



CONTINUUM AUDIO  
INTERVIEW AVAILABLE  
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# Approach to the History and Evaluation of Vertigo and Dizziness

By Terry D. Fife, MD, FAAN, FANS

## ABSTRACT

**PURPOSE OF REVIEW:** This article reviews a method of obtaining the medical history of patients presenting with dizziness, vertigo, and imbalance. By combining elements of the history with examination, the goal is to identify patterns and an effective differential diagnosis for this group of patients to help lead to an accurate diagnosis.

**RECENT FINDINGS:** Studies over the past dozen years have changed the historical approach to patients with dizziness from one based primarily on how the patient describes the sensation of dizziness. This older approach can lead to misdiagnosis, so a preferred method puts greater emphasis on whether the dizziness is acute or chronic, episodic or continuous, or evoked by or brought on by an event or circumstance so that a pattern may be derived that better narrows the differential diagnosis and focused examination can further narrow to a cause or causes.

**SUMMARY:** Dizziness is a common symptom of many possible causes. This article will help clinicians navigate gathering the history and examination to formulate a working diagnosis in patients affected by dizziness.

## CITE AS:

CONTINUUM (MINNEAP MINN)  
2021;27(2, NEURO-OTOLOGY):  
306-329.

Address correspondence to  
Dr Terry D. Fife, Barrow  
Neurological Institute, 240 W  
Thomas Rd, Ste 301, Phoenix,  
AZ 85013, tfife@email.arizona.  
edu.

**RELATIONSHIP DISCLOSURE:**  
Dr Fife reports no disclosure.

**UNLABELED USE OF  
PRODUCTS/INVESTIGATIONAL  
USE DISCLOSURE:**  
Dr Fife reports no disclosure.

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## INTRODUCTION

**D**izziness is frequently encountered in neurology, otolaryngology, and general medical practice, and generates many visits to emergency departments and other medical clinics. The lifetime prevalence of having significant dizziness is between 17% and 30%.<sup>1</sup> The lifetime prevalence of dizziness due to vestibular disease was 7.4% in one population-based survey.<sup>2</sup> A multinational observational registry of patients with some form of a peripheral vestibular symptom found that 65% were female and that those aged 51 to 60 years were the most commonly affected. The age group between 41 and 70 years accounted for almost two-thirds of all patients with dizziness whereas patients older than 70 accounted for 18.4%.<sup>3</sup> Another study in a neuro-otology clinic found that the peak age group for having dizziness was 60 to 69 years, and women were more likely to have nonperipheral vestibular causes of dizziness.<sup>4</sup> This suggests, perhaps contrary to a widely held view, that people 70 years and older are not the age group most commonly seen for peripheral vestibular disorders. Meanwhile, in a study of all referred patients with a chief complaint of dizziness at an academic otolaryngology

neuro-otology clinic where one might think peripheral vestibular causes would be especially common, the researchers found 42% of patients did not have peripheral vestibular disorders. This underscores the breadth of possible causes of dizziness well beyond just inner ear disorders. Furthermore, approximately one in six patients presents with two causes of dizziness at the same time.<sup>4</sup>

**TABLE 1-1** lists these and some additional examples of common types of dizziness that often occur together. The most common combinations of two diagnoses found in a cohort study of an academic otolaryngology clinic were (1) vestibular migraine and benign paroxysmal positional vertigo (BPPV) and (2) vestibular migraine and Ménière disease.<sup>4</sup>

Many patients with chronic dizziness have an expensive and often long sojourn before an accurate diagnosis is made. One survey study found that many

### Common Combinations of Simultaneous or Overlapping Disorders Causing Dizziness or Vertigo

**TABLE 1-1**

| Combined disorders  | Typical associated history   |
|---|--|
| <b>Vestibular migraine and benign paroxysmal positional vertigo (BPPV)</b>          | Episodes of spinning, rocking swaying, tilting, and frequent motion sensitivity with nausea that last minutes to many hours and frequent visually induced vertigo, photophobia, and periodic migraine headaches AND intermittent spells of vertigo triggered by the head tilting back or looking up or when turning in bed   |
| <b>Ménière disease and vestibular migraine</b>                                      | Random prolonged spells of spinning lasting hours and associated with unilateral fluctuating hearing and tinnitus and low-frequency hearing loss on audiometry AND episodes of spinning, rocking swaying, tilting, and frequent motion sensitivity with nausea that last minutes to many hours and frequent visually induced vertigo, photophobia, and periodic migraine headaches |
| <b>Vestibular migraine and persistent postural perceptual dizziness<sup>a</sup></b> | Episodic spells of vertigo, rocking swaying, tilting, floating, and frequent motion sensitivity with nausea, visually induced vertigo, photophobia, periodic migraine headaches, and associated generalized anxiety  |
| <b>Vestibular neuritis and vestibular migraine</b>                                  | Acute onset of spinning vertigo lasting several weeks and gradually abating over time but overtaken by ongoing motion sensitivity with nausea, visually induced vertigo, photophobia, and periodic migraine headaches  |
| <b>Vestibular neuritis and BPPV</b>   | Acute onset of spinning vertigo lasting several weeks and gradually abating over time but punctuated by intermittent spells of vertigo triggered by head tilting back or looking up or when turning in bed   |
| <b>Vestibular neuritis and persistent postural perceptual dizziness</b>             | Acute onset of spinning vertigo lasting days and gradually abating over time but overtaken by constant rocking and/or floating sensations without nausea, with visually induced vertigo and associated generalized anxiety   |
| <b>BPPV and persistent postural perceptual dizziness</b>                            | Intermittent spells of vertigo triggered by head tilting back or looking up or when turning in bed with rocking and/or floating sensations without nausea, with visually induced vertigo and possibly associated generalized anxiety   |
| <b>BPPV and orthostatic intolerance or orthostatic hypotension</b>                  | Intermittent spells of vertigo triggered by head tilting back or looking up or when turning in bed and also intermittent near-fainting lightheaded sensations on standing  |

<sup>a</sup> This combination is fairly common, and determining which disorder accounts for most of the dizziness requires close monitoring of the response to treatments.

were referred to multiple health care providers, but one-third still felt frustrated, misdiagnosed, or misdirected.<sup>5</sup> This article aims to guide practicing clinicians in their approach to patients with dizziness and to use the data from the history and examination to discern a pattern associated with a more specific causative diagnosis.

**CLASSIFICATION OF VESTIBULAR SYMPTOMS**

TABLE 1-2 outlines definitions of common vestibular symptoms as determined by the International Classification of Vestibular Disorders of the Bárány Society.<sup>6</sup> These definitions were developed to standardize nomenclature used in clinical and research communications among those who care for patients with vestibular

**TABLE 1-2** Definition of Common Vestibular Symptoms as Determined by International Consensus<sup>a,b</sup>

| Symptom          | Definition   | Subtypes  |
|------------------|--|---|
| <b>Vertigo</b>   | Sensation of self-motion when no self-motion is occurring or the sensation of distorted self-motion during an otherwise normal head movement | Spontaneous: occurs without obvious trigger<br>Triggered: occurs with an obvious trigger <ul style="list-style-type: none"> <li>● Positional: triggered <i>after</i> changing head position</li> <li>● Head motion: occurs only during head motion</li> <li>● Visually induced: triggered by seeing objects in motion in the visual surround</li> <li>● Sound-induced: triggered by sound</li> <li>● Valsalva-induced: triggered by Valsalva maneuver or straining</li> <li>● Orthostatic: triggered by change in body position from lying or sitting to standing</li> <li>● Other triggered forms</li> </ul> |
| <b>Dizziness</b> | Sensation of disturbed or altered spatial orientation without the feeling of false motion  | Spontaneous: occurs without obvious trigger<br>Triggered: occurs with an obvious trigger <ul style="list-style-type: none"> <li>● Positional: triggered <i>after</i> changing head position</li> <li>● Head motion: occurs only during head motion</li> <li>● Visually induced: triggered by seeing objects in motion in the visual surround</li> <li>● Sound-induced: triggered by sound</li> <li>● Valsalva-induced: triggered by Valsalva maneuver or straining</li> <li>● Orthostatic: triggered by change in body position from lying or sitting to standing</li> <li>● Other triggered forms</li> </ul> |

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and balance disorders. Unlike common teaching in the past, the International Classification of Vestibular Disorders system does not consider vertigo to be a subtype of the broader rubric of dizziness but rather a separate descriptor from dizziness. Vertigo designates the false perception of motion in any direction whereas dizziness designates distortion of spatial orientation without the perception of motion. Thus, by using this construction, a patient with a single disorder, such as BPPV, could simultaneously describe vertigo, dizziness, and postural symptoms even though he or she has a single cause of symptoms. In the International Classification of Vestibular Disorders definitions, the term *vestibular symptoms* does not mean that the symptoms are necessarily caused by or directly related to vestibular structures or physiology; rather, it is meant to be

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| Symptom                         | Definition   | Subtypes  |
|---------------------------------|--|---|
| <b>Vestibulovisual symptoms</b> | Visual symptoms resulting from vestibular dysfunction or from the interaction of the visual and vestibular systems; examples include visual illusions that the environment is tilted or blurring of visual lag during head movements; categorized as <i>external vertigo</i> | <p>External vertigo: illusion that the visual surround is spinning or flowing</p> <p>Oscillopsia: the perception that the visual surround is oscillating or bouncing</p> <p>Visual lag: the illusion that the visual surround lags behind during head movement</p> <p>Visual tilt: the illusion that the visual surround is not true vertical</p> <p>Movement-induced blur</p>  |
| <b>Postural symptoms</b>        | Balance-related symptoms that occur when upright (seated, standing, walking); examples include feeling unsteady, swaying, or rocking only when upright   | <p>Unsteadiness: the feeling of being unstable when seated, standing, or walking</p> <p>Directional pulsion: unsteadiness with a feeling of veering or falling to a particular direction</p> <p>Balance-related near fall: a feeling of imminent or nearly falling due to vestibular symptoms, pulsion, or unsteadiness</p> <p>Balance-related fall: a complete fall due to vestibular symptoms, pulsion, or unsteadiness<sup>c</sup></p> |

<sup>a</sup> Data from Bisdorff A, et al, J Vestib Res.<sup>6</sup>

<sup>b</sup> The use of the term *vestibular symptoms* in this table is meant to refer to the symptoms often used but does not mean to imply that all conditions causing these symptoms have a basis in vestibular pathways or mechanisms.

<sup>c</sup> Not used are terms such as *drop attack*, *otolithic crisis*, *otolith crisis of Tumarkin*. Instead, the consensus panel opted for the terms *balance-related falls* or *near falls*.

a broader term of phenomenological descriptions only some of which have vestibular causes.<sup>6</sup> When terms such as *vestibular disorders, processes, mechanisms,* or *causes* are mentioned, then this does refer to conditions that are due to vestibular structures or mechanisms. Although this set of definitions is intended to permit physicians to speak a common language, it should be expected that patients will continue to describe symptoms in their own way.

**WAYS TO EVALUATE PATIENTS WITH DIZZINESS**

A patient with dizziness can be approached in several ways, and what seems easiest or most useful for each clinician may vary by background, practice setting, specialty, and experience. No system is necessarily ideal for all specialties in all clinical settings. All the approaches are similar and involve data gathering in the history and examination, but which data and which steps follow vary slightly in each approach. Following are four examples:

- 1 Pattern recognition.** This approach focuses on using data from the history and examination to identify patterns (or overlapping patterns) that best fit with a specific cause or causes. The disadvantage is that it takes time to gather details of the history and experience to become familiar with common clinical patterns of disorders that cause vertigo or dizziness. The advantage for the neurologist is that this approach is what neurologists do every day in evaluating patients. For example, neurologists routinely use these methods for evaluating patients with headache, muscle weakness, or unexplained spells. It is the nature of the

**TABLE 1-3 Common Vestibular Disorders Associated With Major Syndrome Categories<sup>a</sup>**

| Syndrome                            | Description  | Examples of disorders   |
|-------------------------------------|--|---|
| <b>Acute vestibular syndrome</b>    | A syndrome of acute-onset, continuous vertigo, dizziness, and unsteadiness lasting days to weeks often associated with nausea, vomiting, nystagmus, and vertigo or dizziness aggravated by head motion in any direction  | Vestibular neuritis, stroke causing vertigo, acute drug toxicity, demyelinating disease vestibulopathy, Wernicke syndrome, selective serotonin reuptake inhibitor (SSRI) or serotonin norepinephrine reuptake inhibitor (SNRI) discontinuation  |
| <b>Episodic vestibular syndrome</b> | A syndrome of recurrent spells of vertigo, dizziness, or unsteadiness lasting seconds to hours, occasionally days. The episodes may be associated with brief periods of nausea, nystagmus, loss of balance, headache, central nervous system symptoms, or hearing symptoms   | Spontaneous: vestibular migraine, Ménière disease, transient ischemic attack (vertebrobasilar insufficiency), vestibular paroxysmia, cardiac causes (aortic stenosis, arrhythmia), episodic ataxias<br><br>Triggered: benign paroxysmal positional vertigo, orthostatic intolerance or hypotension, motion sickness, central positional vertigo |
| <b>Chronic vestibular syndrome</b>  | A syndrome of chronic vertigo, dizziness, or unsteadiness lasting months to years; symptom descriptions may include gait unsteadiness, ataxia, hearing loss, nausea, nystagmus, or oscillopsia; may result from a progressive neurodegenerative disorder, a static deficit in vestibular function, or evolving symptoms between episodic vestibular episodes | Persistent postural perceptual dizziness, bilateral vestibulopathy, late effects of stroke, cerebellar ataxias, posterior fossa neoplasms, chronic visually induced vertigo or dizziness, mal de débarquement   |

<sup>a</sup> Data from Bisdorff A, et al, J Vestib Res<sup>6</sup> and Bisdorff AR, et al, Neurol Clin.<sup>9</sup>

specialty (and other medical specialties as well) to identify patterns of symptoms and signs that point to a specific disorder. This is the approach discussed in this article and is not really dissimilar to the International Classification of Vestibular Disorders approach or those guided by TiTrATE (Timing, Triggers, Associated symptoms and Targeted Examination)<sup>7</sup> or ATTEST (Associated symptoms, Timing, Triggers, Examination Signs and Testing)<sup>8</sup> described later in this list.

- 2 International Classification of Vestibular Disorders classification. This is not so much an approach as it is an organizational scheme to proceed from symptoms and signs to syndromes to mechanisms in the cause of vertigo and dizziness. It consists of using information from the history and examination to place the patient's symptoms into one of three syndrome patterns: (1) acute vestibular syndrome; (2) episodic vestibular syndrome (episodic or triggered); or (3) chronic vestibular syndrome. Once categorized in one of these syndrome patterns, each syndrome being associated with a finite list of specific diagnoses or mechanisms (TABLE 1-3),<sup>6,9</sup> the differential diagnosis can be narrowed and pattern recognition is used to arrive at a specific diagnosis. A limit of this system is that some vestibular diagnoses involve symptoms that cross boundaries between episodic vertigo and chronic vertigo because disorders do not always fit neatly into the syndromic category. In addition, when a patient has more than one form of dizziness simultaneously, which occurs in nearly 20% of cases, this must be recognized and the approach must be applied to each type of dizziness.
- 3 TiTrATE and ATTEST. TiTrATE is an acronym for Timing, Triggers, Associated symptoms and Targeted Examination.<sup>7</sup> ATTEST is an acronym for Associated symptoms, Timing, Triggers, Examination Signs and Testing.<sup>8</sup> These algorithms use memory aids to help clinicians recall which parts of the history are most essential. However, some pattern recognition is needed; pattern recognition is still very helpful for the most common urgent care causes of dizziness such as BPPV, vestibular neuritis, and stroke presenting with isolated dizziness or vertigo. This system was developed as a way of improving diagnosis, particularly for those in primary care and emergency settings who need to keep their history abbreviated.
- 4 Symptom description. This now-outdated approach introduced in the early 1970s is based on categorizing the patient's description of dizziness as fitting into one of four categories: vertigo, presyncope, impaired equilibrium, or nonspecific dizziness. Data over the past dozen years have made apparent that using a symptom characterization as the sole algorithmic branch point leads to more misdiagnosis when compared with using timing and trigger information. This is especially true in the acute care setting such as in emergency departments. The symptom description approach was introduced at a time when it was believed that the most common single cause of dizziness was "hyperventilation," which was said to account for 22% of dizziness. This was tested by having the patient hyperventilate, and, if the sensation resembled his or her dizzy sensation, then "hyperventilation can often thus be diagnosed without further ado."<sup>10</sup> This is no longer considered valid. Although some value remains in obtaining a description of the patient's sensation, its value in diagnosis tended to be overemphasized by using this approach. The description should be combined with other features of the history including onset, duration, triggers, factors that aggravate symptoms, and so on.

It is not necessary to abandon any aspect of the history that a patient can describe consistently and reliably. All aspects of history (symptom description, onset, frequency, duration, and provoking or aggravating circumstances) are prone to being incorrectly conveyed by the patient or incorrectly interpreted by the clinician (CASE 1-1). Clinicians should probe until the question is understood and answered as well as possible. At times, the history may still fit no pattern or simply remain unclear. Limitations in recalling and describing sensations, discerning triggers, and recalling durations are understandably susceptible to some unreliability. Memory is malleable and can be influenced by the questions asked or by friends and family, prior life experiences, anxiety and emotional links to the memory, and other factors.<sup>12</sup> Taking a history is still partly an art. In addition, health care providers must also watch for their own bias errors in concluding a diagnosis (TABLE 1-4).

## KEY POINTS

- Dizziness is a common symptom that occurs at all ages but especially in patients aged between 41 and 70 years.
- Peripheral vestibular disorders are common, but half of patients with dizziness have a nonvestibular mechanism, and approximately one in six patients present with two different causes of dizziness at the same time.
- Many patients with dizziness see multiple health care providers in evaluation of the dizziness and feel frustrated, misdiagnosed, or misdirected.
- Overreliance on a patient's description of the dizziness and using it as the main piece of information to choose among causes leads to mistakes in the diagnosis.
- All aspects of history (symptom description, onset, frequency, duration, and provoking or aggravating circumstances) should be questioned until understood as well as possible because any part of the history can be miscommunicated by a patient or misunderstood by the health care provider.

**CASE 1-1**

A 37-year-old woman presented with dizziness that came on suddenly 4 weeks earlier as a feeling of abrupt intense spinning. She believed the dizziness was related to her neck because she had some neck pain on and off for which she sought chiropractic care in the past that helped. She said the dizziness lasted for minutes to hours, and she had some nausea, as well. At times, she felt like her “head is swimming,” and, when she got up, sometimes she felt off balance or tilted to one side briefly. When asked what she was doing at the time of the first episode, she indicated she was getting out of bed. Another time she had straightened up after putting away some shoes on the closet floor. She also indicated that none of the spells occurred when she had remained completely motionless but seemed triggered by moving her neck.

Examination was normal including tandem gait and no spontaneous or gaze-evoked nystagmus was seen. She had paroxysmal upbeat torsional nystagmus with Dix-Hallpike positioning to the right; that is, nystagmus fast phases caused the upper pole of her eyes to beat toward the right ear during straight-ahead gaze so that the upper pole of the right eye extorted and the upper pole of the left eye intorted. This gave the appearance from the examiner’s viewpoint of counterclockwise torsional nystagmus, characteristic of right posterior canal benign paroxysmal positional vertigo (BPPV).

**COMMENT**

In this case, the patient gave a clear history of a spinning feeling but also of a swimming sensation. This was a clue for a vestibular mechanism; however, as an isolated piece of the history, a vestibular mechanism was suspected but certainly not assured. The patient also thought the neck movement was the trigger, but with further questioning, it was actually head movement relative to gravity that was the trigger, and neck movement, by necessity, also occurred. Finally, the patient gave imprecise durations of the symptoms because she lumped the spinning feeling together with the vague swimming-head after-effect she felt at times when active. This makes the point that patients may have several durations and sensations from the same disorder and may misattribute the triggering circumstance and conflate several sensations, resulting in incorrect timing or duration of dizziness or vertigo. It also points out why examining with the Dix-Hallpike test is advisable in patients with vestibular symptoms even if their history seems to steer one away from the diagnosis of BPPV. Throughout this issue, the term *Dix-Hallpike test* is used rather than *Dix-Hallpike maneuver* (even though both are correct) to distinguish it from treatment maneuvers such as canalith repositioning or the Epley maneuver, which are discussed in more detail in the article “Episodic Positional Dizziness” by Kevin A. Kerber, MD, MS,<sup>11</sup> in this issue of *Continuum*.

## Taking the History

Good history-taking both conveys empathy and gathers important information about the medical symptoms.<sup>13</sup> It is often best to start with an open-ended question about when the symptoms began and what the patient is experiencing. Some patients give a very good and cogent history of their symptoms and prefer to be listened to. If the patient begins by talking about prior tests or what others have told him or her, try to redirect the patient to get the key details of the actual symptoms. Once the current symptoms and symptom evolution up to the time of the visit are obtained, then one can review the test results, prior diagnoses, and treatments tried.

**SYMPTOM DESCRIPTION.** As mentioned earlier, overemphasis of the description of the sensation of dizziness or vertigo can lead to misdiagnosis, especially in the emergency department setting.<sup>14</sup> Nevertheless, one should not ignore this part of the history but rather take it in context along with the other historical information. Patients may describe spinning, whirling, rotational sensations, tilting, sinking, free-falling, or rising, and all these symptoms defined as vertigo imply a higher likelihood of a vestibular process. Such symptoms are quite uncommon in presyncope, for example.<sup>15</sup> Dizziness and lightheadedness, nausea, yawning, visible pallor observed by others, diaphoresis, and a feeling as though one is about to pass out are much more characteristic of cardiovascular causes of dizziness.<sup>15</sup> Patients with vestibular migraine may report a multitude of sensations, including spinning, rocking, floating, motion sickness, and visually induced vertigo and dizziness, whereas patients with Ménière disease are much more likely to describe vertigo as a spinning feeling with nausea and vomiting, often to the point they do not want to even move during attacks.

**SYMPTOM ONSET.** The mode of onset can be helpful in some cases; some causes can lead to abrupt symptoms and some can evolve more gradually, and others may have a clear relation to a specific event. For example, acute vertigo after significant head trauma suggests a traumatic vestibulopathy. When the onset occurs after minor trauma or a mild concussion and symptoms are subjective and

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## Error Avoidance in Diagnosis of Dizziness<sup>a</sup>

TABLE 1-4

| Error                                 | Remedy   |
|---------------------------------------|--|
| Incorrect data                        | Probe the answers and take a careful history. Take a repeat history on follow-up to reaffirm the diagnosis or revise the diagnosis. Recollection is itself influenced by prior questioning as patients begin to think about their previous responses and may give somewhat different answers with sequential questioning.  |
| Lack of familiarity with the patterns | Lack of familiarity with the pattern of a cause or encountering a rare pattern is part of training and experience and why lifelong learning is crucial. Be aware of and keep up with evidence-based guidelines where they exist to expand one's sphere of competence.  |
| Cognitive bias                        | We all have a tendency to draw conclusions that reinforce our first impression or to be influenced by the historian or by the way the history is delivered (eg, haphazard, tangential, fraught with an anxious affect, having an overly inclusive symptom list). Care should be exercised to avoid dismissing patient symptoms because the patient relays the history poorly or with a great deal of superimposed anxiety. |

<sup>a</sup> Errors in identifying the diagnosis can arise from several sources, only some of which are listed in this table.



signs are absent, one must view this with circumspection and in full context. The onset of rocking or swaying dizziness without nausea after a cruise suggests mal de débarquement. Onset of dizziness after turning in bed or tilting the head can suggest BPPV as this may indicate that position changes are actual triggers (see the Triggers section). Abrupt onset of vertigo and loss of equilibrium without known provocation can suggest a vascular mechanism, especially if accompanied by the simultaneous onset of other neurologic features. Symptoms that begin after initiation of a new medication can indicate a medication is the cause (CASE 1-2).

**SYMPTOM PERIODICITY.** It can be helpful to ascertain whether symptoms are episodic or constant or whether the symptoms are constant at a low ebb but periodically intensify and whether they are influenced by a particular circumstance. For example, some patients with vestibular migraine have episodes of vertigo but no symptoms in between. A bit more commonly, however, some patients with vestibular migraine have a continuing susceptibility to motion sickness and visually induced vertigo but try to avoid those triggers and then, in addition, have some periodic spontaneous vertigo spells. Ménière disease is characteristically episodic often with some residual symptoms for a few days after attacks if they are severe, but patients usually recover before the next attack. Occasionally, Ménière disease may cause attacks with enough

## CASE 1-2

A 63-year-old woman reported nearly 1 year of dizziness and feeling unsteady. She described feeling off balance when she was up and walking or with activity on her feet. She had no spinning or sensations of motion and speculated that it may be her vision or her neck, but evaluations by an ophthalmologist and cervical spine MRI performed by an orthopedic surgeon did not confirm her suspicions. She had tried acupuncture, chiropractic treatment, and meclizine. Acupuncture seemed to make her neck feel better, but none of these measures improved the dizziness. She said her dizziness was simply present all the time. She had no focal weakness, numbness, change in hearing, slurred speech, or visual symptoms. She said her neck pain “acts up” sometimes, and she avoided lying on her left side when sleeping. Her past medical history was notable for a 10-year history of type 2 diabetes mellitus and moderate neuropathy.

Examination was normal except for neuropathy in a stocking distribution to the ankles. Dix-Hallpike positioning to the left revealed paroxysmal positional nystagmus, which was treated.

## COMMENT

Many patients try to identify the causes of their symptoms and sometimes develop incorrect conclusions. In this case, the patient gave little reason to suspect benign paroxysmal positional vertigo (BPPV) except that she could not lie on her left side. Her imbalance was probably a combination of mild neuropathy, and her dizziness was likely related to BPPV during motion activities. As in the previous case, Dix-Hallpike positioning can reveal BPPV even when the history reported lacks appropriate timing and triggers.

frequency that patients barely recover from one spell before another occurs. Vestibular neuritis usually has a monophasic course of abrupt severe symptoms with slow and gradual resolution over a week to several months depending on its severity. BPPV is characteristically episodic, but if a patient has some proneness to motion sickness, some degree of dizziness might be reported as seeming to be nearly constant with periodic worsening.

**DURATION OF SYMPTOMS.** Particularly in the case of vertigo, BPPV should be high on the list of possible causes of spells that are shorter than 1 minute. For spells lasting minutes, a transient ischemic attack affecting vestibular structures and vestibular migraine are considerations. For spells lasting 2 to 6 hours, Ménière disease and vestibular migraine can be causes. Chronic symptoms can be seen with persistent postural perceptual dizziness, mal de débarquement, bilateral vestibulopathy, and cerebellar degeneration.

**TRIGGERS.** A trigger should be obvious and reproducible to qualify as a trigger and not just a possible exacerbating circumstance. For example, Dix-Hallpike positioning is a trigger for active BPPV because it results in vertigo most of the time it is performed. However, someone with vestibular neuritis will feel dizzy during the Dix-Hallpike test but will also feel increased dizziness with any kind of head motion until compensation has taken place. The latter is therefore not really a trigger as much as an aggravating circumstance. Vestibular migraine can sometimes be aggravated by fragrances, certain visual stimulation, or excessive head motion, but these do not actually always trigger a spell as much as increase the likelihood of a spell or making ongoing dizziness worse. Dizziness can be triggered by orthostatic postural changes in someone severely prone to orthostatic hypotension or postural tachycardia syndrome (POTS).

**ASSOCIATED FEATURES.** Sometimes the associated features can be characteristic or very helpful. Ménière disease attacks can be preceded by or associated with unilateral ear fullness and muffled hearing and louder low-pitched roaring tinnitus related to one ear. Transient ischemic attacks may be associated with focal hemisensory symptoms, dysarthria, diplopia, or hemiataxia. Superior canal dehiscence syndrome may be accompanied by autophony, a heightened hearing of internal body sounds. Dizziness or postural symptoms from cerebellar ataxia may be accompanied by dysarthria, gait ataxia, and ocular motor abnormalities causing blurry vision during gaze changes. Anxiety is commonly associated with many conditions that lead to dizziness, sometimes as part of the underlying cause but often as a reaction to the symptoms, the loss of control, and feelings of uncertainty about when symptoms will occur or whether they will become severe.

**IMPACT ON QUALITY OF LIFE.** Establishing the impact on the patient's life is important in gauging how aggressive to be in the workup and treatment. Along with this assessment should be a discussion on what the patient expects or would like from the visit. Some patients may have what seems to be fairly minor dizziness, but they are very worried about it due to their own concerns with having something serious. In some of those cases, once they feel it has been adequately established to be benign, they do not want any medication but would consider other approaches. Some patients, particularly those with persistent postural perceptual dizziness (also referred to as PPPD), may have a normal examination but view their lives as severely negatively impacted by the

## KEY POINTS

- History-taking is best started with an open-ended question to allow patients to relay how the symptoms began and what they experience, although patients may need to be redirected in some cases.
- Excessive reliance on the patient's description of dizziness or vertigo leads to mistakes in diagnosis, but some patients accurately describe spinning, whirling, rotational sensations that do indeed imply a higher likelihood of a vestibular process.
- For patients who describe clear vertigo (spinning, whirling, rotation), if spells last less than 1 minute, then benign paroxysmal positional vertigo may be the cause. If the spells last minutes, transient ischemic attack or vestibular migraine should be considered. If the spells last hours, Ménière disease or vestibular migraine may be the cause.
- It is helpful to ask patients about the impact the dizziness or vertigo has on their quality of life and ascertain their goal for the visit and evaluation because some patients just want to be reassured that the cause is benign but can live with the symptom if need be, whereas others are desperate for treatment to relieve the symptoms.

symptoms. Knowing this will help produce a better treatment plan and a more agreeable experience and outcome for the patient (**CASE 1-3**).

**PREVIOUS TREATMENTS TRIED.** In some cases, if no treatment has yet been tried or it is the patient's first medical evaluation, this is a brief conversation. In other cases, many opinions and tests have been offered but no actual treatments have been attempted. If a patient has been treated, the previous treatment can be noted; if it failed in the past, perhaps it should not be repeated. Some patients may have symptoms amenable to physical therapy but say it did not work in the past. It can be helpful to ask the patient to recount what was done by the therapist and how many sessions were attended. Some patients fail to respond because the therapist mistakenly tried to treat for BPPV but the patient actually has vestibular neuritis or another condition. Other patients may not have complied with a reasonable trial because they were not convinced it would help. In other cases, a medication may have been used but at such a low dosage or for such a short therapeutic trial it should be reconsidered.

### CASE 1-3

**A 41-year-old man presented with 3 years of intermittent dizziness and vertigo. He was not really sure when the dizziness began, but the first time he recalled it occurring was when he was exercising vigorously at a gym training for a triathlon and he developed sudden wooziness and a feeling of spatial disorientation that lasted several minutes. He also described an occasional spontaneous sensation of spinning often lasting just seconds, and he had a constant low-grade motion sickness aggravated by repetitive head movements and by seeing pattern and object motion around him. Nausea and a low-grade sense of rocking or swaying dizziness became a daily problem, and he took ondansetron most days to mitigate this. He stopped the training he used to do because of the dizziness and nausea. He had a history of migraine and got about six severe migraine headaches per year and much more frequent lower-level headaches that he worked through.**

**Examination was normal. Brain MRI, a hearing test, and videonystagmography were all unremarkable.**

### COMMENT

This is most likely vestibular migraine, although rocking/swaying and visually induced dizziness as the patient described can also be seen with persistent postural perceptual dizziness. He also had so-called quick spins (that is, fragments of vertigo consisting of a partial turn for a brief moment that then stops without continuing but may recur many times). He also had significant nausea and migraine headaches. This patient's quality of life was diminished by the symptoms, and despite 3 years of seeing physicians, he had not received a diagnosis or been tried on any therapy aside from ondansetron. After discussion with the patient about migraine treatment options, nortriptyline was started and titrated to 75 mg daily with significant improvement so that he was able to return to all previous activities.

## Examination

Note whether the patient came in with a walking assistive device and observe whether the device is worn from use and inquire how long has the patient used it and why. For example, a cane may be used due to knee arthritis or perhaps because of imbalance or recurrent falling. Consider whether the reported severity of vertigo, dizziness, or imbalance is proportional to observed balance and function. If the patient reports constant nausea and inability to walk, does that fit with his or her appearance? Does the patient sit straight and use his or her arms to stand, and is it done with little or significant effort? Take note of the interaction with others in attendance and the patient's affect, train of thought, attention to details, and the logic of chronology of history as relayed by the patient.

The neurologic examination of the patient with dizziness or vertigo should include evaluation for potential related signs such as Horner syndrome, hemisensory deficits, unilateral facial weakness, dysarthria, limb ataxia, dysconjugate gaze, head tilt, spasticity, abnormal reflexes, or distal somatosensory deficits, which may be clues to localizing lesions in the cerebellum, brainstem, spinal cord, or peripheral nerves. Such signs can localize the vertigo or dizziness to a central nervous system (CNS) location or explain unrelated comorbid conditions that contribute to imbalance or tendency to fall.

Otосcopy is not part of the standard examination for many neurologists but can be helpful in excluding obvious ceruminous impaction; a bulla or cyst; or perforation, darkness, or coloration changes of the tympanic membrane.

Orthostatic vital signs are indicated when orthostatic hypotension or intolerance is suspected.

## Areas of Special Focus for Patients With Vertigo and Dizziness

Although much can be garnered from the neurologic examination, several areas of the examination (eye movements, speech articulation, cerebellar functions, vestibular function, Romberg sign, gait, and tandem gait) are particularly important in evaluating patients with dizziness or vertigo. A more detailed coverage of the bedside examination is included in the articles "Vestibular Testing" by Timothy C. Hain, MD, and Marcello Cherchi, MD, PhD, FAAN,<sup>16</sup> and "Episodic Positional Dizziness" by Kevin A. Kerber, MD, MS,<sup>11</sup> in this issue of *Continuum*.

**STANCE, GAIT, AND ROMBERG SIGN.** Imbalance and gait difficulty overlap but are not the same. Balance entails the maintenance of steady weight distribution by adjusting the center of gravity in all positions whether stationary or while in motion. Balance uses CNS integration mostly via the cerebellum to maximize control of weight distribution by using somatosensory, visual, and vestibular inputs. Stability in gait requires the integration of balance and motor control but is also influenced by cognitive judgment and anticipation and orthopedic considerations. For patients with dizziness overall, balance is judged by gait speed, gait base, and the ability to walk in tandem and maintain balance during and after quick turns. Other elements of gait, including cadence (steps per minute), stride length, floor clearance, gait ignition, arm swing, and foot strike location, may provide additional information. Examination of gait should be included whenever possible in patients with dizziness or vertigo.

Some patterns can steer toward a possible diagnosis. Falls during Romberg testing may indicate bilateral vestibulopathy or somatosensory dysfunction. A

wide-based gait may be seen in chronic communicating hydrocephalus, ataxic neuropathy, or hypothyroidism but is most characteristic of cerebellar dysfunction. Patients with acute vertigo (less than 10 days from onset) due to vestibular neuritis commonly have a slight change in gait mechanics, including a longer stride length and longer stance time using the ipsilesional leg and an overall tendency to minimize head movements,<sup>17</sup> although this usually

### CASE 1-4

A 57-year-old man was in good health until about 8 months earlier when he developed a feeling of spinning when getting out of bed. He staggered to the bathroom, the vertigo continued, and, within 20 minutes, he started vomiting. He stayed home from his job as a claims inspector for a commercial trucking company and sat, hoping it would resolve, but, after an hour, he went to a local emergency department. There, a head CT, a head and neck CT angiogram, ECG, and laboratory values were all negative, and he was sent home with instructions to see an otolaryngologist the next day. He saw an otolaryngologist 9 days later, had a normal audiogram, and was told he may have a viral inner ear infection and that it would resolve. In the weeks that followed, he noticed some improvement, but his improvement plateaued and eventually worsened such that he developed continual dizziness. He described no nausea but felt a continuous swaying and floating sensation that seemed to be constant and diminished his ability to focus at work. He was bothered by environments with fluorescent lights whenever in crowds or stores where there seemed to be too much visual stimulation. A trial of 15 sessions of vestibular physical therapy 4 months after the onset of vertigo did not help. He was functioning at work but felt he could not go on living like this and was desperate. Brain MRI was normal. Videonystagmography that had been performed 4 weeks after the onset of vertigo showed 38% right reduced vestibular responses and another videonystagmography 5 months after the onset of vertigo was normal.

Examination was normal, including head impulse testing. When exposed to an optokinetic strip, he appeared to be bothered by it, although his optokinetic responses were normal.

### COMMENT

This patient most likely has had two types of dizziness. He had vestibular neuritis at the outset that accounted for his early symptoms, but, over time, the dizziness changed from vertigo and dizziness with quick movements to a feeling of constant swaying and floating. One could speculate that he did not compensate for the unilateral vestibular loss, but this is less likely because vestibular function by head impulse testing clinically normalized after several months as occurs in nearly half of cases of vestibular neuritis. The dizziness that has bothered him for the past 5 to 6 months is persistent postural perceptual dizziness, a condition that sometimes follows a vestibular event. Despite a normal examination, this patient was greatly troubled by the ongoing dizziness, which is common in persistent postural perceptual dizziness as well.

normalizes in 6 to 9 weeks after onset as the patient achieves compensation.<sup>18</sup> This manifests as a gait that appears cautious, a bit slower, and with some minimization of normal head movements (**CASE 1-4**).

Between bouts of vertigo due to BPPV and Ménière disease and with vestibular migraine and most cases of PPPD, the gait is either normal or mostly normal with some guardedness. Most patients with PPPD have a normal gait or occasionally appear to walk cautiously.

**EYE MOVEMENTS, NYSTAGMUS.** Examination of eye movement abnormalities can be very helpful in localization. For patients with dizziness and vertigo, eye movement findings can help in clarifying the cause of vertigo. For example, lateropulsion (or ipsipulsion) of saccades, in which saccades toward the side of the lesion tend to overshoot the target whereas saccades to the opposite side tend to undershoot the target, is a feature seen in lateral medullary syndrome (Wallenberg syndrome). Saccadic dysmetria, in which bidirectional overshooting of the target and occasional undershooting occurs, localizes to the cerebellar vermis and fastigial nucleus. Skew deviation of the eyes is characterized by acquired misalignment of the eyes due to a disturbance of the otolith ocular pathways that project to the interstitial nucleus of Cajal. Skew deviation is more common in central vestibular lesions but may be seen in peripheral vestibular lesions, as well. Skew deviation from peripheral vestibular lesions has a small amplitude and abates within days as compensation of the acute peripheral vestibular asymmetry occurs.<sup>19</sup> The article “Acute Vestibular Syndrome” by Kristen K. Steenerson, MD,<sup>20</sup> in this issue of *Continuum* discusses skew deviation further in the context of acute continuous vertigo.

Nystagmus is a hallmark sign of vestibular disorders. Nystagmus may be categorized in many ways, but for practical clinical purposes, it may be spontaneous, gaze related, or positional.<sup>21</sup> If nystagmus is present in primary gaze, then it is considered spontaneous nystagmus. If nystagmus is not spontaneous but occurs with a gaze to any direction, then it is gaze related or gaze evoked. If it is not spontaneous or gaze-related nystagmus but position changes induce the nystagmus, it is positional nystagmus.

Examination for nystagmus can provide valuable information in patients with acute, episodic, and chronic vestibular syndromes.

**SPONTANEOUS NYSTAGMUS.** Spontaneous downbeating nystagmus should be considered of central origin and localizes to the cerebellar vermis and cervicomedullary junction. The most common conditions causing it are cerebellar ataxias, less commonly Chiari malformation or multiple sclerosis. Spontaneous upbeating nystagmus is encountered much less frequently but may be seen in cerebellar ataxias, multiple sclerosis, Wernicke syndrome, autoimmune encephalitis, or lesions of the medial dorsal medulla. Spontaneous horizontal nystagmus can be central or, more commonly, a result of peripheral vestibular lesions. In peripheral vestibular horizontal nystagmus, the nystagmus stays in one direction. That is, if it is spontaneous right-beating nystagmus, it intensifies with gaze in the direction of the fast phase (to the right) and diminishes or goes away with gaze to the left. Although clinicians test for this by assessing the effect of gaze direction changes, it is still a form of spontaneous nystagmus. When the nystagmus adheres to this pattern, referred to as the

## KEY POINTS

- Spontaneous downbeat nystagmus should be considered a central finding that localizes to the cerebellar vermis or cervicomedullary junction.
- In peripheral vestibular horizontal nystagmus, the nystagmus stays in one direction, intensifying with gaze in the direction of the fast phase and diminishing or abating with gaze in the direction away from the fast phase of nystagmus.

*Alexander law*, it indicates the nystagmus is of peripheral vestibular origin. When the spontaneous nystagmus is right beating and changes to left beating with gaze to the left, it is very likely central in origin.

**GAZE-EVOKED NYSTAGMUS.** Gaze-evoked nystagmus occurs when no spontaneous nystagmus is present but when the patient gazes to the right, right-beating nystagmus occurs, and when the patient looks to the left, left-beating nystagmus occurs. Gaze-evoked nystagmus occurs when the gaze-holding mechanism is impaired so that the elastic forces of the orbits draw the eyes to drift back toward midline, which is then followed by a corrective saccade. For horizontal gaze holding, the neural integrator is mediated by the nucleus prepositus hypoglossi and medial vestibular nucleus, as well as by inputs from the flocculonodular lobe of the cerebellum. For vertical and torsional gaze holding, the interstitial nucleus of Cajal in the midbrain mediates gaze holding. Gaze-evoked nystagmus is a CNS finding and should be distinguished from so-called *end point nystagmus*. End point nystagmus occurs when the patient attempts to look at the extremes of horizontal gaze, usually approximately 45 degrees from the vertical meridian, and a small degree of nystagmus occurs.<sup>22</sup> Pathologic gaze-evoked nystagmus is usually evident by 30 degrees of eccentric gaze or less.

**POSITIONAL NYSTAGMUS.** Positional nystagmus is key to diagnosing the most common cause of recurrent vertigo, BPPV. In patients with dizziness or vertigo, examination with Dix-Hallpike positioning elicits paroxysmal positional nystagmus that is a combination of upbeating and torsional nystagmus with the top pole of rotation (fast-phase) beating toward the downward ear, indicating BPPV related to the posterior semicircular canal of the ear that is downward. That is, for right posterior canal BPPV, the nystagmus is counterclockwise from the examiner's perspective (top pole nystagmus beats torsionally toward the right ear) and upbeating from the patient's perspective.

Positional testing may also result in horizontal nystagmus, which is most commonly a variant of BPPV related to one of the lateral canals. Positional downbeating nystagmus may be seen with variants of BPPV, but central causes should be considered if it does not resolve with positioning treatments. Static positional nystagmus is common and may be seen with video goggles that allow visual fixation to be removed. Spontaneous static positional upbeating nystagmus less than approximately 6 degrees per second is seen in vestibular migraine but may occasionally occur in other conditions and so should not be considered reliably localizing or indicative of a central versus peripheral etiology.

Examination for vestibular function helps identify the absence or presence of peripheral vestibular function on one or both sides ([CASE 1-5](#)).

### Synthesis of History and Examination

Once relevant information from the history, observations, and examination is compiled, the next steps are integration and discernment of patterns or possible patterns to generate a differential diagnosis ([TABLE 1-5](#)).<sup>23-26</sup> As already mentioned, sometimes symptoms and signs are from two or more conditions that cause dizziness or vertigo with features of both disorders at the same time ([TABLE 1-1](#)).

## Laboratory Testing

Blood tests for vertigo are rarely helpful as a matter of routine but may be indicated in some cases. Patients taking antiepileptic drugs that may account for dizziness or abnormal eye movements may need drug levels assessed. For patients with impaired balance, vitamin B<sub>12</sub>, methylmalonic acid, hemoglobin A<sub>1c</sub>, and thyroid function studies may be warranted. For patients with possible orthostatic dizziness, a complete blood cell count and comprehensive metabolic panel may be ordered. In patients with bilateral fluctuating hearing with or without vertigo and in whom luetic otitis or autoimmune inner ear disease is suspected, an antinuclear antibody screen, erythrocyte sedimentation rate, and

## CASE 1-5

**A 48-year-old man was seen for recurrent falling and constant dizziness. His past medical history was notable for type 2 diabetes mellitus, hypertension, and chronic renal failure due to autosomal dominant polycystic kidney disease. He said the symptoms had been ongoing for the past 4 months since he was hospitalized for peritonitis complicating his peritoneal dialysis. He recalled developing some dizziness in the hospital but was discharged to an extended care facility as antimicrobial therapy was continued. When it came time for discharge, it was apparent he could not walk well even with a cane, whereas he had been walking independently before hospitalization.**

**An otolaryngologist found normal hearing on his audiogram and felt it was not likely an otologic issue. Brain MRI was normal. The patient had 12 sessions of home physical therapy but still felt dizzy and off balance. When standing, he felt severely off balance, saying, "I don't know where I am in space," but this improved if he touched something with his hand. When he walked or rode in a car, his vision jostled and he had trouble focusing until he stopped moving. He had no nausea or spinning sensation, and he was not aware of much change in his hearing.**

**Examination revealed evidence for neuropathy to the midlegs. His head impulse test was abnormal to the right and left. No nystagmus was detected. Dynamic visual acuity was impaired. Romberg sign was consistently positive. Bilateral vestibulopathy was suspected and confirmed by videonystagmography that showed severely reduced caloric vestibular responses (summed caloric responses of 11 degrees per second).**

This is a case in which the history and bedside examination strongly pointed to bilateral vestibulopathy, most likely caused by an aminoglycoside such as gentamicin that was likely given as treatment of the peritonitis. Gentamicin is more selectively vestibulotoxic, so hearing was not significantly affected. This case also points out how having concurrent neuropathy with attendant somatosensory loss severely reduces the ability to walk because vestibular and somatosensory signals are important sensory inputs for balance, so when the patient's vision was removed by closing his eyes, he consistently fell.

## COMMENT



**TABLE 1-5 Clinical Features of Some Disorders Causing Dizziness and Vertigo**

| Common dizziness/vertigo descriptions                       | Onset  | Timing/duration        | Triggers  | Associated features  | International Classification of Vestibular Disorders syndrome <sup>a</sup> | Disorder                             |
|---|--|------------------------|---|--|--|--------------------------------------|
| <b>Vestibular disorders</b>                                 |  |                        |   |  |  |                                      |
| Spinning, rotating, whirling, tilting, floating, or falling | Abrupt   | 5–60 seconds           | Tilting head back, rolling in bed, straightening after bending                | Occasionally nausea, nystagmus with Dix-Hallpike test on affected side   | Episodic vestibular syndrome   | Benign paroxysmal positional vertigo |
| Spinning, whirling, rotating, tilting                       | Abrupt or evolving over 30 minutes with some variability | Days to weeks          | No reliable trigger, 15% with antecedent upper respiratory infection symptoms | Worse with any head motion, nausea, direction-fixed nystagmus (early on), abnormal head impulse test to the side affected                                | Acute vestibular syndrome  | Vestibular neuritis                  |
| Spinning, whirling, rotating, tilting                       | Abrupt or evolving over 30 minutes with some variability | Days to weeks          | No reliable trigger, 15% with antecedent upper respiratory infection symptoms | Worse with any head motion, nausea, acute unilateral hearing loss, direction-fixed nystagmus (early on), abnormal head impulse test to the side affected | Acute vestibular syndrome  | Labyrinthitis                        |
| Severe spinning, whirling, rotating, imbalance              | Abrupt or evolving over 30 minutes with some variability | 30 minutes to 12 hours | No reliable trigger in most cases   | Unilateral tinnitus and hearing loss that may fluctuate on the affected side; worse during head motion; low-frequency hearing loss on the affected side  | Episodic vestibular syndrome   | Ménière disease                      |

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| Common dizziness/<br>vertigo descriptions  | Onset  | Timing/<br>duration  | Triggers                                     | Associated<br>features   | International<br>Classification<br>of Vestibular<br>Disorders<br>syndrome <sup>a</sup> | Disorder   |
|--|--|--|--|--|--|--|
| Reduced equilibrium, unsteadiness worse during movements                           | Usually insidious, occasionally more abrupt depending on mechanism             | Continuous symptoms worse during head movements or in darkness   | No trigger                                   | Bilateral abnormal head impulse test, positive Romberg sign, reduced dynamic visual acuity   | Chronic vestibular syndrome  | Bilateral vestibulopathy   |
| Spinning, tilting, oscillopsia, floating sometimes induced by sounds <sup>23</sup> | Insidious but occasionally patients describe a sensation of "popping" at onset | Spinning, tilting, oscillopsia, floating may last seconds to minutes recurrently; autophony, <sup>b</sup> tinnitus, ear fullness, and hearing may be fairly continuous | Sounds may trigger spells of worse symptoms  | Autophony, unilateral ear pressure or fullness and hearing reduction, tinnitus; occasionally nystagmus can be induced by noise or vibration on examination | Episodic vestibular syndrome   | Superior canal dehiscence  |
| <b>Hemodynamic disorders</b>   |  |  |  |  |  |  |
| Near-faintness, "about to pass out," "lightheadedness" <sup>24</sup>               | Abrupt, usually when standing  | Minutes, may be recurrent on standing; may culminate in syncope or abate in minutes  | Most events occur or are evoked when upright | Pallor, diaphoresis, nausea, may culminate in syncope, symptoms relieved by lying flat; abnormal orthostatic heart rate/ blood pressure                    | Episodic vestibular syndrome   | Orthostatic dizziness (orthostatic intolerance, orthostatic hypotension) |

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| Common dizziness/vertigo descriptions  | Onset  | Timing/duration  | Triggers             | Associated features   | International Classification of Vestibular Disorders syndrome <sup>a</sup> | Disorder                                 |
|--|--|--|----------------------|---|--|--|
| <b>Central nervous system and related disorders</b>  |  |  |                      |   |  |  |
| Spinning, tilting, rocking, floating, visually induced vertigo/dizziness, motion sensitivity | Abrupt or more gradual, sometimes discrete spells, sometimes constant but varying in intensity | Spinning, tilting, rocking, floating may vary from brief quick spins lasting a few seconds recurrently to spells lasting minutes to much of the day; visually induced vertigo/dizziness, motion sensitivity may be nearly constant | No reliable triggers | Migraine headache history, periodic photophobia or phonophobia; examination is usually normal   | Episodic vestibular syndrome   | Vestibular migraine                      |
| Rocking, swaying, floating, no spinning, and minimal or no nausea <sup>25</sup>              | Often insidious, may ensue after a vestibular disorder or emotional event                      | Often constant, varying in intensity; occasionally comes and goes for hours or days at a time  | No trigger           | Sometimes less severe when distracted; minimally affected by head movements; disability and distress seem out of proportion to normal examination | Chronic vestibular syndrome  | Persistent postural perceptual dizziness |
| Vertigo, poor equilibrium or ataxia  | Abrupt   | Usually resolves in 5–20 minutes   | No trigger           | Transient nystagmus and ataxia but resolve after minutes  | Acute vestibular syndrome  | Transient ischemic attack                |

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| Common dizziness/<br>vertigo descriptions  | Onset   | Timing/<br>duration  | Triggers   | Associated<br>features  | International<br>Classification<br>of Vestibular<br>Disorders<br>syndrome <sup>a</sup> | Disorder  |
|--|---|--|------------|---|--|---|
| Vertigo, poor equilibrium or ataxia  | Abrupt  | Continues, may see some gradual improvement in days to months depending on size and location of infarction | No trigger | Nausea, poor gait balance; possibly gaze-evoked nystagmus, diplopia, dysarthria, normal head impulse test, possibly hemiataxia and other central nervous system signs <sup>26,c</sup> | Acute vestibular syndrome  | Stroke  |
| Impaired balance worse with head movement, less commonly with position changes <sup>27</sup> | Usually insidious; abrupt onset may occur with cerebellar stroke or hemorrhage or with episodic ataxias or acute cerebellitis | Continuous for degenerative and lesion-based cerebellar disease  | No trigger | Balance worsens with fatigue, alcohol, sedation; gaze-evoked or vertical nystagmus; limb and truncal ataxia, dysarthria, abnormal pursuit, and saccadic eye movements                 | Chronic vestibular syndrome  | Cerebellar dizziness (from a variety of cerebellar disorders) |

<sup>a</sup> By using the International Classification of Vestibular Disorders construct of syndromes, the term *vestibular* does not necessarily mean the conditions under each rubric have an actual vestibular mechanism of origin.

<sup>b</sup> Autophony is the abnormally increased perception of one's own internal body sounds (one's own voice, blinking, heart sounds, etc).

<sup>c</sup> Cerebellar stroke confined to the cerebellar tonsil, flocculus, nodulus, superior vermis, and cerebellar peduncles may present only with vertigo and imbalance lacking other prominent signs.<sup>26</sup>

tests for syphilis can be obtained. Patients presenting with ataxia should have a test for anti–glutamic acid decarboxylase 65 (GAD65) antibodies and possibly vitamin E level. Although anti-GAD65 antibodies are associated with stiff person syndrome, in some people high levels of anti-GAD65 antibodies may manifest primarily with cerebellar ataxia with imbalance and “dizziness,” oftentimes with downbeating nystagmus that is amenable to treatment with immunotherapy (eg, mycophenolate, cyclosporine, rituximab, cyclophosphamide). For anyone suspected of having dizziness in association with Wernicke syndrome, assessment and possible empiric treatment for this condition should be considered.

### CASE 1-6

A 52-year-old woman was previously seen for benign positional vertigo on and off for the past 4 years. She had recently seen the physical therapist who treated her and said no benign paroxysmal positional vertigo could be found at that time. The patient had recurrent, mostly random spells of dizziness that did not seem clearly triggered by anything, lasted a few minutes, and mild to severe wobbliness, sometimes being so severe that she had to sit down or would fall. The last severe spell occurred while standing in the produce section of a grocery store, and she had to sit on the floor. It lasted 3 to 4 minutes, and she became sweaty and felt a little queasy but denied a feeling of spinning. She had been in good health and took only a thyroid supplement, estrogen, and rosuvastatin. Recent head CT was normal, and ECG, complete blood cell count, and comprehensive metabolic panel ordered by her primary physician were all normal.

On examination, her sitting blood pressure was 96/62 mm Hg with a heart rate of 93 beats/min; on standing, it was 94/63 mm Hg with a heart rate of 96 beats/min, and she had no symptoms. The remainder of the neurologic examination was normal, and cardiac auscultation was also normal. Postural hypotension was suspected, and a tilt-table study was performed. On the test, she had no change in heart rate or blood pressure until 19 minutes of head-up tilt at which time she exhibited a systolic blood pressure drop to 61 mm Hg, and she passed out while strapped to the tilt table. Her blood pressure normalized soon after resumption to a recumbent position.

She responded to fludrocortisone, liberalized dietary sodium intake, and pyridostigmine. The pyridostigmine was successfully discontinued after 6 weeks, and the episodes did not return. Over the subsequent year, she was able to discontinue the fludrocortisone as well.

### COMMENT

This case illustrates delayed orthostatic hypotension, a nonvestibular cause of recurrent dizziness. In this case, routine orthostatic vital signs did not reveal immediate changes in blood pressure. Rather, the decline in blood pressure occurred after a time in the upright position. The delay in onset made the episodes seem entirely random. However, as she described the circumstances of the episodes, they were all while she was upright.

## Cardiac and Hemodynamic Testing

If cardiogenic near-syncope is suspected, an ECG, echocardiogram, Holter monitoring, and cardiology referral can be obtained. Tilt-table testing can be helpful in patients with unexplained syncope but also in those with suspected recurrent, unexplained near-syncope dizziness. Patients in whom recurrent spells of dizziness occur mostly or exclusively when upright may have dizziness caused by orthostatic hypotension. Delayed orthostatic hypotension (defined as having onset beyond 3 minutes of standing or head-up tilt) may be missed by routine orthostatic vital signs but can be detected by a tilt-table test. In one study, only 46% of patients demonstrated orthostatic hypotension within the first 3 minutes, and 39% did not exhibit orthostatic hypotension until more than 10 minutes of head-up tilt,<sup>28</sup> so a tilt-table study for this indication should assess vital signs and symptoms for 30 minutes. Nearly one-third of patients who have delayed orthostatic hypotension have a 10-year mortality of approximately 29%, and nearly one-third eventually develop an  $\alpha$ -synucleinopathy such as Parkinson disease, dementia with Lewy bodies, or multiple system atrophy (CASE 1-6).<sup>29</sup>

## Vestibular Testing

Vestibular testing is performed to determine the functionality and integrity of the peripheral vestibular apparatus and pathways, so it should generally be done to confirm or refute a hypothesis. Of course, like many other tests in medicine, it can be sometimes helpful in situations in which the cause is very uncertain to at least ascertain that the vestibular structures and reflexes remain intact. The role of diagnostic vestibular testing is discussed in detail in the article “Vestibular Testing” by Timothy C. Hain, MD, and Marcello Cherchi, MD, PhD, FAAN,<sup>16</sup> in this issue of *Continuum*.

## Imaging Studies

Imaging may be indicated when the cause of dizziness is uncertain or the examination reveals findings of CNS dysfunction. For patients with dizziness and vertigo, a noncontrast head CT has a very low yield of identifying a cause when patients with headache, trauma to the head and neck, altered mental status, focal neurologic deficits, or recent head or neck surgery are excluded. In one prospective analysis of patients presenting to an emergency department with dizziness, none of the 200 studies found a causative lesion, and head CT in this setting was deemed not cost effective.<sup>30</sup> Temporal bone CT is indicated to identify lesions such as cholesteatoma or lesions within the labyrinth, including canal dehiscence. Brain MRI without contrast is a reasonable first step, and MRI with and without contrast is warranted if a vestibular schwannoma or other structural lesion of the cerebellopontine angle is a consideration.<sup>31</sup> Head and neck CT angiography (CTA) or head and neck magnetic resonance angiography (MRA) may be appropriate when dizziness or vertigo may have a vascular cause.

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## CONCLUSION

Dizziness and vertigo by newer definitions are distinct and separate descriptors (TABLE 1-2). Vertigo is not considered a subset of dizziness but a separate symptom descriptor that indicates illusory motion. Dizziness describes a feeling

## KEY POINT

- Delayed orthostatic hypotension (having onset beyond 3 minutes after standing or head-up tilt) may be missed by routine orthostatic vital signs but can be detected by a tilt-table test.

of impaired spatial orientation without the illusion of motion. The causes of dizziness and vertigo are many, but peripheral vestibular disorders are common. A thorough history is paramount in making a diagnosis, and, in most cases, the examination and laboratory testing are confirmatory rather than primary diagnostic tools. All aspects of the patient's history have importance, and no reliably relayed part of the history should be ignored. Key examination focus points include eye movements and especially observations of nystagmus and bedside tests of vestibular function and symmetry. Derivation of a logical differential diagnosis requires recognizing patterns or near patterns in the clinical history, which is something neurologists do routinely. The following articles in this issue guide the evaluation of this group of patients, many of whom can enjoy substantial improvement in their quality of life by proper evaluation and treatment.

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