

CHAPTER 4

Dizziness

Dizziness is a common, multifaceted, yet often elusive symptom. In its broadest sense, it is a feeling of uncertainty about one's physical and spatial orientation and motion in space. Spatial orientation is maintained by continuous sensory monitoring. These include visual, vestibular proprioceptive pressure sensation. Input from these systems provides an image of our position and motion in space and enables the patient to maintain balance. When these systems provide an invalid orientation system of our position in space patient reports "dizziness". The feeling is nonphysiologic because the patient is not actually having his or her environment moved but is receiving false or conflicting information about their position or motion relative to the environment. For example, when getting off a merry-go-round, a person feels as if he or she is still moving when, in fact, this is not the case. In this case, the limits have been exceeded (physiological dizziness) or when there is pathological dizziness the sensory systems provide inadequate or incorrect sensory input. There are many abnormal sensations that patients describe as dizziness; the first task of the physician, therefore, is to elucidate exactly what the patient means when reporting dizziness.

TERMINOLOGY

The general symptom of uncertainty of position and motion in space can be separated into five major categories each with its own diagnostic significance. The physician must inquire specifically about each in order to firmly establish the type of dizziness the patient is experiencing.(Box 4-1)

In general, dizziness is of a benign etiology but at times can signal significant neurologic or otologic disease.

PATHOPHYSIOLOGY

In order to understand the various clinical syndromes associated with *dizziness*, it is useful to have some basic understanding of the symptom pathophysiology. Dizziness arises when orientation in space is disturbed. Knowing where the body is in relation to the environment and being able to move efficiently throughout that environment without feeling dizzy requires the integration of a set of delicate sensing devices (i.e. proprioceptive, visual, and vestibular) that feed information about the environment and a motor system that responds to the sensory information to maintain equilibrium. Whenever any portion of the system is damaged or gives false information, the system (cerebellar, basal ganglia, corticospinal system) becomes unbalanced and there is a mismatch between the actual orientation of the body in space and what the patient perceives. This mismatch produces a false sense of motion that results in the patient's feeling of dizziness or vertigo.

BOX 4-1

1. *Vertigo*. This is a spontaneous feeling or illusion of movement. This is usually a rotational sensation in which either the patient or the environment is moving. Vertigo represents an illusion of movement. This may occur in horizontal (patient spinning around environment) or vertical plane (rocking up and down as on a boat). True vertigo is usually due to dysfunction of the vestibular system—peripheral labyrinth, vestibular nuclei in the pons, and the flocculus of the cerebellum. Vertigo is usually accompanied by staggering or reeling gait, nausea and vomiting.
2. *Syncopal sensation or presyncope*. This is the sudden feeling of faintness that occurs when circulation to the brain is inadequate. This is due to diffuse cerebral ischemia with inadequate oxygenation of the brain secondary to hypotension, cardiac arrhythmia, stenosis of cerebral vessels, or other circulatory factors. Syncopal sensations may also be due to hyperventilation with reduction of carbon dioxide blood levels and constriction of cerebral blood vessels.
3. *Dysequilibrium*. An imbalance may occur when the patient is standing or walking; this feeling of being off-balance is often called dizziness by the patient. Imbalance or dysequilibrium can be caused by a large number of motor and sensory disorders, e.g., hemiparesis, Parkinson's disease, hydrocephalus, spasticity from myelopathy, sensory loss in the legs from neuropathy or spinal cord disorder, and cerebellar disorders.
4. *Visual-induced dizziness*. A change in visual acuity due to ocular pathology or a recently modified eyeglass prescription may cause dizziness owing to an altered vestibulo-ocular reflex system. This dizziness and feeling of spatial disorientation may be accompanied by occipital or base of skull headache of the muscle contraction type.
5. *Lightheadedness or giddiness*. This is an all-inclusive category for other vague or nondescript sensations of uneasiness in the head. Patients may also complain of a floating or spinning sensation inside the head. It is important to recognize that this symptom is not true vertigo and therefore not necessarily of vestibular origin. This type of complaint is often attributed to emotional causes, but many patients probably have subtle vestibular dysfunction, circulatory problems, sensory disturbances, or visual disturbances.

Of the various sensors, the eyes are the most important, because vision is the basic orienting sense. Even though primary visual disorders infrequently cause dizziness, unless the patient has developed diplopia, a person has only to close one eye and passively move the open eye by applying gentle pressure to the side of the eyeball to appreciate what can happen when the visual information received from the eye is not what the brain expects. How visual input affects balance and orientation is complex and involves integration of vision and eye movement. If such integration did not take place, every eye movement would be interpreted by the brain as a sudden movement of the environment. Most eye movement is initiated in the cortex and then relayed to the vestibular nuclei and lateral gaze center in the pons. From this point, eye movements are integrated by the medial longitudinal fasciculus to the nuclei of the extraocular muscles. Simultaneous with the corticovestibular discharge is what has been called a corollary discharge to the visual cortex to prepare it for the imminent movement. This prevents the potential disorienting effect of the change in visual information.

The second critical sensor of body orientation is the labyrinth. Two types of sensors are present in each labyrinth: (1) the saccule and utricle, which are static receptors to relay information in reference to gravity, and (2) the cristae in the semicircular canals, which are kinetic receptors that sense angular or rotational movement. The labyrinths are in a tonic balance, and dysfunction in one or both will distort information about balance and body movement. This imbalance in labyrinthine input produces a sensation of movement when none is present (i.e., vertigo).

Nerve impulses from the labyrinth are carried by the vestibular portion of the eighth cranial nerve and terminate in the vestibular nuclei and flocculus of the cerebellum. From the vestibular nuclei there are efferents: (1) to the medial longitudinal fasciculus to integrate eye movements; (2) to the cerebellum for balance and limb control; (3) to reticular formation to maintain or stir arousal to prepare for the movement; and (4) to the spinal cord via the vestibulospinal tract to control muscle tone for balance maintenance. Some vestibular efferents project to the cortex in the deep posterior temporal lobe. The major clinical significance of these temporal lobe projections is that when they are stimulated by an epileptic discharge, vertigo will result.

The third major sensor is the proprioceptive system. This system brings in information concerning position and movement of the joints. Particularly rich in proprioceptive receptors are the zygapophyseal joints of the upper cervical spine; these provide all-important information about head position and movement. Touch, pressure, and auditory input also are involved in the overall concept of spatial orientation but much less so than the three principal sensors. In conjunction with this complex sensory system, a responsive motor system is also needed in order to make the necessary postural adjustments to maintain balance. An impaired motor system will produce imbalance but alone should not produce true vertigo.

CLINICAL EVALUATION

The neurologic history and examination must be expanded to assess the complaint of dizziness properly.

History

The physician must review the primary complaint several times in order to establish what the patient is actually experiencing when complaining of dizziness. A series of specific questions should be asked about the symptom. Box 4-2 lists such questions.

Once the full character of the patient's complaint has been delineated, other pertinent medical history can be explored (Box 4-3).

Examination

Standard physical and neurologic examinations should be performed; particular attention should be paid to cerebellar testing and the cranial nerve examination. Look for nystagmus; rotatory, vertical or horizontal. Check finger to nose to examiner's finger; patients with vestibular disease will past point toward the affected side because the healthy vestibular apparatus is tonic and forces the response to the weak side in the same way in which the eyes are forced to that side (i.e., the slow phase of the nystagmus). The patient may also turn to one side if asked to close eyes and take 50 steps in place. After performing the routine examination, the physician should carry out a series of maneuvers in an attempt to reproduce the patient's symptoms. After each maneuver, the patient is asked if this mimics the patient's complaint. The patient is assessed for nystagmus, past pointing, and imbalance while symptomatic. The

different maneuvers are carried out to attempt to reproduce exactly what the patient means by dizziness.

BOX 4-2

1. Is the problem acute or chronic?
2. Was the onset sudden or gradual?
3. Are the symptoms constant or intermittent? If they are intermittent, what will precipitate an exacerbation?
4. Do nausea and vomiting accompany the dizziness?
5. Are the symptoms exacerbated by motion?
6. Are the symptoms exclusively related to the position change or to the assumption of a specific position?
7. Is hearing impaired during the attack?
8. Is tinnitus present?
9. Is there an accompanying sensation of fullness in one or both ears?
10. Do any other neurologic symptoms accompany the dizziness, such as diplopia, weakness, perioral numbness, dysphagia, dysarthria, ataxia, or headache?
11. Does the patient feel off-balance? Is there a tendency to fall to one side?
12. Are symptoms worse with the eyes opened or closed?

BOX 4-3

1. Is cardiac disease or other chronic medical illness present?
2. Does the patient have a decrease in vision, hearing, or peripheral sensation in legs or arms?
3. Has there been any recent viral illness?
4. Does the patient have an ear or sinus infection?
5. Has there been recent head trauma? If so, is it related to the symptoms?
6. Has there been neck trauma, or does the patient have neck pain or stiffness?
7. Has the patient been exposed to any toxic substance (e.g., pesticides, hydrocarbon vapors)?
8. Is the patient on any medications? Many medications cause dizziness as serious potential adverse effect.
9. Does the patient use alcohol? What effect does its use have on the dizziness?
10. Are there psychiatric or emotional problems?

Dizziness Simulation Battery Maneuvers

Box 4-4 details the seven maneuvers carried out in Dizziness Simulation Battery tests developed by Drachman.

BOX 4-4

1. Blood pressure and pulse rate change from supine to erect posture. Pressure is checked with initial rise and again in 3 minutes to see if the patient compensates for the normal initial drop. Observe for orthostatic hypotension.
 2. Hyperventilation for 3 minutes. This normally causes lightheadedness.
 3. Valsalva maneuver. The patient squats 30 seconds, then strains 15 seconds. This will usually simulate syncopal sensation.
 4. Carotid sinus stimulation. Gentle unilateral noncompressive stimulation is provided for 10 seconds with careful pulse monitoring. This may induce bradycardia and syncope.
 5. Rapid turns when walking and rapid passive head movements by the examiner (flexion and extension as well as rotation) are performed. If vertigo is elicited only when the neck is twisted and not with a change in body position with the neck immobile (e.g., lying down or full body turn), the problem is not due to labyrinth disease but this suggests cervical spine disease or vascular compression of the carotid or vertebral arteries.
 6. Nylen-Bárány or Hallpike maneuver. Seat the patient on the examining table or hospital bed, hold the patient's head in both hands and then gently but rapidly lower the patient backward until the head is 45° below the edge of the table or bed. While lowering the patient back, the examiner simultaneously turns the patient's head to the side. This maneuver will test for positional vertigo.
 7. Spinning. The patient is seated in a swivel chair that is rapidly spun around (10 times in 20 seconds). This will produce true vertigo in most normal patients.
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DIAGNOSTIC ASSESSMENT

Based on the clinical features, it is possible to characterize the type of disorder causing the complaint of dizziness and to determine the diagnostic studies that are most useful. If dizziness is associated with an illusion of motion, this is most likely due to a primary vestibular disorder. If there is tinnitus and autonomic dysfunction (e.g., perspiration, nausea, etc.) without hearing loss or neurologic dysfunction, this indicates a peripheral vestibular or labyrinthine disorder. If symptoms due to peripheral vestibular disorder do not resolve rapidly, referral to an otologist is warranted. Further testing including hearing tests with pure tone audiography (impaired with cochlear disorders) and speech discrimination (impaired with retrocochlear disorders) are indicated. If vertigo is associated with cochlear hearing loss pattern, Ménière's disease is suggested. If vertigo is not associated with hearing loss or tinnitus, the patient may have a drug-induced or toxic disorder involving the vestibular system.

If vertigo is not associated with tinnitus and the neurological examination is normal, it is important to perform hearing tests and caloric testing. Based on results of auditory or vestibular function tests, it should be possible to differentiate labyrinthine and cochlear disorders from retrolabyrinthine and retrocochlear (lesions affecting eighth nerve, brain stem, cerebellum) conditions. Electronystagmography demonstrates nystagmus by recording eye movement deflections by a patient and may detect nystagmus too subtle to be visualized by clinical observation alone. Electronystagmography may also help to diagnose dysfunction of the vestibular system and to determine if the lesion is in the peripheral or central portion of the system.

Retrolabyrinthine disorders include (1) mass lesions affecting the eighth nerve and

cerebellopontine angle, (2) brain stem (including multiple sclerosis) and cerebellar lesions, (3) vascular lesions (aneurysm, angiomas), ectatic vessels of vertebrobasilar arterial system, and (4) petrous bone apex and tentorial lesions. If an eighth nerve or a cerebellopontine angle lesion is suspected, contrasted MRI or CT scan should be performed. MRI scanning also delineates brain stem, cerebellum and petrous apex better than CT. If a retrolabyrinthine or retrocochlear lesion is suspected and imaging procedures are negative, brain stem (auditory) evoked potentials may be helpful in delineating the location of the lesion and can help to differentiate cochlear from retrocochlear hearing loss. In cochlear disorders, the patient has reduced pure tone audiogram; whereas in retrocochlear lesions, there is more difficulty understanding speech such that patient has marked difficulty understanding speech especially when talking on the telephone, or when in a crowded noisy room.

If a patient's complaint of dizziness is not associated with an illusion of motion but is more of a vague lightheadedness, this is frequently not a vestibular dysfunction and in such cases, vestibular and auditory tests are not needed. If the symptoms seem secondary to anxiety, the dizziness can often be reproduced by hyperventilation. If there are associated cardiac symptoms, medical-cardiac evaluation may be required. If symptoms are precipitated by standing, postural hypotension is suggested. In the elderly patient there may be multiple factors including sensory loss, hearing decrease, visual decrease, medical problems and also the effects of medications, on the vestibular and nervous system.

CLINICAL SYNDROMES

Labyrinthine or Peripheral Vestibular Disorders

In general, labyrinthine or peripheral vestibular disorders are characterized by intense vertigo. The vertigo is dramatically exacerbated by movement. The onset is occasionally so sudden and severe that the patient may fall to the floor. Nausea and vomiting are common. Nystagmus is usually present and is rotational or horizontal but rarely vertical. Ocular fixation may suppress both vertigo and nystagmus. Because of the tonic effect of the labyrinth, slow phase of nystagmus is toward the side of damaged labyrinth. Nystagmus decreases with gaze toward impaired side and increases with gaze toward intact side. On finger-to-finger testing, patient will past-point toward damaged side and will fall to that side on tandem walking. Symptoms are worse when lying with involved side down. Caloric testing shows decreased response on affected side. The condition may be episodic; in fact, any patient with episodic vertigo most likely has peripheral rather than central (i.e., brainstem/cerebellar) disorder.

Acute Peripheral Vestibulopathy (Acute Vestibular Neuronitis, Acute Labyrinthitis)

Acute peripheral vestibulopathy occurs most commonly in young adults. The onset is acute in 75% of the cases. Vertigo, nausea, oscillopsia (experience of objects moving in the environment due to marked coarse nystagmus), imbalance, and motion sensitivity are present. Nystagmus due to peripheral labyrinthine or vestibular disorder may be horizontal or rotatory but rarely vertical (this indicates brain stem disease). The patient frequently is sweaty and has skin pallor. Tinnitus and sensation of fullness in the ear are experienced by some patients. Posterior headache and fatigue may also be present in the recovery period following vertigo episode. The etiology is uncertain, but approximately 50% of the patients report recent upper respiratory tract infection. The severe symptoms last from a few days to about a week, and mild, gradually diminishing uneasiness typically continues for 2 to 6 weeks. Rapid movement, strenuous

exercise, emotional stress, and alcohol consumption can exacerbate symptoms. Treatment is symptomatic, with bed rest and anticholinergic medication every 4 to 6 hours affording some relief (scopolamine, 0.6 mg; promethazine [Phenergan], 25-50 mg; meclizine or cyclizine [Antivert, Bonine], 25-50 mg; diphenhydramine [Benadryl] 25-50 mg; or dimenhydrinate [Dramamine], 50 mg). Side effects include sedation and dryness of mucous membranes. In intractable cases diazepam (Valium), lorazepam (Ativan), and methylphenidate (Ritalin) have been used. The goal of these therapies is to restore the balance between impulses arriving in the brain stem. Vertigo is result of vestibular mismatch from the two ears – sedatives dampen firing rate of over-firing side and stimulants increase firing rate from the under-active vestibular system. In some cases symptoms will recur 6 weeks after the initial bout.

Benign Recurrent Vertigo

Benign recurrent vertigo is an uncommon condition that occurs both in children (ages 3-7) and adults. In children, it is characterized by abrupt onset and short duration (often less than 1 minute). The child looks pale during the attack and may vomit. The attacks recur from daily to semiannually, with an average of 8 to 12 per year. Caloric testing is usually abnormal. Spontaneous remission occurs. In adults the condition tends to be familial. The attacks last hours rather than seconds, the condition tends to be chronic, and caloric testing is normal.

Infection

The labyrinth can be affected by either systemic illness (particularly viral disease such as mumps, measles, rubella, and herpes zoster) or as an extension of otitis and mastoiditis. In chronic suppurative otitis (an increasingly rare disease) labyrinthine fistulas may develop. When this occurs, any abrupt rise in middle ear pressure from sneeze or cough will trigger sudden severe attack of vertigo. In syphilitic labyrinthitis, there is progressive impairment of vestibular and auditory function to cause vertigo and deafness.

Benign Positional Vertigo [BPV]

BPV is one of the most common causes of vertigo. This presents as dizziness or vertigo of abrupt onset and frequently is misdiagnosed as a serious condition such as stroke or multiple sclerosis. The symptoms may be frightening to the patient and trigger emergency department visit; however, the lack of neurological dysfunction makes serious neurological illness unlikely. These patients experience vertigo when they place their heads in a specific position. Most commonly symptoms occur when the patient lies down or turns over in bed. Symptoms begin after a 2 to 10-second latency period and usually last about 20 seconds. These patients may also report more constant background dizziness, which is different from vertigo. The symptoms can be reproduced with the Nysten-Bárány maneuver and during the period of vertigo, nystagmus is present. The response will fatigue with repeated testing and subsequently remains resistant to further change for many hours. For some patients, repeated self-produced attacks become a satisfactory method of treatment. Most cases are idiopathic, but predisposing factors include head trauma, advancing age, inactivity, and ear disease. Age of onset is usually in the late 50's. The etiology is uncertain, but probably is due to a loose otolith or loose calcified particles in the ampullae of the semicircular canals (usually in the posterior canals)—so-called cupulolithiasis. Treatment consists of repositioning exercises with the goal of moving the free-floating particulate material from the posterior semicircular canal to an alternative less sensitive position within the vestibular labyrinth. This provides rapid and complete relief of the vertigo. BPV does not respond to vestibular suppressants or antiemetics as do other forms of vertigo. If

symptoms recur, consider perilymphatic fistula and refer to ENT.

Ménière's Disease

Ménière's disease is relatively common and is characterized by an excess of endolymph with resultant distention of the membranous labyrinth. Onset is usually in the 40s. The classic syndrome consists of recurrent attacks of acute vertigo, tinnitus, decreased hearing and full feeling in the affected ear. In initial attacks, vertigo may be the only symptom. The frequency of attacks varies and long periods of quiescence are not uncommon (e.g., 2-3 yrs). The caloric response is decreased on the side of the disease. The hearing loss is sensorineural with cochlear features. This cochlear pattern of hearing loss is characterized by low-frequency hearing loss with preserved speech (word) discrimination. The hearing impairment is usually unilateral but may be bilateral. When bilateral, the hearing loss may be asymmetric. This cochlear pattern is contrasted to the findings in retrocochlear lesions such as cerebellopontine angle tumors. In these cases, there is unilateral high-frequency hearing loss and impaired speech discrimination. If there is any question, the differentiation between a cochlear and retrocochlear lesion can be made by brain stem (auditory) evoked potentials and other hearing tests. Tinnitus may persist between attacks, and in some cases hearing loss precedes vertiginous attacks. With repeated attacks, hearing is gradually lost, with low-frequency tones being the first to be lost. The labyrinth can eventually be destroyed and at that point, clinical symptoms disappear. The disease has a familial tendency and is bilateral in 10% to 20% of the cases. Diagnosis is usually made by history; however, documentation of fluctuating low-frequency hearing loss is diagnostic. Medical treatment with anticholinergics, diazepam, and diuretics has not been very rewarding, and many patients are treated with surgical procedures that destroy the labyrinth or cut vestibular portion of eighth nerve. Abstaining from smoking, drinking, and stressful situations is helpful.

Ototoxic Substances and Metabolic Etiologies

Aminoglycoside antibiotics (kanamycin, streptomycin, and gentamicin), phenytoin, phenobarbital, quinidine, diazepam, aspirin, and alcohol are all known to have vestibular toxicity. Usually prompt withdrawal will reverse the problem, but unfortunately this is not effective in every case. In some alcoholic patients with or without Wernicke-Korsakoff syndrome dizziness may be a prominent symptom. Hypothyroidism may also cause vestibular dysfunction.

Vertigo Due to Migraine

Some patients with basilar migraine will experience vertigo as an aura or concurrent symptoms with their migraine headache. At times, vertigo may be the only symptom of the migraine attack—the so-called migraine equivalent or migraine accompaniment.

Disease of the Eighth (Acoustic) Nerve

Acoustic neuromas (neurinomas, schwannomas) or granulomatous disease such as sarcoid will occasionally affect the eighth nerve. Vertigo, when present, is persistent yet much less severe than in labyrinthine disease. Because of its close association with the auditory nerve, hearing is usually impaired. Because eighth nerve is joined by seventh nerve along its course through petrous bone, facial weakness may be observed. Tinnitus is usually present, but fullness in the ear is not. Motion sensitivity is not common but can be present. The neoplasm initially

widens the internal acoustic meatus. Diagnosis is established by CT/MRI.

Tumors in the Cerebellopontine Angle

The eighth nerve is often involved in mass lesions in this area, but vertigo is seldom symptom. Hearing loss and decreased facial sensation and reduced facial (upper and lower) motility are often present. Cerebellar signs and eventually fifth, sixth, ninth, and tenth nerve dysfunction occur in larger lesions. Diagnosis of these tumors is established by CT/MRI and treatment is surgical. Consider this lesion if patient has history of neurofibromatosis.

Central Lesions

Lesions involving flocculonodular lobe of the cerebellum or vestibular nuclei in pons produce vertigo. Ischemia, with or without infarction or hemorrhage in these areas frequently produces vertigo. Multiple sclerosis plaques and mass lesions also produce vertigo, but they present a more subacute course. Vertigo may actually be the initial symptoms in some patients with multiple sclerosis; however, the patient usually has other associated neurologic signs on examination. There are several clinical features that differentiate central lesions. Most important is the presence of other signs and symptoms of brain stem or cerebellar damage (Horner's syndrome, diplopia, weakness with upper motor neuron signs, sensory signs, and cerebellar findings). Nystagmus is vertical or horizontal and not usually rotatory. Ocular fixation will not suppress the vertigo or the nystagmus. On Nylen-Bárány maneuver, nystagmus appears as soon as the head is positioned and, unlike labyrinthine-induced nystagmus, is prolonged and nonfatiguing. Positional exacerbation may occur but is not as dramatic. With structural lesion such as brain stem infarct, symptoms usually last several months, but they can be present up to a year. The best symptomatic treatment of central lesions is with diazepam (Valium). Anticholinergics may help, but their efficacy is marginal. MRI scanning is the preferred imaging study when central lesion is suspected. CT scans do not usually adequately image the brainstem and cerebellum and are also very insensitive in diagnosing multiple sclerosis.

Dizziness as a symptom of cerebrovascular disease has been misunderstood and greatly overdiagnosed. This is particularly true of transient ischemic attack (TIA). Fisher has reviewed his experience, and his statistics are useful in helping place the symptom in its proper perspective. First, documented vertigo in ischemia or completed stroke from carotid or middle cerebral lesions is very rare. In posterior cerebral artery occlusion, there is a 25% incidence of vertigo, but the symptom is usually not severe. Basilar artery occlusion renders 75% of its victims dizzy, and in 25% it is the first symptom. Finally, 80% of the patients with vertebral artery occlusion experienced dizziness, along with the other symptoms and signs of Wallenberg's syndrome. As a general rule, dizziness is only found in vertebrobasilar vascular disease not carotid disease, and is always accompanied by other brain stem or cerebellar signs. Dizziness alone, particularly if it is recurrent and has been present for months or even years, should *not* be attributed to vertebrobasilar insufficiency. One final comment on dizziness in vascular disease: potentially treatable cerebellar hemorrhage often presents as vertigo. For this reason, any older patient, especially the hypertensive person who is seen for the sudden onset of vertigo, should be examined very carefully for cerebellar signs and undergo computed tomography as soon as possible. In evaluating patients with dizziness, CT/MRI are not indicated unless there are accompanying other neurological symptoms or signs, major cerebrovascular risk factors, or findings of central disorder on the electro-nystagmogram.

Epileptic Dizziness

Some seizure patients will experience true vertigo as an initial symptom of their seizure or as the sole manifestation of their epilepsy. Epileptic dizzy spells usually begin suddenly and only last for a few seconds. There is no exacerbation with position change. Nausea is common. In most cases, epileptic focus is demonstrated in posterior temporal region on electroencephalogram. Most patients have other manifestations of a partial complex seizure disorder and not vertigo as the sole manifestation of their epileptic disorder.

Medical Illness

Dizziness, at times, vertiginous, is known to occur in diabetes mellitus, thyroid disease, and other endocrine diseases. Hypotension (often secondary to medications) and a multiple cardiac diseases are causes of presyncopal variety of dizziness.

Sensory or Multisensory Disorder

Because spatial orientation is directly related to the information received through the sensors, any imbalance, disruption, or distortion of input can lead to a feeling of spatial disorientation, dysequilibrium, or dizziness. Severe proprioceptive loss such as that seen in tabes dorsalis or severe peripheral neuropathy will often cause dysequilibrium but rarely vertigo unless the patient is in the dark. The elderly often suffer with multisensory deficit—decreasing vision, hearing, and proprioception. These factors coupled with slower righting reflexes produce cautious, uncertain gait (presbystasis). Minor missteps or rapid movement can trigger imbalance, lightheadedness, and vertigo. Treatment for this condition basically falls on the patient, who must learn to be cautious, not to move quickly, and to carefully focus the eyes in the proper direction before starting off. Some patients benefit by dragging a long cane along so they always feel and hear where the ground is located. The elderly often take medications, which affect blood pressure, or have cardiac conditions that allow variations in cardiac output; therefore, it is the combined effect of multiple factors that leads to dizziness in these persons.

Cervical Spine Disease

Experimental studies have shown that stimulation of proprioceptive fibers in the zygapophyseal joints of upper cervical spine can produce vertigo. The extrapolation from these studies is the hypothesis that cervical spondylosis and arthritis of the facet joints may cause irritation of the receptors for proprioception and thus induce clinical vertigo. If cervical vertigo is suspected, anti-inflammatory medications are often useful in treatment of the condition.

Psychogenic Dizziness

Many patients with emotional problems experience dizziness; this can be lightheadedness, giddiness, or vertigo. This type of problem is most commonly seen in anxiety states and panic attacks, but it is also present in depression, hysteria, and schizophrenia. The symptoms are often vague. In some patients, hyperventilation is major factor in their lightheadedness sensation. The vestibular system in the brain stem has rich interconnections with the reticular formation, so it is not surprising from an anatomic point of view to find the heightened arousal state of anxiety associated with vertigo and lightheadedness. In anxiety, eye and head movements tend to be furtive, thus producing rapidly changing visual input that further taxes integrative capacity of

orientation mechanisms. These and probably other physiologic and chemical changes are responsible for complaint of dizziness in anxious or depressed patients.

Other Disorders

There are always patients who cannot be neatly classified or diagnosed. In some, they may have partial syndromes or a combination of many factors. The examiner must also realize that there is a great deal of variation in sensitivity of vestibular apparatus in normal population. Some patients are sick after one turn of a merry-go-round, whereas others can fly into space or barrel-roll high-speed aircraft all day without slightest degree of disorientation.

SUMMARY

Dizziness is a common, yet complicated complaint to evaluate. Exact characterization of the complaint separates the principal types of dizziness: vertigo, presyncope, imbalance and nonspecific light-headedness. True vertigo is usually due to a disturbance in the labyrinthine system either peripherally or centrally. Separating central from peripheral lesions is very important for the peripheral diseases tend to be benign whereas the central lesions are often destructive (e.g., tumor, stroke, demyelinating plaque).

Suggested Readings

Evaluation of Dizzy Patient

Drachman DA: *A 69 year old man with chronic dizziness*, JAMA 280:2111, 1988.

Froehling DA and Silverstein MD: *Does this dizzy patient have a serious form of vertigo*, JAMA 271:385, 1994.

Gizzi M: *The diagnostic approach to the dizzy patient*, Neurologist 4:138, 1998.

Hoffman RM: *Evaluating dizziness*, Am J Medicine 107:468, 1999.

Baloh RW and Honrubia V: *Clinical Neurophysiology of the Vestibular System*. F.A. Davis Co., Philadelphia, 1979.

Drachman DA and Hart CW: *An approach to the dizzy patient*, Neurology 22:323-334, 1972.

Furman JM and Jacob RG: *Psychiatric dizziness*, Neurology 48:1161, 1997.

Hotson JR and Baloh RW: *Acute vestibular syndrome*, NEJM 339:680, 1998.

Kroenke A and Valvassori G: *Benign positional vertigo*, Neurology 37:371, 1987.

Korgeorgos J, Scott DF, Swash M: *Epileptic dizziness*. Br Med J 282:687-689, 1981.

Kumar A, Valvassori G: *An algorithm for neurologic disorders*. Neurol Clin North Am 2:779-796, 1984.

Positional Vertigo

Baloh RW and Honrubia V: *Benign positional vertigo*, Neurology 37:371, 1987.

Furman JM and Cass SP: *Benign paroxysmal positional vertigo*, NEJM 241:1590.

Vertigo Due to Neurological Disease

Baloh RW: *Stroke and vertigo*, Cerebrovascular Disease, 2:3, 1992.

Grad A and Baloh RW: *Vertigo of vascular origin*, Arch Neurology 46:281, 1989.

Korgeorgos J, Scott DF, and Swash M: *Epileptic dizziness*, Br Med J 282:687, 1981.

Kumar A, Torok N: *Neurological diagnosis of intracranial lesions*. Otorhinolaryngology 30:138-140, 1982.

Treatment of Dizziness

Hain TC: *Treatment of vertigo*, The Neurologist 1:125, 1995.

Tinnitus

Marion MS and Cevette MJ: *Tinnitus*, Mayo Clinic Proceedings 66:614, 1991.

Meniere Disease

Shea JJ: *Classification of Meniere disease*, American Journal Otolaryngology 14:224, 1993.

Slatter YWHR: *Medical treatment of menicre disease*, Otolaryngol Clin North Am 30:1027, 1997.