INTRODUCTION

Dizziness accounts for 3.3% to 4.4% of ED visits.¹⁻³ This translates into more than 4.3 million ED patients with dizziness or vertigo annually in the United States⁴ and probably 50 to 100 million worldwide.

Dizziness means different things to different people. Patients may describe feeling dizzy, lightheaded, faint, giddy, spacey, off-balance, rocking, swaying, or spinning. Expert international consensus definitions for vestibular⁵ and related symptoms⁶ are shown in Box 1. Although historically much has been made of the distinction between the terms dizziness and vertigo, current evidence (described by Kerber and Newman-Toker (Pitfalls article) elsewhere in this issue) suggests the distinction is of limited clinical usefulness. This article does not make a distinction between these terms unless specifically noted.

KEY POINTS

- The prevailing diagnostic paradigm for diagnosing emergency department (ED) patients with dizziness is based on dizziness symptom quality or type.
- Recent research suggests that the logic underlying this traditional approach is flawed.
- A newer approach based on timing and triggers of the dizziness likely offers a better diagnostic approach, especially in an unselected ED dizziness population.
- This new approach uses timing-trigger categories to define targeted bedside history and physical exam techniques to differentiate benign from dangerous causes.
- Evidence-based eye movement exams accurately discriminate BPPV (Dix-Hallpike test) and vestibular neuritis (HINTS test) from dangerous central mimics such as stroke.
- Future research should seek to prospectively study the new approach to dizziness for its overall diagnostic accuracy, resource efficiency, and impact on health outcomes.

KEYWORDS

- Dizziness
- Vertigo
- Stroke
- Vestibular diseases
- Diagnosis
- Medical history taking
- Physical examination
- Emergency departments
The differential diagnosis of dizziness is broad, with no single cause accounting for more than 5% to 10% of cases. This article focuses on the most common and most serious causes of new-onset dizziness in adults. More than 15% of patients presenting with dizziness to an ED have dangerous causes. Sometimes, a serious cause is obvious based on the presentation (eg, dizziness with fever, cough, and hypoxia due to pneumonia). Other times, dangerous conditions can present with isolated dizziness that mimics benign problems. Misdiagnosis in this latter group is not uncommon, even when patients are evaluated by neurologists.

An important clinical goal is to distinguish serious from benign causes using the fewest resources possible. On average, however, diagnosing dizziness consumes disproportionate resources through extensive testing and hospital admission. Indiscriminate application of CT, CT angiography (CTA), and MRI has low yield and low value in this patient population, yet brain imaging for dizziness continues to increase steadily over time. Annual spending on patients with dizziness in US EDs is now $4 billion, with another $5 billion spent on those admitted. Previously, the evidence base for diagnosing patients with dizziness was limited. A proliferation of recent research, however, has supplied clinicians with high-quality data to guide bedside diagnosis and management, particularly with regard to identifying cerebrovascular causes. This article proposes a new diagnostic paradigm based on symptom timing and triggers, derived from recent advances in evidence-based, targeted bedside examinations for specific dizziness subpopulations. New, acute dizziness presentations are focused on, and discussions about treatment are limited except where specifically relevant to initial ED management.

NEW DIAGNOSTIC APPROACH

Accumulating evidence over the past decade suggests using a different approach based on the timing and triggers for dizziness symptoms rather than type. Timing refers to the onset, duration, and evolution of the dizziness. Triggers refer to actions, movements, or situations that provoke the onset of dizziness in patients who have intermittent symptoms.

A timing and triggers history in dizziness results in 6 possible syndromes (Table 1). This conceptual approach has been endorsed by an international committee of

<table>
<thead>
<tr>
<th>International consensus definitions for major vestibular symptoms</th>
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<tbody>
<tr>
<td>Dizziness is the sensation of disturbed or impaired spatial orientation without a false or distorted sense of motion. This includes sensations sometimes referred to as giddiness, lightheadedness, or nonspecific dizziness but does not include vertigo.</td>
</tr>
<tr>
<td>Presyncope (also near-syncope or faintness) is the sensation of impending loss of consciousness. This sensation may or may not be followed by syncope. When patients report “lightheadedness,” it should be classified as presyncope, dizziness, or both.</td>
</tr>
<tr>
<td>Syncope (also faint) is transient loss of consciousness due to transient global cerebral hypoperfusion characterized by rapid onset, short duration, and spontaneous complete recovery. Syncope usually leads to loss of postural control and falling.</td>
</tr>
<tr>
<td>Vertigo is the sensation of self-motion (of head/body) when no self-motion is occurring or the sensation of distorted self-motion during an otherwise normal head movement.</td>
</tr>
<tr>
<td>Unsteadiness is the feeling of being unstable while seated, standing, or walking without a particular directional preference. This sensation has previously been called disequilibrium or imbalance.</td>
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The differential diagnosis of dizziness is broad, with no single cause accounting for more than 5% to 10% of cases. This article focuses on the most common and most serious causes of new-onset dizziness in adults. More than 15% of patients presenting with dizziness to an ED have dangerous causes. Sometimes, a serious cause is obvious based on the presentation (eg, dizziness with fever, cough, and hypoxia due to pneumonia). Other times, dangerous conditions can present with isolated dizziness that mimics benign problems. Misdiagnosis in this latter group is not uncommon, even when patients are evaluated by neurologists.

An important clinical goal is to distinguish serious from benign causes using the fewest resources possible. On average, however, diagnosing dizziness consumes disproportionate resources through extensive testing and hospital admission. Indiscriminate application of CT, CT angiography (CTA), and MRI has low yield and low value in this patient population, yet brain imaging for dizziness continues to increase steadily over time. Use of brain imaging varies 1.5-fold across hospitals without differences in the detection of neurologic causes. Annual spending on patients with dizziness in US EDs is now $4 billion, with another $5 billion spent on those admitted. Previously, the evidence base for diagnosing patients with dizziness was limited. A proliferation of recent research, however, has supplied clinicians with high-quality data to guide bedside diagnosis and management, particularly with regard to identifying cerebrovascular causes. This article proposes a new diagnostic paradigm based on symptom timing and triggers, derived from recent advances in evidence-based, targeted bedside examinations for specific dizziness subpopulations. New, acute dizziness presentations are focused on, and discussions about treatment are limited except where specifically relevant to initial ED management.
specialists tasked with formulating vestibular research definitions\(^\text{27}\) (described by Bisdorff, Staab, Newman-Toker (International Classification of Vestibular Disorders) elsewhere in this issue). Each syndrome suggests a specific differential diagnosis and targeted bedside examination, which are described further. The TiTrATE acronym stands for timing, triggers, and targeted examinations. The Triage–TiTrATE–Test method (Fig. 1) results in a new diagnostic algorithm (Fig. 2). This article focuses on the 4 acute

<table>
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<th>Table 1</th>
<th>Timing-and-trigger–based vestibular(^a) syndromes</th>
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<tbody>
<tr>
<td>Timing</td>
<td>Obligate Triggers(^b) Present</td>
</tr>
<tr>
<td>New, episodic</td>
<td>t-EVS (eg, BPPV)</td>
</tr>
<tr>
<td>New, continuous</td>
<td>t-AVS (eg, post gentamicin)</td>
</tr>
<tr>
<td>Chronic, persistent</td>
<td>Context-specific chronic vestibular syndrome (eg, uncompensated unilateral vestibular loss, present only with head movement)</td>
</tr>
</tbody>
</table>

Abbreviations: t-EVS, triggered episodic vestibular syndrome; s-EVS, spontaneous episodic vestibular syndrome; t-AVS, traumatic/toxic acute vestibular syndrome; s-AVS, spontaneous acute vestibular syndrome.

\(^a\) Note that the use of the word vestibular connotes vestibular symptoms (dizziness, vertigo, imbalance, or lightheadedness and so forth) rather than underlying vestibular causes (eg, BPPV or vestibular neuritis).

\(^b\) Trigger for nonspecific forms refer to obligate triggers (episodic), exposures (acute, continuous), and contexts (chronic) that sharply distinguish these forms from their spontaneous counterparts. Spontaneous causes, as defined in this article, sometimes have underlying predispositions or precipitants, but these are not only-and-always associations.


Evidence-Based Diagnosis of Dizziness

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**Fig. 1.** The Triage–TiTrATE–Test approach to diagnosing dizziness and vertigo. The TiTrATE acronym stands for timing, triggers, and targeted examinations. (Adapted from Neuro-ophthalmology Virtual Education Library. Available at: http://novel.utah.edu/newman-toker/collection.php. Accessed April 15, 2015; with permission.)
syndromes and does not discuss the 2 chronic syndromes. Some patients with a chief symptom of dizziness have prominent associated features, suggesting a likely diagnosis (Table 2). The emphasis is on those with isolated dizziness or vertigo. Isolated excludes major medical or general neurologic symptoms but includes headache and the typical otologic (eg, hearing loss, tinnitus, or ear fullness), autonomic (eg, nausea/vomiting), or balance (eg, gait unsteadiness/ataxia) accompaniments normally encountered in patients with acute vestibular symptoms.

FOUR VESTIBULAR SYNDROMES

The 4 key vestibular syndromes in ED patients presenting recent intermittent or continuous dizziness are described: triggered episodic vestibular syndrome (t-EVS), spontaneous episodic vestibular syndrome (s-EVS), traumatic/toxic acute vestibular syndrome (t-AVS), and spontaneous acute vestibular syndrome (s-AVS). The word vestibular refers to vestibular symptoms (dizziness, vertigo, unsteadiness, and lightheadedness), not underlying vestibular causes. For t-EVS and s-AVS, the focus is targeted bedside examination, emphasizing eye movements (Tables 3 and 4). For s-EVS and t-AVS, the focus is targeted history taking (see Table 4). Although full details for individual diseases are presented in other articles in this issue, this article summarizes key aspects related to early differential diagnostic considerations.

Episodic Vestibular Syndrome

The episodic vestibular syndrome (EVS) involves intermittent dizziness lasting seconds, minutes, or hours. Episode duration is more important than total illness duration. Most such patients have multiple, discrete episodes spaced out over time. Relapsing and remitting symptoms lasting weeks at a time, such as sometimes seen in multiple sclerosis, should not be considered in this category. EVS is divided into triggered and spontaneous forms; each is discussed.

Triggered episodic vestibular syndrome

Approach Episodes of the t-EVS are precipitated by some specific obligate action or event. The most common triggers are head motion or change in body position (eg, arising from a seated or lying position, tipping the head back in the shower to wash one’s hair, or rolling over in bed). Uncommon triggers include loud sounds or Valsalva maneuvers, among others.5 Attacks usually last seconds to minutes, depending on the underlying cause. Because some vestibular forms are provoked repetitively and...
frequently or patients’ nausea can linger between spells, some patients may overstate episode duration. This can usually be sorted out by careful history taking.

It bears emphasis that clinicians must distinguish triggers (head or body motion provokes new symptoms not present at baseline) from exacerbating features (head or body motion worsens preexisting baseline dizziness). Head movement typically exacerbates any dizziness of vestibular cause (benign or dangerous, central or peripheral, or acute or chronic). The concept that worsening of dizziness with head motion equates with a peripheral cause is a common misconception.28

The goal of physical examination in t-EVS is to reproduce a patient’s dizziness to witness the corresponding pathophysiology (eg, falling blood pressure on arising or abnormal eye movements with Dix-Hallpike testing). A caveat for postural symptoms is that orthostatic dizziness and orthostatic hypotension are not always related.29,30 Orthostatic hypotension may be incidental and misleading, especially in older patients taking antihypertensive medications.31 Conversely, dizziness on arising without systemic orthostatic hypotension may indicate hemodynamic transient ischemic attack (TIA) from hypoperfusion distal to a cranial vascular stenosis32 or, alternatively, intracranial hypotension.33 Neurologic evaluation is probably indicated for patients with reproducible and sustained orthostatic dizziness but no demonstrable hypotension or benign paroxysmal positional vertigo (BPPV).

Prototype t-EVS causes are BPPV and orthostatic hypotension. Dangerous causes include neurologic mimics, known as central paroxysmal positional vertigo (CPPV) (eg, posterior fossa mass lesions34), and serious causes of orthostatic hypotension,35 such as internal bleeding. All are associated with episodic positional symptoms but can be readily distinguished from one another using targeted bedside history and examination. Orthostatic hypotension causes symptoms only on arising, whereas BPPV causes symptoms both on arising and on lying back or when rolling in bed.36

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Prominent associated symptoms, signs, or laboratory results that may be available at the initial triage step to inform diagnosis in dizziness/vertigo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptom or Finding</td>
<td>Diagnoses That Are Suggested by the Finding</td>
</tr>
<tr>
<td>Altered mental status</td>
<td>Wernicke encephalopathy; stroke; encephalitis; seizure; intoxication with alcohol, illicit drugs, carbon monoxide; hypertensive encephalopathy</td>
</tr>
<tr>
<td>Transient loss of consciousness</td>
<td>Arrhythmia; acute coronary syndrome; aortic dissection; pulmonary embolism; vasovagal syncope; hypovolemia; stroke; subarachnoid hemorrhage; seizure</td>
</tr>
<tr>
<td>Headache</td>
<td>Stroke; craniocervical vascular dissection; meningitis; carbon monoxide exposure; vestibular migraine; high or low intracranial pressure; subarachnoid hemorrhage</td>
</tr>
<tr>
<td>Neck pain</td>
<td>Craniovascular vascular dissection (especially vertebral artery)</td>
</tr>
<tr>
<td>Chest/back pain</td>
<td>Acute coronary syndrome; aortic dissection</td>
</tr>
<tr>
<td>Abdominal/back pain</td>
<td>Ruptured ectopic pregnancy; aortic dissection</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>Pulmonary embolism; pneumonia; anemia</td>
</tr>
<tr>
<td>Palpitations</td>
<td>Arrhythmia; vasovagal syncope; panic disorder</td>
</tr>
<tr>
<td>Bleeding or fluid losses</td>
<td>Hypovolemia; anemia</td>
</tr>
<tr>
<td>New/recent medication use</td>
<td>Medication side effects or toxicity (eg, gentamicin)</td>
</tr>
<tr>
<td>Fever or chills</td>
<td>Systemic infection; encephalitis; mastoiditis; meningitis</td>
</tr>
<tr>
<td>Abnormal glucose</td>
<td>Symptomatic hypoglycemia; diabetic ketoacidosis</td>
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<table>
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<tr>
<th>Vestibular Condition</th>
<th>Test Maneuver</th>
<th>Nystagmus Duration</th>
<th>Trajectory/Direction</th>
<th>Variation in Direction</th>
</tr>
</thead>
<tbody>
<tr>
<td>t-EVS(^a) (episodic nystagmus triggered by specific positional maneuvers)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posterior canal BPPV</td>
<td>Head hanging with 45° turn to each side (Dix-Hallpike test)</td>
<td>5–30 s(^b)</td>
<td>Upbeat-torsional(^c)</td>
<td>Direction reversal on arising</td>
</tr>
<tr>
<td>Horizontal canal BPPV</td>
<td>Supine roll to either side (Pagnini–McClure maneuver)</td>
<td>30–90 s(^b)</td>
<td>Horizontal</td>
<td>Spontaneous reversal during test</td>
</tr>
<tr>
<td>CPPV</td>
<td>Any (usually head hanging)</td>
<td>5–60 s(^b) (sometimes persistent if position is held)</td>
<td>Any (usually downbeat or horizontal)</td>
<td>Any (often direction fixed)</td>
</tr>
<tr>
<td>s-AVS(^a) (spontaneous nystagmus that may be exacerbated nonspecifically by various head maneuvers)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vestibular neuritis or labyrinthitis</td>
<td>Gaze testing(^d)</td>
<td>Persistent</td>
<td>Dominantly horizontal</td>
<td>Direction fixed (acutely)</td>
</tr>
<tr>
<td>Stroke</td>
<td>Gaze testing(^d)</td>
<td>Persistent</td>
<td>Any (usually dominantly horizontal, occasionally vertical or torsional)</td>
<td>Direction fixed or direction changing with gaze position</td>
</tr>
</tbody>
</table>

Italic: very likely peripheral nystagmus; Bold: very likely central nystagmus; Roman: indeterminate nystagmus (other eye movement features may be diagnostic).

\(^a\) Only 2 syndromes (t-EVS and s-AVS) are shown in this table because the other 2 syndromes (s-EVS and t-AVS) lack characteristic, diagnostic patterns of nystagmus.

\(^b\) BPPV nystagmus usually begins after a delay (latency) of a few seconds, peaks in intensity rapidly, then decays monophasically as long as the head is held stationary. In the horizontal canal variant, the nystagmus may be biphasic, with a spontaneous direction reversal after the initial nystagmus, even if the head is held motionless. CPPV may begin immediately or after a delay, may decay or persist, and may or may not change direction during testing.

\(^c\) Torsion with the 12-o’clock pole (top) of the eye beating toward down-facing (tested) ear, sometimes referred to as geotropic (i.e., toward the ground).

\(^d\) In the AVS, gaze testing is useful but positional tests are not. With peripheral lesions, nystagmus should increase in intensity when a patient’s gaze is directed toward the fast phase of the nystagmus and should not reverse. With central lesions, this same pattern may occur, but more than one-third of the time, the nystagmus reverses direction when the patient’s gaze is directed away from the fast phase of the nystagmus (i.e., is direction changing with gaze position).

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Targeted Examination</th>
<th>Benign Disorder</th>
<th>Dangerous Mimic</th>
<th>Safe-to-Go Features</th>
</tr>
</thead>
</table>
| t-EVS    | Orthostatic vitals; positional tests for nystagmus | BPPV | Posterior fossa mass | • No pain, auditory, neurologic symptoms, or syncope  
• Symptoms not limited to arising and occur when tipping head forward/back or rolling in bed  
• Asymptomatic with head stationary, symptoms reproduced by specific positional tests (see Table 3)  
• Characteristic, canal-specific, peripheral-type nystagmus on positional tests (see Table 3)  
• Therapeutic response to canal-specific repositioning maneuvers (posterior canal: modified Epley maneuver; horizontal canal: Lempert roll [barbecue] maneuver) |
| s-EVS    | Head, neck, and cranial nerve history; ear, hearing history | Vestibular migraine or Menière disease | TIA | • No cardiorespiratory symptoms or transient loss of consciousness  
• No diplopia or other dangerous D symptoms (dysarthria, dysphagia, dysphonia, dysmetria)  
• No papilledema, Horner syndrome, cranial nerve signs (eg, facial palsy, especially if headache present)  
• No sudden, severe, or sustained pain (especially located in the posterior neck)  
• Strong/long past history of dizziness episodes (at least 5 spells over >2 years)  
• Clear precipitants (eg, stress, food, visual motion) for multiple episodes or ABCD² risk score ≤3  
• Migraine: history of migraine headache; classic visual aura or photophobia with most attacks  
• Ménière: history of unilateral fluctuating hearing loss or tinnitus with most attacks |
<table>
<thead>
<tr>
<th>s-AVS</th>
<th>HINTS; ear, hearing examination</th>
<th>Vestibular neuritis</th>
<th>Stroke c</th>
</tr>
</thead>
</table>

- Maximum 1 prodromal spell <48 h before onset
- No excessive vomiting or gait disorder
- No pain, auditory, neurologic symptoms
- No papilledema, Horner syndrome, cranial nerve signs (eg, facial palsy), especially if headache present
- Stands and walks unassisted (even if unsteady or wide based, unable to perform tandem gait)
- HINTS plus hearing/ear examination—SEND HIM ON HOME:
  - SEND—straight eyes (no vertical ocular misalignment, also known as skew), no deafness
  - HIM—head impulse misses (unilateral abnormal impulse opposite nystagmus direction)
  - ON—one-way nystagmus (unidirectional nystagmus worse in gaze toward fast phase)
  - HOME—healthy otic and mastoid examination (pearly tympanic membrane with no pimplies, pus, or perforation; no pain on palpation of the mastoid)

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a Vestibular disorders are highlighted because ED physicians have a high degree of comfort diagnosing other benign causes of isolated t-EVS (eg, orthostatic hypotension) and isolated s-EVS (eg, vasovagal syncope). Dangerous nonvestibular, non-neurologic causes (principally for s-EVS) are rarely isolated (see Table 2).

b Only 3 syndromes (t-EVS, s-EVS, and s-AVS) are shown in this table because the other syndrome (t-AVS) is typically diagnosed based largely on exposure history.

c Findings on the HINTS examination that suggest stroke are given the acronym INFARCT (impulse normal, fast-phase alternating, and refixation on cover test). Thus, if an s-AVS patient has any 1 of these 3 eye signs (bilaterally normal head impulses; direction-changing, gaze-evoked nystagmus; or vertical skew deviation), stroke is likely.

and CPPV can be distinguished based on characteristic eye examination differences on standard positional tests for nystagmus, including the Dix-Hallpike test (see Table 3).  

**Diseases**  
BPPV is the most common vestibular disorder in the general population, with a lifetime prevalence of 2.4% and increasing incidence with age. In the ED, it is probably the second most common cause, accounting for approximately 10% of ED dizzy presentations. It results from mobile crystalline debris trapped in 1 or more semicircular canals (canaliths) within the vestibular labyrinth. Symptoms and signs vary based on the canal(s) involved and whether the crystals are free-floating or trapped. Classic symptoms are repetitive, brief, triggered episodes of rotational vertigo lasting more than a few seconds but less than 1 minute, although nonvertiginous symptoms of dizziness or even presyncope is frequent.  

The diagnosis is confirmed by reproducing symptoms and signs using canal-specific positional testing maneuvers and identifying a canal-specific nystagmus (see Table 3). Because the offending canal(s) are generally not known in advance, multiple diagnostic maneuvers are typically performed. Proven bedside treatments to displace the offending crystals (canalith repositioning maneuvers) are also canal specific. As discussed previously, BPPV mimics include orthostatic hypotension and CPPV. Patients with atypical nystagmus forms (eg, downbeat or horizontal) on Dix-Hallpike testing usually have CPPV, and some cases are due to posterior fossa tumors or strokes. CPPV includes common, benign causes, such as intoxication with alcohol or sedative drugs, but such patients are more apt to complain of continuous, persistent dizziness exacerbated (rather than triggered) by position change and are usually readily diagnosed based on context and other signs of intoxication.  

Orthostatic hypotension is common, accounting for 24% of acute syncopal spells. Classic symptoms are brief lightheadedness or a feeling of near-syncope on arising, but vertigo is common and underappreciated. Orthostatic hypotension is caused by numerous conditions that produce hypovolemia, cardiac dysfunction, or reduced vasomotor tone. The most common causes are medications and hypovolemia.  

The primary dangerous concern is internal bleeding. Strong bedside predictors of moderate hypovolemia from blood loss are postural dizziness so severe as to prevent standing and a postural pulse increment greater than 30 beats per minute, but the sensitivity of these findings is only 22%. Furthermore, the benign postural orthostatic tachycardia syndrome produces similar clinical findings. Heart rate is not a consistent predictor of serious disease; absence of tachycardia or even relative bradycardia can occur in catastrophic conditions, such as ruptured ectopic pregnancy. Coexistent chest, back, abdominal, or pelvic pain should suggest intrathoracic or intra-abdominal emergencies. Dangerous diseases presenting severe orthostatic hypotension but sometimes lacking overt clues include myocardial infarction, occult sepsis, adrenal insufficiency, and diabetic ketoacidosis.  

**Spontaneous episodic vestibular syndrome**  
**Approach**  
Episode duration for s-EVS varies, ranging from seconds to a few days, but a majority of spells last minutes to hours. Patients are often asymptomatic at the time of ED presentation. Because episodes cannot usually be provoked at the bedside (as they can with the t-EVS), evaluation relies almost entirely on history taking. The frequency of spells varies from multiple times a day to monthly, depending on the cause. Although precipitants may exist (eg, red wine prior to vestibular migraine), many spells occur without apparent provocation. This differs from BPPV and other diseases with obligate, immediate triggers. Diagnosis may be clear-cut in typical
cases. Unfortunately, classic features, such as frank loss of consciousness in vaso-
vagal syncope, headache in vestibular migraine, and fear in panic attacks, are absent in 25% to 35% of cases. Atypical case presentations probably contribute to diagnostic confusion in patients with such transient neurologic attacks.

Prototype s-EVS causes include common benign, recurrent disorders, such as vestibular migraine, vasovagal syncope, and panic attacks. Although Ménière disease is often mentioned as a common cause of s-EVS, its estimated population prevalence (0.1%) is much lower than that of the 3 other episodic disorders. Principal dangerous causes are cerebrovascular (vertebrobasilar TIA and subarachnoid hemorrhage), cardiorespiratory (cardiac arrhythmia, unstable angina, and pulmonary embolus), and endocrine (hypoglycemia). Temporary or intermittent carbon monoxide exposure is a rare serious cause.

**Diseases**

Patients with Ménière disease classically present with episodic vertigo accompanied by unilateral tinnitus and aural fullness, often with reversible sensorineural hearing loss. Only 1 in 4 initially presents with the complete symptom triad, and nonvertiginous dizziness is common. Patients with suspected Ménière disease should generally be referred to an otolaryngologist, but care must be taken to avoid missing TIA mimics with audiovestibular symptoms.

Vestibular migraine (previously called migrainous vertigo, migraine-associated vertigo, or migraine-associated dizziness) is a newly described form of migraine. It is related to basilar-type migraine, but episodes lack a second defining brainstem symptom, such as diplopia, quadriparesis, or paresthesias. The 2 migraine types may exist along a continuum. With a population prevalence of approximately 1%, vestibular migraine is a common cause of s-EVS. A definite diagnosis of vestibular migraine requires greater than or equal to 5 attacks with vestibular symptoms, a history of migraine headaches, and migraine-like symptoms with at least half the attacks.

Episode duration ranges from seconds to days. Nystagmus, if present, may be peripheral, central, or mixed-type. Headache is often absent. When headache does occur, it may begin before, during, or after the dizziness and may differ from the patient’s other typical migraine headaches. Nausea, vomiting, photophobia, phonophobia, and visual auras may occur. There are no pathognomonic signs or biomarkers, so diagnosis is currently based on clinical history and exclusion of alternative causes.

An episode similar to prior spells with long illness duration, migraine features, no red flags, and low vascular risk is sufficient for diagnosis without testing (see Table 4).

Reflex syncope (also called neurocardiogenic or neurally mediated syncope) usually has prodromal symptoms, typically lasting 3 to 30 minutes. Dizziness, the most common prodrome, occurs in 70% to 75% and may be of any type, including vertigo. Although rarely seen in clinical practice, central forms of nystagmus may be identified during provocative testing, suggesting a TIA-like mechanism producing central vertigo. In reflex syncope, episodes of near-syncope (no loss of consciousness) substantially outnumber spells with syncope, so many patients likely present with isolated dizziness. The diagnosis is readily suspected if classic contextual precipitants (eg, pain/fear for vasovagal syncope and micturition/defecation for situational syncope) are present, but these are absent in atypical forms, including those due to carotid sinus hypersensitivity. Diagnosis is based on clinical history, excluding dangerous mimics (especially cardiac arrhythmia), and, if clinically necessary, can be confirmed by formal head-up tilt table testing.

Panic attacks, with or without hyperventilation, are often accompanied by episodic dizziness. Dizziness begins rapidly, peaks within 10 minutes and, by definition, is
accompanied by at least 3 other symptoms. There may be a situational precipitant (eg, claustrophobia), but spells often occur spontaneously. Fear of dying or going crazy are classic symptoms but are absent in 30% of cases. Ictal panic attacks from temporal lobe epilepsy generally last only seconds, and altered mental status is frequent. Hypoglycemia, cardiac arrhythmias, pheochromocytoma, and basilar TIA can all mimic panic attacks presenting with dizziness; each can produce a multi-symptom complex with neurologic and autonomic features.

The most common dangerous diagnoses for s-EVS are TIA and cardiac arrhythmias. In 1975, a National Institutes of Health consensus report on TIA recommended that isolated dizziness or vertigo not be considered a TIA, a pronouncement that has been widely accepted. Recent data, however, contradict this classic teaching. Multiple studies show that dizziness and vertigo, even when isolated, are the most common premonitory vertebrobasilar TIA symptoms and are more frequent in the days to weeks preceding posterior circulation stroke.

TIAs can present with isolated episodes of dizziness weeks to months prior to a completed infarction. Dizziness is the most common presenting symptom of vertebral artery dissection, which affects younger patients, mimics migraine, and is easily misdiagnosed. Dizziness and vertigo are the most common symptoms in basilar artery occlusion and are sometimes early and isolated. Because approximately 5% of TIA patients suffer a stroke within 48 hours and rapid treatment reduces stroke risk by up to 80%, prompt diagnosis is critical. Patients with posterior circulation TIA have an even higher stroke risk than those with anterior circulation spells. The presence of 3 or more vascular risk factors or an ABCD$^2$ score greater than or equal to 4 is a predictor of TIA in patients with s-EVS, although high-risk vascular lesions may predict stroke risk more accurately than risk factor-based scoring.

Cardiac arrhythmias should be considered in any patient with s-EVS, particularly when syncope occurs or when exertion is a precipitant, even if the lead symptom is true spinning vertigo. Although some clinical features during the attack may increase or decrease the odds of a dangerous cardiac cause, additional testing (eg, cardiac loop recording) is often required to confirm the final diagnosis.

**Acute Vestibular Syndrome**

The acute vestibular syndrome (AVS) involves acute, persistent dizziness lasting days to weeks, sometimes with lingering sequelae thereafter. Temporal evolution at onset and in the first week is more important than total illness duration. Most such patients have a monophasic course with an early peak in symptom severity, rapid improvement in symptoms over the first week, and gradual recovery over weeks to months. Unusual cases resolve in less than 48 to 72 hours. AVS is divided into postexposure (traumatic/toxic) and spontaneous forms; each is discussed.

**Traumatic/toxic acute vestibular syndrome**

**Approach** Sometimes AVS results directly from trauma or a toxic exposure (t-AVS). The exposure history is usually obvious. The most common causes are blunt head injury and drug intoxication, particularly with medications (eg, anticonvulsants) or illicit substances affecting the brainstem, cerebellum, or peripheral vestibular apparatus.

Most patients experience a single, acute attack resolving gradually over days to weeks once the exposure has stopped. Depending on the nature of the trauma or toxin, other symptoms, such as headache or altered mental status, may predominate. Rotatory vertigo, spontaneous nystagmus (looking straight ahead), and head-motion
Intolerance may be absent or unimpressive if the pathologic effects are bilateral and relatively symmetric, as with most toxins.

**Diseases** Blunt head trauma, blast injuries, whiplash, and barotrauma may cause direct vestibular nerve injury, labyrinthine concussion, or mechanical disruption of inner ear membranes, resulting in an AVS presentation. Care should be taken not to miss a basal skull fracture or traumatic vertebral artery dissection. Traumatic brain injury may cause the postconcussion syndrome. Patients typically present with a combination of dizziness, headaches, fatigue, and minor cognitive impairments, with dizziness the most common symptom in the first 2 weeks after injury.

Anticonvulsant side effects or toxicity is a frequent cause of dizziness and vertigo in the ED and may present with an acute clinical picture. Carbon monoxide intoxication is an uncommon but important cause to consider. Aminoglycoside toxicity is a well-known cause of acute bilateral vestibular failure. Gentamicin produces profound, permanent loss of vestibular function with relatively spared hearing, and toxicity may occur after even a single antibiotic dose. Although this problem is often discovered during the course of an inpatient admission, patients may develop symptoms later and present to the ED. Patients usually present with predominantly gait unsteadiness and oscillopsia (bouncing vision) while walking.

**Spontaneous acute vestibular syndrome**

**Approach** Classic AVS is defined as the acute onset of persistent, continuous dizziness or vertigo in association with nausea or vomiting, gait instability, nystagmus, and head-motion intolerance that lasts days to weeks. Patients are usually symptomatic at the time of ED presentation and focused physical examination is usually diagnostic. Patients generally experience worsening of AVS symptoms with any head motion, including provocative tests (eg, Dix-Hallpike test). Contrary to conventional wisdom, these exacerbating features do not suggest an etiologic or anatomic diagnosis and must be distinguished from head movements that trigger dizziness. This common source of confusion probably contributes to misdiagnosis of a peripheral problem or positional vertigo when dizziness worsens with head movement or testing. The difference is that a patient with s-AVS is dizzy at rest and feels worse with any head motion, whereas a patient with t-EVS is normal at rest and specific head motions induce transient dizziness. This means that positional tests, such as Dix-Hallpike test, should not be applied to AVS patients but reserved for use in EVS.

The prototype s-AVS cause is vestibular neuritis (often incorrectly called labyrinthitis), an acute peripheral vestibulopathy without hearing loss. The primary dangerous mimic is ischemic stroke in the lateral brainstem, cerebellum, or inner ear. Cerebellar hemorrhages rarely mimic a peripheral vestibular process. Uncommon dangerous causes are thiamine deficiency and listeria encephalitis.

Although it is often assumed that strokes usually exhibit neurologic features, obvious focal signs are present in fewer than 20% of stroke patients with s-AVS. Patients are usually symptomatic at initial assessment and often have diagnostic eye signs. Strong evidence suggests that a physical examination clinical decision rule using 3 bedside eye examination findings (HINTS—head impulse test, nystagmus type, and skew deviation; see Table 4) rules out stroke more accurately than early MRI. Importantly, the mere presence of nystagmus (found in both neuritis and stroke) is not as useful as the nystagmus attributes, which help differentiate the 2 (see Table 3).

Eye movement tests have excellent performance characteristics in the hands of neuro-otologists, and similar findings have been replicated by multiple investigative...
teams around the world. Nevertheless, care should be taken before applying these tests in routine ED practice, because interpretation differs between experts and novices and limited instruction may not always be sufficient to yield optimal results. More extensive training with subspecialists directly observing trainees and providing immediate feedback may facilitate skill-building at tertiary care institutions with access to such expertise, but new technologies may offer more widely available help in the near future. Recent studies have found accurate diagnosis using a portable video-oculography device that measures key eye movements quantitatively. Such devices could eventually make subspecialty-level expertise in eye movement assessment widely available for diagnosis or training, although artifacts and related issues with quantitative recordings still currently require expert interpretation.

Neuroimaging studies are often insufficient to accurately diagnose s-AVS cases. CT, the most commonly applied test, is useful to detect (or rule out) brain hemorrhages but is far less helpful for investigating suspected ischemic strokes. Retrospective studies suggest CT may have up to 42% sensitivity for ischemic stroke in dizziness. In prospective studies, however, CT has even lower sensitivity (16%) for detecting early acute ischemic stroke, especially in the posterior fossa (7%). CT should, therefore, not be used to exclude ischemic stroke in s-AVS. Lack of understanding of CT’s limitations for assessment of dizziness may lead to CT overuse and misdiagnosis. Less well known is that even MRI with diffusion-weighted imaging (DWI) misses 10% to 20% of strokes in s-AVS during the first 24 to 48 hours. When smaller strokes (<1 cm in diameter) present with s-AVS, early MRI sensitivity is only approximately 50%. Repeat delayed MRI-DWI (3–7 days after onset of symptoms) may be required to confirm a new infarct. Routine MRI in all ED dizziness also has a low yield. Imaging only older patients with vascular risk factors is a common practice, but the countervailing concern is that young age predisposes to missed stroke. Stroke risk in patients presenting isolated s-AVS and no vascular risk factors is still approximately 10% to 20%, and 1 in 4 strokes occurs in a patient under age 50. Overreliance on youth, low vascular risk, normal neurologic examination, and normal CT likely explains the high odds of missed stroke in isolated dizziness, particularly among younger stroke victims.

Diseases Vestibular neuritis is a benign, self-limited condition affecting the vestibular nerve. Some cases are linked to specific causes (e.g., multiple sclerosis), but most are idiopathic and possibly related to herpes simplex infections. Although vestibular neuritis is usually a monophasic illness, 25% of cases have a single brief prodrome in the week prior to the attack and others have recurrences months or years later. MRI with or without contrast is normal and unnecessary. Diagnosis is based on nystagmus type and vestibular reflexes. Early treatment with oral or intravenous steroids is supported by some evidence but remains controversial.

When hearing loss accompanies vertigo in a neuritis-like s-AVS presentation, the syndrome is known as viral labyrinthitis, although cochleovestibular neuritis might be more appropriate. This benign presentation must be differentiated from bacterial labyrinthitis, a dangerous disorder resulting from spread of middle ear or systemic infection that may lead to meningitis if left untreated. Even in the absence of systemic or local (otitis or mastoiditis) infection, however, this presentation should be viewed suspiciously, because inner ear strokes typically present this way and may often be the cause of s-AVS with hearing loss in the ED. The prevalence of stroke in ED dizziness is 3% to 5% and probably less for those with isolated dizziness. Among ED dizzy patients, those with AVS
are a high-risk subgroup for stroke (approximately 25% of s-AVS cases). Posterior circulation stroke typically presents with s-AVS, sometimes after a series of spontaneous episodes in the preceding weeks or months (ie, TIAs, usually from posterior circulation stenosis, culminating in stroke). Almost all of these strokes (96%) are ischemic. Most are initially associated with minor neurologic disability that recovers well, absent recurrent stroke. Delays in prompt diagnosis and treatment, however, can result in serious permanent disability or death. Although most such patients are not thrombolysis candidates by current guidelines, they may benefit from early secondary prevention treatments and interventions to prevent posterior fossa stroke complications.

**BEDSIDE APPROACH SUMMARY**

For the usual ED patient with isolated dizziness or vertigo that is not obviously of traumatic or toxic cause, the goal for the syndrome-specific targeted examination is to firmly diagnose the specific benign conditions described previously. A majority of cases with initial diagnostic uncertainty are due to common cardiovascular (medication-induced orthostatic hypotension and vasovagal syncope), psychiatric (panic disorder), or vestibular (BPPV, vestibular migraine, and vestibular neuritis) disorders. These benign conditions can each be diagnosed confidently at the bedside using a syndrome-targeted history and examination. Patients whose presentations are atypical or whose targeted examination findings are suspicious for dangerous underlying causes should undergo appropriate laboratory tests, imaging, or consultation.

Bedside examinations for benign vestibular disorders probably deserve special attention in emergency medicine education and in developing decision support tools. Confusion over the conduct of these examinations may stem from the fact that a given clinical feature (eg, upbeat-torsional nystagmus) predicts a benign condition in one syndrome (t-EVS, indicating typical posterior-canal BPPV) but a dangerous one in another (s-AVS, indicating a brainstem stroke). Thus, it is crucial to identify the timing-and-trigger syndrome before targeting the examination, something seldom done in current practice, and, unfortunately, often omitted in prominent textbooks and journal articles. Key criteria that define typical benign vestibular disorder cases and differentiate them from dangerous neurologic causes are shown in Tables 3 and 4.

**SUMMARY**

The prevailing diagnostic paradigm for diagnosing ED patients with dizziness is based on dizziness symptom quality or type. Recent research suggests that the logic underlying this traditional approach is flawed. A newer approach based on timing and triggers of the dizziness likely offers a better diagnostic approach, especially in an unselected ED dizziness population. Using this approach allows targeted bedside examinations of proven value to be used effectively. Future research should seek to prospectively study the new approach to dizziness for its overall diagnostic accuracy, resource efficiency, and impact on health outcomes.

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**APPENDIX**

In the spirit of the flipped classroom, the guest editors of this *Neurologic Clinics of North America* issue on “Emergency Neuro-Otology: Diagnosis and Management of Acute Dizziness and Vertigo” have assembled a collection of unknown cases to be accessed electronically in multimedia format. By design, cases are not linked with specific articles, to avoid untoward cueing effects for the learner. The cases are real and are meant to demonstrate and reinforce lessons provided in this and subsequent articles. In addition to pertinent elements of medical history, cases include videos of key examination findings.

A hyperlink to a URL is provided for a secure server that houses the 10 cases. Note that additional cases may be added over time. Simply click on a case you wish to view and advance at your leisure. The purpose of the cases is illustrative, and there are no quizzes. Please click on this link to be directed to the interactive cases:

https://connect.johnshopkins.edu/vertigooverview/

The authors hope you enjoy the final result.

**REFERENCES**


