An Approach to Dizziness

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Dizziness is a vague and ubiquitous symptom which frequently frustrates and perplexes the clinician. Dizziness in the broad sense implies any unpleasant sensation of disturbed relationship to surrounding objects. A number of synonyms such as faintness, light-headedness, giddiness, and swaying, although descriptive, are often no more specific. Problems in diagnosis, therefore, are due not only to the ambiguity of the term but also to the wide spectrum of disorders in which dizziness may be a prominent symptom. Although disorders of the vestibular system usually receive primary attention, it should be kept in mind that many neurologic, cardiovascular, psychiatric, and other disorders are not infrequently associated with dizziness.

The term vertigo, on the other hand, is specific and may be distinguished from other forms of dizziness in that it implies a definite rotational sensation or illusion of motion. Although it is often impossible to make a differentiation between vertigo and other types of dizziness in the individual patient, the distinction is important as vertigo specifically reflects dysfunction in the vestibular system.

The purpose of this paper is to review the anatomy and physiology of the vestibular system, to discuss the most common clinically significant causes of dizziness, and finally, to consider the practical office evaluation of the dizzy patient.

The Vestibular System. A basic understanding of the anatomy and physiology of the vestibular system is necessary in order to understand the procedures employed to test its integrity as well as the clinical manifestation of its dysfunction. The vestibular system includes the end organ (semicircular canals and otolith system), a portion of the eighth cranial nerve, and the vestibular nuclei in the brain stem.[1] Fibers from the vestibular nuclei project to the cerebellum, skeletal musculature, extraocular muscles, and undoubtedly to the cerebrum. The precise connections to the cortex are unknown, although vestibular representation in the posterior temporal lobes is suspected. The vestibular apparatus serves as the primary organ of equilibrium combining with visual and other sensory stimuli to provide sensation of motion and spatial orientation. The vestibular system also functions in the control of skeletal muscle tonus and in stabilizing the eyes during head movements. In the steady state, there is equal tonic neural input from the two end organs. With rotational movement, the semicircular canal in the plane of movement is stimulated, leading to a change of neural activity in the brain stem, which is projected to the cortex to produce the appropriate conscious sense of rotation. A disease state which changes the firing frequency of the end organ, or the neural input from the brain stem, causes vertigo. In such cases the cerebral cortex is seemingly deceived by the brain stem input and interprets it as rotational movement.[2]

Vestibular dysfunction may be classified as peripheral or central, the dividing line being where the eighth nerve enters the brain stem. Peripheral disease is generally felt to be most common and is not infrequently associated with a hearing disturbance due to the close anatomical relationship between cochlear and vestibular portions of the inner ear. A patient with unidirectional jerk nystagmus, vertigo in the direction of the first component, past pointing, and falling in the direction of the slow component is probably suffering from acute dysfunction of the end

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organ on the side of the slow phase. Bidirectional nystagmus varying with direction of gaze, vertical spontaneous nystagmus, past pointing, and falling toward the fast phase points to brain stem disease.[2] Vertigo is usually more intense with peripheral disorders and is commonly associated with nausea and vomiting.

Positional vertigo refers to the vertigo and accompanying nystagmus which occurs after the head is placed in a particular plane. Peripheral and central forms of positional vertigo have also been described, based on the characteristics of the nystagmus induced.[2] In the peripheral type, a latency of 3 to 40 seconds is observed between attaining the precipitating head position and the onset of nystagmus. If the head remains in this position, the nystagmus will decrease or fatigue after 30 to 60 seconds. Gradual habituation of the nystagmus occurs with repetition of the aggravating position and reproducibility is poor at any given examination. With central pathology, the onset of nystagmus is immediate and fatigue and habituation do not occur. Again, vertigo is generally more severe with peripheral disorders.

Otolaryngologic Causes of Dizziness. A variety of otologic disorders ranging from impacted cerumen, and otitis media to otosclerosis and trauma to the labyrinths may produce dizziness. This discussion will be limited to the more common and clinically significant disorders.

Meniere's disease or endolymphatic hydrops is predominantly a disease of middle-age or older, and is unilateral in 90% of cases. The onset is usually insidious, frequently beginning with tinnitus, fullness in the ears, and fluctuating hearing loss. The hallmark of the disease is episodic vertiginous spells usually lasting between 2 and 24 hours. During the attack, pain or numbness may be noted in the affected ear as well as diminution of auditory acuity. Spells recur at irregular intervals varying from days to years.[3]

The majority of patients with positional vertigo suffer from *benign positional vertigo.*[4] In this type, hearing and caloric responses are normal and transient symptoms occur only with assumption of a certain head position. The greatest incidence is between 40 and 60 years of age and symptoms often clear spontaneously within one year. The etiology is unknown.

Acute labyrinthitis may occur as a result of acute or chronic otitis media and is manifested by vertigo, nystagmus, severe hearing loss, nausea, and vomiting. *Toxic labyrinthitis* is usually secondary to ototoxic antibiotics such as streptomycin. Typical vertigo may not occur since both labyrinths are equally depressed. The primary manifestation may be difficulty walking with staggering and inability to stay on course. Hearing loss and high-pitched tinnitus may or may not occur.

Vestibular neuronitis or viral labyrinthitis is primarily a disease of young adults which frequently exists in epidemic form. This disease may be defined as a sudden vestibular crisis lasting days or weeks with partial or total loss of vestibular function in one ear without auditory involvement, and without recurrence.[3] The prolonged period of the attack and the absence of hearing loss and recurrence distinguish vestibular neuronitis from Meniere's disease.

Neurologic Causes of Dizziness. *Acoustic neuromas* are the most common of the cerebellopontine angle tumors comprising approximately 5% of all intracranial tumors. Early symptoms are usually vestibular and cochlear, but the latter usually appear first. The onset of unilateral high-frequency hearing loss and high-pitched tinnitus is often insidious. Vertigo is usually not a prominent symptom and most patients complain of transitory, but progressive, vague dizziness and unsteadiness which is aggravated by movement.[5] This may be explained by the slow expansion of the tumor which allows time for the vestibular nuclei to compensate for the disturbance.[6] Associated signs of involvement of the seventh, sixth, and fifth cranial nerves and cerebellum are usually late, although such signs should be sought and are diagnostic when present.

Although the sudden onset of dizziness is a frequent symptom of *vertebro-basilar cerebrovascular disease,* it rarely occurs in isolation without other symptoms of neurologic dysfunction such as diplopia, dysarthria, facial numbness, or hemiparesis. C. M. Fisher points out that dizzy spells that continue more than six weeks without accompanying symptoms are almost never cerebrovascular in nature.[7] There seems to be an unwarranted tendency to attribute mild giddy spells in elderly persons to "hardening of the arteries."

Dizziness is a frequent and often early symptom in *multiple sclerosis.* Like cerebrovascular disease, however, other signs and symptoms such as visual blurring, diplopia, ataxia, paresis, and paresthesias are almost always present. According to some authors, multiple sclerosis is the most common cause of the central type of positional nystagmus.[6] A unique

feature in this disease is the frequent dissociation of vertigo and nystagmus.

Vertiginous symptoms may occur as ictal phenomena in patients with *seizures of temporal lobe origin.* In such cases consciousness is usually impaired, momentary amnesia is often noted, and nystagmus does not occur. Some patients complain of being hurled into space as if in a tornado.

Cardiovascular Causes of Dizziness. The cardiovascular causes of dizziness have in common the production of symptoms by arrest or diminution of cerebral perfusion. With *orthostatic hypotension,* the patient experiences light-headedness, unsteadiness, and occasionally syncope upon suddenly assuming an erect position. This syndrome may be idiopathic, however, it is often secondary to prolonged bed confinement, anti-hypertensive agents, or impairment of sympathetic vasomotor reflex activity. Such attacks are usually accompanied by pallor, tachycardia, and sweating as well as the obvious drop in blood pressure. *Cardiac disorders* ranging from mechanical obstruction to alterations in cardiac rate or rhythm can impair cerebral blood flow and may produce vertigo and dizziness. In aortic stenosis, the onset of symptoms is usually related to sudden position change. In addition, almost any arrhythmia may produce vertigo and dizziness. The patient may state in such cases that his dizzy episode is associated with a detectable change in cardiac rhythm. *Overactive autonomic reflexes* may also result in dizziness followed by syncope. Hypotension and bradycardia may be triggered by an emotional or painful stimulus as in vasovagal syncope or be precipitated by micturition, paroxysmal coughing, or compression of the carotid sinus in the neck. An impending feeling of syncope characterizes dizziness of cardiovascular etiology.

Anemia and *hypoglycemia* should also be considered among the systemic causes of dizziness.

Other Causes of Dizziness. Dizziness is a common symptom in psychiatric disorders. These patients usually complain of a vague sensation of light-headedness, which tends to be continuous rather than episodic and is associated with symptoms of anxiety, depression, or other hypochondriacal complaints. *Hyperventilation syndrome* is a very common cause of dizziness, and the majority of these patients suffer from emotional problems. Acute attacks of hyperventilation produce relatively circumscribed episodes of light-headedness, frequently with circumoral and digital paresthesias and tightness in the chest. Positional vertigo occurring only after hyperventilation has also been reported.[8] The hypocapnia produced by hyperventilation leads to cerebral vasoconstriction and a reduction in cerebral blood flow.

Drachman and Hart described the occurrence of dizziness among patients with *multiple sensory deficits.*[8] The abnormalities comprising this syndrome consisted of two or more of the following: visual impairment (usually cataracts), peripheral neuropathy, vestibular deficits, cervical spondylosis, and orthopedic abnormalities interfering with ambulation. Typically, patients were elderly diabetics, who only complained of light-headedness when walking and particularly when executing a turn. Dizziness in this group was most closely reproduced by walking and turning the head when standing. Drachman and Hart postulate that the deprivation of accurate sensory information produces disorientation which is referred to as dizziness by its sufferers.[8]

There is considerable disparity between studies regarding the frequency of various causes of dizziness.[6] Most reports deal with a selected and limited segment of the dizzy population. The data compiled by the Northwestern University Dizziness Clinic perhaps best reflect the incidence of various types of dizziness in the general population.[8] This study consisted of 125 patients with the complaint of dizziness of any type without any selection. A reasonably certain diagnosis was claimed in 91% of cases (Table). Disorders of the peripheral vestibular system proved to be the most common cause of dizziness in this study. These patients complained of rotational vertigo, frequently associated with nausea and vomiting, occurring in the absence of evidence of neighboring brain stem deficits. Among the 38% of patients with peripheral vestibular disorders, benign positional vertigo occurred twice as frequently as any other ves-

Table
Major Causes of Dizziness*

Peripheral Vestibular Disorders	38%
Hyperventilation Syndrome	23%
Multiple Sensory Deficit	13%
Psychiatric Disorders	9%
Uncertain Diagnosis	9%
Brain stem Cerebrovascular	5%
Cardiovascular	4%
Neurologic Disorders (others)	4%

* Total is greater than 100% because several patients had more than one diagnosis.

tibular disorder. Meniere's disease and vestibular neuronitis each made up 4% of this total. Hyperventilation and multiple sensory deficits together accounted for over one third of the patients. The relative infrequency of cerebrovascular disease presenting with symptoms of dizziness is pointed out in the 5% figure. These patients, in keeping with C. Miller Fisher's statement,[7] all had evidence of additional neurologic involvement. The absence of cases of seizure and acoustic neuroma indicates that these are rare but nonetheless important causes of dizziness.

Evaluation. On the basis of the preceding discussion of the pathophysiology and causes of dizziness, we will now consider the clinical evaluation of the dizzy patient. Many sophisticated and expensive neurologic and otologic procedures are available. We shall concentrate, however, on practical office techniques and on indications for specialist referral.

The single most important aspect in the evaluation of a patient with dizziness is a good history, properly interpreted. Drachman and Hart[8] have noted that the patient's subjective experience of dizziness can often be separated into one of four types: 1) a definite rotational sensation, 2) a sensation of impending faint, 3) dysequilibrium or loss of balance with little or no head sensation, and 4) vague lightheadedness other than vertigo, syncope, or dysequilibrium.[8] Inquiries need to be made regarding precipitating factors such as relation to posture and motion, the intermittency or constancy of symptoms, and associated symptoms such as hearing loss, tinnitus, nausea or vomiting, and loss of balance.[9] The occurrence of other otologic symptoms (discharge, fullness, or pain in ears) and neurologic symptoms such as disturbances of vision, speech, motor, and sensory function should be sought. Past medical history including significant medical illnesses and use of medications must be obtained.

In view of the wide variety of disorders in which dizziness may be present, a complete physical examination is necessary. In addition, special attention should be directed to certain aspects of the otologic and neurologic examination. The external auditory canal should be checked for impacted cerumen or other abnormalities, and the eardrum carefully examined for evidence of fluid level, hyperemia, bulging, or perforation. Disturbances of hearing may become evident during conversation with the patient. Attention should be paid to the level of voice required for communication, the need for repetition, and the degree to which lip reading is depended upon. Although not as sensitive as standard audiometry, the classic Weber and Rinne tests using an audiometric tuning fork in the 500 Hz range are most informative. Certain aspects of the neurologic examination should be emphasized. The patient should be observed for past pointing. In this test the patient stands, extends both arms, points with both index fingers at the examiner's index fingers held at shoulder height, and then closes his eyes. Drift of the hands consistently in either direction is abnormal.[6] With peripheral disease, the arms will deviate in the direction of the slow phase of the nystagmus. Peripheral disturbances will be aggravated by testing the patient with his head inclined over one or the other shoulder. Past pointing in the direction of the fast phase or in the absence of nystagmus indicates central vestibular pathology.

Disturbances in gait and posture may be seen in patients with proprioceptive, cerebellar, and vestibular disorders. The Romberg test is classically positive in patients with proprioceptive defects; the direction of falling is usually variable. With peripheral vestibular disease, the Romberg test is also positive; however, the direction of fall is consistently to one side and is influenced by changing the position of the head. In cerebellar disease, station and gait are not remarkably influenced by closing the eyes, and ataxia is generally in evidence.

Because of the ever-present concern of the insidious cerebellopontine angle tumor, careful examination of the fifth and seventh cranial nerves should be carried out. The symmetry and strength of the facial musculature and muscles of mastication need to be evaluated. The corneal reflex is simple to test and is most informative. While the patient is looking upward and to one side, the physician touches the cornea on the opposite side with a piece of cotton. If blinking is absent in both eyes, this indicates a loss of sensation in the cornea tested; if blinking is absent only in the eye tested, the homolateral facial nerve may be impaired. Impairment of the homolateral corneal reflex is frequently noted in cerebellopontine angle tumors.

One of the most important components of the evaluation is the dizziness simulation battery. This battery exposes the patient to a number of situations that commonly trigger dizziness in an attempt to simulate the patient's own symptom. These maneuvers include: blood pressure determinations lying and standing, a standard Valsalva maneuver for 15 seconds, head turning with eyes open or closed, a sudden

turn when walking, hyperventilation for three minutes, and the Nylen-Bárány test.[8] The latter maneuver is a test for positional vertigo. The patient is abruptly moved from a seated to a supine position with the head hanging 45° backwards, and turned to one side and then the other. The patient is observed for vertigo and nystagmus, noting the onset, duration, and direction of nystagmus.

Another simple technique which can be performed in the office setting is the caloric test. Not only can vestibular function be assessed by this procedure but also the patient can compare the sensation produced with his own symptoms. One must first make certain that the ear canals are free of debris and that the drums are intact. If available, the use of 15–20(+) lenses or Frenzel glasses will inhibit visual fixation and magnify the eyes for better observation. With the patient recumbent and the head elevated 30°, 3 to 5 cc of ice water are slowly poured into the ear. The eyes should then be observed for nystagmus which should be horizontal with the quick component to the opposite side. How well the two sides agree is most important. A unilateral absent or hypoactive response may be the product of Meniere's disease, vestibular neuronitis or acoustic neuroma. Perversion of nystagmus, such as absence of the quick component, horizontal nystagmus, or a prolonged hyperactive response point to brain stem involvement. It should be emphasized, however, that one should not try to read too much into the findings obtained by the caloric testing.

Routine laboratory studies in the dizzy patient should include: a complete blood count, sedimentation rate, Fasting Blood Sugar (FBS), blood urea nitrogen (BUN), thyroid function studies, electrocardiogram, and skull x-rays with views of the internal auditory canals.[8,9]

A more extensive evaluation is often needed. Patients with objective otologic abnormalities including hearing loss should receive prompt referral to an otologist for audiologic testing and further diagnostic studies. The presence of abnormalities on neurologic examination or the history of ataxia, headaches, disturbance of consciousness, or visual disturbance would necessitate neurologic consultation. Even in those patients with transient symptoms or in whom a benign etiology has been demonstrated, careful follow-up is advisable.

Treatment. The treatment of the dizzy patient obviously depends on the underlying cause. Since a benign condition underlies the majority of dizziness cases, an important aspect of therapy in these patients is reassurance. Patients with peripheral vertigo should be informed about vestibular habituation. In other words, the nervous system will ordinarily adapt to an imbalance between the two end organs and vertigo will ultimately cease. In addition, since visual fixation has an inhibitory effect upon vestibular symptoms, vertiginous patients should resist the tendency to close their eyes and should fix a nearby object.[2] The prophylactic use of drugs for symptoms of dizziness has not been very gratifying. Most of the information on treatment is largely anecdotal. Vasodilators such as nicotinic acid and betahistine are often recommended; however, I am unaware of any data that clearly support their efficacy. Valium® may be beneficial since it apparently exerts some selective sedative effect on the vestibular nuclei.[10]

Meclizine in doses of 25 mg three times a day is perhaps the most popular and effective medication in the treatment of recurrent or continuous vertiginous symptoms. Cohen and DeJong[11] demonstrated in a double-blind crossover study that meclizine was significantly more effective than a placebo in the treatment of vertigo. There was, in addition, no difference in the response of patients with disease of the peripheral or central vestibular systems.

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