Chapter 25
Dizziness

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Introduction

A dizzy patient presenting to a busy clinician poses a unique diagnostic challenge. Fortunately, a focused, structured evaluation can guide a provider through the most common differentials in order to establish a clear diagnosis and develop an effective treatment plan. It should be noted that about 5% of primary care visits deal with a chief complaint of dizziness and more than 50% of patients with dizziness seek help from their primary care clinician [1]. The causes of dizziness remain constant across a wide spectrum of practice settings [2].

Dizziness can be classified into four main categories: vertigo, presyncope, disequilibrium, and lightheadedness. Making a diagnosis can initially seem challenging due to the often vague, nonspecific, or inconsistent nature of the patients' reporting. In addition, some causes of dizziness can share similar features and precipitants. A broad differential consisting of both malignant and benign causes coupled with nonspecific patient descriptors can lead to diagnostic frustration.

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Despite these barriers, the cause of the dizziness is revealed by the patient's history in more than 50% of cases [3].

The patient's initial, raw, unguided description of their symptoms is the most important step in determining the cause of their dizziness. For this reason, it is imperative to obtain an unguided, pure description of what the patient is actually experiencing. This can be achieved by starting with open-ended, non-leading questions such as: "Without using the word 'dizzy' tell me what you are feeling" or "When you say you feel dizzy, what exactly do you mean?" After the provider has cast the net widely, wait and listen as the diagnostic clues come to the surface.

Patients with vertigo tend to report a spinning sensation or fictitious sense of motion. If such a patient also has migraine symptoms (photophobia, phonophobia, aura, or typical headache), vertiginous migraine should also be considered, affecting 3% of the population and 10% of migraineurs [1]. Patients with presyncope may report a feeling of pending loss of consciousness or "blackout." Patients with disequilibrium may report being "off-balance" and "feeling wobbly" or describe other perturbations in balance and coordination. Feelings of lightheadedness are often very vague, imprecise, and difficult for patients to articulate without using the word "dizzy." They may describe feeling disconnected from the environment, a sense of floating or giddiness; it is this abstract nature of symptom reporting that in itself is actually diagnostic. It should be noted that psychiatric causes can be identified in many of these patients as well [4] (Fig. 25.1).

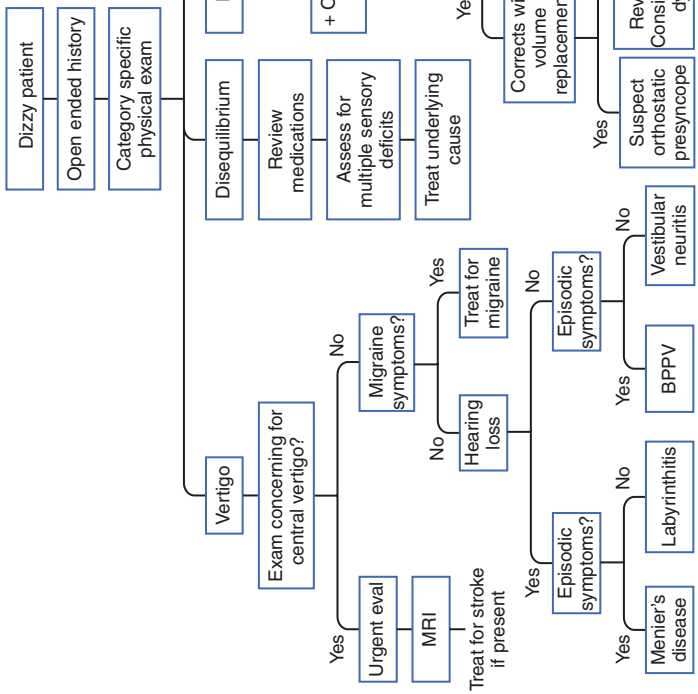

FIGURE 25.1 Approach to patient with dizziness

Decision-Making/Differential Diagnosis

Vertigo

Vestibular dysfunction accounts for more than 40% of dizzy episodes. Here the subtypes include benign paroxysmal positional vertigo (BPPV), vestibular neuritis (viral infection that involves the vestibular nerve), labyrinthitis (postinfectious inflammation of the labyrinthine organs), and Meniere's disease (increased endolymphatic fluid in the inner ear). Central vertigo (brainstem stroke, posterior circulation insufficiency) and migrainous vertigo, as previously noted, are also important diagnostic considerations. Vertigo can be caused by disturbance of the peripheral vestibular system (vestibular portion of the eighth cranial nerve and vestibular labyrinth) or by lesions within the central nervous system (brainstem or cerebellum). Nausea and vomiting are typical with acute vertigo, but can be mild or short lived in BPPV. Interestingly, peripheral vertigo tends to cause more severe nausea and vomiting than central causes. Movement worsens all types of vertigo, and even though patients with peripheral vertigo may not want to move, they are usually able to walk. In comparison, patients with central vertigo experience greater impairment in gait and posture and are often very ataxic and unable to walk [5].

Most patients have benign disorders of the peripheral vestibular system and dysfunction of either the vestibular nerve (vestibular neuritis) or the labyrinth (labyrinthitis). A few (less than 5%) may have serious strokes of the cerebellum or brainstem, which may rapidly cause coma and death from acute hydrocephalus or brainstem compression. For this reason, it is imperative to make a clinical distinction between central and peripheral vertigo [6].

Central vs. Peripheral Vertigo

Severe vertigo may occur in the setting of acute peripheral and central lesions. However, there are some clinical clues that favor a central cause over a peripheral cause. An acute vertebrobasilar stroke is almost always accompanied by other signs and symptoms of brainstem ischemia such as diplopia, dysphagia, weakness, or numbness. A history of atherosclerotic cardiovascular disease and associated risk factors should heighten a clinician's concern for vertebrobasilar ischemia. Findings on the physical exam that suggest stroke in dizzy patients include normal bilateral vestibulo-ocular reflexes (noted on head impulse test), skew deviation, (described later in the chapter), abnormal visual tracking (saccadic pursuit), and direction-changing nystagmus. Visual fixation can suppress nystagmus from the peripheral lesion but not from a central cause. In peripheral lesions, the predominant direction of nystagmus remains the same in all directions of gaze; nystagmus that changes direction with gaze shifting is suggestive of a central abnormality. Other features that suggest a peripheral cause include falling in the opposite direction to nystagmus and horizontal/torsional nystagmus. In summation, the probability of stroke is increased in the setting of severe truncal ataxia, normal vestibulo-ocular reflex, skew deviation, saccadic pursuit, direction-changing nystagmus, and nystagmus that is not suppressed with visual fixation (see Table 25.1) [5].

Table 25.1 Findings concerning for central vertigo

Severe ataxia
Direction-changing nystagmus
Nystagmus not affected by fixation
Saccadic pursuit
Skew deviation
Nystagmus does not fatigue with repeated dix-Hallpike maneuver

BPPV

BPPV is the most common form of peripheral vertigo and tends to occur when the patient moves the head in a particular position as in turning over in bed, looking up, getting up, or lying down in bed. It is believed to be caused by calcium debris within the posterior semicircular canal (canalithiasis). The semicircular canals detect angular head accelerations. Debris in the canal causes inappropriate movement of the endolymph causing the erroneous sensation of spinning when the head shifts with respect to gravity. Most causes of BPPV are idiopathic or related to minor head injury; however, many different precipitants have been proposed. Patients tend to experience recurrent episodes of vertigo that last 1 min or less. Associated symptoms include nausea and vomiting. Hearing loss is usually absent and patients have no other neurologic complaints. Episodes may recur for weeks without therapy. Provoking symptoms and nystagmus with maneuvers such as the Dix-Hallpike maneuver support the diagnosis. With this maneuver, nystagmus and vertigo usually last less than 30 s; after it stops and the patient sits up, the nystagmus will recur but in the opposite direction. Repeating the maneuver on the same side will diminish the intensity of the nystagmus. Further testing is not indicated in typical BPPV, and neuroimaging is reserved for patients with symptoms that are not typical or those who present with other red flags [7].

Meniere's Disease

Meniere's disease is believed to be caused by abnormal fluid balance in the inner ear and associated with episodic vertigo (rotational nystagmus with episodes lasting up to 24 h), tinnitus (low pitch), and sensorineural hearing loss. It is unclear why fluid builds up in the endolymphatic spaces of the inner ear. The diagnosis is made based on clinical features as no specific testing is confirmatory. Audiometry should be performed in all patients with suspected disease. Brain MRI is

often indicated to rule out CNS tumors (acoustic neuroma) and vascular malformations. Patients should be referred to ENT early in the disease process as even though vertigo attacks may be controlled in most patients, hearing loss can be progressive.

Labyrinthitis/Vestibular Neuritis

Vestibular neuritis is believed to be an acute viral or postviral inflammatory process involving the vestibular portion of the eighth cranial nerve. It should be noted that the majority of patients do not necessarily report symptoms of a preceding viral illness [1]. It typically presents as an acute vertigo with nausea, vomiting, and gait impairment. In isolated vestibular neuritis, auditory function is preserved, whereas if the patient has unilateral hearing loss, the condition is defined as labyrinthitis. Patients with unilateral sensorineural hearing loss should be referred for audiometry. If no identifiable cause is found for this defect, imaging of the posterior fossa and internal auditory canal should be considered. There are no confirmatory tests for vestibular neuritis or labyrinthitis, and the diagnosis is made on a clinical basis. Patients with inconsistent exam findings, older patients, and those with new headache or any focal neurologic signs should undergo CNS imaging to rule out acute central vascular events of the brainstem and cerebellum [5].

Migrainous Vertigo

Migrainous vertigo affects about 3% of the population and 10% of migraineurs. Although this is a diagnosis of exclusion, it should be considered in patients who have a history of migraine or present with headache with associated migrainous features and associated symptoms (aura, photophobia, phonophobia, etc.). As with any vertiginous patient, those with red flag symptoms should be referred for imaging to exclude a central cause.

Presyncope

Presyncope is the prodromal symptom of impending loss of consciousness and is more common than actual syncope. The evaluation of these patients is very similar to patients who experience true syncope. As with dizziness, an open-ended history is essential to pursuing the appropriate diagnostic steps. Orthostasis is the most common cause of presyncope. It usually occurs when a patient is in an upright or seated position or when transitioning from a supine to standing position. Orthostasis can be caused by intravascular volume depletion, medications, and autonomic dysfunction [1]. Patients with vasovagal or neurocardiogenic presyncope may report a prodrome described as feelings of warmth, lightheadedness, diaphoresis, nausea, and visual darkening. Witnesses of the event may also describe pallor. Such episodes may be precipitated by specific situations (coughing, sneezing, micturition, stressful events, etc.). Syncope that occurs while supine, with exertion, or suddenly (without prodrome) should raise clinical suspicion for malignant cardiac arrhythmias (ventricular tachycardia, high-grade heart block, prolonged sinus pauses) or structural cardiac abnormalities (severe aortic stenosis or hypertrophic obstructive cardiomyopathy). All patients should be assessed for cardiac history or risk factors and should undergo orthostatic vital sign measurement and a careful review of their medication list. In addition, ECG testing should be done to detect anomalies such as prolonged QT interval, heart block/conduction disturbance, and tachy–/bradyarrhythmias [8]. Indwelling cardiac devices should be checked to ensure proper function. Additional testing including ambulatory cardiac monitoring, event monitoring, and/or echocardiography will depend upon the clinical scenario.

Disequilibrium

Disequilibrium is a sense of imbalance that occurs with walking or standing. It represents a disturbance in balance or coordination which leads to ambulatory impairment.

Determining the cause of dizziness in older patients can be challenging because it is often multifactorial, as patients often have several comorbid disorders and are on many medications which serve as possible contributors. The most common cause of disequilibrium in this population is multiple sensory deficit syndrome wherein multiple issues impair the patient's ability to ambulate unassisted [9]. Visual impairment, deafness, peripheral neuropathy, muscle weakness, and deconditioning can all contribute to dysfunction. This patient's gait is usually hesitant and apprehensive. Metabolic disease such as hypothyroidism, hypoglycemia, anemia, and adrenal insufficiency may be associated with disequilibrium. Movement disorders such as early Parkinson's disease may manifest with disequilibrium prior to the development of tremor and other characteristic features [9].

Lightheadedness

Psychiatric disorders (anxiety, depression, somatization disorder, personality disorder) and fibromyalgia are often the primary cause of nonspecific dizziness. Nonspecific dizziness may also be caused by hyperventilation. Purposeful hyperventilation, where the patient is positioned in a supine position and asked to breathe deeply and rapidly (30-times per minute) through their mouth, can be diagnostic if the technique recreates the patient's symptoms [1]. Using this technique can be reassuring and this revelation may provide therapeutic value.

Key History and Physical Exam

The most important step in determining the cause of the patient's dizziness is to set an environment conducive for the delivery of an unbiased description of what the patient is actually experiencing. As the clinician patiently listens, they are receiving clues that will help them categorize the patient's symptoms into one of the four major categories of dizziness.

In addition, as with any symptom evaluation, the duration, characteristics, precipitating/alleviating factors, and associated symptoms or features such as hearing loss should be discussed. The history should also include a review of the patient's medications as well as a dietary review for substances containing caffeine (which may lead to tachyarrythmias) or alcohol (which through direct toxicity or abrupt discontinuance can precipitate symptoms).

Duration of symptoms can assist with narrowing down the cause of vertigo. For example, BPPV and Meniere's disease tend to cause episodic vertigo, whereas persistent symptoms are more likely caused by vestibular neuritis or labyrinthitis. It should be noted that even when a vestibular lesion is permanent, vertigo subsides over days to weeks as the CNS adapts to the defect. As a result, vertigo is never a permanent or continuous symptom [10]. Some patients reporting long duration of symptoms are often described as having a continuous predisposition to vertigo or are describing a nonvestibular type of dizziness. In addition, patients should be asked about and evaluated for hearing impairment as hearing loss and tinnitus are suggestive of peripheral lesions and can be seen in labyrinthitis and Meniere's disease. These symptoms are not seen in vestibular neuritis or BPPV [1].

The goal of the physical exam is to reproduce the patient's symptoms and make note of any abnormal findings. The special additional maneuvers/tests should be decided upon based on the working differential diagnosis derived from the patient's history. In general, all patients should undergo an assessment for positional changes in blood pressure to detect orthostatic hypotension as well as an otologic (to evaluate for OME [otitis media with effusion]/AOM [acute otitis media]), cardiac, and neurological evaluation [10].

Dix-Hallpike Maneuver

The Dix-Hallpike maneuver should be performed in all patients with suspected vertigo as this test is 50–88% specific for BPPV [11]. These maneuvers are performed with the goal

of reproducing vertigo and nystagmus and are more useful in patients who do not already have symptoms and nystagmus at rest. This maneuver is initiated with the patient in a seated position. The examiner rotates the patient's head 45° to one side and then rapidly places the patient in a supine position while allowing the head to hang about 20° over the end of the table. The examiner should focus on the patient's eyes for about 30 s to evaluate for nystagmus. If no nystagmus is appreciated, the patient is returned to an upright position and observed for 30 s for nystagmus, and then the maneuver is repeated with the head turned to the opposite side. There is usually a latency of a few seconds before the patient develops nystagmus and symptoms. In such cases the nystagmus usually lasts less than 30 s. After the patient sits up, the nystagmus may recur in the opposite direction. If nystagmus is precipitated with this maneuver, it should be repeated on the same side. In BPPV the intensity and duration of the nystagmus should diminish with each repetition [11]. It should also be noted that the affected ear is the one that is down facing on provocation of nystagmus with this maneuver.

Nystagmus

The presence of nystagmus suggests that the dizziness is vertigo. Nystagmus is a rhythmic oscillation of the eyes. One function of the vestibular system is to maintain gaze during head movement through the vestibular ocular reflexes. A unilateral lesion leads to pathologic asymmetry in the vestibular system. This results in a slow drift of the eyes away from the target followed by a fast corrective movement in the reverse direction. The eyes will beat in the direction of the fast phase. This spontaneous nystagmus will continue until normal vestibular function is restored or until the CNS adapts to the lesion [6]. In a peripheral lesion, the fast phase is away from the affected side, and nystagmus increases in frequency and amplitude with gaze toward the side of the fast phase. When there is suspicion of a central cause, a detailed exam should be performed to search for cranial nerve abnor-

malities, motor or sensory changes, dysmetria, or abnormal reflexes. However, the absence of other neurologic signs does not exclude a central process.

Hearing Evaluation

There are several options for evaluation of hearing in the office setting. A rough evaluation for asymmetric hearing impairment can be performed by having the patient repeat words that are softly whispered into each ear or by having the patient close his or her eyes and identify the ear near which the examiner is rubbing fingers together. The Weber and Rinne test can distinguish between conductive hearing loss (CHL) and sensorineural hearing loss (SNHL). In the Weber test, a vibrating tuning fork is placed in the midline of the forehead. In normal hearing, the sound should be heard equally in both ears; however, in conductive hearing loss, the sound lateralizes to the affected ear, and in sensorineural hearing loss, the sound lateralizes to the unaffected ear. The Rinne test compares air conduction of sound vs. bone conduction of sound. In this test, the vibrating tuning fork is placed and held on the patient's mastoid process; when the sound can no longer be heard in that position, the tuning fork is then moved directly in front of the ipsilateral ear. In a normal exam the patient should be able to hear the tuning fork again, demonstrating that air conduction is greater than bone conduction. In a positive test the patient cannot hear the tuning fork again when it moved from the mastoid process to directly in front of the ear. This result demonstrates that bone conduction is greater than air conduction, suggesting conductive hearing loss. For example, on evaluation with the Weber test, if the sound is heard equally in both ears, the exam is normal. However, if the sound is heard louder in the right ear, this could indicate either CHL in the right ear or SNHL in the left ear. In the same patient, the Rinne test is then performed on the right ear; if bone conduction is greater than air

conduction, then CHL is confirmed in the right ear; however, if the Rinne test is normal on the right ear (air conduction > bone conduction), SNHL of the left ear is suspected.

Head Impulse Test, Skew Deviation, Saccadic Pursuit, and Direction-Changing Nystagmus

The head impulse test demonstrates integrity of the vestibulo-ocular reflex. The examiner sits in front of the patient and places hands on each side of the patient's head. The patient should focus on the clinician's nose and the clinician focuses on the patient's eyes. If the reflex is intact, the patient's eyes can remain focused on the clinician's nose during rapid head movements to both sides. If there is impairment in the peripheral vestibular system, the vestibulo-ocular reflex will be abnormal causing the eyes to move away with head movement toward the affected side which is followed by the patient's eyes quickly moving back to the clinician's nose (corrective saccade). In patients with acute vertigo, a normal vestibulo-ocular reflex bilaterally is suggestive that the cause of dizziness is central. Skew deviation is demonstrated when one eye is vertically aligned higher than the other, a sign of cerebellar or brainstem disease. Abnormal visual tracking is detected by asking the patient to follow a slowly moving target such as the clinician's finger both horizontally and vertically, while keeping the head still. The pursuit should be smooth. However, with cerebellar or brainstem disease, quick catch-up movements are noted and are called saccadic pursuit [10, 12].

Many patients with acute vertigo will have spontaneous nystagmus when looking straight ahead. In both central and peripheral vertigo, the nystagmus will worsen when a patient looks in the direction of the quick component (fast/corrective phase). In peripheral disease when the patient looks in the opposite direction (slow component), the nystagmus will disappear or diminish. However, in some patients with stroke,

when looking to the opposite side, the nystagmus can reverse denoting a direction-changing nystagmus. In peripheral disease nystagmus diminishes during fixation on an object; in central disease the nystagmus is unchanged [6].

Romberg Testing

Romberg testing can stimulate disequilibrium. The patient stands upright, feet together, with arms at sides and then asked to close his or her eyes. Symptoms of tilting or falling with eyes closed may suggest disordered proprioceptive and vestibular function. Symptoms with eyes open or closed may suggest cerebellar disease [12].

Other Diagnostic Testing

Other studies such as laboratory tests (CBC, serum chemistry, thyroid function tests) and radiography are not often beneficial and have low diagnostic yield without other supporting findings. MRI of the brain is indicated in patients with findings suggestive of central vertigo or acoustic neuroma. CT scans are less sensitive for detecting brainstem pathology but can be performed with thin cuts through the brainstem/posterior fossa. MRA sensitivity and specificity exceed 95% in detecting stenosis or occlusion of the posterior circulation [10, 12].

Treatment

Successful treatment of dizziness is predicated on a meticulous evaluation to determine the disease process responsible for the patient's symptoms. Most treatment strategies are based on relieving the underlying cause of the symptoms (see Table 25.2).

Table 25.2 Treatment pearls for the dizzy patient

Type of dizziness	Management
Migrainous vertigo	NSAIDS
	Triptans
	Evaluate need for prophylaxis
Disequilibrium	Reduce polypharmacy
	PT/OT referral
	Vision/hearing screening
	Ambulatory assist device
Meniere's disease	Audiometry
	Diuretics
	Referral to ENT
Labyrinthitis	Audiometry
	Steroids × 10 days
	Supportive therapy
BPPV	Epley maneuver
	Antihistamines if frequent attacks
	Vestibular rehab
Vestibular neuritis	Steroids × 10 days
	Supportive therapy
	Vestibular rehab
Lightheadedness	Treat underlying disorder
	Controlled breathing
	Behavioral health referral

BPPV

The majority of patients with BPPV achieve remission with particle repositioning maneuvers such as the Epley maneuver, where the goal is to clear debris from the semicircular canal. It should be noted that these maneuvers may be effective when the history is highly suggestive of BPPV even when no nystagmus is appreciated. Some studies suggest that a single maneuver is effective in 85% of patients. The Epley maneuver is performed by having the patient sit upright on the exam table with head rotated to the right (affected ear); the patient is then laid in a supine position with the head hanging over the end of the table. The patient's head is then rotated to the left, and the patient's head and body are rotated an additional 90° until the patient's nose is angled toward the ground. This position is held for 30 s and the patient is briskly returned to a seated position. This technique is repeated until no nystagmus can be detected. Videos of this sequence can be seen on NEJM clinical videos or on youtube.com. Modified versions of the Epley maneuver can be taught to patients for self-treatment at home. Even after an effective maneuver is performed, patients may have milder symptoms for several hours to days. Medications are not useful for the brief episodes of vertigo associated with BPPV; however, when there is a high frequency of episodes, antihistamines and antiemetics can provide relief. Patients may also benefit from a referral for vestibular rehabilitation [13].

Vestibular Neuritis/Labyrinthitis

To relieve the suspected postviral inflammatory process involved in vestibular neuritis and labyrinthitis, patients should be prescribed a 10–14-day steroid taper. Antiviral agents have not been demonstrated to hasten recovery and are therefore not recommended. Patients should also be provided with other supportive care which may include volume replacement for severe nausea and emesis, antiemetics, and in some cases a referral for vestibular rehabilitation [1].

Meniere's Disease

The abnormal fluid balance of this condition can be managed based on the severity of disease. All patients should be referred for an ENT evaluation. Noninvasive treatments include salt restriction, diuretics, antihistamines, vestibular suppressants, and antiemetics. Patients should also be referred for vestibular rehabilitation. Patients with refractory or severe disease despite medical therapy may be candidates for invasive therapies such as intratympanic gentamicin or glucocorticoids and, in some cases, surgical therapy [1].

Disequilibrium

The evaluation of a patient with disequilibrium involves looking for other disorders that are contributing to the symptoms as treatment is focused on addressing the issues that underlie this condition. This may involve withdrawal of precipitating medications, referral for ophthalmologic evaluation and corrective lenses for visual impairment, providing an ambulatory assistive device (cane or rolling walker), physical therapy evaluation, and the treatment of any metabolic, neurologic, or movement disorders that were unveiled during the evaluation.

Presyncope

The evaluation of patients with presyncope is the same as for patients who experienced true syncope. As with all other causes of dizziness, the history guides the clinician to the appropriate exam and relevant testing needed to secure the diagnosis. Any potentially offending medications that can safely be tapered or discontinued should be a priority. Patients with orthostatic hypotension can benefit from volume replacement if they are volume depleted. Refractory orthostasis should prompt the clinician to consider autonomic dysregulation. In addition to management of underly-

ing metabolic and endocrine disorders, medications such as midodrine or fludrocortisone can be initiated. Patient education with behavior modification should be provided to patients with a neurocardiogenic process. Patients with atherosclerotic cardiovascular disease history or risk factors, those with sudden or exertional syncope, and those who experience syncope in a supine position should undergo thorough evaluation and treatment for potentially malignant causes [13].

Lightheadedness

Hyperventilation syndrome often coupled with psychiatric disorders (anxiety and depression) is the main contributor to the vague and imprecise symptoms of lightheadedness. As previously noted, if the hyperventilation provocation test successfully recreates the patient's symptoms, this can also be therapeutic and reassuring to the patient [1]. Conscious breathing exercises can control future events. If a mood disorder is revealed during symptom evaluation, this should be treated with the appropriate agents, and the patient can also be referred to the relevant behavioral health specialist.

Clinical Pearls

- The patient's initial, raw, unguided description of symptoms is the most important step in determining the cause of dizziness.
- Movement worsens all types of vertigo, and even though patients with peripheral vertigo may not want to move, they are usually able to walk. In comparison, patients with central vertigo experience greater impairment in gait and posture and are often very ataxic and unable to walk.
- All patients with Meniere's disease should be referred to ENT early in the disease process as even though vertigo attacks may be controlled in most patients, hearing loss can be progressive.

- Orthostasis can be caused by intravascular volume depletion, many medications, and autonomic dysfunction.
- Visual impairment, deafness, peripheral neuropathy, muscle weakness, and deconditioning can all contribute to disequilibrium.
- The hyperventilation test for suspected lightheadedness can be both therapeutic and diagnostic.

Do Not Miss This!

- These are findings on the physical exam that suggest stroke in dizzy patients: normal bilateral vestibulo-ocular reflexes (noted on head impulse test), skew deviation, abnormal visual tracking (saccadic pursuit), and direction-changing nystagmus.
- Syncope that occurs while supine, with exertion, or suddenly (without prodrome) should raise clinical suspicion for malignant cardiac arrhythmias (VT, high-grade heart block, prolonged sinus pauses) or structural cardiac abnormalities (severe aortic stenosis or HOCM).

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