

The Dizzy Patient: How You Can Help

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Painstaking elucidation of a patient's symptoms is the key component of the diagnostic workup for dizziness and vertigo. A rational, straightforward, and cost-effective approach that uses minimal, selective diagnostic testing can get to the root of an individual's specific problem. Dizziness is a nonspecific complaint that can have different meanings in different patients, which makes careful analysis of the patient's symptoms the most critical component of the diagnostic workup (Table 1). When they are questioned about the specific sensations that characterize their "dizziness," patients will typically describe one of the following situations. Light-headedness and imbalance that occur when assuming an upright posture This common complaint of presyncope is usually attributable to cerebral hypoperfusion when patients rise from a sitting or supine position. It is typically worse in the morning after prolonged bed rest. Patients do not experience these symptoms when they assume a supine position. These periods of cerebral hypoperfusion may result from obstruction in the carotid and vertebrobasilar circulations, typically secondary to atherosclerosis. More commonly, symptoms result from a dysautonomia, which prevents an appropriate cardiovascular response to changes in posture. Dysautonomias are most commonly associated with antihypertensive or antiarrhythmic therapy (eg, β -blockers, calcium channel blockers, α -blockers, angiotensin-converting enzyme inhibitors, amiodarone).¹ Primary dysautonomias (such as Shy-Drager syndrome) are rare; they are suggestive of multisystemic autonomic dysfunction. Diagnosis of this form of dizziness, which is more common in the elderly, is usually straightforward. Symptoms occur only when the patient rises; typically, there is a history of cardiovascular disease and/or diabetes mellitus. Bedside examination (lying and standing blood pressure testing with a postural drop of 20 mm Hg systolic and/or 10 mm Hg diastolic) may confirm orthostatic hypotension; however, the lack of this physical finding should not rule out the diagnosis if the patient's history is highly suggestive. Frequently, autonomic function testing using a tilt table will elicit symptoms accompanied by a drop in blood pressure that cannot be demonstrated at the bedside. A stenotic lesion can be ruled out with transcranial Doppler echocardiography or magnetic resonance angiography of the neck. Treatment of presyncope may be as simple as advising the patient to rise slowly, to squeeze his or her legs together before rising, or to wear support hose. Altering medications or adjusting the dosages also may help. Under certain circumstances, pharmacotherapy may prove beneficial.² Objective imbalance Patients may equate an inability to maintain normal gait with dizziness, even if they are not suffering from true rotatory vertigo. This includes ataxia; however, this article does not specifically address ataxia. Vague sensation of light-headedness, subjective sensations of imbalance These complaints, which can be characterized by their imprecision, are consistently among the most common that clinicians encounter when evaluating dizziness. Patients describe a variety of sensations, including feeling far away or somewhat detached from themselves or their environment, a tightness or fullness in their heads, or a sensation of imbalance that never progresses to the point at which they actually fall or veer. These symptoms are all characteristic of psychogenic dizziness. Barber³ has noted that this diagnosis is suggested during the first 5 to 10 minutes of the office visit if the patient has no specific organic complaints. Eliciting a history from these patients can be frustrating; often, they cannot describe their symptoms precisely. Rather than feeling frustrated, you can feel encouraged because the nonspecific, nonphysical nature of the complaints leads to a specific diagnosis. The physical examination is usually normal in these patients, except that hyperventilation typically reproduces their symptoms. Psychogenic dizziness represents a chronic anxiety disorder with associated panic and/or phobic disorders. Although the patient's history may strongly suggest psychogenic dizziness, a full neurologic history taking and physical examination must be performed, and selective tests such as electronystagmography also are frequently ordered. This is done to reassure the clinician and—perhaps even more important—the patient that no organic disease is present. Treatment of these patients incorporates typical therapeutic strategies used in the treatment of anxiety disorders.

Slowly increasing doses of selective serotonin reuptake inhibitors are the mainstay of treatment, often coupled with psychotherapeutic approaches such as cognitive behavioral therapy. Central Vertigo True vertigo is an illusion that the environment is moving (typically, rotating or spinning). The sensation is usually accompanied by nausea. Vertigo may be of central (brain stem or cerebellum) or peripheral (inner ear or vestibular nerve) origin. Once you have determined that the patient is suffering from vertigo, the next step is to establish whether this represents central or peripheral disease (Table 2). The history and physical examination are the most useful tools in determining the site of a suspected lesion. Central vertigo Disorders of the lower brain stem and cerebellum-including ischemia, demyelination, migraine, and rarely, neoplasm-are responsible for central vertigo. Ischemia The patient presenting with vertigo resulting from vertebrobasilar insufficiency, a transient ischemic attack, or a cerebral vascular accident involving the brain stem will typically present with associated symptoms that may include diplopia, dysarthria, dysphagia, drop attacks, paresthesias, and loss of motor function. Cerebellar dysfunction may be more difficult to rule out based on history because symptoms of a cerebellar stroke can sometimes closely resemble those associated with peripheral vestibular disorders. However, on physical examination, patients with a cerebellar stroke demonstrate difficulty in rapidly alternating supination and pronation of the hands and may perform poorly on finger-to-nose testing (dysdiadochokinesia). Patients who display these signs and/or who have significant risk factors for ischemic disease, such as smoking, hypercholesterolemia, or diabetes mellitus, should undergo appropriate radiologic studies in order to rule out a cerebral vascular accident. Multiple sclerosis (MS) Vertigo is the initial complaint in approximately 5% of patients with MS and eventually is observed in up to 50% of those with this disorder.⁴ Migraine This common cause of central vertigo will be discussed below.

Peripheral Vertigo Unlike vertigo of central origin, vertigo originating from dysfunction of the inner ear or eighth cranial nerve has few associated symptoms. When these symptoms are present, they are typically related to auditory dysfunction. Thus, peripheral vertiginous disorders are best classified based on the duration of the actual vertigo attacks, as well as on the presence or absence of symptoms of unilateral auditory dysfunction. Determining the duration of actual vertigo-as distinct from constitutional symptoms connected with the event (eg, nausea or fatigue)-is critical to establishing the diagnosis. Rarer causes of peripheral vertigo are discussed in the Box. Episodes that last for seconds Patients with benign positional vertigo (BPV) experience episodes of vertigo lasting less than 1 minute that are brought on by a rapid head movement in a nonaxial plane (eg, rolling over in bed or looking up rapidly). As soon as the patient steadies himself, the vertigo resolves. BPV is the most common peripheral vestibular disorder. It is typically idiopathic, but it may occur because of head trauma or subsequent to a vestibular neuronitis or labyrinthitis (see below). BPV is thought to result from the accumulation of organic debris (canaliths) within one of the semicircular canals of the inner ear-typically, the posterior canal.⁵ The diagnosis of BPV can be made from the history; it can be confirmed by the Dix-Hallpike (or Barany) maneuver. This consists of moving the patient from a sitting to supine position, with his head turned and hanging over the head of the bed or table so that the affected ear faces the floor. The elicitation of vertigo and nystagmus with the patient in this position confirms the diagnosis of BPV. The prognosis for this disorder is excellent, since the natural course is spontaneous remission. However, the duration of the symptomatic period varies and may persist for months. During this time, the patient may be incapacitated because of recurrent episodes of vertigo and the fear associated with these unpredictable attacks. Recently, a safe, simple, and effective treatment-the Epley canalith repositioning maneuver-has been developed. This technique incorporates positional maneuvers performed at the bedside that cause the canaliths to fall out of the semicircular canal and into the labyrinthine vestibule, where they cause no adverse effects. This treatment eliminates vertigo in more than 90% to 95% of cases and allows the patient to resume a normal lifestyle.⁵ (Nevertheless, BPV tends to recur, and although the Epley maneuver eliminates the acute episodes, it does not prevent recurrences, which may occur months to years after the initial diagnosis.) Episodes that last for minutes or hours There are no peripheral vestibular disorders that cause episodes of vertigo lasting for minutes. The only disorder that typically causes vertigo of such a duration is vertebrobasilar insufficiency (discussed above). If vertigo lasts for minutes and is accompanied by central neurologic symptoms, evaluation of the posterior fossa circulation, typically with arteriography, is warranted. Two disorders cause episodes of vertigo that last for hours: Meniere disease and migraine. These entities are differentiated by the presence or absence of auditory dysfunction. Episodes of vertigo lasting for hours, fluctuating and progressive sensorineural hearing loss, and tinnitus constitute the trio of symptoms that defines Meniere disease. Aural fullness or pressure is commonly reported as well. Meniere disease is an idiopathic disorder that typically occurs in patients aged between 30 and 60 years. It ultimately affects both ears in 45% of patients.⁶ No

definitive theory for the pathogenesis of this disorder exists. Although it has generally been attributed to the accumulation of fluid within the inner ear (endolymphatic hydrops), the validity of this theory has recently been questioned. The history strongly suggests the diagnosis. Physical examination may reveal evidence of hearing loss on tuning-fork testing, as well as signs of peripheral vestibular loss (eg, poor performance on tandem gait assessment with eyes closed-although in older patients, this examination is less valid). The typical disease course consists of clusters of vertiginous episodes separated by periods of remission. Hearing loss and tinnitus are usually exacerbated during the vertiginous episodes. In most patients, the disease "burns out," and they are left with chronic moderate to severe hearing loss, tinnitus, and imbalance, particularly in the dark. The treatment of Meniere disease is controversial^{7,8}; a comprehensive discussion is beyond the scope of this article. Note that this condition has an extraordinarily high (60% to 80%) short-term response rate to nonspecific (placebo) therapies. The medical management of this disorder has focused on vestibular suppressants (eg, diazepam or meclizine) to control vertigo and diuretics and a low-sodium diet to decrease hydrops. Of these therapies, only vestibular suppressants have proved effective in controlling vertigo; none has been shown to improve auditory function. Nonetheless, many of these agents remain in use; their beneficial effects are likely attributable to the nonspecific therapeutic responses seen in patients with Meniere disease. Long-term use of vestibular suppressants should be discouraged because their use prevents accurate diagnosis and treatment, prevents central compensation to a peripheral vestibular loss, and may predispose elderly persons to falling. Surgical therapies include those that attempt to preserve hearing (endolymphatic shunt, gentamicin perfusion, or vestibular nerve section) and those in which hearing and vestibular function are lost (ie, labyrinthectomy, which is performed only when the patient's hearing is virtually nonexistent). Indications for these surgeries are controversial; discussions of the options may be found elsewhere.^{7,9,10} Vertigo lasting for hours without significant auditory symptoms is most commonly migrainous in origin (85% to 90% of cases).¹¹ The typical presentation is recurrent episodes of true vertigo occurring in a patient with a personal or strong family history of migraine. The vertiginous episode need not be directly associated with the headache. There is considerable confusion concerning this disorder, which has been referred to variously as recurrent vestibulopathy, benign recurrent vertigo, or vestibular Meniere disease, and disagreement still exists about its cause. Vestibular suppressants are recommended to suppress acute vertigo. Patients experiencing frequent attacks should be treated with a migraine prophylaxis. In a small percentage of patients (5% to 10%), the episodes of vertigo serve as a prelude to the development of full-blown Meniere disease, which usually occurs within 1 to 2 years after the initial episode. An equal number of patients have no history of migraine and will never have Meniere disease. This idiopathic disorder is best referred to as recurrent vestibulopathy.⁹ Vestibular suppressant medications are the mainstays of treatment for these 2 groups of patients. Episodes that last for days to weeks Vestibular neuronitis is a common and frightening disorder that often precipitates a visit to the emergency department. It is characterized by an acute onset of vertigo associated with nausea and vomiting, but no symptoms of auditory or CNS dysfunction.¹² The lack of associated symptoms, the absence of dysdiadochokinesia, and the fact that symptoms do not recur are key factors that help differentiate this relatively benign disorder from other neurovestibular pathologies. The vertigo slowly remits over a period of days to weeks. Vestibular suppressants are the mainstay of treatment for vestibular neuronitis; corticosteroids may reduce the duration and severity of symptoms if administered soon after the onset of the episode.¹³ Labyrinthitis is a rare condition in which there is inflammation within the inner ear resulting in vertigo lasting for days and hearing loss in the affected ear. Viral causes are treated with vestibular suppressants and corticosteroids. MRI with enhancement is required to rule out a retrocochlear lesion (eg, an acoustic neuroma). Bacterial labyrinthitis most frequently results from spread of bacteria (typically, *Streptococcus pneumoniae*) to the inner ear from the meninges during an episode of meningitis. Meningitic labyrinthitis is the most common cause of acquired deafness in children. Administering corticosteroids along with antibiotics at the time of onset of meningitis diminishes the incidence and severity of the hearing loss. Very rarely, a bacterial infection will spread to the inner ear from an otitis media. A previous version of this article appeared in the January 2001 issue of *Consultant*. It has been updated and revised for *Applied Neurology*. Michael J. Ruckenstein, MD, is associate professor in the Department of Otorhinolaryngology: Head and Neck Surgery at the Hospital of the University of Pennsylvania in Philadelphia. REFERENCES 1. Mathias CJ. Orthostatic hypotension: causes, mechanisms, and influencing factors. *Neurology*. 1995;45(4 suppl 5):S6-S11. 2. Robertson D, Davis TL. 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