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The Timing-and-Triggers Approach to the Patient With Acute Dizziness

December 2019
Volume 21, Number 12

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Abstract

Acute dizziness is a common presentation in the emergency department. Due to newer research, the diagnostic approach to dizziness has changed, now focusing on its timing and triggers of instead of the patient's symptom quality (vertigo versus lightheadedness). Each timing-and-triggers category has its own differential diagnosis and diagnostic approach, which will aid emergency clinicians in distinguishing benign causes of dizziness from life-threatening causes. Brain imaging, even with magnetic resonance imaging, has important limitations in ruling out stroke presenting with dizziness. Benign paroxysmal positional vertigo can be treated with repositioning maneuvers at the bedside, offering cost-effective management options.

Case Presentations

The day shift signs out to you a 44-year-old previously healthy man. He is currently at CT. His dizziness started 6 hours previously and has been present ever since. He describes unsteadiness and "feeling like I am drunk," and has vomited 3 times. He denies headache or neck pain, weakness, or numbness. His vital signs are normal. There is some left-beating horizontal nystagmus in primary gaze and in leftward gaze. The head impulse test is normal. The sign-out is that if his CT scan is normal, he can go home with meclizine and follow-up with his PCP in 2 days. That sounds reasonable, but you wonder if there is something else that needs to be considered...

The 70-year-old woman in room 3 complains of "lightheadedness" that has been going on for 5 days. It goes away at times, and gets worse when she gets out of bed. The dizziness has woken her from sleep several times. She has hypertension and high cholesterol. Her vital signs are normal. Sitting up in the stretcher, she is asymptomatic but feels apprehensive about moving her head. There is no nystagmus in primary gaze. You wonder if you should order a CT or if there is a better diagnostic test...

In room 7, there is a 58-year-old diabetic man whose triage chief complaint was syncope. On further questioning, he is reporting vertigo that is so severe it made him ease himself to the ground. There was no trauma, and it began abruptly 3 hours prior. Fingerstick glucose is 110 mg/dL. There is nystagmus on primary gaze that beats to the right, and when he looks to the right, the amplitude of the nystagmus increases. He is very nauseous and has vomited 3 times. A head impulse test is positive. Skew deviation is absent and he is mildly unsteady but can walk unassisted. You wonder if this could be stroke and whether the stroke team should be activated...

Selected Abbreviations

AICA	Anterior inferior cerebellar artery
AVS	Acute vestibular syndrome
BPPV	Benign paroxysmal positional vertigo
ac-BPPV	Anterior canal BPPV
hc-BPPV	Horizontal canal BPPV
pc-BPPV	Posterior canal BPPV
CPPV	Central paroxysmal positional vertigo
EVS	Episodic vestibular syndrome
s-EVS	Spontaneous episodic vestibular syndrome
t-EVS	Triggered episodic vestibular syndrome
HINTS	Head impulse–nystagmus–test of skew
HIT	Head impulse test
PICA	Posterior inferior cerebellar artery
SCA	Superior cerebellar artery
TIA	Transient ischemic attack
VOR	Vestibulo-ocular reflex

Introduction

It is unusual to work a shift in the emergency department (ED) without seeing at least 1 patient with the complaint of dizziness. The challenge with these patients is due, in part, to the fact that the traditional diagnostic paradigm, which was created nearly 50 years ago, is deeply flawed and leads to confusion. Newer studies strongly suggest that a different diagnostic paradigm based on "timing and triggers" of the dizziness rather than the traditional "symptom quality" (or the "What do you mean, 'dizzy'?") approach is a better approach.

Compounding this problem is the fact that many physicians—and even some general neurologists—have an incomplete understanding of the basic physical examination findings that are useful in evaluating the dizzy patient. Nystagmus, in particular, is poorly understood by many clinicians, and the head impulse test (HIT) has only recently been introduced to emergency medicine practice. The goal of this article is to bridge this knowledge gap and to review the tools and techniques that are available to assist clinical decision-making in the dizzy patient.

Based on the current literature and clinical experience, this issue of *Emergency Medicine Practice* presents a new, algorithmic approach to the diagnosis of acute dizziness. Although this approach to the dizzy patient takes a few extra minutes up-front, it will save time and expense later. More importantly, confidently making a correct diagnosis in a timely fashion may improve patient outcomes, such as reducing falls due to dizziness and improving long-term vestibular function.[1-3] In the case of transient ischemic attack (TIA), starting acute treatments reduces the outcome of stroke.[4,5]

Critical Appraisal of the Literature

A literature search was performed in PubMed and the Cochrane Database of Systematic Reviews. PubMed was searched using the terms *vertigo, dizziness, disequilibrium,* OR *lightheadedness* (limited to title or abstract), limited to the English language, up to November 1, 2018. Relevant Cochrane reviews in the ear, nose, and throat (ENT) and neurology sections were searched. This yielded 22,697 titles (PubMed) and 6 Cochrane reviews. No emergency medicine guidelines exist; however, the American Academy of Neurology[6] and the American Academy of Otolaryngology-Head and Neck Surgery[7] published practice guidelines on benign paroxysmal positional vertigo (BPPV) that have some overlap with emergency medicine practice.

Importantly, one study analyzed the strength of the evidence base in the literature on dizziness and found it to be weak.[8] Of the literature that does exist, most studies were done in settings or by subspecialists

that render them not relevant to ED practice. Therefore, I have used judgment to identify the very small proportion of articles relevant to the management of the acutely dizzy patient by emergency clinicians. Additional references from these articles were identified.

An important first step in critically appraising the literature on dizziness is to analyze the landmark article by Drachman and Hart published in 1972 in the journal *Neurology*.[9] This article influenced subsequent medical literature and practice over the ensuing decades, and it forms the foundation of the "symptom-quality" approach to dizziness that is taught across specialty lines. The authors (a neurologist and an ENT specialist) established a "dizziness clinic" to which patients were referred. The patients underwent 4 half-days of evaluation, including history and detailed physical examination. A diagnosis was assigned by the lead author. Methodologic limitations of this study included:

- **Small number of patients:** Only 125 patients were enrolled over a 2-year period, of whom 21 were rejected for inadequate data and another 9 for lack of a diagnosis. Only 95 patients completed the study.
- **Highly selected patient population**: Recruited patients had to be fluent in English and available (and well enough) to return on 4 additional half-days for further testing in a clinic. These were not typical ED patients with dizziness, many of whom would have general medical conditions or be too sick (or die) to return for multiple repeat clinic visits.
- **Lack of independent verification of the diagnosis:** The lead author assigned a diagnosis without any external verification. To some extent, circular reasoning was applied, in that a peripheral vestibular disorder was typically assigned to patients with rotatory nystagmus.
- **No long-term follow-up of patients:** In addition to the lack of verification of the diagnosis, no follow-up was done, adding further ambiguity to the initial diagnostic accuracy.

Nonmethodologic limitations included:

- **Lack of any brain imaging:** Neither computed tomography (CT) nor magnetic resonance imaging (MRI) was available in 1970-1972.
- **Some important diagnoses were not recognized at that time.** Vestibular migraine was not an established diagnosis. Posterior circulation transient ischemic attack (TIA) presenting as isolated dizziness was not considered to occur.

The paradigm of "symptom quality" has never been prospectively validated, and the subjects of this study are not representative of ED patients with dizziness. Although the article was an important contribution in its time, it is fatally flawed. Newer evidence shows that its inherent logic is wrong.

Etiology, Relevant Anatomy, Physiology, and Pathophysiology

The precise cause of dizziness from general medical causes is often unclear, but may be mediated by neural dysfunction due to factors such as fever, hypotension, anemia, medication side effects, or electrolyte or glucose abnormalities. For patients with dizziness due to vestibular and neurological causes, the mechanism of the dizziness is more clear-cut; understanding the anatomy and physiology is key.

Starting with the end organs in the labyrinth and working centrally, the peripheral vestibular apparatus (organ of balance) and the cochlea (organ of hearing) lie in the temporal bone. (**See Figure 1.**) The vestibular apparatus includes 3 paired semicircular canals that sense rotational motion and the utricle and saccule that sense linear motion. (**See Figure 2, page 4.**) These structures are connected to each other and 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. As fluid moves in a semicircular canal, it displaces the cupula, which generates the sense of motion.

To view color images of the figures in this issue, scan the QR code with an enabled device.

Figure 1. Inner Ear Anatomy

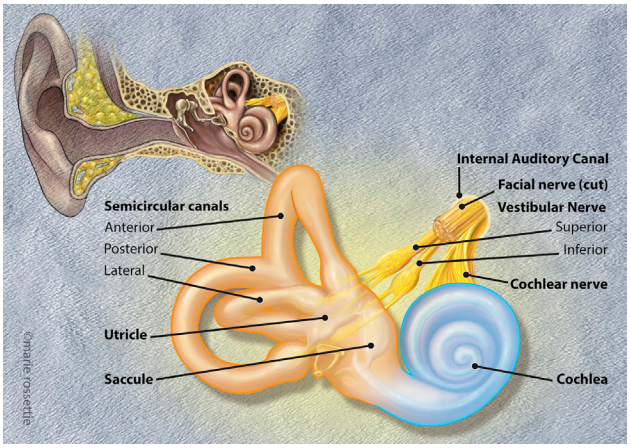

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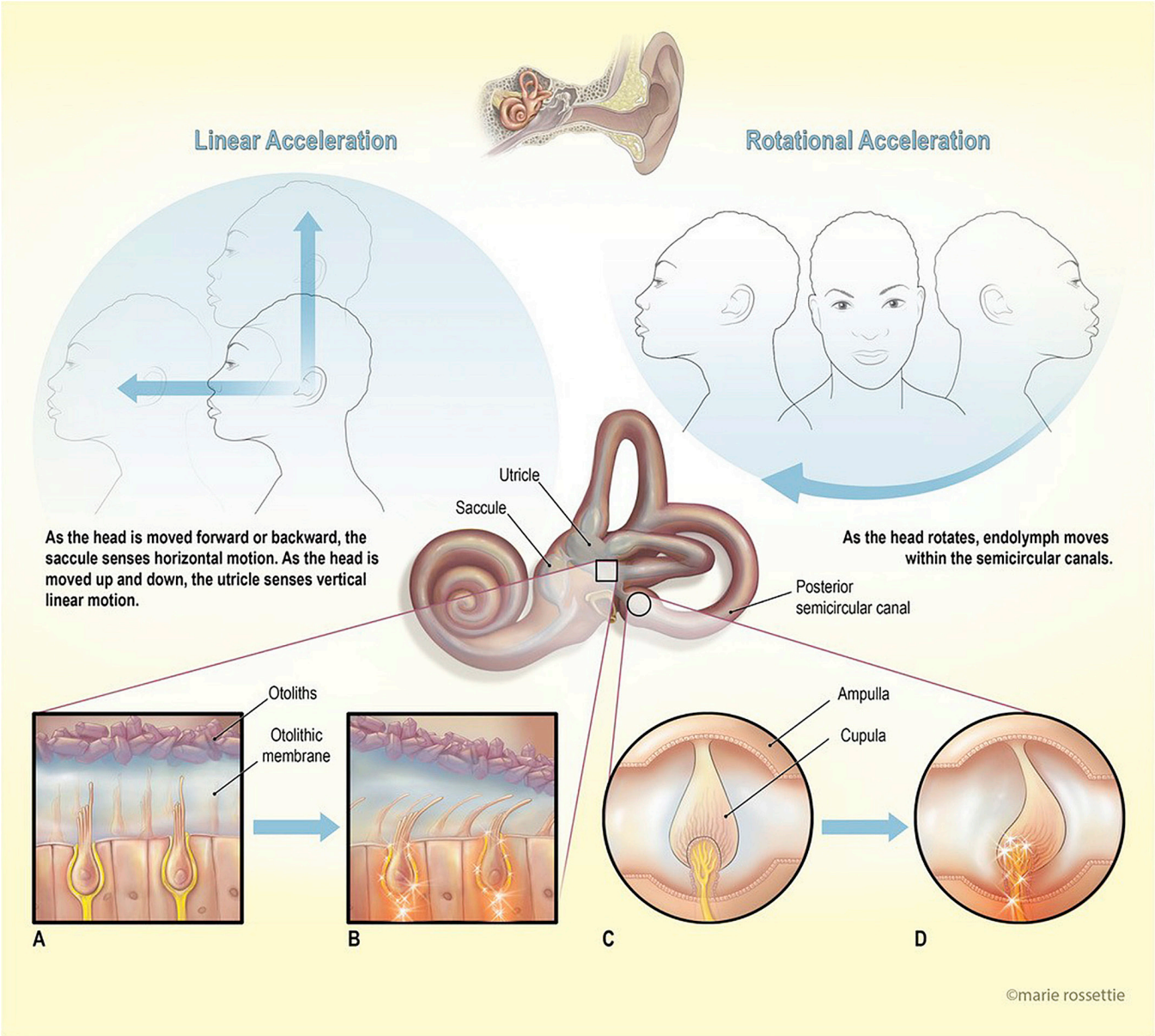
Figure 2 shows that, with linear head movement, gravity causes the heavier otoliths to move, displacing the hair cells in the utricle (vertical) and saccule (horizontal) movement. With angular motion, fluid motion displaces the cupula, which lies within the dilated end-portion of the semicircular canals (ampulla). This displacement of the cupula is also transduced into electrical energy. The electrical impulses from the hair cells of the utricle, saccule, and the cupulae of the semicircular canals are then transmitted to the brainstem via the vestibular nerve.

The pathophysiology of BPPV is that dislodged otoliths from the utricle migrate into one of the semicircular canals; most commonly, these are the posterior canals, because they are the most dependent, with respect to gravity.[10] Free-floating otoliths can then move in the canal, simulating motion that is not actually occurring. In a small proportion of patients with BPPV, the otoliths become "stuck" on the cupula, known as a *heavy cupula*. Gravity causes a pulling on the cupula that also falsely simulates motion. **(See Figure 3, page 5.)**

The eighth cranial nerve is actually 2 nerves: the vestibular nerve, which innervates the peripheral vestibular labyrinth; and the cochlear nerve, which innervates the hearing apparatus. These 2 nerves enter the skull via the internal acoustic canal (with the

Figure 2. Vestibular Anatomy and Physiology



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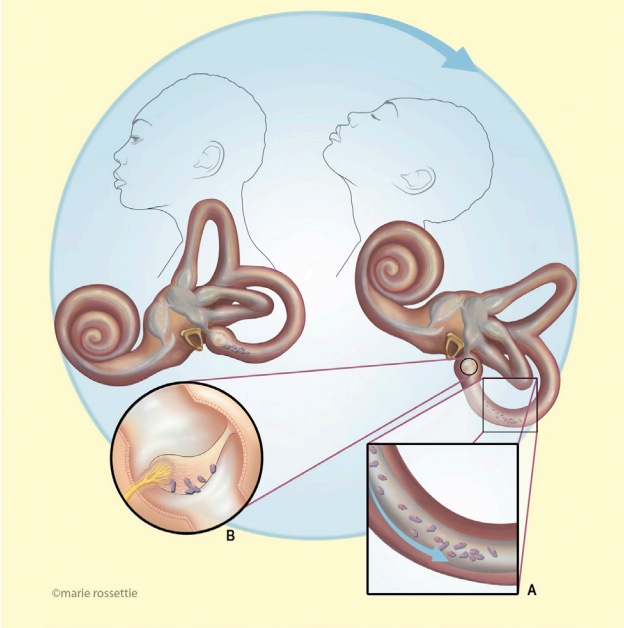
facial nerve and labyrinthine artery). Signals from the vestibular labyrinth travel through the vestibular nerve and enter the brainstem. After traversing the lateral pons (nerve root entry zone), they synapse in the vestibular nuclei of the pons and upper medulla. The vestibular nuclei have extensive connections with the cerebellum, oculomotor system, cortex, and spinal cord. The connections with the oculomotor system contain a reflex arc—the vestibulo-ocular reflex (VOR)—that helps to maintain one's gaze when the head is moving, an important survival trait. This VOR is clinically tested by the horizontal head impulse test (HIT), which was described in 1988.[11] This arc does not loop through the cerebellum, which is why the HIT is "negative" in patients with cerebellar stroke. However, because of the connections between the vestibular nuclei and the cerebellum, the cerebellum does have an important modulating function on the VOR.

Blood supply is via paired vertebral arteries that ascend in the neck and fuse to form the basilar artery. Just prior to fusing, they give off the posterior inferior cerebellar artery (PICA). A large branch of the mid-basilar is the anterior inferior cerebellar artery (AICA). The superior cerebellar artery (SCA) is a major branch just before the basilar artery splits into the terminal branches of the posterior cerebral arteries.

The PICA nourishes the lateral medulla. The AICA nourishes the lateral pons, where the vestibular nerve root entry zone is into the brainstem. (**See Figure 4.**) A branch of the AICA, the labyrinthine artery, supplies the peripheral labyrinth. This explains why strokes of the lateral pons and the labyrinth are associated with a positive or (falsely) "reassuring" HIT.

Figure 3. Mechanisms of Benign Paroxysmal Positional Vertigo



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Differential Diagnosis, Diagnostic Approach, and Misdiagnosis

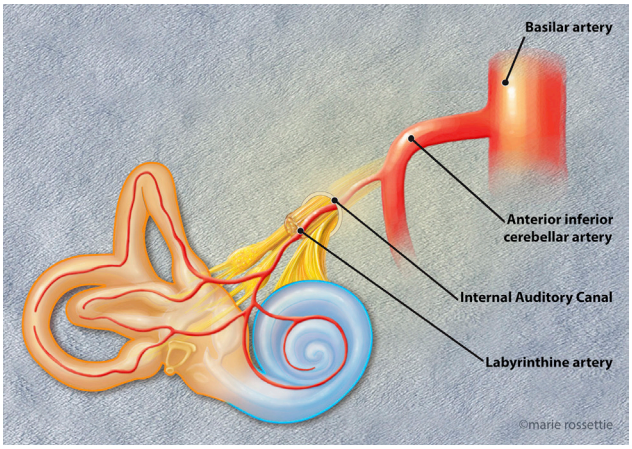
The list of causes of dizziness is too long to be clinically useful and is not the best way to approach the patient diagnostically. Rather, one must develop an organized method to sort through the various causes of dizziness. In an analysis of 9472 patients from a large National Hospital Ambulatory Medical Care Survey (NHAMCS) database of ED patients,[12] the causes of dizziness listed in the charts by the attending emergency physicians were as follows:

- General medical conditions (toxic, metabolic, and infectious): 49%
- Otologic or vestibular conditions: 33%
- Cardiovascular causes: 21%
- Respiratory conditions: 12%
- Neurological diseases: 7%
- Cerebrovascular causes: 4%

Predefined "dangerous" diagnoses (mostly serious cardiovascular, cerebrovascular, and general medical conditions) accounted for 15% of cases, and patients aged > 50 years were more than twice as likely as younger patients to have a dangerous diagnosis.[12]

Rather than basing a differential diagnosis on the descriptive word a patient uses ("vertigo," "lightheadedness," or "imbalance," which is the traditional symptom-quality approach), a more logical strategy is based on the timing and the triggers of the dizziness.[10,13-16]

Figure 4. Cerebrovascular Anatomy of the Labyrinth



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Symptom-Quality Approach

The traditional diagnostic approach is to ask the patient, "What do you mean, 'dizzy'?" According to this scheme, patients will select (1) vertigo, (2) lightheadedness, (3) disequilibrium, or (4) none of the above. The first group will have a vestibular problem, the second group has a cardiovascular or general medical problem, the third group has a neurological problem, and the last group usually has psychiatric diagnoses.[9]

For the traditional symptom-quality approach to dizziness to work, 2 things must be true: First, patients should be able to consistently select a single "type" of dizziness. Second, each dizziness type should be tightly associated with a list of specific diagnoses. However, neither of these propositions is true. In a study of ED patients with dizziness, researchers presented a list of questions about the type of dizziness and the timing and triggers of the dizziness.[17] Then, they asked the same questions again an average of 6 minutes later, but in a different sequence. Fifty percent of the time, patients changed the type of dizziness that they had selected just minutes before and, frequently, they selected 2 or even 3 different dizziness types. On the other hand, they were far more consistent answering questions about the timing of the dizziness and the things that triggered the dizziness.

Second, the use of one term or another (eg, *vertigo* or *lightheadedness*) is not tightly associated with a specific differential diagnosis. In a study of 1666 patients with acute dizziness presenting to an ED, the use of the word *vertigo* was not associated with a cerebrovascular diagnosis.[18] In another study of 59 patients with BPPV (the prototypical peripheral vestibular diagnosis), 16 of the patients (27%) reported "dizziness" and not "vertigo."[17] Elderly patients were 3 times more likely to report nonvertiginous dizziness.[19] In another review of patients with cardiovascular conditions with dizziness, nearly 40% reported vertigo (rather than lightheadedness, which would be expected using the traditional paradigm).[20]

Timing-and-Triggers Approach

One would never base the differential diagnosis of a patient with chest pain or headache solely on the descriptive word that the patient uses. A timing-and-triggers approach is really no different from the way doctors take a history on any other patient. For chest pain, one establishes the onset, the evolution, its constant or intermittent nature, and things that trigger it or alleviate it. It is less important that a patient says that her chest pain is "sharp" or "dull" or even "tearing" rather than whether it has been present intermittently, occurs only with exertion, and is relieved by rest. In addition, the review of symptoms identifies the context that alters the differential diagnosis. Dizziness associated with back pain, fever, and dysuria suggests pyelonephritis. Dizziness with vomiting and diarrhea after eating a bad-tasting burrito suggests gastroenteritis. Dizziness with melena that is worse when standing up suggests a gastrointestinal bleed with volume depletion.

To help remember the key elements of the timing-and-triggers assessment approach, I have created the mnemonic "ATTEST."

ATTEST
A = **A**ssociated symptoms
TT = **T**iming and **T**riggers
ES = **E**xamination **S**igns
T = (confirmatory) **T**esting

The ATTEST approach leverages the history and clinical examination to assess the dizzy patient in a systematic way so as to not miss important diagnostic information. The ATTEST approach will be discussed in greater detail in the "Emergency Department Evaluation" section (page 8). Although this approach has not been prospectively evaluated in routine ED practice, it is consistent with the way other chief complaints are approached, and it is rooted in vestibular neurological physiology and pathophysiology, which is far more consistent with the existing evidence base.[21]

The review of systems and the basic context (eg, age and past medical history such as vascular risk factors) are as important with a dizzy patient as with any other ED patient. Asking about timing and triggers yields 3 acute vestibular categories that are tightly associated with a specific differential diagnosis.[10,13,22,23] (**See Table 1, page 7.**)

Patients will have one of several syndromes: (1) the acute vestibular syndrome (AVS), (2) the spontaneous episodic vestibular syndrome (s-EVS), and (3) the triggered episodic vestibular syndrome (t-EVS). Patients with the AVS have sudden or rapid onset of dizziness that is continuously present; associated with nausea, head motion intolerance, and often (but not always) nystagmus.[23] Patients with the s-EVS have intermittent episodes of dizziness that come "out of the blue," without any trigger at all. Patients with the t-EVS have very brief episodes of dizziness that are reliably triggered by something; most often, this is movement of the body (such as standing up) or movement of the head (such as turning over in bed).[10]

For practical purposes, in ED patients with the AVS, the major distinction to be made is neuritis versus stroke. This is because approximately 95% of AVS patients have one of those two diagnoses;[24] 2% to 3% will have an initial presentation of multiple sclerosis.[24,25] The remainder may have one of a long list of very uncommon diagnoses.[24] One important uncommon cause is Wernicke encephalopathy (thiamine deficiency).[26]

For patients with the s-EVS, the most common

diagnosis is, by far, vestibular migraine, but the important serious diagnosis is TIA. Although posterior circulation TIA presenting as isolated dizziness was long thought to not exist, mounting evidence demonstrates that it does.[27-31] In a study of 1141 stroke patients, brief episodes of symptoms occurring within the 2 days prior to the stroke that could be ascribed to posterior circulation ischemia were 36 times more likely in patients who had vertebrobasilar strokes compared to those with anterior circulation strokes.[28]

For patients with the t-EVS, BPPV and non–life-threatening causes of orthostatic hypotension are the common benign causes; central paroxysmal positional vertigo (CPPV) and serious causes of orthostasis are the life-threatening causes. CPPV is caused by small lesions (mass, multiple sclerosis, or tumor) in the region of the fourth ventricle, which can mimic BPPV.[32,33]

Misdiagnosis

Misdiagnosis of patients with acute dizziness—especially misdiagnosis of cerebellar and brainstem stroke—remains an important issue for which there are multiple reasons. The problem is not restricted to emergency clinicians. In a German study of 475 ED patients with dizziness assessed by a neurologist, nearly 50% of diagnoses were changed by a second neurologist (who was blinded to the initial diagnosis) on follow-up.[34] Importantly, evolution of the clinical course over time played a factor in misdiagnosis in 70% of the incorrectly diagnosed patients.[34]

Another reason for misdiagnosis is the "needle in the haystack" phenomenon. Very few ED patients with dizziness are having strokes. In a study of 1666 adult ED patients with dizziness, < 1% of those with isolated dizziness had a cerebrovascular cause.[18] Multiple other studies found that, of ED patients with dizziness who are discharged with a peripheral vestibular diagnosis, < 0.5% (range, 0.14%-0.5%) are subsequently hospitalized with a stroke.[35-38] Although low, this number is higher than for non-dizzy control patients. These studies look forward, prospectively analyzing all dizzy patients (the whole "haystack"), and in the case of dizziness, they show very low misdiagnosis rates.

Other studies look backward, retrospectively analyzing patients who are diagnosed with posterior strokes (focusing only on the "needles"). In some of these "look backward" studies, between 28% and 59% of patients with cerebellar strokes are missed in the ED.[39-41] Risk factors for stroke misdiagnosis include younger age, vertebral dissection as a cause, and a presentation of dizziness.[42-44] In a study of 240 patients with cerebellar strokes, 10% (25 patients) presented with isolated dizziness that mimicked peripheral lesions.[45] Misdiagnosis rates look very different, depending on whether the study is a "look forward" or a "look backward" study.[46]

Table 1. Timing-and-Trigger-Based Vestibular[a] Syndromes in Acute Dizziness and Their Corresponding Differential Diagnosis

Syndrome	Description	Common Benign Causes	Common Serious Causes	Important Rare Causes
AVS	Acute, continuous dizziness lasting days, accompanied by nausea, vomiting, nystagmus, head-motion intolerance, and gait unsteadiness	• Vestibular neuritis • Labyrinthitis	• Posterior circulation ischemic stroke	• Multiple sclerosis • Wernicke encephalopathy • Drug/medication side effects or toxicity
s-EVS	Episodic dizziness that occurs spontaneously, is not triggered,[b] and usually lasts minutes to hours	• Vestibular migraine • Ménière disease	• Posterior circulation TIA	• Cardiac dysrhythmia • Pulmonary embolism • Panic attack
t-EVS	Episodic dizziness triggered by a specific, obligate trigger (typically a change in head position or standing up), and usually lasting less than 1 minute	• BPPV • Orthostatic hypotension caused by benign problems	• CPPV • Orthostatic hypotension due to serious medical illness	• Superior canal dehiscence • Postural tachycardia syndrome • Panic attack • Vertebral artery rotation (Bow Hunters syndrome)

Note: This table lists the most common diseases causing these presenting syndromes and is not intended to be exhaustive.

[a]The word *vestibular* here connotes vestibular symptoms (dizziness or vertigo or imbalance or lightheadedness, etc), rather than underlying vestibular diseases (eg, BPPV or vestibular neuritis).

[b]Dizziness is "triggered" (not dizzy at baseline, dizziness develops with movement), as in position vertigo due to BPPV. This must be distinguished from dizziness that is "exacerbated" (dizzy at baseline, worse with movement); such exacerbations are common in AVS, whether peripheral (neuritis) or central (stroke).

Abbreviations: AVS, acute vestibular syndrome; BPPV, benign paroxysmal positional vertigo; CPPV, central paroxysmal positional vertigo; s-EVS, spontaneous episodic vestibular syndrome; t-EVS, triggered episodic vestibular syndrome; TIA, transient ischemic attack.

Kiersten L. Gurley, Jonathan A. Edlow. Acute dizziness. *Seminars in Neurology.* 2019;39(01):27040. © Georg Thieme Verlag AG. Used with permission.

Patients with anterior circulation strokes often present with lateralizing weakness, which receives more attention in the National Institutes of Health Stroke Scale (NIHSS), prehospital stroke scales, and high-impact medical literature[47] than posterior circulation strokes. The latter are misdiagnosed more than twice as often as anterior circulation events.[42] Our use of stroke heuristics emphasizes lateralizing deficits, which are often subtle or absent in patients with posterior circulation strokes.

A recent review concluded that overreliance on the symptom-quality method, lack of familiarity with the eye movement examination, overweighting of age and other traditional vascular risk factors, and overreliance on CT scanning are major factors related to misdiagnosis.[16]

Lack of familiarity with some of the eye findings is also an important factor in misdiagnosis. Nystagmus, in particular, is often underutilized or poorly understood by emergency clinicians but may be diagnostically essential. In a study of 1091 dizzy patients in United States EDs, physicians used templates to document the presence or absence of nystagmus in 887 (80%) of the cases.[48] Nystagmus was documented as present in 185 (21%).[48] Of these 185 patients, information regarding the nystagmus that was sufficient to be diagnostically useful was recorded for only 10 (5.4%). Of patients given a peripheral vestibular diagnosis, the nystagmus description conflicted with the final diagnosis in 81%.[48] The simple presence or absence of nystagmus is important, but the details of the nystagmus are far more important in informing the diagnosis.

Finally, CT scanning is of very limited utility in posterior circulation stroke. In a large Canadian study of ED patients with dizziness who were discharged with a benign ICD-9 "dizzy" diagnosis and followed for 30 days, patients who returned with a stroke were 2.3 times more likely to have had a CT on the first visit, suggesting that physicians were correctly identifying worrisome patients but were applying the wrong diagnostic test.[49]

Prehospital Care

Other than a fingerstick glucose test, there are no important prehospital interventions for the vast majority of dizzy patients. The only relevant prehospital issue has to do with emergency medical services' recognition of posterior circulation stroke, which can be difficult enough in the ED, let alone in an ambulance. Some preliminary data suggest that, in patients with acute-onset dizziness, adding finger-to-nose testing to a prehospital stroke scale may improve stroke recognition in these patients. Educational efforts may help increase prehospital recognition of posterior circulation stroke.[50] Since there is always a transfer of a patient to a stretcher, some formal gait assessment may also be useful in this regard, but this has not been studied. As with any potential stroke patient, defining the last time known normal is important.

Emergency Department Evaluation

History and vital signs will usually identify the 50% of patients whose dizziness is caused by some general medical cause. The particular descriptive word used by the patient (eg, "lightheadedness" or "vertigo" or "imbalance") to describe their dizziness is not useful diagnostically and should not, by itself, drive the workup.

The algorithmic approach to the evaluation of the dizzy patient should be begin with ATTEST. **(See the Clinical Pathway for the ATTEST Approach to Emergency Department Patients With Acute Dizziness, page 9.)** The first 3 letters in the ATTEST mnemonic (**A**ssociated symptoms, **T**iming, and **T**riggers) refer to historical information: "What happened?" "When?" "Is the dizziness continuous or intermittent?" "Are there associated symptoms?" "What is the broader context?"

Consider medical causes of the complaint, including:

- Fever, dysuria, and back pain, suggesting infection
- Heavy use of ibuprofen (or other nonsteroidal anti-inflammatory drug) and black stools, suggesting gastrointestinal bleeding
- New antihypertensive or anticonvulsant medication use, suggesting medication side effect
- Moderate-mechanism motor vehicle crash, suggesting cervical artery dissection versus cupulolithiasis versus intercranial bleed
- Abdominal pain, vaginal bleeding, and positive pregnancy test, suggesting an ectopic pregnancy.
- Chest pain and dyspnea, suggesting myocardial ischemia or pulmonary embolus
- Flank and back pain, suggesting aortic vascular complications

Each situation suggests a diagnosis or group of diagnoses that would require confirmatory testing. Similarly, the vital signs inform this diagnostic process; ie, is there fever, tachycardia, hypotension, or hypoxia? Thus, the first diagnostic step in the diagnosis of the patient with acute dizziness is simply to take a history and review the vital signs, just as with any other patient. If a general medical diagnosis is likely, I recommend a brief diagnostic "STOP," which takes less than 1 minute to perform.[10,13]

In order to identify patients who might potentially be mimicking a general medical condition, the 3 components of the "STOP" are: (1) a quick test for worrisome nystagmus (described in detail on page 10), (2) arm dysmetria, and (3) truncal ataxia. (To test

for truncal ataxia, simply have the patient sit up in the stretcher without grabbing hold of the side rails.) If the "STOP" test is reassuring, then proceed with treatment for the presumed condition. If it is worrisome, consider various vestibular or central conditions.

Acute Vestibular Syndrome

If the history does not suggest a general medical condition (or if the "STOP" is worrisome), then the next question to pose is, "Is the dizziness persistently present and still present at the time of evaluation in the ED?" A "yes" answer identifies patients with the AVS, who have the abrupt or rapid onset of dizziness that has lasted hours to days and is still present at the time of examination, even when the patient is lying still. The dizziness may decrease when lying still and worsen with head movement, a common occurrence that does not mean that the dizziness has a peripheral cause.

Although the strict neuro-otology definition of the AVS includes the presence of nystagmus, some patients who otherwise fulfil the AVS definition (such as many with cerebellar stroke), do not. The presence or absence of nystagmus is a key distinction because it affects how one interprets the HIT.[13,14,22,23]

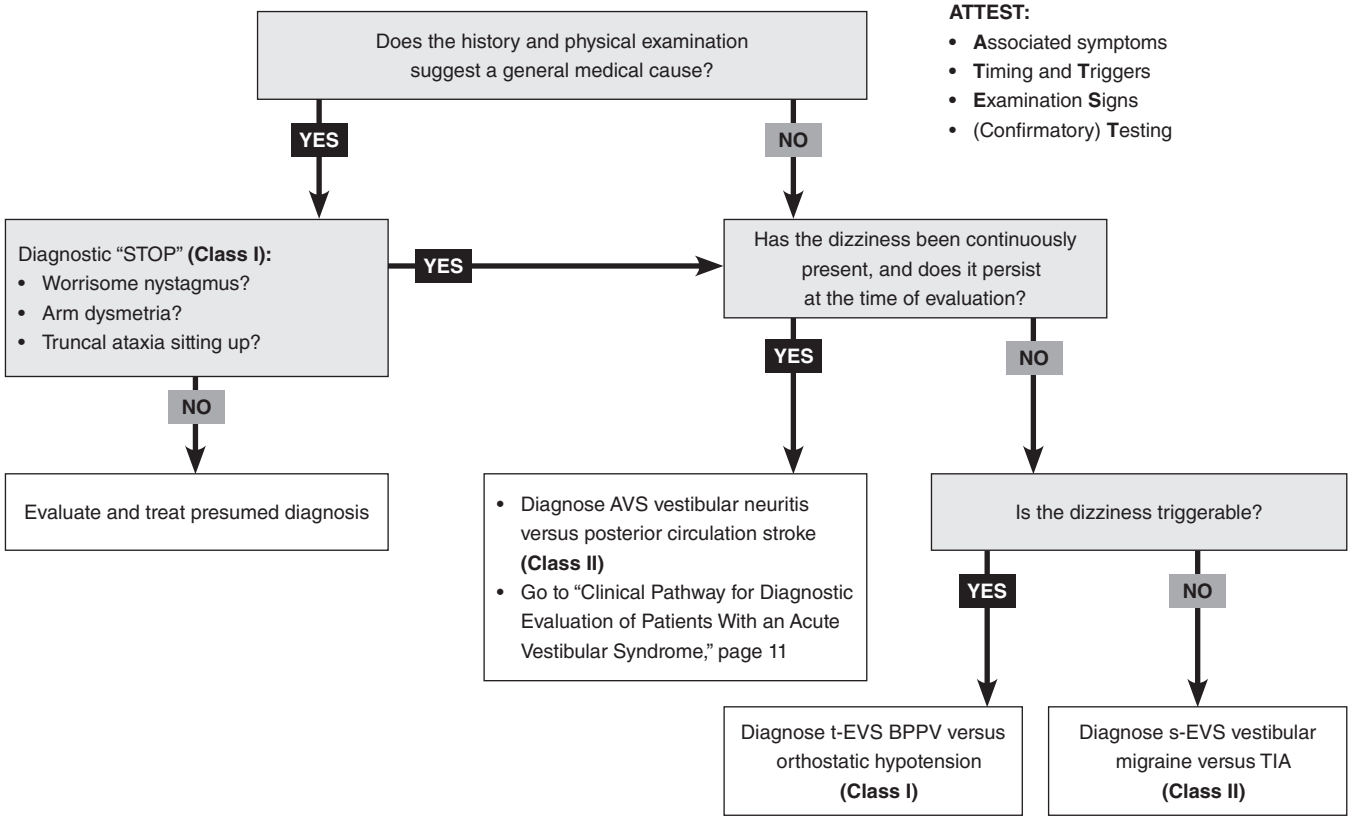
Head Impulse–Nystagmus–Test of Skew (HINTS) Testing

Because, by definition, these patients are acutely symptomatic, one can use physical examination to distinguish between central cause (stroke) and peripheral cause (neuritis), referred to as *head impulse–nystagmus–test of skew* (HINTS) testing. (Note that the HINTS acronym is distinct from the acronym for the head impulse test, or HIT.)

An important caveat is that most of the studies that examine the utility of HINTS have been done with neuro-otologists performing the examinations.[51,52] One study conducted by stroke neurologists showed that non–subspecialists can be trained to use the HINTS examination effectively.[53] Another European study of specially trained emergency physicians (12 hours of special training using Frenzel lenses to interpret the eye findings) also provided evidence of its effective use in the ED.[54,55]

Because HINTS has not been validated in routine practice, 2 additional components must be added to the examination of patients with the AVS:

Clinical Pathway for the ATTEST Approach to Emergency Department Patients With Acute Dizziness



Abbreviations: AVS, acute vestibular syndrome; BPPV, benign paroxysmal positional vertigo; s-EVS, spontaneous episodic vestibular syndrome; t-EVS, triggered episodic vestibular syndrome; TIA, transient ischemic attack.

For Class of Evidence definitions, see page 11.

a targeted posterior circulation examination and testing of the gait.[13,23] When performing the physical examination, I therefore pose 5 questions to be asked, in the following sequence:

1. Is there a central pattern of nystagmus?
2. Is skew deviation present?
3. Is the HIT worrisome for a central process (ie, absent corrective saccade)?
4. Are there central nervous system findings on the targeted posterior circulation examination?
5. Can the patient sit up or walk without assistance?

None of these tests is 100% sensitive, so if the answer to *any one of the questions* is "yes," the patient has a central process, likely stroke, and should be admitted to the hospital for further workup.[13,14,22,23] If the answer to *all 5 questions* is "no," then the patient likely has neuritis and can be safely discharged with outpatient follow-up. **Table 2** summarizes the HINTS elements of the physical examination for AVS, in addition to the 2 components for posterior circulation examination and gait testing. The 5 test elements of HINTS testing, in order, are:

Test 1: Nystagmus Testing

The acronym (HINTS) notwithstanding, I do not start with the HIT, but rather with nystagmus. There are several reasons for this. First, nystagmus testing is easy for the patient. Second, if there is no nystagmus, then interpretation of the HIT is problematic, since it has been validated only in patients with nystagmus. Third, if there is no nystagmus, it makes the vestibular neuritis and labyrinthitis very unlikely (in patients presenting in the first 2 to 3 days of their illness). Finally, if there is nystagmus that is of a central type, whatever the results of the remainder of the examination, this is a patient who must be assumed to be having a stroke.

To test for nystagmus, first simply ask the patient to open his eyes and look forward. Observe whether there is any jerk nystagmus, in which the eyes drift in one direction, then snap quickly back. By convention, it is the rapid phase for which the nystagmus is named. If a patient looks forward and his eyes drift to the left, then snap back to the right, he has a *right-beating horizontal nystagmus*. This is usually very easy to see, especially in the first 2 to 3 days of the patient's onset of symptoms. Next, ask the patient to follow the examiner's finger, going 30° to 40° to the right, then to the left. This is called *gaze-evoked nystagmus*. Also look for *vertical* or *pure torsional nystagmus*. In patients with the AVS, nystagmus that is vertical, torsional, or that changes direction with the direction of gaze is *central*.[10,14]

Test 2: Skew Deviation Testing

Next, I check for skew deviation using the alternate cover test, which is also very easy for the patient. One simply stands in front of the patient, instructing him to focus on your nose. Alternately cover one eye

Table 2. Summary of Useful Physical Examination Findings in Symptomatic Patients With the Acute Vestibular Syndrome

Examination Component	Peripheral (ALL must be present to diagnose vestibular neuritis)	Central (ANY ONE of these findings suggests posterior fossa stroke)
Nystagmus (straight-ahead gaze and rightward and leftward gaze)	Dominantly horizontal, direction-fixed, beating away from the affected side[a]	Dominantly vertical and/or torsional or dominantly horizontal, direction-changing on left/right gaze[b]
Test of skew (alternate cover test)	Normal vertical eye alignment and no corrective vertical movement (ie, no skew deviation)	Skew deviation (small vertical correction on uncovering the eye)[c]
Head impulse test	Unilaterally abnormal with head moving toward the affected side (presence of a corrective refixation saccade toward the normal side)[d]	Usually bilaterally normal (no corrective saccade)
Targeted neurological examination (see text, page 13)	No cranial nerve, brainstem, or cerebellar signs	Presence of limb ataxia, dysarthria, diplopia, ptosis, anisocoria, facial sensory loss (pain/temperature), unilateral decreased hearing
Gait and truncal ataxia	Able to walk unassisted and to sit up in stretcher without holding on or leaning against bed or rails	Unable to walk unassisted or sit up in stretcher without holding on or leaning against bed or rails

[a]Inferior branch vestibular neuritis will present with downbeat-torsional nystagmus, but this is a rare disorder. From the emergency medicine perspective, vertical nystagmus in a patient with an acute vestibular syndrome should be considered to be central (a stroke).

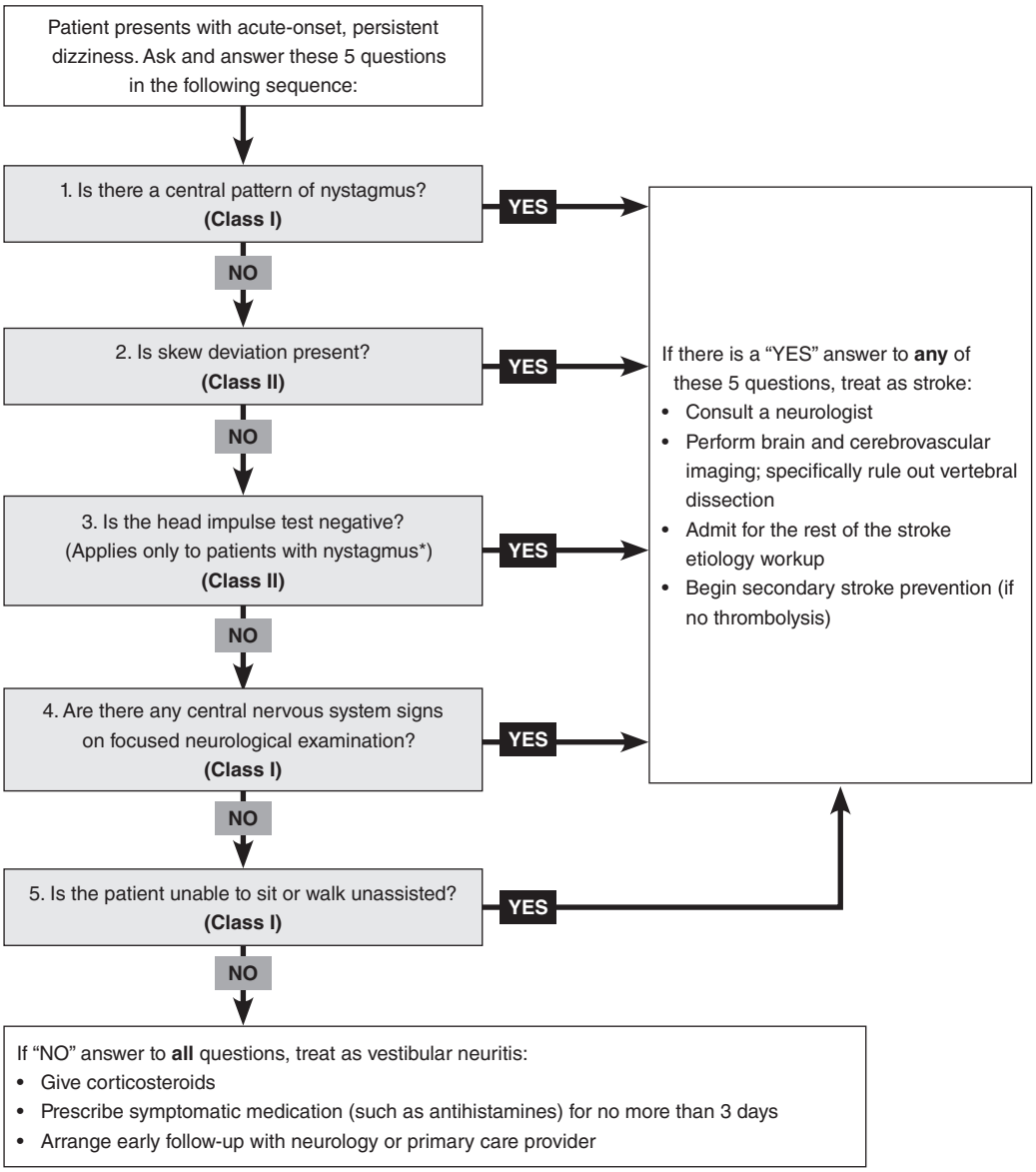
[b]More than half of posterior fossa strokes will have direction-fixed horizontal nystagmus that, alone, cannot be distinguished from that typically seen with vestibular neuritis.

[c]Many patients with posterior circulation stroke will have no skew deviation; so, on this criterion alone, cannot be distinguished from vestibular neuritis.

[d]Strokes in the anterior inferior cerebellar artery territory may produce a unilaterally abnormal head impulse test that mimics vestibular neuritis, but hearing loss is usually present as a clue. If a patient has bilaterally abnormal head impulse test, this is also suspicious for a central lesion if nystagmus is present (as may be seen in Wernicke syndrome).

Kiersten L. Gurley, Jonathan A. Edlow. Acute dizziness. *Seminars in Neurology.* 2019;39(01):27040. © Georg Thieme Verlag AG. Used with permission.

Clinical Pathway for Diagnostic Evaluation of Patients With an Acute Vestibular Syndrome



*In patients without nystagmus, the head impulse test may give misleading results; the focused neurological examination and gait assessment become more important in this group. (See page 13, "Test 4: Targeted Examination," and "Test 5: Gait Testing.")

Class of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

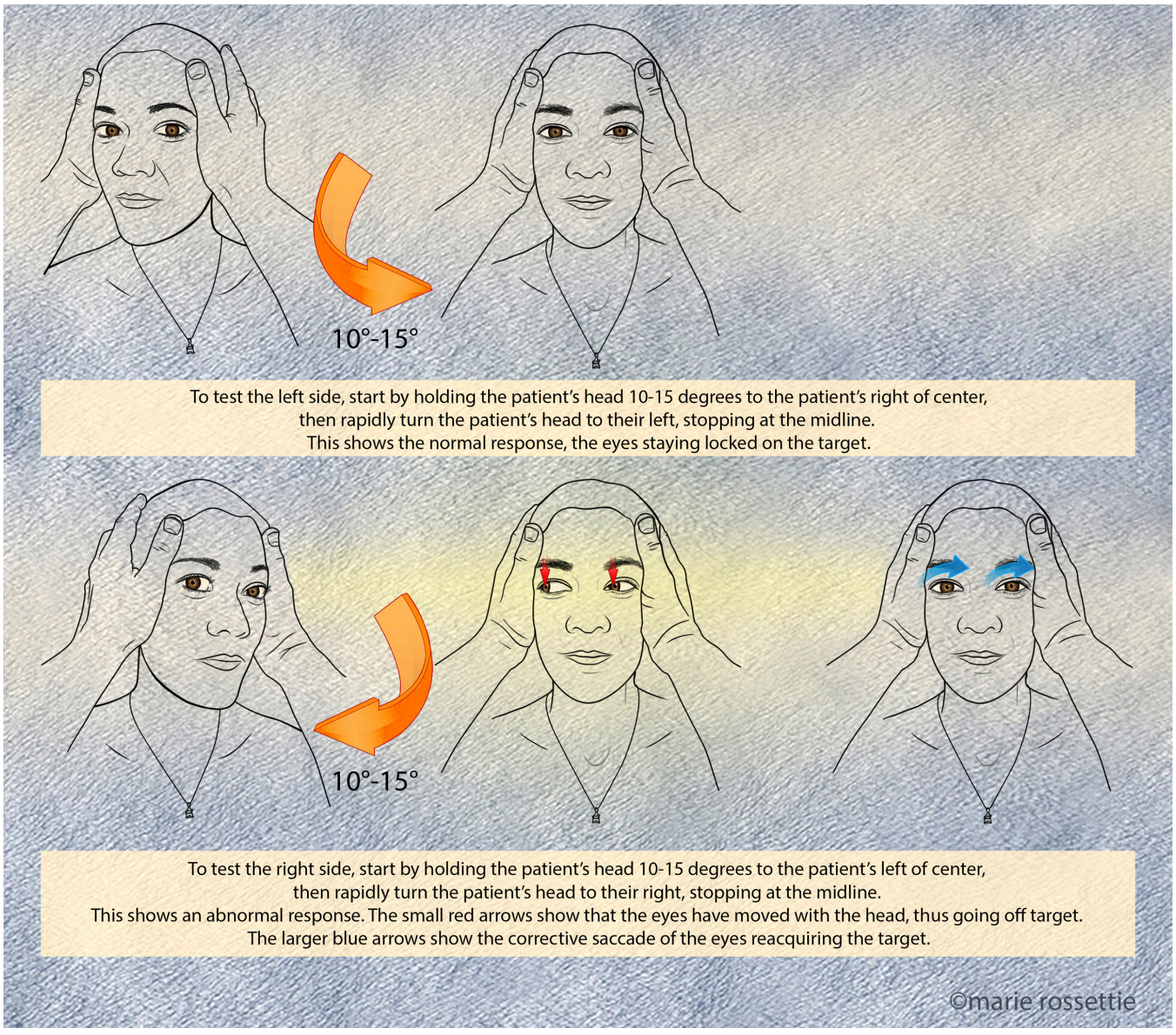
Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

and then the other, multiple times, moving from one eye to the next every second or so. The presence of skew deviation—a small vertical correction in the eye when it is uncovered—indicates a brainstem localization. Although uncommon exceptions exist, from the perspective of an emergency clinician, it is safest to assume that skew deviation is always due to a central problem. It is easiest if one focuses on one or the other eye (it does not matter which), because each will display the vertical correction (one going down while the other goes up).

Test 3: Head Impulse Testing

The third component of the examination is the HIT. (**See Figure 5.**) Again, the patient is instructed to relax his head and neck and to focus on the examiner's nose. The examiner grasps the patient's head on both sides and very rapidly snaps it in one direction or the other over a very small arc (only 10° to 15°).[11,13,14,23] Ideally, hold the head 10° to 15° from the midline and then move it very quickly to the midline. The "normal" or "negative" HIT (when the eyes remain focused on the examiner's nose) is worrisome for stroke, whereas the "abnormal" or "positive" HIT (when the eyes move with the head and then snap back in one corrective saccade to the examiner's nose) is reassuring for neuritis. Therefore, use of the words "normal," "abnormal," "negative," and "positive" to describe the HIT is ambiguous, since the "negative" test is worrisome and the "positive" test is reassuring. It is best to simply state whether a corrective saccade is "absent" or "pres-

Figure 5. Head Impulse Test



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ent."[51,56] Approximately 10% of HITs in which there is a reassuring corrective saccade are false-positives due to strokes,[51] usually of the AICA territory or the labyrinth itself.[23]

Test 4: Targeted Examination

The fourth component of the AVS examination is to perform a targeted examination to detect any central nervous system findings due to posterior circulation ischemia. In addition to a general motor and sensory examination, this examination targets the cranial nerves, cerebellar function, and visual fields. The latter is not in the posterior fossa, but tests the occipital cortex, which is nourished by the posterior cerebral artery, the terminal branches of the basilar artery. This should not require more than a few minutes, but it must be done systematically. Any (new) abnormality indicates a central finding and would therefore be inconsistent with neuritis. For example, anisocoria and ptosis (Horner syndrome) suggest a lateral medullary infarct. Another detail is that the unilateral facial sensory loss in lateral medullary stroke involves pain and temperature, not light touch, which is the usual modality tested by most non–neurologists. It is important to recognize that acute hearing loss, which is traditionally associated with a peripheral process, can also occur with an acute cerebrovascular event involving either the AICA or labyrinthine artery.[23]

Test 5: Gait Testing

Finally, even if all of the first 4 tests are reassuring, the gait must be tested in patients with an AVS. If a patient is unsafe on his feet, he cannot be discharged safely from the ED. In addition, the greater the degree of gait abnormality, the more likely it is that the cause of an AVS is stroke.[57] In a series of 114 patients with an AVS (67% with neuritis, 33% with stroke), most patients with neuritis were able to walk independently, whereas most patients with stroke could not.[57] In fact, two-thirds of the stroke patients could not even stand up independently. Importantly, all of the 10 patients with AICA stroke (whose HIT can be misleading) had severe gait instability.[57]

Spontaneous Episodic Vestibular Syndrome

Patients with the s-EVS report one or more episodes of dizziness of variable duration not triggered by head or body-position changes. Because patients with the s-EVS are, by definition, no longer symptomatic and are not triggerable, physical examination is not useful to distinguish the most common diagnoses, which are vestibular migraine and posterior circulation TIA. Diagnosis relies on history and epidemiologic context.[10] If a patient with vestibular migraine or TIA was still symptomatic at the time of evaluation, he would present and be evaluated as if he had an AVS, just as a patient with an anterior circulation TIA who still had symptoms at the time of presentation would be assumed to be having a stroke.

Specific criteria exist for diagnosis of vestibular migraine.[58] (**See Table 3.**) There is a strong female predominance for vestibular migraine (5:1).[58] Patients with vestibular migraine have multiple episodes of dizziness, and headaches may occur before, during, or after the dizzy episodes.[59] When headaches do occur, they are usually (but not always) similar to migraines that occur without the dizziness. The duration of the dizziness is variable and, by definition, can last 5 minutes to 72 hours,[60] although rarely the duration is even shorter.[58] Because migraine is a central phenomenon, the associated nystagmus can be of a central type.[61]

Up to half of patients who have posterior circulation TIAs have isolated, transient dizziness.[28] Other symptoms include typical posterior circulation symptoms related to the long tracts that pass through the brainstem, cranial nerve dysfunction, or visual field cuts due to posterior cerebral artery ischemia of the visual cortex. Contrary to conventional wisdom, short-term stroke risk may be higher with posterior circulation TIA than with anterior circulation TIA.[27,62]

Recognizing that none of these elements can be used in a binary, yes/no fashion, factors that suggest vestibular migraine over TIA include younger age, more frequent attacks over a longer period of time, other migraine-related symptoms (such as headache, phonophobia, photophobia), and absence of traditional vascular risk factors.

Patients with Ménière disease (which was relatively uncommon in an ED series of dizzy patients) also present with s-EVS and will usually have ringing or buzzing in the ear and, over time, progres-

Table 3. Diagnostic Criteria for Vestibular Migraine[10]

- At least 5 episodes of vestibular symptoms[a] of moderate[b] or severe intensity, lasting between 5 minutes and 72 hours
- Present or previous history of migraine with or without aura (according to the International Classification of Headache Disorders)
- One or more of the following migraine features occurring 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, aggravation by routine physical activity
  - Photophobia or phonophobia
  - Visual aura
- No other vestibular explanation

[a]Spontaneous, positional, or visually induced vertigo; head-motion-induced dizziness with nausea.

[b]Vertigo is "moderate" if it interferes with but does not preclude daily activities, and is "severe" if it prohibits daily activities.

sive hearing loss.[63] Treatment is symptomatic and patients should be referred to an ENT specialist.

Triggered Episodic Vestibular Syndrome

The physical examination is very helpful in patients with a t-EVS and will often establish a specific diagnosis. **(See Table 4.)** Although the utility of orthostatic vital signs has been traditionally downplayed in emergency medicine, a patient with dizziness when standing up who develops symptoms and orthostatic vital signs on standing up is highly likely to have orthostatic hypotension as a cause of the dizziness, and the evaluation is directed at finding the underlying cause.

BPPV should be suspected in patients with very brief episodes of dizziness, generally lasting less than a minute. Brief episodes of dizziness that wake a patient up from sleep are nearly always BPPV.[64-67] One study showed a positive likelihood ratio of 60 for a BPPV diagnosis if dizziness occurred with lying down or turning in bed.[66]

In patients with suspected BPPV, bedside testing can confidently establish the diagnosis. The most commonly affected canal is the posterior canal (pc-BPPV) which is usually tested by the Dix-Hallpike maneuver. If this test is negative on both sides, then the horizontal canal (hc-BPPV) is tested by the supine head roll test. In pc-BPPV, the nystagmus is typically up-beating and torsional, and in hc-BPPV it is horizontal and direction-changing. This illustrates how the interpretation of nystagmus differs from the AVS (where torsional or direction-changing = worrisome diagnosis) from t-EVS (where torsional for pc-BPPV and horizontal direction-changing for hc-BPPV = benign diagnosis).

Occasionally, BPPV patients have no nystagmus.[68-70] Possible causes are a small number of otoliths in the canal, use of vestibular suppressants at the time of diagnosis, or small-amplitude nystagmus that the examiner is not perceiving due to visual

Table 4. Benign Paroxysmal Positional Vertigo Physical Examination, Type of Nystagmus, and Therapeutic Maneuvers

Canal Involved, Mechanism (Proportion of BPPV Cases)	Provocative Diagnostic Maneuver/Test	Expected Type of Nystagmus[a]	Therapeutic Maneuvers
pc-BPPV (80%-85%)	Dix-Hallpike	• Up-beating (from patient's perspective) and torsional[b]	• Epley maneuver • Alternative: Semont maneuver
hc-BPPV (15%-20%) (also called lateral canal BPPV)			
Canalolithiasis (majority of horizontal canal cases)	Supine head roll	• Geotropic (beats towards the floor) horizontal that is transient[c] • Occurs on both sides, but is more intense on the affected side	• Lempert log roll ("barbecue") maneuver • Alternative: Gufoni maneuver
Cupulolithiasis (minority of horizontal canal cases)	Supine head roll	• Apogeotropic (beats toward the ceiling) horizontal, which is persistent • Occurs on both sides, but is more intense on the healthy, unaffected side	• Gufoni maneuver
ac-BPPV (~1%-2%) (also called superior canal BPPV)	Dix-Hallpike	• Down-beating vertical nystagmus[d]	• Can use Epley maneuver, but this form of BPPV usually resolves spontaneously

[a]Although the Dix-Hallpike 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 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.

[b]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.

[c]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 cause 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.

[d]Downward-beating nystagmus can be observed with ac-BPPV. However, because ac-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.

Abbreviations: ac, anterior canal; BPPV, benign paroxysmal positional vertigo; hc, horizontal canal; pc, posterior canal.

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fixation by the patient.

Some patients with hc-BPPV will have spontaneous (or more persistent) nystagmus that is normally not seen with BPPV.[71,72] This occurs because, depending on the orientation of the patient's head, otoliths in the horizontal canal may be moving in a patient sitting up and looking forward.

Finally, very rarely, patients with CPPV caused by structural lesions adjacent to the fourth ventricle (usually a tumor, multiple sclerosis plaque, or small brainstem stroke) will exhibit nystagmus or other features that are atypical for BPPV.[33,73] (**See Table 5.**) These patients will often have some symptoms (such as headache) that patients with BPPV never have, or they do not respond as expected to a repositioning maneuver. They may have physical findings that localize to the brainstem or cerebellum that patients with BPPV do not have, or they may exhibit nystagmus in the absence of movement or dizziness.

Diagnostic Studies

Diagnostic studies for patients who are suspected of having some general medical condition causing their dizziness (toxic, metabolic, or infectious) should be ordered based on the suspected diagnosis; ie, blood glucose for suspected hypoglycemia, stool guaiac and hematocrit for suspected gastrointestinal bleeding, etc. In patients with the AVS, the physical examination should reliably distinguish between stroke (or other central causes) and vestibular neuritis or labyrinthitis.

A key point is that not only is imaging not required but, in fact, has serious limitations in patients with a posterior circulation stroke. The "reassurance" that a negative CT scan in a dizzy patient excludes a central cause is false reassurance.[49] CT scanning is notoriously unreliable for any acute ischemic stroke and is even worse in the posterior circulation. A negative CT scan should never be relied on, by itself, to exclude posterior circulation ischemic stroke, especially patients presenting with an AVS.[74-79] In 2 large series of consecutive ED patients with dizziness who had CT performed, 0/344 (0%) and 7/448 (1.6%) had emergent findings on CT relevant to the dizziness.[78,79] Furthermore, intracranial hemorrhage rarely presents as an isolated AVS. In a series of 595 cases of intracranial hemorrhage, the only patient who had an isolated AVS also had cerebellar dysmetria and rotatory nystagmus on examination.[80] Conversely, in the series of 448 patients presenting with dizziness, only 2 (0.5%) had an intracranial hemorrhage. However, if associated headache is prominent or there are findings that suggest CPPV, CT may be justified.

Importantly, early MRI, and even diffusion-weighted imaging (DWI)-MR, can miss stroke in these patients if it is performed in the first 48 hours. In a meta-analysis of 3236 patients with acute ischemic stroke, nearly 7% had a negative DWI-MR, and this was strongly associated with a posterior circulation location.[81] The proportion of falsely negative DWI-MR (when performed in the first 72 hours in patients with the AVS) ranges from 12% to 18%,[51,52,82,83] and approaches 50% in small strokes (< 10 mm in axial diameter).[52] Another large study found that number was 4%, but it is not clear what percentage of the patients in the whole group had transient versus persistent symptoms.[84] Importantly, in all of these series, large-vessel disease was common. MRI with DWI-MR performed after 72 hours should reliably diagnose stroke.

In patients with s-EVS, there is no specific test that distinguishes vestibular migraine from posterior circulation TIA, and decision-making must be individualized based on history, epidemiology, and context. In patients with t-EVS who likely have BPPV, no diagnostic testing beyond physical examination is needed. A therapeutic canalith repositioning maneuver might be considered a diagnostic test. (**See Table 4, page 14.**)

Table 5. Characteristics of Patients With Triggered Episodic Vestibular Syndrome That Suggest a Central Mimic (CPPV) Rather Than Typical BPPV[10]

1. Presence of symptoms or signs that are not seen in BPPV:
   - Headache
   - Diplopia
   - Abnormal cranial nerve or cerebellar function
2. Atypical nystagmus characteristics or symptoms during positional tests:
   - Down-beating nystagmus[a]
   - Nystagmus that starts instantaneously, persists for longer than 90 seconds, or lacks a crescendo-decrescendo pattern of intensity
   - Prominent nystagmus with mild or no associated dizziness or vertigo
3. Poor response to therapeutic maneuvers:
   - Repetitive vomiting during positional maneuvers
   - Unable to cure patient with canal-specific canalith repositioning maneuver[b]
   - Frequent recurrent symptoms

[a]Down-beating nystagmus can be seen with anterior canal BPPV. However, because BPPV of this canal is rare and because down-beating nystagmus is most often the result of central structural lesions, it is safer for emergency clinicians to consider this finding to be *always* worrisome, prompting imaging and/or specialty consultation or referral.

[b]Modified Epley maneuver or equivalent for posterior canal BPPV; Lempert ("barbecue") maneuver or equivalent for horizontal canal BPPV.

Abbreviations: BPPV, benign paroxysmal positional vertigo; CPPV, central paroxysmal positional vertigo.

Treatment

Treatment of patients with general medical causes of dizziness or orthostatic hypotension will depend on the specific diagnosis. In patients with an AVS due to stroke, treatment depends on the mechanism (small-vessel disease versus large-vessel occlusion versus dissection). Some patients with severe gait instability may be candidates for intravenous thrombolysis even if their NIHSS score is low. These decisions have to be made on a case-by-case basis. Patients with vestibular neuritis are treated with corticosteroids.[3] This is typically a 10-day taper that starts at 60 mg oral prednisone per day.

In patients with s-EVS, treat posterior circulation TIA just as you would a patient with an anterior circulation TIA; ie, by administering antiplatelet treatment. One expert source recommends simple aspirin (barring a contraindication) for patients with an $ABCD^2$ score of < 4, and dual antiplatelet therapy with both aspirin and clopidogrel if the $ABCD^2$ score is ≥ 4.[85] (An $ABCD^2$ score calculator is available at: www.mdcalc.com/abcd2-score-tia) Treat patients with a cardioembolic cause with full anticoagulation. Vestibular migraine is treated as with any patient with migraine.[58-60] All of these patients should have neurology follow-up.

Strong evidence supports treating BPPV with a canalith repositioning maneuver, not with meclizine.[7] Data show that emergency clinicians can effectively use these maneuvers, but often do not.[86,87] Testing beyond physical examination is also frequently done, though this is almost never productive.[86-89] Emergency clinicians should think of BPPV as they do nursemaid's elbow—a diagnosis suspected by history and confirmed and treated with a bedside maneuver, without imaging. Some of the most common maneuvers for canalith repositioning are included in the "Helpful Links" section on page 19.

Finally, an acutely dizzy patient may also be dehydrated due to either vomiting, decreased oral intake, or both, so hydration with fluids is an important adjunct to ensure environmental safety.

Risk Management Pitfalls for Dizziness in the Emergency Department

(Continued on page 17)

1. **"I thought that because the dizziness got worse with head movement, it had to be peripheral."**
   This is a common misconception. Dizziness at rest in a patient with a cerebellar stroke or tumor often intensifies with head motion. It is crucial to distinguish dizziness that is triggered by movement (no dizziness at rest, but dizziness develops with movement) versus dizziness that is exacerbated by movement (dizziness is present at rest, but worsens with head movement).

2. **"The negative CT ruled out a stroke!"**
   Brain CT is a great test for hemorrhage, but a terrible test for posterior circulation infarction. Hemorrhage is a very uncommon cause of isolated dizziness without other symptoms or signs. One should never rely on a negative head CT to exclude a cerebellar or brainstem infarct.

3. **"I ruled out a posterior circulation TIA because isolated dizziness is never due to ischemia; other brainstem findings will always be present."**
   This is a misconception that stems from old expert opinion dating back to the mid-1970s. Newer studies make it clear that isolated dizziness is the most common transient symptom that precedes posterior circulation stroke and occurs in approximately 8% of these patients.

4. **"The patient had acute dizziness for 24 hours. The neuroradiologist read the MRI with diffusion-weighted images as negative, so there is no way this is a stroke."**
   It is important to emphasize that in the first 48 to 72 hours after a posterior circulation ischemic stroke that presents with isolated dizziness, diffusion-weighted MRI will miss as many as 1 in 5 patients. There is a reluctance by physicians, in general, to accept that an MRI can be negative in any acute stroke, but the data are clear.

5. **"The patient had a bad headache and said he had some transient double vision, but the Dix-Hallpike test was positive on both sides. I gave him meclizine for his BPPV."**
   There are some symptoms that never occur with BPPV—including headache and double vision. One can never make a diagnosis of BPPV in a patient with severe headache or diplopia (even if transient). As well, the treatment for BPPV is a canalith repositioning maneuver such as the Epley maneuver, not meclizine.

Special Populations

Children can have any of the same conditions as adults, although all of them are far less common in the pediatric population. The literature base for children is thin, but there is no reason to think that the physiologic underpinnings for diagnosing the vestibular diseases or central nervous system processes would be any different than in adults. The same is true for pregnant women.

The only significant special population is the geriatric group. The important difference in this group is that elderly patients with BPPV less commonly report vertigo and more commonly report nonspecific lightheadedness or dizziness than younger patients do.[2,19]

Controversies and Cutting Edge

Use of the HINTS examination by emergency clinicians in routine practice has never been validated. Learning and performing the HINTS examination may take some time and commitment from the emergency clinician. If emergency clinicians who use HINTS encounter ambiguous findings, one should probably err on the side of patient safety (over-calling something central rather than peripheral).

Investigators are studying the use of a portable goggle device with an embedded infrared device to record the eye movements when the patient is being taken through each of the component movements of the HINTS examination. Proof of concept exists with results that come out as worrisome for a central event or reassuring for a peripheral event.[90] Studies are currently ongoing to investigate this approach. Another way to implement the same concept; ie, "exporting" specialist expertise to nonspecialist clinicians is with telemedicine. The AVERT (Acute Video-Oculography for Vertigo in Emergency Room for Rapid Triage) study is currently enrolling patients to test this hypothesis. Details are available at: https://clinicaltrials.gov/ct2/show/NCT02483429

Another recent study of 86 patients presenting with s-EVS showed that perfusion-weighted MRI can help make a stroke diagnosis.[91] Inclusion criteria were rapid onset of vertigo/dizziness, resolution

Risk Management Pitfalls for Dizziness in the Emergency Department

(Continued from page 16)

6. **"The patient had both hearing loss and dizziness, so it has to be a peripheral problem – right?"**
   The classic teaching that coexistence of an acute hearing loss plus dizziness is always a peripheral lesion is wrong. A stroke of the AICA territory or of the labyrinthine artery can cause a stroke of the lateral pons or of the vestibular labyrinth and cause both hearing and balance findings.

7. **"The patient was only 32 years old with no vascular risk factors, so there's no way that this is a stroke."**
   Young patients have strokes! Mechanisms that are more common in young stroke patients include arterial dissection and cardioembolism (especially through a patent foramen ovale), but they also can have large-vessel disease. Young age is associated with stroke misdiagnosis.

8. **"The patient felt very dizzy on gait examination, but had no nystagmus at all, so I ruled out cerebellar stroke."**
   Only about half of patients with cerebellar strokes have nystagmus, so its absence in no way rules it out. In fact, the absence of nystagmus makes acute vestibular neuritis or labyrinthitis extremely unlikely and probably increases the probability of stroke in a patient with an AVS without nystagmus.

9. **"I know HINTS testing and the HIT was unequivocally positive. I saw a corrective saccade, so the problem must be vestibular neuritis."**
   No single component of the HINTS testing rules out stroke. Although the HIT is the most sensitive of all of the components of HINTS, it still only has a sensitivity of about 85%. It is also important to recognize that this sensitivity was done in studies by neuro-otologists, and the sensitivity in routine emergency medicine practice is not known.

10. **"The HINTS testing was worrisome, but the neurologist said to do a MRI and if it was negative, to discharge the patient and he would see him on Monday."**
    Neurologists are the usual consultant for an acutely dizzy patient, but many neurologists still use the outmoded "symptom-quality" approach to dizziness and some are unfamiliar with newer data about the HINTS testing being more sensitive than early MRI. If the physical examination suggests a central cause, it trumps a "negative" MRI.

of symptoms within 24 hours, and no previous history of recurrent vertigo. Of the 86 patients, 23 were still symptomatic at the time of presentation, so the HINTS testing was done and meaningful. Of the 63 asymptomatic patients, 32 had strokes, of which DWI-MR was negative in half. In 9 of these 63 patients (14%), perfusion-weighted MRI showed cerebellar hypoperfusion as a cause of the event. The duration of the dizziness in these patients ranged from several minutes (50%) to several hours (50%).

Disposition

The disposition of patients with the AVS depends on 2 factors. First is a high risk of fall, even if the patient has a benign cause of dizziness that normally would result in discharge. A component of this risk is due to dehydration that results from nausea and decreased oral intake and/or vomiting. This is an especially important issue for elderly patients and for those who live alone.

The second factor is the underlying diagnosis. Patients with general medical causes that require admission should be admitted, whereas if the underlying diagnosis is a condition that can be dealt with as an outpatient, this should be done. Patients with an acute stroke should be admitted for investigations of the underlying vascular lesion and treatment of the stroke. The disposition of patients with TIA depends on the resources in the ED. The disposition depends more on how quickly the vascular workup can occur and less on the specific location of where those tests are done (ie, possibly an observation unit).[92]

Summary

Using an algorithmic approach to the acutely dizzy patient, emergency clinicians can often confidently make a specific diagnosis that leads to correct treatment, thus reducing the misdiagnosis of cerebrovascular events. Emergency clinicians should try to become familiar with an approach that exploits timing and triggers as well as some basic rules about nystagmus. The gait should always be tested for all patients who might be discharged. CT scans are extremely unreliable to exclude posterior circulation stroke presenting as dizziness. Importantly, early MRI (within the first 72 hours), even with DWI, will miss 10% to 20% of these cases as well.

Case Conclusions

You are NOT OK with the plan for discharge if the man's CT is normal. His CT was normal, but sensitivity of noncontrast head CT in early posterior circulation stroke is very low and a negative CT should never reassure physicians that they have ruled out ischemic stroke. The absence of a report of "vertigo" is diagnostically meaningless. Although his nystagmus is consistent with a peripheral problem, it is also consistent with a central problem, so completing the bedside examination for a patient with an AVS is important. Calling the HIT "normal" is also problematic. "Normal" means the absence of a corrective saccade, which in the setting of the AVS is worrisome for stroke. Better terminology would be that HIT is "worrisome" or "reassuring," and better yet, "absence or presence of a corrective saccade." Since physical examination is more sensitive than even early MRI for posterior circulation stroke presenting as isolated dizziness, this patient was admitted for a stroke workup.

For the 70-year-old woman with lightheadedness, there were elements of her history that suggested BPPV. First, the dizziness was intermittent. Second, intermittent dizziness that awakens patients from sleep is nearly always BPPV. The absence of nystagmus and symptoms at rest were also very consistent with BPPV. Older patients with BPPV often do not report vertigo. The best test is a Dix-Hallpike test to look for pc-BPPV, and if that is negative, a supine head roll to test for hc-BPPV. In her case, the Dix-Hallpike was positive and you successfully treated her with an Epley maneuver, thus avoiding the need for imaging, blood tests, and neurology consultation.

For the diabetic man with severe vertigo who was triaged as syncopal, the most sensitive way to exclude stroke in the ED is physical examination. Going through the 5 questions for patients with an AVS—nystagmus, skew deviation, the HIT, presence of other findings on neurological exam, and gait testing—will make this determination with a high level of confidence, assuming the clinician is comfortable with these components of the examination. In this patient, the results of these 5 components were all reassuring. You diagnosed vestibular neuritis, started prednisone, and discharged him with neurology follow-up. The important thing is to know that brain imaging is not the best way to exclude stroke in an early-presenting patient with an AVS.

Time- and Cost-Effective Strategies

- In patients with the AVS with nystagmus, use the physical examination to identify patients with a peripheral cause (usually vestibular neuritis or labyrinthitis) who can be discharged without the need for time-consuming consultation or brain imaging.
- Understand that transient nontriggered dizziness may be caused by posterior circulation TIA and that a TIA evaluation may prevent a stroke in these patients. ***Risk Management Caveat:*** Traditionally, isolated dizziness was thought not to be due to a TIA, but newer data show that this is incorrect. Early evaluation, diagnosis, and treatment of TIA (identifying large-vessel disease and cardioembolic sources) can reduce the risk of future disabling stroke by as much as 80%.
- Knowing how to diagnose and treat BPPV is

not only great medical care, but it saves time, money, and leads to greater patient and physician satisfaction. ***Risk Management Caveat:*** BPPV is the most common vestibular problem that physicians encounter. Learning how to identify and treat this condition is not only very satisfying, but saves a lot of time and money, obviating the need for expensive consultation and imaging. Care must be taken to be sure that the patient meets the diagnostic criteria for BPPV. Ideally, these patients can be treated in the ED not only to improve their symptoms but also to reduce complications, such as falls.

Helpful Links

The following are links to videos of some of the various tests and maneuvers for diagnosing and treating dizziness:

Dix-Hallpike Maneuver:
- www.youtube.com/watch?v=wgWOmuB1VFY
- www.youtube.com/watch?v=J3S4Dh8BU10

Epley Maneuver:
- www.youtube.com/watch?v=9SLm76jQg3g
- www.youtube.com/watch?v=J3S4Dh8BU10

Lempert ("barbecue") maneuver:
- www.youtube.com/watch?v=mwTmM6uF5yA

Foster half-somersault maneuver for pc-BPPV:
- www.youtube.com/watch?v=Wez9SZJ7ABs

Semont (liberatory) maneuver:
- www.youtube.com/watch?v=A72UjulJSzE
- www.youtube.com/watch?v=pK9qaprUU64

Gufoni maneuver:
- www.youtube.com/watch?v=DgKaWSuvpRs
- www.youtube.com/watch?v=5FfzQQ5d060

Key Points

- Use a timing-and-triggers (rather than a symptom-quality) approach to the diagnosis of patients with dizziness.
- Each acute timing-and-triggers category (AVS, s-EVS, and t-EVS) is tightly associated with a specific differential diagnosis.
- Approximately 95% of patients with the AVS have either neuritis (vestibular neuritis or labyrinthitis) or posterior circulation stroke.
- In patients with the AVS presenting in the first 48 hours, bedside examination can help to distinguish between neuritis and stroke with greater sensitivity than MRI.
- The major differential diagnosis of patients with s-EVS is vestibular migraine and posterior circulation TIA. Because these patients are asymptomatic and the dizziness cannot be triggered, physical examination is not helpful.
- Use physical examination to diagnose patients with t-EVS.
- Because BPPV is so common and easily treatable, learn how to diagnose this condition by physical examination and treat with a bedside repositioning maneuver.
- CT scan is a poor test to exclude posterior circulation stroke acutely and should never be relied upon in this setting.
- Approximately 10% to 20% of patients with the AVS who present within 48 hours of onset will have a falsely negative MRI, even with diffusion-weighted imaging.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study is included in bold type following the references, where available. The most informative references cited in this paper, as determined by the author, are noted by an asterisk (*) next to the number of the reference.

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CME Questions

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1. Approximately what percentage of patients with acute dizziness will change their "type" of dizziness when re-questioned an average of 6 minutes later?
a. < 5%
b. 10%-15%
c. 50%
d. > 80%

2. Patients with an acute onset of dizziness that is persistently present and present at the time of examination in the ED have which of the following vestibular syndromes?
a. Acute vestibular syndrome
b. Triggered episodic vestibular syndrome
c. Spontaneous episodic vestibular syndrome
d. Chronic vestibular syndrome

3. Patients who are asymptomatic at rest, but with brief episodes of dizziness (usually lasting less than a minute) that CAN be provoked at the bedside have which of the following vestibular syndromes?
a. Acute vestibular syndrome
b. Triggered episodic vestibular syndrome
c. Spontaneous episodic vestibular syndrome
d. Chronic vestibular syndrome

4. Patients who are asymptomatic at rest, but with episodes of dizziness that CANNOT be triggered at the beside have which of the following vestibular syndromes?
a. Acute vestibular syndrome
b. Triggered episodic vestibular syndrome
c. Spontaneous episodic vestibular syndrome
d. Chronic vestibular syndrome

5. In patients with the acute vestibular syndrome without nystagmus, the head impulse test has a sensitivity of approximately what percentage?
a. 10%
b. 30%-40%
c. Unknown sensitivity in patients without nystagmus
d. 80%-90%

6. In patients with the acute vestibular syndrome, nystagmus findings that suggest a central cause include all of the following EXCEPT:
a. Uni-directional horizontal nystagmus
b. Torsional nystagmus
c. Vertical nystagmus
d. Direction-changing (gaze-evoked) horizontal nystagmus

7. When performing the head impulse test, it is important to rapidly turn the patient's head approximately how many degrees in order to maximize safety?
a. 15°
b. 30°-40°
c. 45°
d. 60°

8. **In a patient with the acute vestibular syndrome, the presence of anisocoria strongly suggests which of the following diagnoses?**
   a. Cerebellar infarct
   b. Cerebellar hemorrhage
   c. Lateral medullary infarct
   d. Midbrain infarct

9. **In a patient with possible BPPV, the most likely physical examination maneuver to help confirm the diagnosis is:**
   a. Head impulse test
   b. Skew deviation
   c. Finger-to-nose testing
   d. Dix-Hallpike maneuver

10. **Which statement about brain imaging in patients with acute dizziness is TRUE?**
   a. In patients with BPPV, a brain MRI can help establish the diagnosis.
   b. In patients with posterior circulation stroke, the CT is diagnostic in > 75% of cases.
   c. In patients with posterior circulation stroke, early MRI with diffusion-weighted images will establish the diagnosis in nearly all patients.
   d. In patients with vestibular migraine, neither CT nor MRI is indicated.

In upcoming issues of
Emergency Medicine Practice....

- NSTEMI
- Diabetic Emergencies
- Acid-Base Abnormalities
- Gastroenteritis

CME Information

Date of Original Release: December 1, 2019. Date of most recent review: November 10, 2019. Termination date: December 1, 2022.

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Target Audience: This enduring material is designed for emergency medicine physicians, physician assistants, nurse practitioners, and residents.

Goals: Upon completion of this activity, you should be able to: (1) demonstrate medical decision-making based on the strongest clinical evidence; (2) cost-effectively diagnose and treat the most critical presentations; and (3) describe the most common medicolegal pitfalls for each topic covered.

Objectives: Upon completion of this article, you should be able to: (1) explain the timing-and-triggers approach to the diagnosis of patients with dizziness; (2) describe the differential diagnosis of each of the vestibular syndromes; (3) recognize a posterior circulation transient ischemic attack in patients presenting with transient episodes of dizziness; and (4) utilize bedside physical examination maneuvers to diagnose and treat acute dizziness, when appropriate.

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Commercial Support: This issue of *Emergency Medicine Practice* did not receive any commercial support.

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Emergency Medicine Practice (ISSN Print: 1524-1971, ISSN Online: 1559-3908, ACID-FREE) is published monthly (12 times per year) by EB Medicine (PO Box 1671, Williamsport, PA 17703).