Problems in Family Practice

Vertigo: A Physiological Approach

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An understanding of vestibular function and a careful history will quickly separate patients with disease of the vestibular system from others. Further history and physical examination, often coupled with audiometric and vestibular function tests, will aid in localizing the lesion to the central or peripheral portions of the system and in determining the affected side. Medical management, which is often nonspecific and involves considerable psychological support, usually suffices.

Dizzy patients are no rarity in any primary care practice; yet, the evaluation and management of these patients are, for many physicians, frustrating and unrewarding. Several reasons are apparent: first, the vestibular system and associated sensory systems comprise a "sixth sense" which is important to normal function but which, unlike the other senses, largely escapes conscious scrutiny. We are rarely aware of vestibular sensation during normal function, and we often find ourselves unable to describe what we feel when the system malfunctions. Secondly, the vestibular system interacts with the visual, somatosensory, motor, and autonomic systems at a subconscious level; thus, it is not surprising that a disorder in one system may produce a constellation of symptoms (eg, vertigo, ataxia, nausea, and oculomotor difficulties during an acute episode of Ménière disease). Thirdly, most patients with dizziness have benign, self-limited disorders; surgical and autopsy material is rarely available to provide histopathologic insight into these disorders. Fourthly, the vestibular end organ is, like the cochlea, essentially inaccessible to examination during life; if the otologist could see the cochlea and semicircular canals the way his ophthalmologic colleagues see the retina, life would be much simpler. Finally, a number of patients have dizziness which is functional. Management of these patients can be time consuming and frustrating.

The good news is that dizzy patients can be managed in a satisfying way. It usually is not difficult to separate functional symptoms from organic disorders, vestibular from nonvestibular symptoms, central from peripheral pathology, the potentially serious from the innocuous. A small number of well-defined syndromes will accommodate most patients with true vertigo.[1] The key is a good history and an understanding of vestibular physiology.[2] Physical examination and laboratory tests are usually confirmatory. A physiological approach is essential for reasons suggested above;

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we are not as intuitively familiar with our vestibular sense as with hearing or vision, and we are severely limited in our ability to study the pathological anatomy of the vestibular system. This paper will discuss both the normal and disordered physiology of the vestibular system, a few common specific disorders, a scheme of evaluation, and the basic elements of therapy for vestibular disorders. The focus will be on peripheral vestibular function and dysfunction.

Physiology

The peripheral vestibular system consists of a paired set of end organs and the vestibular portions of the eighth nerves. On each side, there are three semicircular canals which are mutually orthogonal, ie, each canal defines a plane which is at right angles to the planes defined by the other two canals. Each semicircular canal has a dilated ampulla, within which lie the hair cells, embedded in a gelatinous structure called the cupula. These hair cells are anatomically and phylogenetically similar to the hair cells of the cochlea. When the head is rotated in the plane of a semicircular canal, the inertia of the endolymphatic fluid causes a deflection of the cupula. In fact, the endolymph and cupula tend to remain stationary while the head moves, but it is the relative movement which is important. The hair cells are displaced, and the nerve fibers innovating these hair cells subsequently display either an increased or decreased firing rate. This signal passes to the vestibular nuclei and forms the basic substrate of vestibular sensation.

It must be stressed that this part of the system is elegantly adapted to the perception of rotational head movements, not of position, linear movement, or linear acceleration. Introspection will confirm that the natural movements of the head are all rotational rather than translational. The three semicircular canals on each side are capable of detecting rotational movements in any plane. Theoretically, each side should be capable of doing the job alone, giving the system the kind of redundancy which is so common in paired systems throughout the body.

The second part of the peripheral vestibular system consists of the otolith organs, the utricle and saccule. Each of these organs lies in the vestibule itself, a nearly spherical space connecting the semicircular canal fluid spaces with those of the cochlea. These sense organs also contain hair cells, but the hair cells are embedded in a gelatinous material containing numerous calcium crystals called otoconia. The otoconia are more dense than the hair cells or the surrounding fluids. Thus, hair cell deflection can occur, not only by inertia (during linear acceleration) as occurs in the semicircular canals, but also by gravity alone. Sensory input from these organs also travels in the vestibular nerves to the vestibular nuclei.

From the vestibular nuclei, the system makes complex afferent and efferent connections, including direct projections to the nuclei of the extraocular muscles, and to and from the cerebellum and spinal cord for control of posture and muscle tone. Poorly traced connections exist to the cerebral cortex, subserving both the perception of head movement and modulation of vestibular function by higher centers. Finally, there are extensive projections to the reticular activating system, by which vestibular input is distributed to autonomic centers.

Obviously, the vestibular system plays a role in the maintenance of balance, sensing position, movement, and rotation of the head. However, this is an incomplete picture in two ways (Table 1). First, other systems are important in equilibration. Our eyes obviously provide considerable information about our position and movement in space. In addition, both proprioceptors in the joints and touch and pressure receptors in the skin provide considerable clues. The maintenance of balance, then, depends on the interplay of the three systems: vestibular, visual, and proprioceptive/exteroceptive. In general, a person deprived of one of these three systems can still function fairly well. However, a patient with either absent vestibular function or severely disturbed posterior column function (as in pernicious anemia or tabes dorsalis) will have severe difficulties when deprived of visual input, as in the dark.

Balance is not the only, or even necessarily the most important, function of the vestibular system. The maintenance of stable visual fields is probably of equal importance. For each rotational head movement, the semicircular canals on each side send a neural message to the vestibular nuclei,

Table 1. Physiology of Vestibular and Associated Systems

Structure	Function
Semicircular canals*	Stabilize visual image during head movements Balance
Utricle, Saccule*	Balance
Visual system	Smooth pursuit eye movements Saccades Balance
Proprioceptors, Exteroceptors	Balance

*and associated neural pathways

which is in turn relayed to the oculomotor nuclei, resulting in an exactly equal and opposite rotational movement of the eyes. For head movements of moderate size and velocity, this compensation is so nearly perfect that relative to the outside world, the eyes do not move at all. Two simple examples will confirm this fact. If one index finger is held at arm's length directly in front of the eyes, and the head is shaken briskly from side to side as if to say "no," the finger will appear stationary without any blurring. To prove that this is not due to eye movement compensation by visual feedback, simply perform the control experiment: move the index finger back and forth at the same velocity with which the head was previously shaken. The eyes are completely unable to follow the finger, which will appear blurred or multiple. The second illustration is even more telling. A movie taken with a hand-held camera from a moving vehicle will, when projected, appear to bounce up and down so that even the horizon is unsteady. Contrast this with the stable visual image of the observer himself riding in the car. The difference is simply that the observer has a vestibular system to correct for head movements, and the camera does not.

This vestibulo-ocular reflex is only one of several important mechanisms of eye movement control. The eyes are capable of tracking by visual feedback, although these "smooth pursuit" eye movements are much slower than the vestibulo-ocular movements. A third kind of eye movement which is important to understanding of vestibular physiology, and particularly vestibular testing, is the saccade. This is an extremely rapid eye movement (up to 500 degrees per second), which normally occurs whenever the eyes shift targets without a change in focal plane. Most of us execute several consecutive saccades while reading a line of print, for example. The saccade is also used by the central nervous system as a corrective or compensatory eye movement. For example, when the eyes are attempting to follow a moving object whose velocity exceeds the range of smooth pursuit tracking, such as a tennis ball, a saccade is used to move the eyes rapidly from the point at which the object is lost to the expected region of the object's arrival. Also, when vestibulo-ocular eye movements exceed about 15 to 20 degrees excursion, the saccade will return the eyes to a more central direction of gaze. This is the basis for the fast phase of nystagmus.

Pathophysiology

As it is rarely possible to obtain anatomical or biochemical data in vestibular disease, it is necessary to turn to studies of disordered function. One obvious question comes to mind: what are the ef-

fects of total bilateral loss of vestibular function? This occurs occasionally from disease processes and was fairly common in the early days of streptomycin usage when its ototoxicity was not well known. These patients actually function fairly well, with minimal ataxia, when they have adequate visual and proprioceptive input. They have severe problems with balance when in the dark, and they cannot swim safely because so much proprioceptive input is lost in the water. Such patients frequently complain of oscillopsia, a symptom in which the visual image cannot be held stable when the head is moving. This is similar to the example given earlier of the movie taken from the window of an automobile. Patients with bilateral vestibular loss do not have vertigo. In fact, they are immune to motion sickness.

Unilateral vestibular loss, if it has been very gradual or if there has been time for central compensation, is usually less of a problem. The vestibular end organs on either side of the body are usually sufficient for eye movement control and balance. An exception to this rule is the elderly patient, who presumably lacks the ability to compensate at a central level for the loss of function.

An acute vestibular loss, such as might occur after vascular occlusion or head trauma, is a far different matter. The patient will generally be extremely ill, with vertigo, nausea, and vomiting, and a strong desire to lie perfectly still. There will be a brisk nystagmus beating to the intact side, and the patient will past-point to the side of the lesion. If the Romberg test is performed, the patient will fall towards the side of the lesion. Within a matter of hours, the central nervous system, if intact, will begin to compensate. Inhibitory input from the cerebellum will essentially "turn off" the opposite labyrinth, so that for a short period there is essentially a total bilateral vestibular loss of function. Over a period of many days to a few weeks, the central nervous system manages to deactivate circuits from the injured labyrinth, and the inhibitory "clamp" that had been placed on the normal side is then released. Once this phase of compensation is complete, the patient achieves the usually asymptomatic state of the chronic unilateral vestibular loss as described above. However, if the loss of function on the diseased side has been less than complete, compensation may be less satisfactory. A dead labyrinth is easier to live with than one which is severely diseased.

Vertigo and nystagmus are the hallmarks of acute peripheral vestibular disorders. Either may occur in central nervous system disease as well, but the presence of both severe vertigo and brisk nystagmus simultaneously usually indicates peripheral disease. Conversely, the patient who does not have nystagmus during a dizzy spell or who has nystagmus without any vertigo at all usually does not have a peripheral vestibular disorder. Vertigo is a hallucination of movement which arises because of a conflict in information from different sensory systems. Typically, the disordered labyrinth is telling the central nervous system that the head is moving while the visual and proprioceptive senses deny this. Naturally, the vertigo is worse with the eyes closed and the loss of visual input. Vertigo must be clearly separated from other symptoms which patients often include under the semantic umbrella of "dizziness." These include light-headedness, weakness, numbness, blurred vision, and vague dissociative feelings.[3]

Nystagmus always accompanies vertigo in acute peripheral vestibular disorders. Vestibular nystagmus is a periodic to and fro movement of the eyes which has a slow component (vestibulo-ocular) in one direction and a fast component (corrective saccade) in the opposite direction. Vestibular nystagmus occurs within the plane of one of the semicircular canals. Thus, it is either horizontal or rotatory-oblique (the superior and posterior semicircular canals are oriented in oblique planes which are midway between the sagittal and coronal planes). A vertical nystagmus can arise only from a disorder of the central nervous system, including portions of the brainstem and cerebellum. It is worth noting here that any nystagmus observable with the eyes open and at midline gaze (to rule out physiological end gaze nystagmus) is pathological. While nystagmus is named by its fast component, it is the slow component which represents vestibulo-ocular activity.

Common Disorders

Ménière disease is common but is certainly very much overdiagnosed. It should not be a wastebasket diagnosis for patients with vague

dizziness. Ménière disease is characterized by recurrent episodes of vertigo, fluctuating hearing loss, and either tinnitus or ear pressure or both. The vertigo may last minutes or hours but not seconds or days. During an attack, the patients are often prostrate and extremely ill. These symptoms are unilateral in 80 percent of cases, and the clinical course is usually one of prolonged periods of exacerbation and remission. About two thirds of cases will eventually "burn out" spontaneously, leaving the residual sensorineural hearing loss. The documentation of a fluctuating hearing loss on audiometry is essential to making a diagnosis of a classic Ménière disease. Most patients are in early or mid adult life when the symptoms begin; onset in childhood or old age is very rare.[4] Men are slightly more often affected than women. Psychological factors have been discussed; patients with Ménière disease tend to be rigid, highly controlled, and conformist individuals.

The histopathology is well defined: there is endolymphatic hydrops, with gross dilatation of all the endolymphatic spaces, secondary labyrinthine membrane ruptures, sense organ destruction, and fibrosis. This picture may also be seen in tertiary syphilitic involvement of the inner ear. For this reason, serologic tests (FTA-ABS—fluorescent treponemal antibody-absorption test—is preferred) are mandatory in evaluating suspected cases of Ménière disease. The well-known histopathology of Ménière disease has suggested therapeutic modalities as well. Both low salt diet and diuretics have shown some benefit. In addition, endolymphatic sac decompression via a mastoid operation appears to be effective in selected cases.[5] However, the evaluation of therapeutic modalities in this disease is made rather difficult by the unpredictable natural history of the disease and by the necessarily subjective criteria for improvement.

Benign positional vertigo (BPV) is nearly as common as Ménière disease but is much less well appreciated. The patient with benign positional vertigo has severe bouts of vertigo brought on by sudden changes in the head position. Most often the patient relates a history of turning over in bed or getting in or out of bed as the precipitating factor. The acute historian will have identified the specific position which reliably elicits an attack. The symptoms are short lived, never lasting more than a minute, and fatigability is noted. If one tries deliberately to elicit several attacks successively, each one will be weaker than the last until no vertigo can be elicited at all. There is occasionally a history of prior head trauma, but past history is otherwise unremarkable. If a complete audiovestibular work-up discloses no other abnormalities, the prognosis is excellent. Most patients improve spontaneously over a period of months or years. Only a few of these patients have died of other causes and had their temporal bones examined microscopically. Some of these have demonstrated an interesting pattern of otolith degeneration, and in some cases, the otoliths have migrated in the endolymph and become embedded in the cupula of the posterior semicircular canal. Thus, cupulo-lithiasis has been suggested as a synonym for benign positional vertigo. Rarely, patients with BPV are sufficiently disabled to require surgery. Singular neurectomy, which denervates the posterior semicircular canal, is the procedure of choice.

Labyrinthitis is a popular diagnosis for a short-lived solitary episode of vestibular illness without other apparent cause. Although there is no good supporting pathological or serological data, it seems reasonable to assume that, especially when the vertigo accompanies an otherwise typically viral illness, a virus is to blame. Other forms of labyrinthitis can occur. Purulent labyrinthitis, although uncommon, may develop as a complication of either meningitis or otitis media. This would present with the symptoms of an acute unilateral vestibular failure, as described in the previous section. Because of the continuity of the inner ear fluids, a diffuse labyrinthitis would be expected to impair cochlear and vestibular function alike; a hearing loss should accompany the vestibular symptoms.

In contrast to this, a syndrome of sudden, acute, and permanent vestibular loss without hearing loss is not infrequently seen. Most otolaryngologists call this *vestibular neuronitis*, assuming that the absence of auditory symptoms must imply a disorder of the vestibular portion of the eighth nerve. However, a vascular accident affecting only the vestibular portion of the internal auditory artery could also explain these symptoms. These patients are usually vertiginous for a couple of weeks and then compensate as expected. It is important to do a thorough work-up to rule out other causes of loss of vestibular function, such as in-

fection and neoplasm. The most common neoplasm in this region is the acoustic neurinoma, a benign tumor arising in the internal auditory canal and extending into the cerebellopontile angle. Most of these tumors actually arise on the vestibular rather than the auditory branch of the eighth nerve, but because of their very slow growth, there is usually not much, if any, vertigo. Thus, the first symptom is usually tinnitus or hearing loss. However, there are exceptions to this rule, and any sudden auditory or vestibular loss should be investigated with the possibility of acoustic neurinoma in mind. Surgical removal is much less risky when these tumors are small.

Many *drugs* affect the vestibular system. Most members of the aminoglycoside antibiotic family, notably streptomycin, kanamycin, gentamicin, and tobramycin, are ototoxic. Most are more toxic to the vestibular than to the auditory end organ. Since the damage is usually gradual and bilaterally symmetrical, symptoms tend to be minimal. A mild ataxia may be the only indication of labyrinthine toxicity. Serial vestibular testing in monitoring the use of these antibiotics is not practical, so it is probably best to simply observe the patients for ataxia, test their hearing periodically, and be aware of the fact that these complications are dose related. Especially in patients with compromised renal function, antibiotic blood levels should be obtained to avoid excessive concentrations. Other antibiotics (vancomycin; chloramphenicol and polymyxin when used topically), antineoplastic drugs (especially cisplatin and nitrogen mustard), quinine derivatives, and many chemicals (eg, carbon monoxide, mercury, gold, lead, arsenic, alcohol, tobacco, aniline dyes) are ototoxic. Aspirin, furosemide, and ethacrynic acid may each cause reversible hearing loss. There is some evidence that different ototoxic agents may have additive toxicities and that these agents may potentiate the damaging effects of excessive noise on the inner ear as well.

Many drugs with central nervous system effects, such as the major and minor tranquilizers, will cause dizziness and ataxia but rarely true vertigo. These drugs will, however, frequently cause nystagmus of a CNS type, and it is important that these drugs be withheld for a period of a few days prior to vestibular testing. If this is impossible, then the interpretation of the results is complicated.

Alcohol has a unique effect on the vestibular system. In addition to its obvious effects on the central nervous system, the alcohol diffuses into the inner ear fluids, slightly decreasing their specific gravity. Since the cupulae of the semicircular canals are gelatinous and absorb the alcohol slowly, a density gradient is created, causing the cupulae to function as gravity receptors. This causes the well-known positional vertigo and nystagmus of acute alcohol intoxication. Eventually the fluids and the gelatinous material of the cupula come into equilibrium, but when alcohol diuresis takes place, there is a subsequent phase where the cupulae are less dense than the fluids, resulting in a positional nystagmus of opposite direction.

Of the myriad of disorders which may affect vestibular centers in the brainstem and cause vertigo, the most common are *multiple sclerosis* (MS) and *vascular disorders*. Nystagmus was part of the classic triad of Charcot in multiple sclerosis. As is well known, multiple sclerosis is characterized by neurologic signs involving scattered areas of the central nervous system with a waxing and waning clinical course in a young adult. In addition to vertigo resulting from vestibular nucleus involvement, there is often internuclear ophthalmoplegia, causing dysconjugate eye movements. Vestibular function tests, as will be described later, may also detect abnormalities of eye movement such as this.

Vascular insufficiency anywhere in the vertebrobasilar territory may also cause vertigo. The reader is referred to standard neurology texts for descriptions of the posterior-inferior cerebellar artery occlusion syndrome. Transient vertebrobasilar insufficiency may occur in the presence of vertebral artery occlusive disease or in a subclavian steal syndrome. Usually there will be central nervous system symptoms in addition to vertigo, such as dysarthria, dysphagia, and visual difficulties.

Head trauma can injure either the peripheral or central vestibular system, or both. Permanent peripheral vestibular damage occurs most often in a transverse temporal bone fracture in conjunction with total hearing loss and facial nerve paralysis on the same side. Several studies have suggested that even very minor head trauma can cause significant long-term vestibular dysfunction. Many patients with "post concussion" syndromes have definite abnormalities on vestibular testing. This is important both in a medico-legal sense and in the counseling of these patients, since the physician is

often tempted to consider these syndromes functional in nature.

Vertigo caused by a *seizure disorder* is quite rare, but is probably a common cause of vertigo in children.[6]

Work-Up

In evaluating a patient who is "dizzy," the first step is to determine whether there is vertigo or not; ask the patient to describe his symptoms without using the word "dizzy." Those patients who do not have vertigo can then be worked up for whatever their true symptom is. For those patients who do have vertigo, an attempt must be made to characterize the disorder as being either peripheral or central. Sometimes these categories can be further subdivided into end organ, neural, brainstem, and cerebellar.

The most important elements of the history are severity, temporal pattern, and associated symptoms. Vertigo of peripheral origin tends to be severe but brief, and the associated symptoms are usually otologic in nature (eg, hearing loss, tinnitus, and ear pressure). Conversely, vertigo of central origin is more commonly associated with other central nervous system signs and frequently is less severe. Any vertigo or dizziness which is continuous for more than two weeks should be strongly suspected of being of central origin.

Physical examination should include a search for nystagmus. Since nystagmus is inhibited by visual fixation, an attempt should be made to prevent this. The best way is to use illuminated Frenzel lenses, but 10 to 20 diopter cataract lenses will work equally well to impair the patient's ability to focus while allowing the physician to observe the eyes at will. If there is a history of positional nystagmus, the Hallpike maneuvers should be carried out. The patient is placed in a sitting position, and with the head supported, rapidly carried into a supine position, first with the head hanging straight back and then on subsequent trials, with the head turned to the right and then to the left. In cases of benign positional vertigo (see preceding section), there will be a brisk rotatory nystagmus present in one or more of these positions, beginning about 5 to 15 seconds after the position is assumed. Physical examination should also include ophthalmoscopy, cranial nerve examination, examination of the tympanic membranes, and standard tests of cerebellar function and gait. Blood pressure should be measured (in both arms if vestibrobasilar insufficiency is suspected), and bruits searched for in the neck.

In all cases where the symptoms point clearly to a vestibular disorder, auditory and vestibular tests should be performed. Children with vertigo should also have neurologic evaluation for seizure disorder. A complete audiogram with air and bone conduction testing, speech discrimination testing, and often special tests, is mandatory. Vestibular function can be tested in a variety of ways. Although the physiological stimulus for the vestibular system is head movement and especially rotation, tests based on this principle have not been widely used clinically. Accurate rotational devices are difficult and expensive to build, and interpretation of these tests is somewhat difficult since both labyrinths are being simultaneously stimulated. Most clinicians use caloric testing to assess the integrity of the vestibular system. When water, either warmer or colder than body temperature, is irrigated against the eardrum, the temperature changes are conducted inward towards the lateral semicircular canal. A convection current causes fluid movement in the canal with deflection of the cupula, simulating the response to horizontal rotation. The normal individual experiences vertigo, and nystagmus is observed (beating toward the irrigated ear for a warm irrigation, away for a cold irrigation). A simple office or bedside caloric test uses 3 cc of ice water irrigated against the tympanic membrane over a 15- to 20-second period. The patient must be supine but with the head elevated 30 degrees so that the horizontal semicircular canals are vertically oriented, permitting a maximum convection current. Nystagmus is then observed with the eyes opened, preferably behind Frenzel or cataract lenses, and the direction and duration of nystagmus observed. The two sides can then be compared. It is also useful to know if the caloric test reproduces the patient's symptoms.

Although a simple caloric test can provide useful information, particularly if there is a gross asymmetry in function, it has several drawbacks which are circumvented by the electronystagmogram (ENG). Nystagmus viewed with the eyes open is always less intense than with the eyes closed, and the electronystagmogram allows the

Table 2. Differential Diagnosis of Vertigo

	Peripheral	Central
History		
Degree	Severe	Mild
Temporal features	Episodic	Continuous
Associated symptoms	Otologic	Central nervous system
Physical examination		
Nystagmus	Horizontal, oblique	May be vertical
Romberg	+	+
Gait	+	+
Specific cerebellar tests	−	+
Audiogram	+	−
Electronystagmogram		
Caloric test	+	−
Positional nystagmus	+	+
Optokinetic, gaze, tracking	−	+

+ = often abnormal
− = usually normal

recording of eye movement with the eyes closed; the electrodes placed on the temple detect changes in the corneoretinal potential due to eye movement. Duration of nystagmus, as measured in the bedside caloric test, is not the most accurate indication of the activity of the vestibulo-ocular reflex. Slow phase velocity is much more accurate and can be measured directly from the electronystagmogram. The ENG provides a permanent and objective record, for subsequent reference. In addition, it is possible to perform other eye movement tests which are important in the differential diagnosis. Sinusoidal pursuit tracking, optokinetic nystagmus (a nonvestibular eye movement in which the slow component is a visual pursuit movement), gaze nystagmus, and saccades can all be accurately measured from the electronystagmogram. Although the ENG never makes an etiological diagnosis, it is extremely useful in distinguishing central from peripheral disorders and in localizing the side of the disorder (Table 2).

In cases where acoustic neurinoma is suspected (usually because of asymmetry of auditory or vestibular function), a radiographic work-up is obtained. Polytomography of the internal auditory canal is useful to detect erosion, while computerized tomography (CT scan) will detect tumors of moderate size within the cerebellopontile angle. It is important to note that neither of these techniques can rule out a small tumor; CT scan typically misses tumors smaller than 2 cm in diameter. If the clinical features, audiometric (often including brainstem evoked-response testing), and ENG findings are sufficiently suspicious, one proceeds directly to posterior fossa myelography.

The author routinely obtains an FTA-ABS test on any patient with fluctuating or progressive hearing loss to rule out luetic involvement.

Treatment

Obviously, the first priority is to treat the underlying disorder. The specific treatment modalities for Ménière disease and benign positional vertigo have been mentioned. Demonstrable vascular disease, infection, or neoplasm must be managed as indicated.

A variety of supportive measures are of general applicability. A patient suffering an acute vestibu-

lar spell is usually most comfortable at bedrest in a supine position with the head either straight up or turned so that the affected ear is up. The room should be dim and quiet with a minimum of distraction. This can usually be accomplished in the home, although rarely hospitalization is required. Patients with stable bilateral vestibular failure (or even unilateral failure in the elderly) may profit from physical therapy to help them overcome their ataxia and learn to make maximum use of their remaining sensory input. They also need to learn the situations which they must avoid, such as swimming or walking in unfamiliar environments in the dark. Some patients with frequent dizzy spells, especially if there is considerable emotional overlay and fear, can be helped by "deliberate dizziness therapy."[7,8] This is simply a regimen of emotional desensitization, whereby the patient learns to make himself dizzy several times a day in a comfortable and safe environment and gradually can reduce the unpleasantness of his spells. For the patient with benign positional vertigo, this may only require reproducing the offending head positions several times in succession, with the added bonus that there will actually be a refractory period of one or two hours after each session. For patients with other vestibular disorders, vertigo can be produced by ice water irrigation, which the patient can carry out at home.

Drug treatment is largely empirical. The anticholinergic agents have some effectiveness, presumably because of their inhibition of the autonomic response to vertigo. Many sedatives and tranquilizers have antivertiginous effects as demonstrated both in clinical trials and by animal studies in which inhibition of nystagmus can be demonstrated. However, no specific vestibular sedative is known. Finally, the sympathomimetic drugs have a poorly understood effect.

Because of concomitant nausea associated with acute vertiginous episodes, intravenous or intramuscular routes are usually preferred. Diazepam (5 to 10 mg) and droperidol, a phenothiazine closely related to haloperidol (1 to 3 cc), are extremely useful. Atropine given subcutaneously (0.2 to 0.4 mg) can occasionally abort a Ménière attack, and patients can be instructed to carry this medication and administer it themselves. Antiemetic suppositories (prochlorperazine, trimethobenzamide) are occasionally useful. In the prophylaxis of motion sickness, minor sedatives of antihistamine derivation, such as cyclizine and meclizine, are commonly used. These are moderately effective, although they cause some drowziness. Interestingly, in research studies, combinations including sympathomimetic drugs, such as dextroamphetamine/scopolamine and ephedrine/promethazine, have been the most effective in preventing motion sickness.

Rarely, one can exploit the relatively selective vestibular ototoxicity of streptomycin. In cases of bilateral Ménière disease with disabling attacks of vertigo, low doses of intramuscular streptomycin are given with daily monitoring of audiometric and vestibular function. With this method it is possible to achieve a total ablation of vestibular function with minimal or no hearing loss, although the patient is then left with bilateral vestibular failure.

Surgery is a last resort for a few patients. Labyrinthectomy provides almost certain relief of vertiginous spells in patients with unilateral peripheral vestibular disease, at the cost of total unilateral hearing loss. Direct application of either ultrasonic energy, or cryogenic probes to the surgically exposed semicircular canals offers a reasonable chance of vestibular end-organ destruction without total hearing loss, but these methods have proved unpredictable. In patients requiring surgical relief but whose auditory function on the affected side must be preserved, vestibular nerve section is the procedure of choice.[9] This can be accomplished via a combined otologic-neurosurgical approach through the middle cranial fossa.

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