

# Managing Patients With Acute Episodic Dizziness

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## INTRODUCTION

Three percent of emergency department (ED) patients present with dizziness, vertigo, lightheadedness, or imbalance.<sup>1</sup> The descriptive word that the patient uses is not diagnostically meaningful. Contrary to conventional wisdom, the answer to “What do you mean by ‘dizzy?’” should not be used to drive the evaluation. History-taking of a patient with dizziness should be no different from that of any other patient.

The differential diagnosis of a patient with chest pain is not exclusively based on the descriptor that a patient uses (eg, evaluate for only aortic dissection if the patient endorses “tearing” pain or only for pneumothorax or pulmonary embolism if the pain is “sharp”). A patient with tearing chest pain that occurs intermittently with moderate exertion and resolves promptly with rest probably has angina, not aortic dissection. Rather, it is the timing, triggers (for intermittent symptoms), context, and associated symptoms that best inform the differential diagnosis of the dizzy patient.<sup>2-4</sup> One evaluates a dizzy patient with fever, dyspnea, and purulent sputum differently from a dizzy patient with acute neck pain after chiropractic treatment.

Two critical problems with the traditional symptom quality approach (“What do you mean by ‘dizzy?’”) completely undercut its logic. First, patients change their response to that question 50% of the time even when reasked the question minutes later.<sup>5</sup> Second, the words are not tightly linked with a specific differential diagnosis. Patients with stroke often endorse “unsteadiness”<sup>6</sup>; those with cardiovascular causes describe “vertigo” nearly 40% of the time,<sup>7</sup> and others with benign paroxysmal positional vertigo, the prototypical vestibular disease, often describe “lightheadedness,” especially older patients.<sup>8</sup> Thus, the history-taking of a patient with dizziness should be the same as that of any other patient and based on timing, triggers, and context (see diagnostic algorithm, [Figure 1](#)).

Approximately 50% of dizzy patients presenting to an ED have various medical (toxic, metabolic, infectious, or

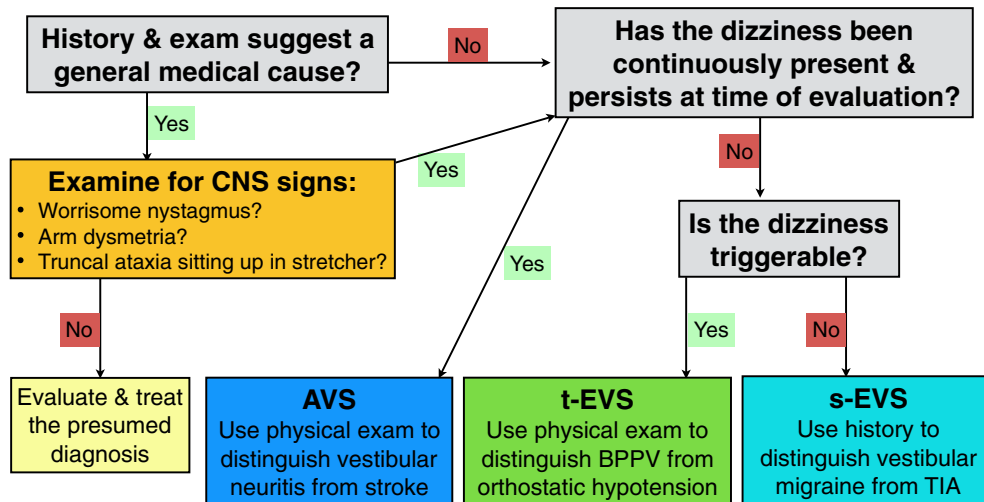
cardiovascular) conditions.<sup>1</sup> Associated symptoms (eg, gastrointestinal bleeding, fever) or context (eg, new antihypertensive medications) often suggest these diagnoses. In the absence of general medical causes for the dizziness, use history to actively categorize the patients into 1 of 3 vestibular syndromes, each of which has a unique differential diagnosis. The 50% of acutely dizzy patients without apparent general medical causes usually fall into 1 of 3 groups based on the onset, episodic or persistent nature of the dizziness, and, if episodic, the presence or absence of triggers.<sup>2,3,9-11</sup>

Acute-onset persistent dizziness, the acute vestibular syndrome, was recently reviewed in *Annals of Emergency Medicine*.<sup>12</sup> Physical examination helps to distinguish vestibular neuritis from posterior circulation stroke, the usual differential diagnosis in this group. Patients with intermittent dizziness whose symptoms have resolved at presentation have 1 of 2 episodic vestibular syndromes. In the triggered episodic vestibular syndrome, episodes of dizziness occur only after some obligate trigger, usually head or body movement, and in the spontaneous episodic vestibular syndrome, dizziness episodes are not triggered by anything. The term “vestibular” denotes the kind of symptom and not its anatomic localization. This article focuses on the management of patients with episodic dizziness, emphasizing the most common (benign paroxysmal positional vertigo) and the most serious (posterior circulation transient ischemic attack) conditions.

## DIAGNOSIS, DIFFERENTIAL DIAGNOSIS, AND MISDIAGNOSIS

A key concept is the distinction between dizziness that is triggered versus exacerbated. Dizziness that is absent at rest and occurs only after a specific movement (triggered) is different from dizziness that is present at rest but increases in intensity with movement (exacerbated). In the first case (eg, benign paroxysmal positional vertigo), asymptomatic patients only experience dizziness with movement (triggered). In the second case (eg, cerebellar stroke) minimally symptomatic patients’ dizziness intensifies with movement (exacerbated).

## Diagnostic Approach to the Acutely Dizzy Patient



The diagnostic approach to acute dizziness should be the same as for any other symptom – based on timing, evolution, triggers of intermittent dizziness, context and associated symptoms, which often suggests a toxic, metabolic, infectious or cardiovascular diagnosis. Prior to treating for these causes, briefly check for CNS findings. Patients without an obvious general medical cause usually fall into one of three categories: the AVS (acute onset persistent, continuously present dizziness), the t-EVS (brief episodes of dizziness caused by some obligate trigger) and the s-EVS (spontaneous episodes of variable duration dizziness not triggered by anything). For each syndrome, only the most common benign and dangerous differential diagnosis is listed

AVS acute vestibular syndrome, s-EVS spontaneous episodic vestibular syndrome, t-EVS triggered episodic vestibular syndrome, CNS central nervous system

**Figure 1.** Diagnostic approach to the acutely dizzy patient.

Many physicians harbor the false notion that dizziness that worsens with movement has a peripheral cause.<sup>13</sup> Patients with persistent dizziness at rest, even if it worsens with movement, have an acute vestibular syndrome, usually caused by either stroke (central) or vestibular neuritis (peripheral).<sup>12</sup> Both types of episodic vestibular syndrome have a specific differential diagnosis (Table 1). The major differential diagnosis of the triggered episodic vestibular

syndrome is benign paroxysmal positional vertigo versus dangerous causes of orthostatic hypotension (serious). The important differential diagnosis of the spontaneous episodic vestibular syndrome is vestibular migraine (benign) versus posterior circulation transient ischemic attack (serious).

Diagnostic challenges for benign paroxysmal positional vertigo (lack of familiarity)<sup>14-17</sup> and posterior circulation transient ischemic attack (difficulty diagnosing transient

**Table 1.** Differential diagnosis of the episodic vestibular\* syndrome.<sup>†</sup>

Syndrome	Description	Common Benign Causes	Important Dangerous Causes	Uncommon Causes
t-EVS <sup>‡</sup>	Episodic dizziness brought on by a specific, obligate trigger (typically a change in head position or standing up), and usually lasting less than 60 s	BPPV Orthostatic hypotension caused by benign conditions	Orthostatic hypotension caused by serious conditions	CPPV Superior canal dehiscence syndrome
s-EVS	Episodic dizziness that occurs spontaneously, is not triggered <sup>‡</sup> Duration is variable, but usually lasts minutes to hours (depending on cause)	Vestibular migraine Meniere's disease	Posterior circulation TIA	Panic attack Vasovagal near syncope Dysrhythmia, PE, ACS, CO toxicity Hypoglycemia

t-EVS, Triggered episodic vestibular syndrome; BPPV, benign paroxysmal positional vertigo; CPPV, central paroxysmal positional vertigo; s-EVS, spontaneous episodic vestibular syndrome; TIA, transient ischemic attack; PE, pulmonary embolism; ACS, acute coronary syndrome; CO, carbon monoxide.

\*“Vestibular” connotes vestibular symptoms (eg, dizziness, vertigo, imbalance, lightheadedness) rather than anatomic vestibular processes (eg, benign paroxysmal positional vertigo, vestibular neuritis).

<sup>†</sup>This table is not intended to be encyclopedic; numerous conditions can cause dizziness.

<sup>‡</sup>By definition, the patient is asymptomatic at rest and the physician is able to clearly trigger the dizziness at the bedside, generally by having the patient move from lying to standing (orthostatic hypotension) or performing various semicircular canal provocative maneuvers such as the Dix-Hallpike's maneuver (BPPV). This must be distinguished from dizziness that is “exacerbated” in a patient who is somewhat dizzy at baseline and worse with movement. Exacerbation of dizziness at baseline commonly occurs with the acute vestibular syndrome, whether peripheral (vestibular neuritis) or central (stroke).

symptoms)<sup>18-20</sup> are well documented. Reasons include absence of guidelines, use of the symptom quality approach (“What do you mean by ‘dizzy?’”) as opposed to one that exploits timing and triggers,<sup>2,3,21,22</sup> and the prevailing paradigm that transient ischemic attack does not cause isolated dizziness.

### TRIGGERED EPISODIC VESTIBULAR SYNDROME

The 2 most common causes of the triggered episodic vestibular syndrome are benign paroxysmal positional vertigo and orthostatic hypotension. Patients with benign paroxysmal positional vertigo (especially older ones) frequently endorse dizziness or lightheadedness (not vertigo),<sup>8</sup> and those with orthostatic hypotension often complain of vertigo (not lightheadedness).<sup>23,24</sup> Although both conditions can be precipitated by standing up, benign paroxysmal positional vertigo frequently occurs at night while one is turning over in bed, whereas orthostatic hypotension should never occur while one is recumbent. In fact, transient episodes of dizziness that waken a patient from sleep are almost always due to benign paroxysmal positional vertigo. Beyond these common causes, another rare cause of triggered episodic vestibular syndrome is the superior canal dehiscence syndrome, in which thinning of the bone of the superior semicircular canal leads to sound- or pressure-triggered (in the middle ear) dizziness, hyperacusis, and pulsatile tinnitus,<sup>25</sup> and odd symptoms such as hearing sounds of one’s chewing, footfalls, or even eyes moving in the orbits may falsely suggest a psychiatric issue. These patients should be referred to an otorhinolaryngologist.

### BENIGN PAROXYSMAL POSITIONAL VERTIGO

With a lifetime prevalence of 2.4%, benign paroxysmal positional vertigo is common.<sup>26,27</sup> Because their symptoms are significant and frightening, patients with benign paroxysmal positional vertigo frequently come to an ED. Besides turning over in bed, other common triggers of benign paroxysmal positional vertigo include head movements associated with getting in and out of bed and bending the head forward or backward. Brief episodes of severe dizziness last less than 1 minute. However, some patients describe ongoing symptoms, likely caused by anxiety about movement, anticipation of dizziness, or persistent nausea that outlasts the dizzy spells. Although these patients can superficially mimic an acute vestibular syndrome (“ongoing dizziness”), careful history-taking usually uncovers the true episodic nature of the symptoms. In ambiguous cases, positional testing for benign paroxysmal positional vertigo (usually not recommended for patients with the acute vestibular syndrome) will establish the correct diagnosis and avoid an unnecessary stroke evaluation.

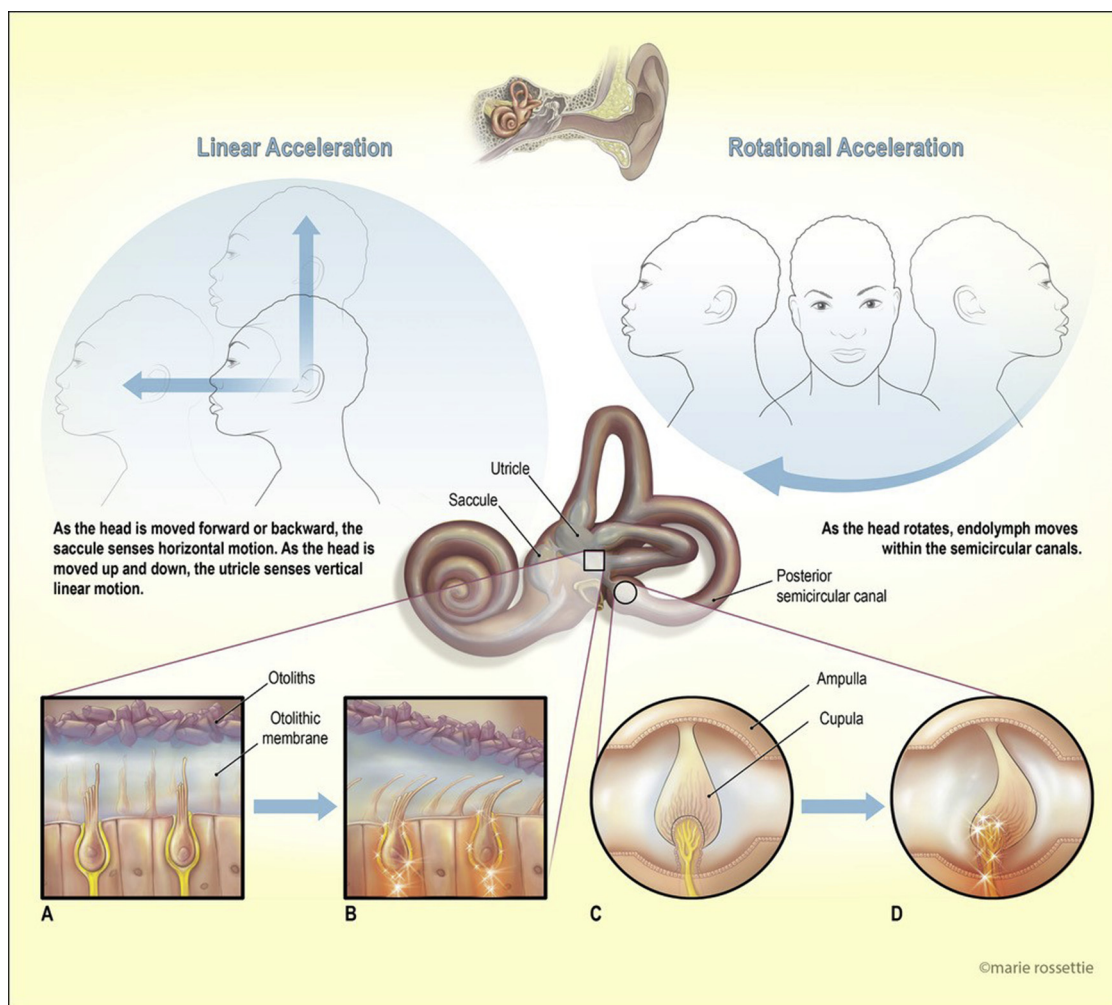
Knowing normal vestibular anatomy and physiology helps in understanding benign paroxysmal positional vertigo (Figure 2). The utricle and saccule sense linear acceleration, and 3 paired semicircular canals sense angular rotation. All are filled with endolymph. Hair cells in the utricle and saccule are covered by a gelatinous otolithic membrane in which calcium carbonate particles (otoliths) are embedded. Linear acceleration is sensed when these otoliths shift position with motion. At the medial end of each semicircular canal, a dilated ampulla contains a membrane called the cupula. Angular rotation of the head causes movement of endolymph within the semicircular canals, the flow of which deflects the cupula. Mechanical energy (of the otoliths and cupula) is translated into electrical energy (in the hair cells) that travels to the brainstem through the vestibular nerve.

Otoliths sometimes become dislodged in the utricle because of head trauma or for unclear reasons. In benign paroxysmal positional vertigo, these loose otoliths can fall into one of the semicircular canals.<sup>28</sup> As the head moves with respect to gravity, the displaced otoliths move freely within the canal (canalolithiasis) or (much less commonly) become “stuck” to the cupula, deflecting it (cupulolithiasis). Both situations falsely simulate rotational movement (Figure 3).

Because of its anatomically dependent position, posterior canal benign paroxysmal positional vertigo accounts for 80% to 85% of cases (Table 2). Free-floating otoliths in the horizontal canal (sometimes called the lateral canal), account for most other cases. Superior canal (sometimes called the anterior canal) benign paroxysmal positional vertigo is rare. This article focuses on posterior canal and horizontal canal benign paroxysmal positional vertigo canalolithiasis, the 2 most common forms.

When benign paroxysmal positional vertigo is suspected, first test for posterior canal benign paroxysmal positional vertigo on both sides by performing the Dix-Hallpike’s test, which will have a positive result on one side and negative on the other (except in the rare instance of bilateral posterior canal benign paroxysmal positional vertigo). Patients with a positive Dix-Hallpike’s result will have dizziness and usually nystagmus visible with the naked eye.<sup>29-32</sup> Patients with a history suspicious for benign paroxysmal positional vertigo but negative Dix-Hallpike’s maneuvers can be tested for horizontal canal benign paroxysmal positional vertigo with the supine head roll test (Table 2).<sup>4</sup> Other than nystagmus, patients with benign paroxysmal positional vertigo do not have headache, decreased hearing, diplopia, or any other neurologic signs or symptoms.

If either diagnostic maneuver has a positive result, treat the specific canal with a canalith repositioning maneuver



**Figure 2.** Vestibular anatomy and physiology.

(eg, Epley's maneuver) or refer the patient to a physician or physical therapist who can.<sup>27</sup> Personal experience<sup>4</sup> and published data show that emergency physicians can successfully treat benign paroxysmal positional vertigo in the ED.<sup>33</sup> Video clips of these diagnostic and therapeutic maneuvers are readily found on the Internet.

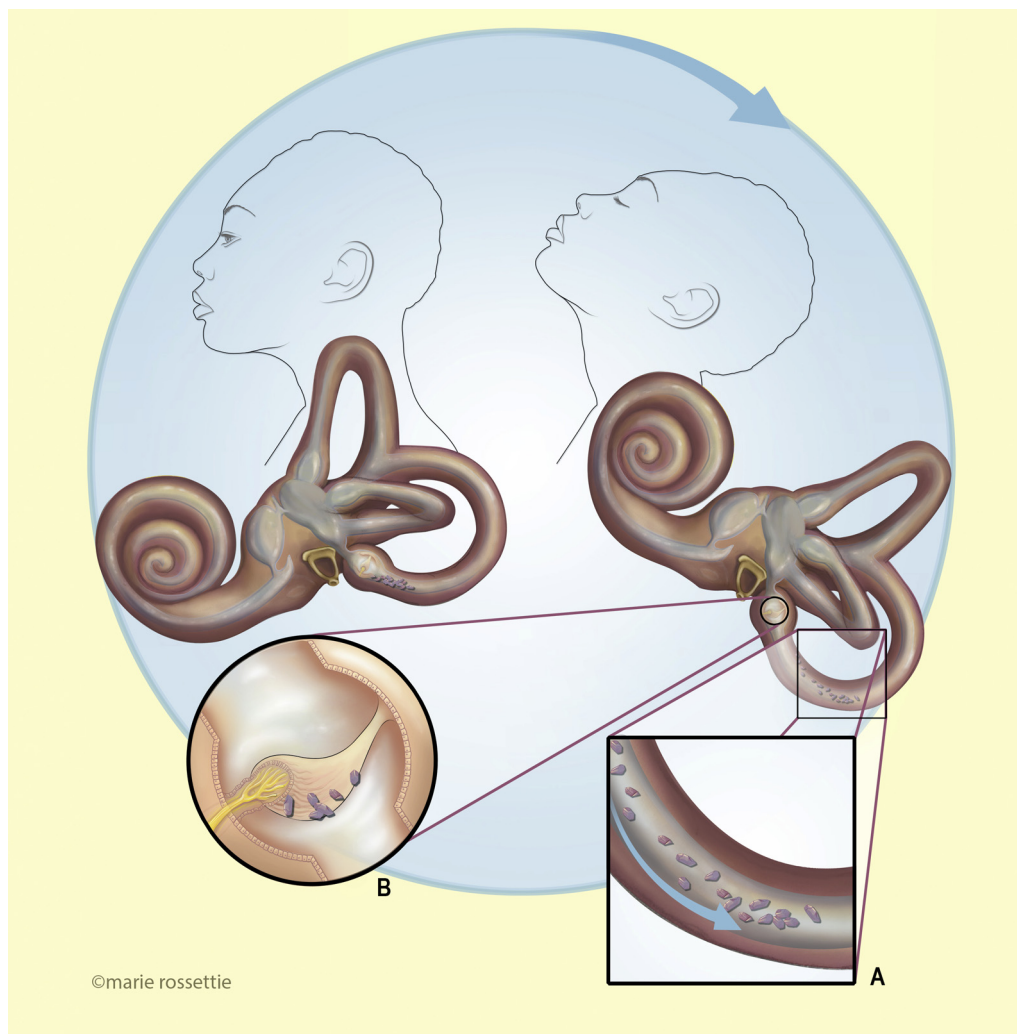
Consultation and testing are rarely necessary, adding time and cost without benefit. In one retrospective study of 193 consecutive adults referred for benign paroxysmal positional vertigo, 136 (71%) had at least one test conducted (none of which were diagnostic).<sup>17</sup> Brain imaging was conducted for 106 patients (76 magnetic resonance imaging [MRI] and 32 computed tomography). Like nursemaid's elbow, benign paroxysmal positional vertigo is a condition suggested by history that is treated with a physical maneuver (without diagnostic testing).

However, if the history suggests benign paroxysmal positional vertigo but the maneuvers do not work, several possible explanations exist (Table 3). One rare cause is central

paroxysmal positional vertigo, in which structural lesions mimic benign paroxysmal positional vertigo; various clinical clues suggest central paroxysmal positional vertigo (Figure 4).<sup>34,35</sup> If the provocative maneuvers do not show typical posterior or horizontal canal benign paroxysmal positional vertigo, consider obtaining neurologic consultation or MRI, the timing of which (during the ED visit versus later on an outpatient status) will depend on the circumstances.

### ORTHOSTATIC HYPOTENSION

Because orthostatic hypotension is core emergency medicine, only a few aspects will be mentioned. First, although it is defined as a decrease in systolic blood pressure by more than 20 mm Hg or in diastolic pressure of more than 10 mm Hg on standing for 2 to 3 minutes, a recent large study (N=11,429) reported that measurements at 1 minute (as opposed to measurements taken after 1 minute) better correlated with both dizziness and adverse outcomes (falls, fractures, motor vehicle crashes, and mortality).<sup>36</sup>



**Figure 3.** Benign paroxysmal positional vertigo mechanisms. A, typical mechanism; canalolithiasis. B, uncommon mechanism; cupulolithiasis (see text).

Second, patients with orthostatic hypotension often describe vertigo, not lightheadedness.<sup>23,24</sup> Third, in one small study (N=30) of orthostatic hypotension, 10 patients actually had transient nystagmus (by video-oculography), potentially caused by global cerebral ischemia.<sup>23</sup>

Obviously dangerous causes of orthostatic hypotension (eg, ectopic pregnancy) would be serious causes of the triggered episodic vestibular syndrome. The diagnostic evaluation is dictated by the individual context. Volume restoration and definitive treatment of the underlying cause will ameliorate the symptoms.

## SPONTANEOUS EPISODIC VESTIBULAR SYNDROME

### Vestibular Migraine

Vestibular migraine is the most common cause of spontaneous episodic vestibular syndrome (Figure 5).<sup>37,38</sup>

Although by definition all patients with vestibular migraine have present or past migraines, the headache and the dizziness do not necessarily co-occur and some patients never have both symptoms simultaneously.<sup>38-40</sup> Photophobia, phonophobia, motion intolerance, and decreased hearing can occur.<sup>38,39</sup> This latter symptom suggests overlap with Meniere's disease. Because migraine is a central process, the nystagmus that is frequently present can be of a central type (direction changing, vertical, or torsional).<sup>41,42</sup> Occasionally, patients with vestibular migraine (10 of 362 patients in a retrospective cohort from a "positional vertigo" clinic) have positional nystagmus that can mimic benign paroxysmal positional vertigo.<sup>43</sup> Because by definition 5 or more episodes are required for definite diagnosis of vestibular migraine, one cannot definitively make the diagnosis after a single episode.

Meniere's disease (recurrent episodes of dizziness, tinnitus, postaural fullness, and hearing loss) also presents as a spontaneous episodic vestibular syndrome.<sup>44</sup> Although

**Table 2.** Physical examination maneuvers and findings that are used to diagnose and treat the typical forms of benign paroxysmal positional vertigo.

Canal Involved, Mechanism (Proportion of BPPV cases)	Provocative Diagnostic Maneuver/Test	Expected Type of Nystagmus*	Therapeutic Maneuver
<b>pc-BPPV (80%–85%)</b>	Dix-Hallpike	Upward beating (from patient's perspective) and torsional <sup>†</sup>	Epley's maneuver Alternative: Semont's maneuver
<b>hc-BPPV (15%–20%) (sometimes called lateral canal)</b>			
Canalolithiasis (majority of horizontal canal cases)	Supine head roll	Geotropic (beats toward the floor) horizontal that is transient <sup>‡</sup> Occurs on both sides, but is more intense on the affected side	Lempert's log roll maneuver Alternative: Gufoni's maneuver
Cupulolithiasis (minority of horizontal canal cases)	Supine head roll	Apogeotropic (beats toward the ceiling) horizontal that is persistent Occurs on both sides, but is more intense on the healthy unaffected side	Gufoni's maneuver
<b>sc-BPPV (≈1%–2%) (sometimes called anterior canal)</b>	Dix-Hallpike	Downward-beating vertical nystagmus <sup>§</sup>	Can use Epley's maneuver, but this form of BPPV usually resolves spontaneously

pc, Posterior canal; hc, horizontal canal; sc, superior canal.

\*Although the Dix-Hallpike's test is fairly specific to pc-BPPV and the supine roll test is fairly specific to hc-BPPV, the maneuvers may sometimes stimulate the other canal. If so, the nystagmus direction will depend on the affected canal, not on the type of maneuver eliciting the nystagmus (eg, if a Dix-Hallpike's test is conducted on a patient with hc-BPPV, the nystagmus will be horizontal, not upward-beating torsional). Also, the nystagmus may be considerably weaker and less obvious than if one were using the "correct" canal-specific maneuver.

<sup>†</sup>On Dix-Hallpike testing, the nystagmus of pc-BPPV will have a prominent torsional component. The 12 o'clock pole of the eye will beat toward the down-facing (tested) ear. On the patient's arising from the down position, the nystagmus will reverse direction because the otoliths are now moving in the opposite direction.

<sup>‡</sup>On supine head roll testing, the nystagmus of hc-BPPV may beat toward the floor (geotropic, usually caused by canalolithiasis) or toward the ceiling (apogeotropic, usually caused by cupulolithiasis). When the other side is tested, the nystagmus will usually beat toward the opposite direction (eg, if right-beating initially with right ear down, then it will usually be left-beating initially with left ear down) because the otoliths are now reversing their direction within the horizontal canal.

<sup>§</sup>Downward-beating nystagmus can be observed with sc-BPPV. However, because sc-BPPV is uncommon and because downward-beating nystagmus is often the result of central structural lesions, it is safer for emergency physicians to consider this a worrisome finding prompting imaging or specialty consultation or referral.

common in specialty clinics, Meniere's disease is an unusual cause of spontaneous episodic vestibular syndrome in the ED. Furthermore, if misdiagnosed, neither vestibular migraine nor Meniere's disease will cause serious short-term consequences.

### Posterior Circulation Transient Ischemic Attack

Posterior circulation transient ischemic attack is an important serious cause of the spontaneous episodic vestibular syndrome. Historically, isolated transient dizziness was not considered to be cerebrovascular.<sup>45</sup>

Mounting evidence shows that this is not true.<sup>18,46-50</sup> In one study of 1,141 prospectively identified stroke patients (759 anterior and 275 posterior circulation), transient episodes of brainstem symptoms in the preceding 2 days were far more likely in patients having posterior circulation strokes (odds ratio 35.8; 95% confidence interval 8.4 to 153.5) compared with those with anterior circulation events.<sup>18</sup> Half of the transient episodes were isolated dizziness. Of the 275 patients with posterior circulation strokes, 23 (8.4%) had preceding brief episodes of isolated dizziness.<sup>18</sup>

**Table 3.** Differential diagnosis of patients whose history suggests benign paroxysmal positional vertigo but for whom Epley's maneuver does not lead to improvement.

Wrong technique	Is the maneuver being performed properly? Review technique to ensure that Epley's maneuver is being properly performed.
Wrong canal	Is the correct canal being treated? Epley's maneuver treats pc-BPPV (85% of cases) but 10%–15% of patients have hc-BPPV, for which a different therapeutic maneuver (Lempert's barbecue roll) is used.
Wrong diagnosis	Does the patient have BPPV? It is key to distinguish patients whose dizziness is triggered (absent at rest and develops with motion, BPPV) vs exacerbated (present at rest, worsens with motion; any cause of acute vestibular syndrome such as neuritis, stroke, or tumor).
Wrong PPV	Could this be a central mimic? Rarely, patients will have CNS structural disease (eg, tumor, stroke) whose presentations may resemble BPPV. These patients will often have suggestive characteristics (see <a href="#">Figure 4</a> ).

PPV, Paroxysmal positional vertigo; CNS, central nervous system.

- 1) Presence of symptoms or signs that are *not* observed in BPPV
  - a) Headache
  - b) Diplopia
  - c) Abnormal cranial nerve or cerebellar function
- 2) Atypical nystagmus characteristics or symptoms during positional tests
  - a) Downward-beating nystagmus\*
  - b) Nystagmus that starts instantaneously, persists for longer than 90 s, or lacks a crescendo-decrescendo pattern of intensity
  - c) Prominent nystagmus with mild or absent associated dizziness or vertigo
- 3) Poor response to therapeutic maneuvers
  - a) Repetitive vomiting during positional maneuvers
  - b) Unable to cure patient with canal-specific canalith repositioning maneuver<sup>†</sup>
  - c) Frequent recurrent symptoms

\*Downward-beating nystagmus can be observed with superior canal BPPV. However, because superior canal BPPV is uncommon and because downward-beating nystagmus is more often the result of central structural lesions, it is safer for emergency physicians to consider this a worrisome finding, prompting imaging or specialty consultation or referral.

<sup>†</sup>Modified Epley's maneuver or equivalent for posterior canal BPPV. Lempert's barbecue maneuver or equivalent for horizontal canal BPPV.

**Figure 4.** Characteristics of patients with triggered episodic vestibular syndrome that suggest a central mimic (central paroxysmal positional vertigo) rather than typical benign paroxysmal positional vertigo.

Newer evidence also challenges the traditional notion that stroke risk after posterior circulation transient ischemic attack is lower than the risk after anterior events. In fact, stroke risk after posterior circulation transient ischemic attack is likely higher than after anterior circulation transient ischemic attack.<sup>46,48,50,51</sup> Vertebral artery stenosis in patients with posterior circulation transient ischemic attack confers an especially high stroke risk.<sup>47,52</sup> Uncommon causes of the spontaneous episodic vestibular syndrome include panic attacks and dysrhythmia (Table 1).

Because by definition patients with the spontaneous episodic vestibular syndrome are asymptomatic at evaluation and the symptom cannot be triggered, physicians must try to distinguish vestibular migraine from

- At least 5 episodes with vestibular symptoms\* of moderate<sup>†</sup> or severe intensity lasting between 5 min and 72 h
- Present migraine or history of migraine with or without aura (according to the *International Classification of Headache Disorders*)
- One or more migraine features with at least 50% of the vestibular episodes
  - Headache with at least 2 of the following characteristics: unilateral location, pulsatile quality, moderate or severe pain, or aggravation by routine physical activity
  - Photophobia or phonophobia
  - Visual aura
- No other vestibular explanation

\*Spontaneous, positional, or visually induced vertigo, head-motion-induced dizziness with nausea

<sup>†</sup>Vertigo is moderate if interferes with but does not preclude daily activities and severe if it prohibits daily activity

Patients with s-EVS caused by vestibular migraine (compared with s-EVS patients with posterior circulation TIA) are more likely to be younger, have a history of migraine, have more vascular risk factors, have longer-duration spells (>1 h), and have more episodes during a longer period. Unfortunately, no one criterion is diagnostic.

**Figure 5.** Diagnostic criteria for vestibular migraine<sup>34</sup> and differences between it and posterior circulation transient ischemic attack.

posterior circulation transient ischemic attack according to history and epidemiologic context (eg, age, vascular risk factors, presence of headache, history of migraine). Physical examination is unhelpful. The treatment of migraine is the same as that for any other migraine patient, as is the treatment of transient ischemic attack.<sup>53</sup>

## CAUTIONS AND PEARLS

The specific word a patient uses to describe his or her dizziness is not diagnostically meaningful. Patients with ongoing dizziness at presentation should be managed as having an acute vestibular syndrome. Dizziness that worsens with movement is not necessarily peripheral and commonly occurs with central causes. The triggered episodic vestibular syndrome caused by benign paroxysmal positional vertigo often occurs at night while the patient is lying in bed, and most patients' disease can be diagnosed

and treated in the ED by using evidence-based diagnostic and therapeutic maneuvers. Testing beyond history and physical examination is rarely indicated. Early diagnosis and treatment of posterior circulation transient ischemic attacks, half of which manifest as brief spontaneous episodes of isolated dizziness, can prevent subsequent strokes.

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