Standardizing your approach to dizziness and vertigo

First, determine whether the sensation the patient is experiencing is dizziness or true vertigo. Then eliminate ominous causes from the array of benign ones.

Dizziness. Vertigo. These 2 terms are often used interchangeably by patients, with the sensations described as imbalance, lightheadedness, disorientation, presyncope, confusion—among others. While dizziness is a broad term that is often used to describe all the aforementioned sensations, including vertigo, true vertigo (a specific type of dizziness) is defined as the perception of movement within one’s visual field while stationary.1 Because patients are not usually aware of the distinction, their reports of signs and symptoms can cause much confusion for health care providers, thereby delaying a diagnosis.

International studies have reported the prevalence of both dizziness and vertigo to be between 15% and 36%.2,3 Over half of all patients with dizziness and vertigo are cared for by the family physician (FP), and the sensations combined account for approximately 5% of all family medicine visits.4,5 Additionally, between 2.5% and 4% of all emergency department (ED) visits stem from complaints of dizziness and vertigo, with an incidence of up to 25% in those >65 years of age.6,7

Causes of dizziness and vertigo are broad, ranging from the benign to the life-threatening. It has been reported that upwards of 50% of patients presenting to the FP’s office for dizziness leave without a diagnosis.8 Given the confusion surrounding the terms and their broad differential, this review aims to provide FPs with the tools to accurately discern benign from ominous causes.

Nonvestibular benign causes vastly outnumber life-threatening ones

Causes of dizziness are classified as either vestibular (these cause true vertigo) or nonvestibular in origin, with nonvestibular causes being more common.7

Nonvestibular etiologies:
Numerous and varied
Nonvestibular causes are broad, spanning many different body...
systems. Cardiovascular causes of dizziness may include orthostatic hypotension, cardiac arrhythmia, myocardial infarction, and carotid artery stenosis. Metabolic causes include complications of diabetes such as hypoglycemia and peripheral neuropathy. Psychiatric conditions such as anxiety, depression, and bipolar disorder can manifest as dizziness, disorientation, or psychogenic vertigo. Medications including nonsteroidal anti-inflammatory drugs, anticonvulsants, antipsychotics, and sedatives can all contribute to dizziness. Other causes of dizziness include Parkinson’s disease, musculoskeletal disorders, and gait disorders. Especially in the elderly, sensory deficit (peripheral neuropathy), poor vision, and polypharmacy (≥5 medications) are common causes of dizziness.

Vestibular etiologies of dizziness = true vertigo

Vestibular causes of a patient’s feelings of dizziness manifest as true vertigo and can be categorized as either central (a dysfunction of one or more parts of the central nervous system that help process balance and spatial information or along the pathway where these sensations are interpreted) or peripheral (a dysfunction of the balance organs of the inner ear) in origin.

- **Central vestibular causes** include vertebrobasilar ischemic stroke, vertebrobasilar insufficiency (transient ischemic attack), vestibular migraines, and meningioma of the cerebellopontine angle and posterior fossa.

- **Peripheral vestibular causes.** Benign paroxysmal positional vertigo (BPPV) represents the most common peripheral diagnosis. It is caused by dislodged otoliths in the posterior semicircular canal. While the majority of BPPV cases are idiopathic in nature, up to 15% may result from previous head injury. Other peripheral vestibular causes include vestibular neuronitis, viral labyrinthitis, Meniere’s disease, vestibular schwannoma, perilymphatic fistula, superior semicircular canal dehiscence (SSCD), and head trauma (basilar skull fracture).

**Start with a history:**

**Is it dizziness or true vertigo?**
The clinical history typically guides the differential diagnosis (FIGURE). Identifying true vertigo from among other sensations helps to limit the differential because true vertigo is caused by vestibular etiologies only. True vertigo is often reported by patients as “seeing the room spin;” this stems from the perception of motion. A notable exception is that patients with orthostatic hypotension will often describe spinning sensations lasting seconds to minutes when they rise from a seated or supine position.

Never depend solely, however, on patient-reported sensations, as not all patients with true vertigo report spinning, and some patients with nonvestibular causes interpret their dizziness as a spinning sensation. Therefore, it is important to tease out specifics about the timing, triggers, and associated symptoms in order to further delineate possible causes (TABLE).

**Make a list of current medications.** Gather a comprehensive list of current medications, especially from elderly patients, because polypharmacy is a major contributor to dizziness in this population. Keep in mind that elderly patients presenting with dizziness/vertigo may have multifactorial balance difficulties, which can be revealed by a detailed history.

**Physical exam: May be broad or focused**

Given the broad range of causes for dizziness, cardiovascular, head/neck, and neurologic examinations may be performed as part of the work-up, as the clinical history warrants. More typically, time is spent ruling out the following common causes.

- **Orthostatic hypotension.** Orthostatic vitals are recommended initially in all patients with dizziness, although these may be normal in patients with orthostatic hypotension. A diagnosis of orthostatic hypotension can be made with systolic blood pressure decreases of 20 mm Hg or diastolic pressure decreases of ≥10 mm Hg within 3 minutes of standing. An increase in heart rate >30 beats per minute after rising from a supine position may indicate autonomic disturbances such as postural orthostatic tachycardia syndrome. However, physical examination findings

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alone are insufficient to make the diagnosis of orthostatic hypotension, and determining the underlying cause of the orthostatic hypotension (dehydration, cardiac dysfunction, pure-autonomic failure, medication adverse effect) is vital.\(^\text{18}\)

**BPPV.** Perform the Dix-Hallpike maneuver (see [https://collections.lib.utah.edu/details?id=177177](https://collections.lib.utah.edu/details?id=177177) for a demonstration of the maneuver) on patients presenting with dizziness with features suggestive of BPPV (eg, attacks of dizziness triggered by head movements).\(^\text{20,21}\)

As BPPV is the most common cause of vestibular dizziness, a negative Dix-Hallpike can be helpful in refining the differential diagnosis.\(^\text{20,21}\) The maneuver begins with the patient seated, looking directly ahead. To test the left side, ask the patient to turn his/her head 45 degrees to the left. Then direct the patient to lie back, so that the patient’s head is off the edge of the examination table and hyperextended, while maintaining the same head orientation. To test the right side, repeat the procedure with the patient turning his/her head to the right.

Torsional nystagmus is necessary for a positive Dix-Hallpike, which is diagnostic for BPPV. The laterality of BPPV can be determined by paying attention to the fast phase of the torsional nystagmus; the superior pole of the eye beats toward the affected side.\(^\text{14}\) The patient may report severe dizziness or vertigo during the Dix-Hallpike, but without torsional nystagmus, the test is negative, and the patient does not have BPPV.\(^\text{14}\)

**Neurologic causes.** Perform a complete neurologic examination in patients who clearly do not have a history of orthostatic hypotension and who have a Dix-Hallpike test that is negative or not indicated.\(^\text{4}\) Also perform cerebellar testing including rapid alternating movements, a finger-to-nose test, and a heel-to-shin test. Round out the

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**FIGURE**

Diagnostic approach to patients presenting with dizziness and/or vertigo

<table>
<thead>
<tr>
<th>False vertigo (paroxysmal positional vertigo)</th>
<th>True vertigo (vestibular)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triggered/ exposure</td>
<td>Continuous</td>
</tr>
<tr>
<td>Continuous</td>
<td>Episodic</td>
</tr>
<tr>
<td>• Medication</td>
<td>• BPPV</td>
</tr>
<tr>
<td>• Polypharmacy</td>
<td>• Vestibular migraine</td>
</tr>
<tr>
<td>•Orthostatic hypotension</td>
<td>• Basilar skull fracture</td>
</tr>
<tr>
<td>Hypoglycemia (diabetes)</td>
<td>• Vestibular insufficiency</td>
</tr>
<tr>
<td>Motion sickness</td>
<td>• SSCD syndrome</td>
</tr>
<tr>
<td>Mal debarquement</td>
<td>• Meningioma</td>
</tr>
<tr>
<td>True vertigo (vestibular)</td>
<td>Continuous</td>
</tr>
<tr>
<td>Continuous</td>
<td>Episodic</td>
</tr>
<tr>
<td>• Cardiac arrhythmia</td>
<td>• Stroke</td>
</tr>
<tr>
<td>• Mood disorder (panic attack)</td>
<td>• Vestibular neuritis</td>
</tr>
<tr>
<td>• Gait/MSK disorder</td>
<td>• Viral labyrinthitis</td>
</tr>
<tr>
<td>• Parkinson's disease</td>
<td>• Vestibular schwannoma</td>
</tr>
<tr>
<td>• Sensory deficit</td>
<td></td>
</tr>
<tr>
<td>• Multiple sclerosis</td>
<td></td>
</tr>
<tr>
<td>• Cerebellar degeneration</td>
<td></td>
</tr>
</tbody>
</table>

BPPV, benign paroxysmal positional vertigo; MSK, musculoskeletal; SSCD, superior semicircular canal dehiscence.
DIZZINESS AND VERTIGO

Up to 15% of cases of benign paroxysmal positional vertigo may result from previous head injury.

### TABLE

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Timing</th>
<th>Trigger</th>
<th>Associated symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orthostatic hypotension</td>
<td>Episodic, lasting seconds to minutes</td>
<td>Postural changes, standing from seated or supine position</td>
<td>Light-headedness, imbalance, confusion, syncope</td>
</tr>
<tr>
<td>Multifactorial imbalance</td>
<td>Episodic, fluctuating severity</td>
<td>With ambulation, may worsen on uneven surfaces</td>
<td>Light-headedness</td>
</tr>
<tr>
<td>Stroke</td>
<td>Continuous, lasting minutes to hours, and may worsen</td>
<td>Spontaneous, associated with atherosclerosis, severe symptoms at onset</td>
<td>Headache, confusion, vomiting, ataxia, diplopia, dysarthria, dyshagia, paresthesia</td>
</tr>
<tr>
<td>Transient ischemic attack</td>
<td>Continuous, lasting minutes to hours</td>
<td>Spontaneous, associated with atherosclerosis</td>
<td>Headache, confusion, vomiting, ataxia, diplopia, dysarthria, dyshagia, paresthesia</td>
</tr>
<tr>
<td>Vestibular migraines</td>
<td>Episodic, lasting minutes to hours</td>
<td>Migraine triggers: stress, fatigue, weather, and food</td>
<td>Headache, head motion intolerance, photophobia, phonophobia</td>
</tr>
<tr>
<td>BPPV</td>
<td>Episodic, lasting seconds, fatigable</td>
<td>Head turning and rolling in bed, possible prior head trauma</td>
<td>Nausea, no associated ear symptoms (patient will deny hearing loss, tinnitus)</td>
</tr>
<tr>
<td>Meniere’s disease</td>
<td>Episodic, lasting 20 minutes to several hours</td>
<td>Spontaneous</td>
<td>Nausea, vomiting, unilateral tinnitus/aural fullness/hearing loss</td>
</tr>
<tr>
<td>Vestibular neuronitis/ labyrinthitis</td>
<td>Episodic, lasting days, fluctuating severity</td>
<td>Spontaneous, associated with viral infection</td>
<td>Nausea, vomiting, unilateral hearing loss (only for labyrinthitis)</td>
</tr>
</tbody>
</table>

BPPV, benign paroxysmal positional vertigo.

### Neurologic Exam

A neurologic exam with an assessment of gait and a Romberg’s test (see https://www.youtube.com/watch?v=U5a4lbwmWo for a demonstration of Romberg’s test). Romberg’s test is performed by having the patient place his/her feet together with hands at sides and eyes closed. The patient is observed for up to a minute, with a positive test denoted by a loss of balance. Abnormal gait may indicate peripheral neuropathy, while a positive Romberg’s test suggests involvement of the proprioceptive receptors and/or their pathway.

#### Central/peripheral vestibular causes

The head impulse, nystagmus, test of skew (HINTS) examination can differentiate between central and peripheral vestibular causes of dizziness and rule out stroke (a central vestibular cause).22 (See https://collections.lib.utah.edu/details?id=177180 for a video demonstrating the steps involved in performing the HINTS examination.) The head impulse (HI) portion of the exam is performed by moving the patient’s head side to side, while having the patient focus on the examiner’s nose. Rapid movements of both eyes (“abnormal” HI) suggest a peripheral etiology, while no eye movement with gaze fixated on the examiner’s nose (“normal” HI) is concerning for stroke or another central cause of vertigo.22

Nystagmus is assessed by having the patient follow the examiner’s finger as it moves in a horizontal direction. Spontaneous hori-
Identifying true vertigo from among other sensations helps to limit the differential because true vertigo is caused by vestibular etiologies only.

Horizontal unidirectional nystagmus suggests a peripheral cause, while vertical or torsional bidirectional (direction-changing) nystagmus points to a central cause. The test of skew is executed by covering and uncovering each of the patient’s eyes, while asking the patient to look ahead. Vertical deviation of the eye after uncovering suggests a central etiology, more specifically one involving the brainstem.

Diagnostic testing/imaging has a limited, but pivotal role

There is a limited role for routine laboratory testing in patients with dizziness. However, for those patients with underlying medical conditions (eg, diabetes), which may contribute to the symptoms, routine blood work can be ordered (ie, finger-stick blood glucose test).

More worrisome suspicions. Patients suspected of cardiac causes should have a full cardiac work-up performed. For suspected stroke, brain tumor, or head trauma, specific computed tomography or magnetic resonance imaging can be arranged. Carotid doppler can be used if dizziness is suspected to be caused by orthostatic hypotension or a vascular cause.

Audiologic and vestibular testing. Audiologic testing is not routinely recommended and is only warranted in instances when patients report hearing loss or changes. Referral to an otolaryngologist for vestibular testing is warranted once life-threatening and alternate etiologies have been ruled out, and a vestibular disorder remains at the top of the differential.

Treatment hinges on cause and may be multifaceted

Treatment hinges on the specific cause of the patient’s dizziness and may involve useful maneuvers, medication, physiotherapy, or perhaps even surgery.

Employ a particle repositioning maneuver for BPPV

A positive Dix-Hallpike test should prompt the use of a particle repositioning maneuver (PRM) to treat BPPV. The goal of PRMs, such as the Epley maneuver (see https://www.youtube.com/watch?v=9SLm76jQg3g for a demonstration of this maneuver), is to move the head in such a way as to return displaced otoliths in the semicircular canal back to the utricle. The Epley maneuver is specific for treating posterior semicircular canal BPPV, which is the most common variant.

Performing the Epley maneuver. To perform the Epley PRM for correction of an otolith in the left posterior semicircular canal, ask the patient to sit and look straight ahead. Lay the patient back, while asking the patient to turn his/her head 45 degrees to the left side. Then ask the patient to turn his/her head 45 degrees to the right side. Instruct the patient to maintain the same 45-degree head orientation, while rolling over to his/her right shoulder, ending in the right decubitus position. Conclude the maneuver by having the patient sit up.

Performing the barbecue roll maneuver. Different PRMs exist to treat less common variants of BPPV, including the “barbecue roll” maneuver for horizontal BPPV (see https://www.youtube.com/watch?v=mwTmM6uF5yA for a demonstration of this maneuver). The barbecue roll maneuver is initiated with the patient looking ahead and lying back. For a left-sided horizontal canal otolith, the patient first turns to the left decubitus position, then moves clockwise to the right decubitus position, stopping at each position for approximately 20 seconds, all while maintaining a straight head position. The patient then turns clockwise into a prone position, pausing, and finally turning into the left decubitus position again. The maneuver is completed with the patient sitting up.

Medications are used to treat symptoms and/or underlying causes

Adjustments in antihypertensives can be made in cases of orthostatic hypotension. Antiemetics (ondansetron, promethazine, metoclopramide), antihistamines (meclizine, dimenhydrinate, diphenhydramine), and benzodiazepines (lorazepam, diazepam) may be used during acute and brief vertiginous episodes to decrease symptom
severity after central causes have been ruled out. However, patients with BPPV should avoid these medications as they may blunt central compensation and increase the risk of falls. Research has shown betahistine to improve vertigo control only in patients with Meniere’s disease and only when taken regularly and prophylactically. Therefore, do not prescribe betahistine for all other causes of dizziness/vertigo.

Consider physiotherapy
All patients with dizziness/vertigo, and particularly those presenting with primary balance concerns, may benefit from vestibular rehabilitation therapy (VRT). This is an exercise-based program focusing on habituation of dizziness and improvement of postural stability. VRT can improve dizziness associated with central and peripheral vestibular lesions, vertigo of uncertain etiology, and psychogenic vertigo. Typically, the vestibular physiotherapist will provide home exercises for the patient, reducing the cost and inconvenience of attending multiple sessions.

Surgery and referrals
Referrals for surgery are rare and are typically reserved for refractory causes of vestibular disease, such as Meniere’s disease, BPPV, SSCD syndrome.

Referral to the ED is warranted for symptom control if an acute vertiginous episode is refractory to initial management. Emergent or urgent neurology consultation is indicated for suspected or confirmed central disorders. Urgent cardiology referral is recommended for patients with symptoms of presyncope/syncope, arrhythmia, or persistent orthostatic hypotension after conservative management. Outpatient referral to an otolaryngologist is warranted if the patient has failed a course of balance physiotherapy, has a persistently positive Dix-Hallpike test after a PRM and vestibular/balance physiotherapy, or has asymmetric hearing loss.

Management starts with primary and secondary prevention
Patient education is essential for avoiding potential triggers of dizziness. Patients with orthostatic hypotension should be educated about the need to correct the underlying mechanism, including the need for adequate hydration and recognition of offending medications and contributory conditions/situations (caffeine, heat, standing quickly). Encouraging balance maintenance through exercise and physiotherapy can help with gait and musculoskeletal disorders, and reducing harmful habits (smoking, poor diet, no exercise) can lead to overall improved cardiovascular health. Advise those with Meniere’s disease to avoid potential triggers such as caffeine, high sodium foods, and alcohol.

References


