nystagmus are monitored in follow-up visits and considered the endpoint of therapy. Monitoring with audiometry between injections to detect potential hearing loss is advised. Total ablation of vestibular function does not appear necessary for this method to be effective because all studies report excellent control of vertigo. Minor was able to control vertigo in 91% of his treatment group with a 3% rate of profound SNHL. A recurrence rate of 22% occurred with control in all but one by further ITAG treatments. These numbers are very similar to vestibular neurectomy results with fewer risks. Continuous delivery methods involve a device that delivers the drug directly to the round window niche (Microwick, Round Window Microcatheter). Direct injection via labyrinthotomy has been tried with significant hearing loss and has fallen out of favor.

### ***Benign Paroxysmal Positional Vertigo (BPPV)***

BPPV is the most common cause of vertigo and results from dysfunction of the posterior semicircular canal (PSCC). Two theories have developed to explain its pathophysiology: cupulolithiasis and canalithiasis. The cupulolithiasis theory proposes that calcium deposits become embedded on the cupula, rendering the PSCC dependent on gravity. In the canalithiasis theory, calcium debris (displaced otoconia) becomes displaced within the PSCC but does not adhere to the cupula. In any case, head movements, particularly looking up, lying down, or rolling over onto the affected ear, result in displacement of the canal “sludge” and resultant symptoms. Several approaches have been developed to treat BPPV, including particle repositioning maneuvers and habituation exercises.

Semont et al adhering to the cupulolithiasis theory proposed a liberatory maneuver as a single treatment alternative. The patient is asked to sit on the side of an examination table with the head turned away from the affected ear. The patient is then quickly moved to the lateral decubitus position, with the head facing the ceiling, and is kept there for 2 to 3 minutes. Then, the patient is moved rapidly through the sitting position and onto the opposite side with the head remaining in the original position (now facing the floor). After a few minutes, the patient is returned to the seated position and is asked to remain upright for 48 hours. The reported cure rates are 84% after one, and 93% following two treatments.

Epley favored the canalithiasis theory and proposed a canalith repositioning procedure inducing gravity-directed movement of particulate matter into the vestibule, where it should not produce symptoms. The patient is initially placed in the Dix-Hallpike position for the affected ear. The head is then slowly taken through extension to the opposite Dix-Hallpike after 2 to 3 minutes. After an equal interval of time in this position, the patient is rolled onto the unaffected shoulder with the head turned toward the floor. After another 2 to 3 minutes, the patient is returned to the seated position. Again, the patient is asked to remain upright for 48 hours after the procedure. Epley reported 80% cure after one treatment and 100% improvement after multiple sessions in 30 patients.

Brandt and Daroff designed habituation exercises requiring the patient to move into the provoking position repeatedly, several times a day. From the seated position, the patient first moves rapidly into the provocative position. After the vertigo stops, the patient returns to the upright, seated position for 30 seconds, then onto the opposite side for 30 additional seconds. This is repeated until the vertigo diminishes. The exercises should be performed every 3 hours until the vertigo resolves for 2 consecutive days. They report a 98% success rate after 3 to 14 days of exercises.

Blakley compared the canalith repositioning maneuver with no treatment, and found 89% of all patients were improved after 1 month with no statistical significance between the 2 groups. The timing of spontaneous remission showed a 50% cure rate after 1 month. Still, advocates of noninvasive treatment techniques claim results superior to these numbers.

### ***Otosyphilis***

Penicillin has been the established treatment of otosyphilis. Intramuscular and intravenous routes are both acceptable. 2.4 million units of benzathine penicillin IM weekly for three consecutive weeks is considered minimal treatment and some would argue that treatment should be extended for one year. If the IV route is chosen, 10 million units of penicillin G per day is given in divided doses for ten days, followed by 2.4 million units of IM benzathine penicillin per week x2. Probenecid increases the half-life and CSF penetration of penicillin and may improve these regimens. Penallergic patients can be dosed with 500mg of tetracycline or erythromycin qid for 30 days. Steroids in addition to penicillin has been shown to improve treatment.

### ***Vertebrobasilar insufficiency (VBI)***

VBI is characterized by vertigo, diplopia, dysarthria, gait ataxia and bilateral sensory and motor disturbance. Symptoms of transient ischemia are alarming but generally benign as there is rich collateral blood supply and a relatively low incidence of stroke. Antiplatelet therapy is warranted usually with aspirin. Ticlid, a platelet aggregate inhibitor, has also been used in these patients, but because of the risk of life-threatening neutropenia, it is only warranted in those patients unable to tolerate aspirin.

### ***Migraine***

Many patients who suffer from migraine have concomitant vertigo and disequilibrium. Furthermore, if their headaches are controlled, they are often asymptomatic from their vertigo. Diagnostic criteria include personal or family history of migraines, motion intolerance, and vestibular symptoms that do not fit other vestibular disorders. Several theories are currently postulated as the mechanism underlying migraine. Symptoms may be of vascular origin, abnormal neural activity in the brainstem, or abnormal voltage-gated calcium channel genes. Treatment includes modifying risk factors, abortive medical therapy, and prophylaxis. These patients should avoid nicotine products, exogenous estrogens, and foods that exacerbate symptoms (red wine, sharp cheese, chocolate, MSG, etc.). Exercise programs and stress reduction are also important. Ergots, sumatriptin, and midrin are helpful in aborting acute attacks. Prophylactic medical therapy can be started if migraines occur several times a month (aspirin, ibuprofen, lithium, calcium channel blockers, amitryptiline and beta blockers).

## **Vestibular Rehabilitation Therapy (VRT)**

The use of exercise in the rehabilitation of patients with vestibular disorders is aimed at promoting vestibular compensation. Many treatment approaches have been designed to promote compensation through habituation. Other approaches are designed to enhance the adaptation of the vestibulo-ocular and vestibulo-spinal reflexes so that less input is required from the vestibular system. Many of these exercises may initially exacerbate the patient's symptoms, so counseling the patient to relieve anxiety and mistrust is important.

Cawthorne-Cooksey exercises were developed in the 1940s and include movements of the head, tasks requiring coordination of the eyes with the head, total body movements, and balance tasks. They recommended performing exercises at tolerable speeds, different positions, and with the eyes open and closed. Also, they had patients perform the exercises in loud and noisy environments, which is often more difficult in patients with vestibulopathies. There are many modified versions of this treatment philosophy in use today.

Candidates for rehabilitation therapy are those with stable lesions of the peripheral or central vestibular system, poor central integration, or abnormal motor function. Many elderly patients with multiple sensorimotor and musculoskeletal deficits fall into this category. The basis for exercise therapy assumes that central compensation following vestibular injury has occurred in an incomplete or inappropriate manner, the patient's symptoms stem from this incorrect compensation and not from fluctuating disease, and customized therapy is more effective than general exercise in changing this compensatory status.

The current common techniques of VRT still include habituation of pathologic responses, postural control exercises, visual-vestibular interaction, and conditioning activities. While standardized exercises are helpful, therapists can individualize treatments by first identifying the pathologic movement that causes the symptoms and developing a list of activities that reproduce the movements. Those movements can be incorporated into normal daily activities so they are reliable and remain interesting to the patient. The exercises should be performed twice daily unless limited by nausea and vomiting. Many patients report improvement within 4 to 6 weeks. However, the longer the problem has existed, the longer the recovery time will be.

Gait exercises can also be added to improve postural control. The starting point may be simple static balance exercises with eyes open and closed, on a stable support surface or on foam. The base of support can be gradually decreased to stress the patient's balance. Practice generally improves overall postural control. The patient also needs the practice of walking in different environments (grass, malls, or at night).

An important point to remember is that the input from two sensory systems is required for adequate postural stability. If there are losses in the visual or somatosensory systems, there is generally a poorer response to VRT. The goal and final level of recovery should be a return to most activities.

## **Conclusion**

Patients with vestibular complaints commonly present to the otolaryngologist. A thorough evaluation and understanding of common vestibular disorders is important in their diagnosis and treatment. After diagnosis and treatment of acute symptoms, weaning of vestibular suppressants and specific pharmacotherapy should be instituted. Patients with persistent complaints or chronic, uncompensated disease will greatly benefit from vestibular rehabilitation instituted early during the course of disease.

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