Dizziness is a nonspecific term that describes an unpleasant sensation of imbalance or altered orientation in space. This common clinical problem affects at least a third of the population in one form or another at some point during their life. The prevalence increases significantly with age and is higher among those with diabetes. In cross-sectional surveys, at least one third of US adults aged 40 years or older (69 million Americans) reported dizziness or postural instability, and those with dizziness had a 12-fold higher risk of falling. Similarly, among adults in the Oslo Health Study, the prevalence of self-reported faintness or dizziness was 29% and was reported more often by women and those older than age 60; however, even in children, dizziness is not uncommon.

Dizziness is often associated with significant comorbidity and serious consequences, including falls, traumatic injuries, and institutionalization. Older adults who report dizziness are more physically frail, have more chronic conditions and sensory impairments, and have a poorer self-perception of their own health than older persons without dizziness. In addition, dizziness causes great psychological distress and greater perceived disability, particularly in the elderly. Furthermore, dizziness results in detrimental effects on quality of life, work, travel, social life, and family life.

Unfortunately, dizziness is commonly misdiagnosed and mistreated in primary care, emergency department, and inpatient hospital settings. Nearly half of emergency department diagnoses in patients evaluated for dizziness are corrected on review, with the diagnoses of stroke and vestibular neuritis most frequently requiring correction.

It is essential to recognize that although dizziness is a common and often disabling medical problem with a significant negative impact on health-related quality of life, it is by no means a homogeneous clinical disorder. It is instead a heterogeneous constellation of clinical syndromes that require distinct diagnostic and management approaches. Dizziness should not be dismissed as an expected feature of aging, nor should it trigger routine batteries of brain imaging and other expensive diagnostic procedures or be treated routinely with empirically administered vestibular sedatives such as meclizine. Indeed, vestibular sedatives are actually effective only for a small minority of patients with dizziness, generally those with acute persistent vertigo, and for some with anxiety-related dizziness. Vestibular sedatives do not generally help patients with benign positional vertigo, sensory or motor disequilibrium, or presyncope.

This article provides a step-by-step approach to the workup that emphasizes a careful history, provocative testing, and a detailed examination, with judicious use of brain imaging and other diagnostic studies (Table 1).

### Key words: dizziness, vertigo, disequilibrium, presyncope, nystagmus

**Table 1 – A step-by-step approach to the workup of dizziness**

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**STEP 1: THE HISTORY**

The most important part of the workup of dizziness is actually the history. For almost all patients, a detailed and thorough history will focus the subsequent physical examination and will help...
the clinician target the use of expensive diagnostic tests, direct the patient to appropriate consultation with a specialist, and choose the therapeutic interventions that will be most likely to resolve the clinical problem.

The specific clinical manifestations of dizziness depend on which category or categories of dizziness exist: vertigo, dis-equilibrium, presyncope, or psychophysiological (psychogenic) dizziness. The proper evaluation and treatment of dizziness rely on accurate recognition of the different categories, despite the vague and sometimes misleading descriptions patients often report about the character of their dizziness. In such cases, the clinician must clarify and reframe those descriptions until he or she understands clearly what the patient is experiencing, including the duration, circumstances, and associated features of the episodes that occur.

Unfortunately, many patients find it difficult to describe their dizziness sensations, and sometimes, because they find the experience so unpleasant, they are unable to attend to the specific clinical features sufficiently to give a clear account. Many resort to calling their experience simply “dizziness” or “spinning,” even when the sensation is not one of turning. The clinician must therefore attempt to “tease out” the essential features that will allow a clear and correct assessment to be made. It helps to ask open-ended questions first, followed by clarifying and discriminating questions, as in the following approach: “Please tell me about your dizziness. Can you describe what an episode of dizziness feels like to you? You mentioned ‘spinning,’ but is it really a feeling like you are actually turning, or is it more of a feeling of unsteadiness or wooziness, or does it feel like you might pass out?”

Depending on the cause, dizziness may be associated with various other symptoms, including audiovestibular symptoms, headache, daytime somnolence, and sleep apnea. To the extent possible, it is important to identify any associated symptoms, recognizing that some of those symptoms may occur with more than one type of dizziness. Headache, for example, can occur with vertigo (eg, migraine, brainstem ischemia), motor dys-equilibrium (such as from cerebellar infarction or hemorrhage), presyncope, and chronic nonspecific dizziness (often associated with anxiety, sleep deprivation, and caffeine overuse/withdrawal).

Vertigo is typically associated with nystagmus (voluntary rhythmic oscillatory eye movements), oscillopsia (an illusory visual disturbance in which stationary objects appear to move as a result of the observer’s head or eye movements), postural imbalance, ataxia, and autonomic symptoms (eg, sweating, pallor, nausea, vomiting). Autonomic symptoms are generally more severe with vertigo of peripheral origin than with vertigo of central origin.

When a patient reports vertigo, it is important to determine how the disorder occurs, that is, whether it manifests as a single prolonged event or a pattern of recurrent events, the duration of the symptoms, and any associated neurologic or audiovestibular symptoms.

Acute monophasic vertigo generally occurs fairly suddenly and generally lasts for at most for a period of hours or days—certainly not weeks, months, or years. Patients can also experience a more protracted disequilibrium, usually lasting days or, at most, a few weeks, but without the vertiginous illusion of movement (eg, spinning). Most such patients with acute persistent vertigo are severely uncomfortable and have prominent examination findings of nystagmus, postpointing, and a Romberg sign. Common accompanying symptoms include nausea, vomiting, and diaphoresis. Evidence of unilateral auditory symptoms (eg, abrupt worsening of hearing, tinnitus) indicates involvement of the inner ear, the eighth cranial nerve, or the brainstem.

Episodic vertigo occurs in recurrent episodes lasting seconds or hours. Episodes that occur in certain positions (known as positional vertigo) or during movement in certain directions (positioning vertigo) are typically brief and last less than 1 minute if they are actually timed. However, patients commonly overestimate the duration of their vertigo episodes, confuse repeated attacks within a bout of vertigo with a single prolonged attack, or confound the vertigo symptom with other symptoms that may persist for longer periods (eg, nausea).

Brief episodes of vertigo lasting less than a minute (when actually timed) and occurring during certain head
movements or changes of position are typical of benign paroxysmal *positioning* vertigo (BPPV), which has also been labeled less accurately as benign *positional* vertigo. The importance of this diagnosis is twofold:

- BPPV does not reflect a serious underlying pathology.
- BPPV can be treated readily by an experienced clinician in a noninvasive in-office procedure.

Some rare CNS conditions can also produce brief episodes of positional vertigo (or, less commonly, positioning vertigo), usually involving the posterior fossa and typically associated with other evidence of CNS pathology revealed by a patient’s history and examination results.

Longer episodes of vertigo accompanying migraine headaches or occurring in patients prone to migraine and lasting minutes or, less likely, hours can occur with migraine-associated vertigo, whereas longer episodes lasting several hours accompanied by auditory symptoms (eg, hearing loss, tinnitus, a feeling of fullness in the ear) are suggestive of Meniere’s disease.

The differential diagnosis of vertigo varies by age (Table 2). Common causes of vertigo in the elderly include BPPV, neurolabyrinthitis, trauma, toxins, migraine, and posterior circulation or labyrinthine ischemia, which is particularly associated with posterior inferior cerebellar artery or anterior inferior cerebellar artery ischemia. Among elderly patients who present with dizziness, vertigo, or imbalance, the proportion of those with cerebrovascular events is small, although a high index of suspicion is necessary, particularly to exclude a posterior circulation cause in the elderly.

The common causes of vertigo in young adults include migraine, Meniere’s disease, viral neurolabyrinthitis, trauma, and toxins. The common causes of vertigo in children with normal tympanic membranes (ie, children who do not have middle ear effusion or otitis media) are migraine and BPPV. The CNS causes of vertigo are uncommon in general clinical practice.

### Disequilibrium

Disequilibrium is a state of altered postural balance that is not due to vertigo. It may occur in static (eg, standing) or dynamic (walking) situations. Patients with disequilibrium often complain of unsteadiness, imbalance, and falls.

Disequilibrium can result either from sensory (“input”) problems, motor (“output”) problems, central problems of sensorimotor integration, or some combination of these. Consequently, there are several subtypes of disequilibrium:

- **Sensory**, due to problems with the input of proprioceptive, vestibular, or visual information.
- **Central**, due to disruption of the normal integration of sensory information or the linkage of sensory information with motor output.
- **Motor**, due to problems with neuromuscular control or mechanical factors.
- **Mixed**.

For example, a patient with diabetes who has severe polyneuropathy may have sensory disequilibrium, as may someone who has undergone cataract surgery, whereas someone who is intoxicated may have problems with sensorimotor integration and motor control (ataxia), and someone with Parkinson’s disease may have a problem with motor control. All of these individuals may report unsteadiness on their feet, or “dizziness,” but the mechanisms are very different, as are the approaches to treatment.

On the sensory (or input) side, optimal balance requires the continuous monitoring of body sway and other orientation information provided by the somatosensory, vestibular, and visual systems. The functional ranges of these systems partially overlap, allowing partial compensation for deficits and distortions. For example, a normal subject can maintain an upright stance either with vision eliminated (ie, with eyes closed), with proprioception disrupted (as from standing on a moving or tilting surface), or with vestibular function distorted (as a result of rotationally induced vertigo). Loss or distortion of input from 2 or more systems is often associated with dizziness, loss of balance, and falls; thus, a patient who has a profound loss of proprioception and sensory disequilibrium, or has uncompensated unilateral vestibular dysfunction and vertigo, may fall if his or her vision is eliminated. This is the basis of the Romberg sign.

Sensory disequilibrium is typically due to proprioceptive impairment (eg, from peripheral neuropathy, or from dysfunction of the dorsal root ganglia or the posterior columns of the spinal cord), but

### Table 2 – Common causes of vertigo by age

<table>
<thead>
<tr>
<th>Age group</th>
<th>Common causes of vertigo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elderly persons</td>
<td>BPPV, neurolabyrinthitis, trauma, toxins, migraine, and posterior circulation or labyrinthine ischemia</td>
</tr>
<tr>
<td>Young adults</td>
<td>Migraine, Meniere’s disease, viral neurolabyrinthitis, trauma, and toxins</td>
</tr>
<tr>
<td>Children (with normal tympanic membranes, ie, those who do not have middle ear effusion or otitis media)</td>
<td>Migraine and BPPV</td>
</tr>
</tbody>
</table>

BPPV, benign paroxysmal positioning vertigo.
Dizziness:
Step-by-Step Through the Workup

it may also be caused by balanced bilateral or compensated unilateral vestibular dysfunction (as caused by aminoglycoside-induced toxicity or the residua of viral neuritis), visual-vestibular mismatch (as caused by impaired vision, ocular misalignment, or use of optic devices [eg, lens implants or new glasses]), or multisensory impairment. Note that although acute unilateral vestibular dysfunction may be considered a form of sensory disequilibrium, this typically presents with vertigo and would therefore be categorized separately. In contrast, patients with chronic unilateral or asymmetric vestibular dysfunction, or symmetric vestibular hypofunction, present with a sensory disequilibrium. Except in cases of visual-vestibular mismatch, patients with sensory disequilibrium generally do worse in the dark and frequently exhibit a Romberg sign on examination.14

Motor (or output) disequilibrium is caused by impaired motor performance, which may be due to either to mechanical factors or to dysfunction of central and peripheral nervous system motor pathways. The central motor pathways that may be affected in patients with motor disequilibrium include the pyramidal, extrapyramidal, and cerebellar systems, whereas the peripheral motor pathways include the peripheral nerves, neuromuscular junctions, and muscles. In practice, though, patients usually complain of dizziness as the primary concern in the setting of motor disequilibrium in which dysfunction involves the extrapyramidal or cerebellar systems, with or without involvement of other systems or pathways. Motor disequilibrium, including that due to cerebellar dysfunction, is generally not exacerbated in the dark or when the eyes are closed.

Presyncope. Presyncope is a syndrome characterized by a sensation of impending loss of consciousness and is commonly associated with generalized weakness, diaphoresis, nausea, and epigastric distress.9 Other associated symptoms may include facial pallor or an ashen-gray appearance, scotomata, visual dimming or gray-out, and, depending on the cause, palpitations, acral and perioral paresthesias, and carpopedal spasms. Symptoms of presyncope, especially those due to orthostatic hypotension, may be exacerbated by exertion (especially after exertion), prolonged standing, increased ambient temperature, and eating. Episodes of presyncope are generally relieved with recumbency.

Presyncope is caused by diffuse and sudden impairment in cerebral metabolism, which may occur in isolation or as a precursor to loss of consciousness (ie, syncope). The sudden cerebral metabolic dysfunction occurs in response to generalized cerebral ischemia or, less commonly, to hypoglycemia or hypoxia. True syncope occurs rarely in patients with hyperventilation, hypoglycemia, or postural orthostatic tachycardia syndrome (POTS).

Orthostatic hypotension is the most common cause, but arrhythmias, orthostatic intolerance (eg, postural orthostatic tachycardia syndrome), hyperventilation, panic attacks, and other conditions can produce presyncope. The independent risk factors for orthostatic hypotension include increasing age, prehypertension or hypertension, and diabetes.15

Presyncope (and syncope) may occur with decreased cardiac output (such as from arrhythmia, obstructive cardiomyopathy, pulmonary emboli), inadequate peripheral vasoconstriction mechanisms (as attributable to vasovagal response, sympatholytic drugs, primary autonomic insufficiency, or central and peripheral nervous system diseases causing orthostatic hypotension), POTS, cerebrovascular constriction (eg, hyperventilation), hypovolemia, mechanical reduction in venous return (such as from cough, micturition), and alterations in the oxygen or nutrient content of the blood (such as those associated with hypoxia, hypoglycemia).

Chronic nonspecific dizziness. In the final category, chronic nonspecific dizziness, the dizzy sensation is often a vague giddiness, which is typically protracted or continuous, varying in intensity, and often associated with periodic exacerbations. Although patients may develop anxiety and depressive symptoms in response to any type of dizziness and the associated fears of falling or injury, anxiety is a significant causal factor for many patients with this category of dizziness. Indeed, chronic nonspecific dizziness is often attributed to impaired central integration of sensory and motor signals in patients who have acute and chronic anxiety, although similar findings can be seen in patients with previous well-compensated vestibular lesions.8,9

Because of the presumed etiologic role of anxiety in at least some of the symptoms in this category of dizziness, other terms used to describe chronic nonspecific dizziness include phobic postural vertigo, psychophysologic dizziness, psychogenic dizziness, and space-motion phobia. In addition, this category is also sometimes called chronic subjective dizziness, because affected patients in this category are least likely to have objective findings on physical examination, which includes a thorough cardiovascular and neurologic assessment.

Although the word “nonspecific” may, admittedly, be inadequate, the term “chronic nonspecific dizziness” at least avoids some of the problems and value-laden concerns related to the other terms. It is also broad enough to encompass almost all of the types of dizziness not addressed by the other categories, yet it is not so generic as to be meaningless.

The specific situations and circumstances that are known to provoke chronic nonspecific dizziness include proximity to crowds, driving, and being in confined places (eg, elevators). In addition, the chronic fluctuating dizzy sensations may be punctuated by episodes of hyperventilation-induced presyncope. Other clinical evidence of coexistent acute and chronic anxiety is typically present, but it is important to recognize that anxiety may also result from other forms of dizziness, because of the sudden, dramatic, and unpleasant associated sensations, and from the fear of falling, injury, or death. These episodes are not associated with facial pallor and are not relieved with recumbency. Women are affected by chronic subjective dizziness about twice as frequently as are men.

STEP 2: PROVOCATIVE TESTING

A thorough history is frequently sufficient to classify a patient’s dizziness into one of the 4 major categories and, perhaps, even to suggest an etiologic di-
agnosis; however, sometimes patients’ descriptions of dizzy sensations are confusing, particularly when the events are episodic. Therefore, provocative testing is often helpful in defining the subjective sensation and the often vague description of dizziness. Provocative testing can be used to produce the physiologic sensations of vertigo or presyncope, which the patient can then compare and contrast with the subjective sensations he or she experiences during a typical spell. In this way, provocative testing can help identify or establish the category (or categories) of dizziness experienced by a patient.

The most commonly employed provocative maneuvers in the assessment of dizziness include rotational testing to induce physiologic vertigo, the Dix-Hallpike test for detecting positioning vertigo, an assessment of orthostatic vital signs, and volitional hyperventilation. Collectively, these maneuvers take approximately 10 minutes to complete. Other provocative maneuvers may be helpful in selected circumstances but are not commonly employed in a primary care setting.8,9

**Rotation-induced vertigo.** Physiologic vertigo can be induced in the office either by rotational or caloric testing. To perform rotational testing in the office, the patient can be seated in a rotary office chair with the head tilted 30 degrees forward, and then rotated carefully 10 times over 20 to 30 seconds. Tilting the head forward 30 degrees places both horizontal semicircular canals parallel to the floor and, therefore, perpendicular to the axis of rotation in the chair. As a result, both horizontal ducts are affected during rotational testing, in which the output from one duct is stimulated, whereas the output from the other canal is inhibited. The mismatch between the resulting vestibular imbalance and visual and somatosensory information produces physiologic vertigo, which generally lasts less than 1 to 2 minutes. During this time, one can observe peripheral vestibular nystagmus and post-pointing. Because of the associated risk of falls and injury, patients should not stand until the vertigo has resolved.

Vertigo can also be produced with caloric testing (eg, by injecting cold or warm water into the patient’s external auditory canal); however, this is generally more uncomfortable for the patient than rotational testing and is also more time consuming and involved for the examiner. Therefore, caloric testing is not recommended for provocative testing to determine the category of dizziness.

**Dix-Hallpike positioning maneuver.** The Dix-Hallpike positioning maneuver tests for positioning-induced nystagmus, particularly that associated with BPPV involving the posterior semicircular duct.16 The patient is instructed to stare off into space and to try to avoid looking at any specific object during the procedure. The patient’s head is turned 45 degrees to one side, and then the patient is rapidly moved backward from a sitting to a head-hanging position. Turning the head to one side places the ipsilateral posterior semicircular duct in a parasagittal plane. When the patient is moved backward, the movement is in the plane of that duct. The examiner maintains the patient in the head-hanging position for approximately 1 minute and observes the patient’s eyes for nystagmus. Anticipation of vertigo, or the actual experience of vertigo, may make patients very anxious. Calm but firm reassurance from the examiner is often necessary to complete the maneuver.

Positioning nystagmus elicited by the Dix-Hallpike maneuver with BPPV involving the posterior semicircular duct is a mixed linear–rotary jerk nystagmus.16 It beats upward and toward the undermost ear and has a latency of 1 to 45 seconds (but typically only a few seconds) and a duration of less than 60 seconds. It lessens or disappears with repetition of the offending head positioning. Nystagmus occurring with any of the following characteristics during Dix-Hallpike testing suggests a central cause and is sufficient to warrant a neurologic consultation:

- The nystagmus begins immediately upon assumption of the head-down position.
- The oscillations have a pendular appearance.
- The oscillations are purely rotary or purely linear.
- The nystagmus continues for more than 1 minute while the head-down position is maintained.

**Orthostatic vital signs (“bedside tilt testing”).** Assessment of orthostatic pulse and blood pressure should be included in the evaluation of a dizzy patient.17 Assessment of pulse and blood pressure with the patient both supine and standing is necessary to diagnose presyncope or orthostatic intolerance caused by either orthostatic hypotension or POTS.

Orthostatic hypotension is defined as a decrease in systolic blood pressure (SBP) of at least 20 mm Hg or a decrease in diastolic blood pressure of at least 10 mm Hg within 3 minutes after a patient stands or within 3 minutes after his or her head is tilted up to at least 60 degrees on a tilt table, regardless of whether the patient is symptomatic or asymptomatic.18,19 Although evidence of orthostatic hypotension is sufficient for some patients to be symptomatic, patients who demonstrate an orthostatic decrease in SBP of more than 40 mm Hg, an orthostatic decrease in mean arterial pressure of at least 20%, or a standing SBP of 90 mm Hg or lower are most likely to be symptomatic.20,21 In patients with supine hypertension, a decrease in SBP of 30 mm Hg or greater “may be a more appropriate criterion” for orthostatic hypotension, because the “magnitude of the orthostatic blood pressure fall is dependent on the baseline blood pressure”; a threshold supine SBP of 160 mm Hg or greater is suggested for the application of this criterion because patients with supine hypertension have a larger orthostatic hemodynamic reserve and demonstrate statistically greater decreases in postural SBP than the general population.

**Hyperventilation-induced presyncope.** Hyperventilation produces presyncope with perioral and acral paresthesias and, potentially, carpopedal spasms, although the latter manifestation is rarely achieved by voluntary hyperventilation. To produce these sensations in the office, the patient is asked to breathe deeply and quickly for 3 minutes through an open mouth with the lips not pursed. This is difficult to do even for cooperative pa-
Clinical Highlights

- Dizziness is often associated with significant comorbidity and serious consequences, including falls, traumatic injuries, and institutionalization. Older adults who report dizziness are more physically frail, have more chronic conditions and sensory impairments, and have a poorer self-perception of their own health than older persons without dizziness.

- Depending on the cause, dizziness may be associated with various other symptoms, including audiovestibular symptoms, headache, daytime somnolence, and sleep apnea. To the extent possible, it is important to identify any associated symptoms, recognizing that some of those symptoms may occur with more than one type of dizziness.

- Drugs associated with dizziness include alcohol and other central nervous system depressant medications, aminoglycoside antibiotics, anticonvulsants, antidepressant medications, antihypertensive medications, chemotherapeutic agents, loop diuretics, and salicylates.

- The most commonly employed provocative maneuvers in the assessment of dizziness include rotational testing to induce physiologic vertigo, the Dix-Hallpike test for detecting positioning vertigo, an assessment of orthostatic vital signs, and volitional hyperventilation.

- Vestibular imbalance is indicated by nystagmus, past-pointing, and postural and gait abnormalities. Clinical disturbances of the vestibulospinal pathways are assessed with several tests, including past-pointing, stance assessment, and the Romberg test.

- Patients who are more likely to have abnormal findings on head CT are those with severe headaches or some neurologic deficits in addition to dizziness, whereas those with isolated dizziness, lightheadedness, or monosymptomatic positional vertigo are very unlikely to have acute, life-threatening abnormalities.

patients, and considerable encouragement from the examiner is often required.

STEP 3: EXAMINATION

In all patients with dizziness or vestibular complaints, careful examination of the eyes, ears, cardiovascular system, nervous system, and vestibular system is indicated. Vestibular imbalance is indicated by nystagmus, past-pointing, and postural and gait abnormalities. Clinical disturbances of the vestibulospinal pathways are assessed with several tests, including past-pointing, stance assessment, and the Romberg test.14

Nystagmus. Because different types of nystagmus have different clinical implications, it is important to carefully characterize nystagmus both by its appearance and by any precipitating and inhibiting factors. For example, nystagmus may be characterized by the symmetry of the oscillations (that is, whether it is pendular or tends to beat preferentially in one direction), by the trajectory of the eye movements (that is, whether it is linear or rotary), by the directionality of the eye movements (that is, whether it is unidirectional or direction-changing as a function of gaze), and by the eliciting circumstances (that is, whether it is spontaneous nystagmus that occurs in the primary position, gaze-evoked nystagmus that is precipitated by a change in eye position within the orb, positional nystagmus that is precipitated by a specific head position, or positioning nystagmus that is precipitated by a direction-specific head movement).

Jerk nystagmus is identified by a clear slow-phase drift in one direction followed by a corrective quick phase in the opposite direction. Jerk nystagmus is traditionally described by the direction of the quick phases (eg, “down beat” nystagmus). In contrast, pendular nystagmus is characterized by smooth sinusoidal oscillations of the eyes.

Nystagmus is evident in approximately 20% of persons who visit the emergency department because of dizziness, but in at least 25% of cases, no further characteristics are recorded.22 In addition, documented descriptions usually conflict with the diagnosis when a peripheral vestibular disorder is diagnosed in the emergency department.22 As a result, documented details do not often allow a meaningful inference concerning the cause of the dizziness.22

Peripheral vestibular nystagmus is a mixed linear–rotary jerk nystagmus that beats in one direction, away from a hypofunctioning labyrinth. Evidence of any of the following characteristics of nystagmus suggests a central cause:

- The oscillations have a pendular appearance.
- The oscillations are purely rotary or purely linear.
- The oscillations change direction as the patient gazes in different directions.
- The oscillations are not suppressed when the patient fixes his or her gaze on a visual target.
- Central neurologic signs or symptoms are evident.

Despite these helpful rules, small cerebellar strokes may mimic labyrinthishine lesions clinically. Therefore, particularly in the elderly, great care should be taken in excluding a central cause for acute vertigo.

Past-pointing. When a patient is assessed for past-pointing, he or she is asked to sit facing the examiner with his or her index finger extended and pointing at, but not touching, the examiner’s extended finger. The patient is then instructed to raise his or her arm to a vertical position with the index finger pointing at the ceiling and subsequently return the arm to the initial position. This is repeated several times with the eyes closed. Consistent deviation of the arm to one side is called past-pointing. If the
eyes are opened or the arm is extended, visual or proprioceptive signals will permit accurate localization of the target even if vestibular function is impaired. For this reason, the standard finger–nose–finger test is not helpful in identifying past-pointing. In acute vestibular lesions, patients past-point toward the affected side; however, the test can be misleading, since CNS compensation rapidly corrects the past-pointing and can produce a drift to the opposite side.

**Stance.** With acute unilateral vestibular lesions, patients have impaired postural control and may sway or fall toward the lesion. Although this is helpful diagnostically, the examiner must take great care when assessing stance and gait in patients with vestibular complaints, as the patients could suddenly fall and injure themselves. The examiner must provide adequate support to prevent falls and injuries. For patients who are unable to maintain their stance without support when their eyes are open, it is unnecessary and potentially dangerous to proceed with a Romberg test or an unsupported assessment of gait.

**Romberg sign.** In the Romberg test, the patient is first asked to stand with eyes open and feet together. If the patient is unable to maintain balance in this position, the stance is widened until this is possible. The patient is then asked to close his or her eyes. Patients with proprioceptive or acute vestibular dysfunction may be unable to maintain this position. Patients with proprioceptive dysfunction may sway (and fall) in any direction. In contrast, patients with unilateral vestibular dysfunction usually sway or fall toward the side of the lesion, particularly if the dysfunction is acute. Because of CNS compensation, the test is less sensitive to chronic unilateral vestibular dysfunction. As with past-pointing, overcompensation may result in falls toward the “good” side.

**STEP 4: DIAGNOSTIC TESTS**

In some cases, additional diagnostic tests will be required. These should be ordered selectively depending on the type of dizziness and suspected underlying etiologies. Unfortunately, studies have suggested that diagnostic testing is often ordered indiscriminately and that the frequency of ordering multiple expensive tests is increasing, particularly in emergency departments.23,24

Brain imaging should not be ordered routinely for dizziness complaints.9,25 The clinical value of CT scans for patients who present to the emergency department with dizziness is very low and appears to have declined over time.23

Patients who are more likely to have abnormal findings on head CT are those with severe headaches or some neurologic deficits in addition to dizziness, whereas those with isolated dizziness, light-headedness, or monosymptomatic positional vertigo are very unlikely to have acute, life-threatening abnormalities.26

With MRI, in particular, structural abnormalities of the brain and neck are common and nonspecific, and routine MRI is unlikely to identify specific etiologies for dizziness.25

Which patients should undergo brain imaging (Table 3)? Certainly those with acute persistent vertigo or motor disequilibrium, without another identified basis, particularly among patients over age 50. Other candidates include patients who have CNS signs or symptoms or a history or clinical evidence of head trauma, alcohol or substance abuse, malignancy (especially cancer associated with a significant potential for CNS metastases), coagulopathy (including that associated with warfarin treatment), or immunodeficiency (AIDS, immunosuppressant medications, etc). Brain imaging is generally not indicated in typical cases of BPPV, in sensory disequilibrium (with peripheral neuropathic findings), in presyncope, or in nonspecific chronic dizziness, psychophysiologic dizziness, or hyperventilation syndromes in the absence of focal neurologic symptoms or signs.

Audiograms should be obtained in all cases in which there are corresponding audio-vestibular symptoms or signs.

Diagnostic studies that may be helpful in patients with disequilibrium vary greatly depending on the identified neurologic or mechanical deficits but may include electromyography and nerve conduction studies, cranial or spinal imaging, and various other blood tests and imaging studies.

Diagnostic studies that may be helpful in selected patients with presyncope and syncope (not attributable to medications or vasovagal reactions) include electrocardiography, Holter monitoring, tilt-table testing, glucose tolerance testing, and studies of autonomic

**Table 3 – Indications for brain imaging in patients with dizziness**

<table>
<thead>
<tr>
<th>Indications for brain imaging</th>
<th>CNS signs or symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute persistent vertigo or motor disequilibrium, without another identified basis, particularly in persons over age 50</td>
<td>CNS signs or symptoms</td>
</tr>
<tr>
<td>Head trauma</td>
<td>CNS signs or symptoms</td>
</tr>
<tr>
<td>Alcohol or substance abuse</td>
<td>CNS signs or symptoms</td>
</tr>
<tr>
<td>Malignancy (especially cancer associated with a significant potential for CNS metastases)</td>
<td>CNS signs or symptoms</td>
</tr>
<tr>
<td>Coagulopathy (including that associated with warfarin treatment)</td>
<td>CNS signs or symptoms</td>
</tr>
<tr>
<td>Immunodeficiency (AIDS, immunosuppressant medications, etc)</td>
<td>CNS signs or symptoms</td>
</tr>
</tbody>
</table>

CNS, central nervous system.

*Brain imaging is generally not indicated in typical cases of benign paroxysmal positioning vertigo, in sensory disequilibrium (with peripheral neuropathic findings), in presyncope, or in nonspecific chronic dizziness, psychophysiologic dizziness, or hyperventilation syndromes in the absence of focal neurologic symptoms or signs.*
and endocrine function. Patients with non-otologic dizziness and lightheadedness who demonstrate normal results from a neuro-otologic evaluation should be considered for head-upright tilt-table testing, even with normal in-office clinical orthostatic blood pressure test results. For patients with orthostatic hypotension in the absence of hypovolemia, the most important diagnostic “test” is a critical review of their medications.

**STEP 5: SPECIALTY CONSULTATION**

Just as identifying the category of dizziness helps target diagnostic testing, it also helps direct specialty consultations when they are needed. For example, patients with vertigo may require consultation with a neurologist, neurosurgeon, or otolaryngologist or possibly a physical therapist, depending on the underlying cause. Similarly, patients with disequilibrium may require the expertise of a neurologist, physiatrist, or physical therapist; those with presyncope may require a cardiologist or neurologist; and patients with chronic subjective dizziness may benefit from a consultation with a mental health specialist.

**CONCLUSION**

Although the term “dizziness,” and a patient’s description of it, can be vague, in most cases a thorough history, provocative testing, and detailed examination will allow distinction of the major categories of dizziness and will often allow a specific etiologic diagnosis as well. Brain imaging and other expensive diagnostic testing should be ordered selectively. These steps will allow directed specialty consultation or appropriate disease-specific treatment to be given.

**REFERENCES:**