Patients with vestibular disorders have a variety of symptoms. The complaint of dizziness can be caused by a variety of disorders including presyncopal faintness, loss of balance, light-headedness, or psychologic disorders and must be differentiated from vertigo or dysequilibrium of vestibular origin. These patients commonly describe a sensation of spinning and may have associated visual or vegetative complaints, especially during acute attacks. The differential diagnosis of vertigo has remained stable over the past several decades but diagnosis and management has improved. With improved understanding of the pathologic processes behind the disorders and renewed interest in vestibular rehabilitation many patients return to fully active lifestyles. With this improvement in medical management, the need to proceed with more aggressive surgical procedures has become a less frequent event.

**Pathophysiology**

The vestibular labyrinth is responsible for detecting both linear and angular head movements. Each is composed of three semicircular canals and two otolithic organs. The semicircular canals - lateral, superior, and posterior - are oriented at right angles to each other and detect rotational movement. The otolithic organs - the utricle and saccule - detect linear acceleration. Stereocilia function as the mechanotransducers of these stimuli and are termed hair cells. In the semicircular canals, the hair cells are organized under a gelatin film called the cupula. Angular acceleration causes deflection of the cupula and hair cells and subsequent hyper or depolarization depending on the direction of movement. In the otolithic organs, the hair cells are attached to a layer of otoconia on the macula. The otoconia remain stationary relative to the head with linear acceleration, which causes deflection and stimulation of the underlying hair cells.

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 this input. 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 manifest symptom. Therefore the goal of treatment is to restore balance between the input systems.

In addition, there is intimate linkage of brainstem pathways that process vestibular and visceral inputs. There is evidence of convergence of the two systems in the nucleus of the solitary tract, the parabrachial nucleus, and the rostral ventrolateral medullary reticular formation. Since the parabrachial nucleus is connected with brainstem sympathetic output pathways, neuroendocrine regions of the hypothalamus, and limbic regions of the forebrain, these findings provide a potential neurologic basis for affective, autonomic, and neuroendocrine manifestations in vestibular dysfunction.
Medical Treatment

Treatment of acute vertigo has two components. First, one must control the acute episode, and secondly, speed the recovery and prevent future episodes. There does not appear to be an ideal drug in the treatment of vertigo. Existing drugs were essentially found during clinical use rather than developed specifically for the treatment of vertigo. Vertigo can be treated symptomatically or specifically. Symptomatic treatment involves controlling the acute symptoms and autonomic complaints. Specific treatment involves targeting the underlying cause of the vertigo. Some common types of vertigo have either established or postulated pathophysiology and lend themselves to specific treatment, others are still unknown and symptomatic control is the only option.

Symptomatic Pharmacotherapy

Vertigo is primarily due to an imbalance between the two vestibular labyrinths whose activity is modulated by the central vestibular system. There are several transmitters in the vestibular nuclei but cholinergic and H1 histaminergic receptors are the main types. Takeda has summarized a model to explain the neurophysiological mechanisms of vertigo. Sensory information from various sources is processed, integrated, and stored, and then compared to sensory information which the programmed system would expect to receive under normal circumstances. With this model a cholinergic system modulates the neural store, and a histaminergic system is used by the "comparator" to stimulate the vomiting center. In addition, GABA neurotransmitters are used as inhibitory signals from cerebellar purkinje cells. An adrenergic system projects from the brainstem to the vestibular nuclei to inhibit vestibular activity. Stimuli from the gastrointestinal tract act on the vomiting center via a serotonergic pathway. The chemoreceptor trigger zone in the area postrema acts on the vomiting center and can be blocked with dopamine (D2) agonists. Lastly, the vestibular nuclei act on the vomiting center via a histaminergic (H1) system. One can clearly see that many pathways and neurotransmitters are involved in causing the vertigo and autonomic complaints. This explains why so many classes of drugs are used in the management of this disorder.

Four general classes of drugs are useful in the treatment of vertigo and its associated autonomic symptoms - anticholinergics, antihistamines, antidopaminergics, and monoaminergics. The most effective single drug for the prophylaxis and treatment of motion sickness is the anticholinergic scopolamine. It was removed from the market because of questions about the delivery system but has now reappeared. Side effects include dry mouth, drowsiness, and blurred vision. Antihistamines include meclizine, dimenhydrinate, and promethazine. They usually last for 4-6 hours except for meclizine which is supposed to remain in the system for 24 hours. They generally have less side effects than the anticholinergics with drowsiness being the most prominent. Some of these agents have some anticholinergic activity with the corresponding side effects of dry mouth and blurry vision. The newer nonsedating antihistamines do not enter the CNS and have no value in the treatment of vertigo and motion sickness. Antidopaminergics such as prochlorperazine and chlorpromazine act at the chemoreceptor trigger zone, reducing neural impulses to the vomiting center. These drugs do not prevent vertigo and motion sickness but may be useful in treating the nausea and vomiting caused by these disorders. Side effects include sedation and the possibility of extrapyramidal symptoms. Monoaminergic drugs include amphetamines and ephedrine. They appear to potentiate the effects of scopolamine and may be used in combination with one of the antihistamines for intense symptoms or in those who do not respond adequately to single-drug therapy. Lastly, the benzodiazepine diazepam act as a vestibular suppressant through the GABAergic system and can also minimize the associated anxiety and panic that occurs with vertigo.

Specific Pharmacotherapy

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 others would argue that treatment should be extended to one year. If the IV route is chosen, 10 million units of penicillin G per day is administered in divided doses for ten days followed by 2.4 million units of IM benzathine penicillin per week for two additional weeks. Probenicid increases the half life and CSF penetration of penicillin and may improve these regimens. Penallergic patients may be dosed with 500 mg of tetracycline or erythromycin qid for 30 days. Steroids in addition to the penicillin has been shown to improve treatment. Prednisone 40 to 60 mg per day for two to four weeks with a taper is considered adequate.
Vertebrobasilar insufficiency (VBI)

VBI is manifested as vertigo, diplopia, dysarthria, gait ataxia, and bilateral sensory and motor disturbance. The 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 has also been used as a platelet aggregate inhibitor in patients suffering from VBI. There is some evidence that it is more effective than aspirin but because of the risk of life-threatening neutropenia it is only warranted in patients unable to tolerate aspirin.

Migraine

This diagnosis is controversial partly because there is not consensus as to the defining criteria. Nevertheless, many patients who suffer from migraine have concomitant vertigo and disequilibrium and 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. Treatment is threefold and includes modifying risk factors, abortive medical therapy, and prophylactic medical therapy. Patients suffering from migraine should avoid nicotine products, exogenous estrogens, and foods known to 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. When migraine occurs several times a month, prophylactic daily medical therapy should be used. Aspirin, ibuprofen, lithium, calcium channel blockers, amitryptiline, and beta blockers have all been found to be effective in reducing the frequency and severity of attack.

Vestibular Neuritis

Vestibular neuritis is characterized by sudden onset vertigo lasting several days and usually no hearing loss. Onset is often preceded by a viral infection of the respiratory or gastrointestinal systems and epidemics are not uncommon. Just as in herpetic lesions of the facial nerve, there are temporal bone studies consistent with viral infection of Scarpa's ganglion in patients with well documented histories of viral neuritis. Ariyasu et al treated patients with methylprednisolone and had relief of vertigo with normalization of the ENG at one month in his treatment group.

Meniere's Disease

The treatment of Meniere's disease or endolymphatic hydrops remains an enigma because the precise etiology of the disease is unknown. Temporal bone studies initially described the finding of hydrops and it was theorized that this was the cause of the subsequent symptoms. Unfortunately, investigators have induced histologic hydrops in animal models without evidence that vestibular symptoms resulted. Despite this, it is still considered the working model and is the basis for most treatment regimens.

Diuretics and dietary salt restriction have been considered the mainstay of treatment for years. It is believed 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 and lead to decreased sodium absorption in the distal tubule of the nephron. Side affects are many and include hypokalemia, hyperglycemia, hyperuricemia, hypotension, and hyperlipoproteinemia. A potassium sparing diuretic such as triamterene can be used in combination (Maxzide or Dyazide) to decrease the side effects.

Carbonic anhydrase inhibitors are used by ophthalmologists to decrease intraocular pressure in treating glaucoma. An analogy drawn between this disease and hydrops led to their use by otolaryngologists. These agents decrease sodium-hydrogen exchange in the renal tubule leading to a diuresis. In addition they decrease the production of CSF. IV therapy can exacerbate symptoms but there is some evidence that oral therapy can reduce hydrops in the short term. Unfortunately again, significant side effects occur in as high as 50% of patients including metabolic acidosis and renal calculi.

Vasodilators are used based on the hypothesis that ischemia of the stria vascularis causes Meniere's. The rationale is to improve the metabolic function of a diseased ear. IV histamine, isosorbide dinitrate, cinnarizine (a calcium antagonist), and betahistine (an oral histamine analogue) have been used with purported success. Betahistine has been extensively studied and used in Europe and has been found to produce vasodilatation of the capillaries, arterioles, and arterial-venous arcades in the stria vascularis and spiral ligament and lower endolymphatic pressure. There has been some short-term relief of vertigo with this agent but no proof exists that any long-term management can be obtained with this drug.
Fifty percent of subjects with clinical evidence of Meniere's disease were found to have antibodies to a 70-kd heat-shock protein in a study by Gottschlich et al. This antibody has been previously associated with autoimmune sensorineural hearing loss and treatment with immunosuppressive agents has gained favor. Systemic and intratympanic glucocorticoids, cyclophosphamide, and methotrexate have all been used by clinicians but this treatment is controversial at best currently.

Despite all of these treatment regimens, it has been well shown that the placebo effect exists in the management of Meniere's disease. Torok performed a retrospective analysis and found that all therapies produced a 60-80% remission rate. Silverstein offered surgery to medical failures and studied the patients that declined more aggressive treatment. 59% had complete control of vertigo at 2 years and 70% at 8 years. He concluded that this was the natural course of the disease. Because of these confounding conditions it has been difficult to determine which treatment regimens alter the course of the disease. Ruckenstein did a retrospective analysis of the literature that was published following Torok’s study and concluded that none of the above treatments provided relief of the symptoms of Meniere's disease. He concluded that all of these therapies were nonspecific and no better than placebo. He did not disparage the use of these agents but instead cautioned that the most benign form of treatment should be utilized in order not to harm the patient. The vestibular suppressants were the only class that were proven to alleviate acute attacks of vertigo in his analysis.

Chemical labyrinthectomy has recently become popular in the treatment of patients with disabling vertigo that is persistent and refractory to medical management. Intramuscular streptomycin was initially used recognizing that it is more vestibulotoxic than cochleotoxic but the diseased ear in unilateral Meniere’s cannot be targeted with systemic therapy. Intratympanic gentamicin allows treatment of unilateral Meniere’s disease without producing systemic toxicity or effects on the opposite ear. The round window membrane serves as the primary route of entry into the inner ear with the annular ligament of the stapes serving as a secondary route. The solution may be used as a stock solution of 40 mg/cc at a pH of 5.4 or buffered to a pH of 6.4 to improve patient comfort. With the patient supine and the head turned away from the treatment ear the tympanic membrane is anesthetized and about 0.5 cc of solution injected into the middle ear. The patient remains in this position for 30 minutes and is instructed not to swallow. Many treatment protocols exist. At UTMB, we routinely schedule the injections weekly until effect has occurred. An audiogram is obtained prior to each injection to ensure that auditory toxicity is not occurring. Patients should report symptoms of acute unilateral vestibular insult with vertigo (usually described as different from that with Meniere’s), imbalance, and a tendency to fall toward the affected ear. These symptoms occur at variable times after the procedure. Clinical signs of vestibular hypofunction such as spontaneous nystagmus or head-shake nystagmus are monitored in followup visits and considered the endpoint of therapy. Cold water caloric can be performed prior to the next injection if there is uncertainty of effect from the prior treatments. Generally, patients are offered the procedure up to six times before concluding failure. Minor was able to control vertigo in 91% of his treatment group with a 3% rate of profound SNHL. There was a recurrence rate of 22% with control of their symptoms in all but one by further ITAG treatments. These numbers are very similar to the results obtained with vestibular neurectomy with far fewer risks.

Benign Paroxysmal Positional Vertigo (BPPV)

BPPV is the most common cause of vertigo. In the past it was explained as cupulolithiasis: calcific deposits became embedded on the cupula, rendering the semicircular canal dependent on gravity. A substantial amount of evidence now exists that the disorder results from canalolithiasis: calcific debris (presumably displaced otoconia) in the semicircular canal but not adherent to the cupula. Head movements, particularly looking up, lying down, or rolling over onto the affected ear, result in displacement of this canal sludge and movement of endolymph away from the cupula exerting a plunger-like effect and pulling of the cupula. Several approaches have been developed to treat BPPV including particle repositioning maneuvers and habituation exercises.

Semont developed the liberatory maneuver as a single treatment alternative. Once the side of involvement is found the patient is quickly moved into the plane of the posterior canal and is kept in that position for 2-3 minutes. The patient is then rapidly moved up through the sitting position and down into the opposite side-lying position with the therapist maintaining the alignment of the neck and head of the body. The patient stays in this position for 5 minutes and then slowly returned to the seated position. They must then remain in a vertical position for 48 hours and avoid the provoking position for one week. Reportedly, it works by floating the debris through the canal and into the common crus and requires only one treatment. Semont reported cure in 84% after one treatment and 93% after two treatments.

Epley proposed a canalith repositioning procedure where the patient is taken rapidly into the Dix-Hallpike position that provokes the symptoms and is kept in that position for 3-4 minutes. The head is then slowly taken through extension and turned to the opposite Dix-Hallpike position. The patient is rolled over onto their side so that the head is turned toward the floor and remains in that position for 3-4 minutes. They are then slowly sat up. Epley
premedicated his patients and used a mastoid vibrator during the treatment which is not considered to be necessary by most clinicians currently. Again the patient is instructed to remain in an upright position for 48 hours and told not to lie on the effected side for 5 days. Epley reported 100% improvement or cure with multiple maneuvers in one patient treatment. This was repeated by Herdmann et al who found 90% were asymptomatic or significantly improved after one treatment.

Brandt and Daroff designed habituation exercises that require the patient to move into the provoking position repeatedly, several times a day. The patient first sits and then moves rapidly into the position causing vertigo. A torsional nystagmus occurs with the vertigo. They stay in this position until the vertigo stops and then they sit up again. They remain in this position for 30 seconds and then move rapidly into the mirror-image position on the opposite side staying there for 30 seconds and then sitting back up. This is repeated until the vertigo diminishes. This is then repeated every 3 hours until the vertigo resolves for 2 consecutive days. Explanations for the success of this maneuver include dislodging of debris in the posterior canal and/or central adaptation reducing the CNS response from the posterior canal. They reported that 98% were asymptomatic after 3-14 days of exercises.

Norre designed habituation exercises that were specific for each patient and were not limited to modifications of the Dix-Hallpike maneuver. These exercises have been advocated for patients who exhibit symptoms of BPPV in planes that are not consistent with the posterior canal and in patients who may have other vestibular disorders overlapping with BPPV. Their results included 32% asymptomatic at one week and 100% asymptomatic at 6 weeks.

Not all researchers have found success in the same percentage of patients as the above authors but there is consistent data showing that the patients treated with any of the methods do better than control groups. In addition, the procedures are relatively quick, easy, and cost-effective to perform. Comparison will show that there is similar success with all approaches but not all are suitable for all patients. Patients who have more anxiety about the treatment and the sensation of vertigo may respond better to the longer term habituation exercises, whereas patients wanting more immediate relief will be better suited for the repositioning procedures. Patients with arthritic diseases and limited mobility also deserve special consideration when planning therapy. In determining immediate "cure", you must remember that this vertigo is fatiguable and some of these patients may recur in the future.

**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 vestibulospinal reflex so that less input is required from the vestibular system. The program is usually begun on an outpatient basis but may be instituted while the patient is still hospitalized. Many of the exercises may initially exacerbate the patient's symptoms, so the patients should be thoroughly counselled to allay any feelings of mistrust. In cases with severe symptoms the patient's exercise protocol may have to be readjusted temporarily.

Cawthorne-Cooksey exercises were developed in the 1940s and include movements of the head, tasks requiring coordination of eyes with the head, total body movements, and balance tasks. They recommended performing these exercises at various speeds, in different positions, and with the eyes open and closed. In addition they had the patients perform these exercises in loud and noisy environments which may be more difficult in the vestibular patient. There are many different VRT protocols in use but the treatment philosophy is remarkably similar to this approach established 60 years ago.

The current common techniques of VRT still include habitation of pathologic responses, postural control exercises, visual-vestibular interaction, and conditioning activities. The therapist must first identify the pathologic movement that causes the symptoms and develop a list of activities that reproduce the movements. These movements can be incorporated into normal daily activities so that they are performed reliably and remain interesting to the patient. They are performed twice daily unless limited by severe nausea and vomiting. If they can stick with the program, many patients report improvement within 4 to 6 weeks.

If postural control is found to be a culprit, this can be added to the exercise program. For example, if a patient is found to depend on somatosensory input despite the availability of visual input, the program may involve exercises that require balancing on thick foam. This can be performed initially with the eyes open and then progress to eyes closed. With visual-vestibular mismatch or bilateral vestibular disorders, exercises may optimize the use of the visual system. Many patients have adopted a sedentary lifestyle in order to avoid their symptoms. In turn, they become deconditioned which may exacerbate their disorder. These patients are designed an appropriate fitness program to match their age and lifestyle. This can incorporate sports that require hand-eye coordination to further improve their condition. One particular activity which may be poorly tolerated in the vestibular patient is swimming because of the relative weightlessness encountered.
A particularly important use of VRT is in the elderly who may have multifactorial balance difficulties. With the impact of complications of falls in the elderly, VRT which may decrease the functional impact of disequilibrium, balance, or gait problems is highly desirable. No studies have shown that the outcome measures of VRT in the elderly are poorer. The only significant difference has been an increased amount of time required to maximize the benefit. It is important to remember though that input from two sensory systems is required for adequate postural stability. If there are losses from the visual or somatosensory systems there is a poorer response to VRT.

VRT has been consistently shown to be an important part of the management of vestibular patients but it is not a cure-all. Most studies show that patients improve on the order of 70-80% but this does not mean to the point of being symptom-free. Cowand et al found that 28% of their patients showed complete resolution of symptoms within one year and 54% improved even if only marginally.

Not all patients are ideally suited for VRT. Patients with stable or slowly progressive vestibular disease are the best candidates. Inappropriate candidates for VRT include those who have fluctuating nonstable vestibular lesions such as with Meniere's disease. If no provocative maneuvers and no postural control abnormalities are found on examination, the patient may not respond favorably as there will not be a treatable component by VRT, these patients may be better managed with other medical or surgical strategies.

Conclusion

Presentation of patients with vestibular complaints is a common occurrence in the otolaryngology clinic. It is often a frustrating encounter as many patients are either poor historians or don't fit squarely into one of the common diagnostic categories. Nevertheless, it is imperative that a thorough evaluation take place so that diagnosis can be established. Many patients have been treated by other physicians with chronic use of vestibular suppressants and no form of rehabilitation. These patients often cling to these prescriptions as they fear the consequences of stopping therapy. After diagnosis and treatment of the acute symptoms, it is now clear that weaning of vestibular suppressants and institution of vestibular rehabilitation is crucial to prevent a chronically uncompensated vestibular patient.

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