

Diagnosing Patients With Acute-Onset Persistent Dizziness



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INTRODUCTION

Three percent of emergency department (ED) patients present with dizziness, vertigo, lightheadedness, or imbalance.¹ These words are not diagnostically meaningful.² Rather, it is the timing and the factors that trigger the dizziness that best inform the differential diagnosis.³ Asking a patient, “What do you mean by ‘dizzy’?” is less important than defining the rapidity of onset, the context, presence of associated symptoms, the intermittent or persistent nature of the dizziness, and triggers of intermittent symptoms.^{2,4-6}

Approximately 50% of dizzy patients have general medical conditions.¹ Associated symptoms (eg, gastrointestinal bleeding) or context (eg, new antihypertensive medications) usually suggest these diagnoses. Some patients endorse brief episodes of dizziness triggered by head or body movement, suggesting benign paroxysmal positional vertigo or orthostatic hypotension. Other patients have short spontaneous episodes of dizziness that are not triggered by anything consistent with posterior circulation transient ischemic attacks or vestibular migraine.

This article focuses on a different group of patients: those with the acute onset of severe persistent and continuous dizziness or vertigo often associated with nausea or vomiting and postural instability that remains present in the ED without an obvious medical cause. Classically, nystagmus is included as a component of the acute vestibular syndrome.⁷ However, not all patients with acute-onset severe persistent dizziness have nystagmus.

To be clear about this distinction, I use the terms “acute vestibular syndrome with nystagmus” and “acute vestibular syndrome without nystagmus.” The distinction is important because the diagnostic approach differs somewhat between these 2 groups. The word *vestibular* refers to the symptoms, not to any anatomic localization. The major differential

diagnosis is vestibular neuritis versus posterior circulation stroke. This article focuses on distinguishing these 2 entities in the ED and uses the term *dizziness* in the general sense.

RELEVANT ANATOMY

The inner ear (or labyrinth) contains the cochlea (end organ for hearing) and the vestibule and semicircular canals (end organs for balance), innervated by the eighth cranial nerve. This nerve has 2 components (cochlear and vestibular) that enter the skull through the internal auditory canal (Figure 1). The vestibular nerve connects to neurons in the brainstem, and then to other parts of the brain, especially the cerebellum.

The posterior inferior cerebellar artery (a branch of the vertebral artery) and the anterior inferior cerebellar artery (a branch of the basilar artery) supply most of this portion of the brainstem and cerebellum. A branch of the anterior inferior cerebellar artery, the labyrinthine artery supplies the end organs (Figure 2). Damage or ischemia to the labyrinth, vestibular nerve, or their central connections can lead to dizziness, deafness, or both.

DIFFERENTIAL DIAGNOSIS

Various general medical and neurologic conditions rarely cause an isolated acute vestibular syndrome. Context (eg, anticonvulsant toxicity) or associated symptoms and signs (eg, confusion in Wernicke’s syndrome) usually suggest the diagnosis.⁸ Some patients with Wernicke’s syndrome present with acute vestibular syndrome without encephalopathy.⁹ High-risk patients should be empirically treated with thiamine.⁸⁻¹⁰ A new multiple sclerosis diagnosis accounts for less than 2% of patients presenting with an acute vestibular syndrome.¹¹

Therefore, for emergency physicians, the crucial differential diagnosis is vestibular neuritis versus stroke. Vestibular neuritis is inflammation of the vestibular portion of the eighth nerve, similar in pathophysiology to Bell’s

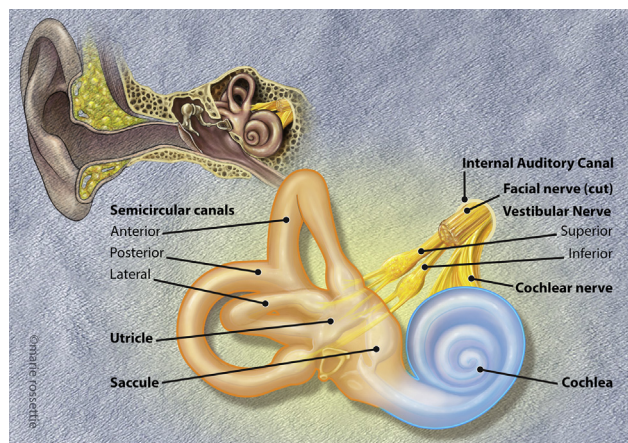


Figure 1. Inner ear anatomy. The structures that traverse the internal auditory canal are the eighth cranial nerve (which includes the cochlear nerve, as well as the superior and inferior vestibular nerves), the facial nerve, and the labyrinthine artery). The vestibular nerves supply the semicircular canals, utricle, and saccule (end organs of balance), and the cochlear nerve supplies the cochlea (end organ of hearing).

palsy.^{12,13} Labyrinthitis is inflammation of both the vestibular and cochlear components of the eighth nerve.

MISDIAGNOSIS

Misdiagnosis of posterior circulation stroke is a needle-and-haystack phenomenon. The haystack (patients with dizziness) is enormous; the needle (ED patients with isolated dizziness caused by a cerebrovascular cause) is tiny, 0.7% in one study of nearly 1,300 patients.¹⁴ The percentage of ED patients presenting with dizziness who

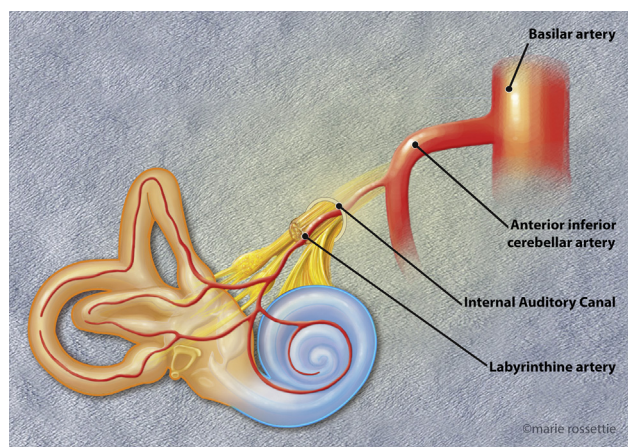
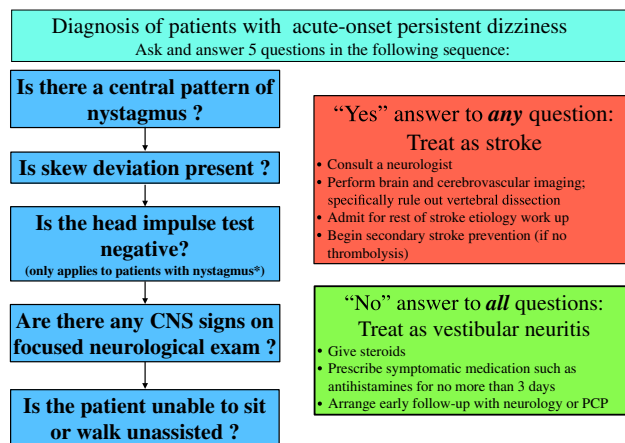


Figure 2. Vascular anatomy to the inner ear and its sources. The vascular supply to the labyrinthine structures, the labyrinthine artery (or internal auditory artery), supplies the cochlea and vestibular apparatus. It is a branch of the AICA, which branches from the midbasilar artery. The vessel exits the skull through the internal auditory canal. AICA, Anterior inferior cerebellar artery.



* In patients **without** nystagmus, the head impulse test may give misleading results; the focused neurological exam and gait assessment become more important in this group (see text)

Figure 3. Diagnostic algorithm for differentiating vestibular neuritis from stroke in patients with AVS. This sequence of testing is largely based on the HINTS model, but the sequence is different (see text for reasons) and there are 2 additional components: targeted neurologic examination that is specifically directed toward the cerebellum and brainstem, and testing for truncal and gait ataxia. I recommend these other components of the diagnostic algorithm because HINTS testing has not been validated when used by emergency physicians under routine circumstances. In one study, adding gait testing to the HINTS examination plus hearing yielded 100% sensitivity for stroke.⁴¹ Furthermore, testing gait in any dizzy patient is important to ensure a safe disposition, even if the cause is peripheral. CNS, Central nervous system; PCP, primary care physician; AVS, acute vestibular syndrome; HINTS, Head Impulse Test, Nystagmus, and Test of Skew.

are discharged with a peripheral vestibular diagnosis and then subsequently hospitalized with a stroke within 7 days is 0.14% to 0.50%, which is very low, but higher than in control nondizzy patients.¹⁵⁻¹⁸

Conversely, 28% to 59% of cerebellar strokes are misdiagnosed in the ED.¹⁹⁻²¹ Risk factors for misdiagnosis of stroke include young age, vertebral dissection as a cause, and a presentation of dizziness.²²⁻²⁴ Ten percent of patients (25/240 consecutive cases) with cerebellar stroke present with isolated dizziness.²⁵ Posterior circulation strokes are missed more than twice as often as anterior circulation strokes.²² Using the traditional “What do you mean by ‘dizzy’?” diagnostic approach rather than one that exploits timing and triggers may contribute to misdiagnosis.

Whatever the cause of misdiagnosis, the personal, economic, and public health effect is potentially large, with estimates of the absolute number of patients harmed ranging from 45,000 to 75,000 per year in the United States.²⁶ One small study reported poor outcomes in patients who received misdiagnoses.²⁷ Another larger retrospective study (N=47 missed strokes of 2,200 total) showed that misdiagnosis for patients with cerebellar

Table 1. Sensitivity of various components of the physical examination for central mechanism in patients with acute vestibular syndrome.

Component of Examination	Sensitivity for Central Cause*	Comments
Nystagmus, %	50–60 ^{28,41}	See Table 2.
Skew deviation, %	25 ³⁴	This finding is not very sensitive but is specific for a central cause, usually in the brainstem. See Table 2.
Head impulse test, % [†]	85–90 ^{28,41,51}	Extremely important to use this test only for patients with AVS with nystagmus. All other patients will have a “negative” test result, which is “worrisome” for a stroke (see text and Figure 3).
Focused neurologic examination, %	65 ^{28,41}	In addition to obvious neurologic findings, it is important to look for subtle findings that can be easily missed.
Gait or truncal ataxia, %	65 ⁴¹	This is an essential test for patients with dizziness. Some patients without the first 4 findings may be unable to sit up or stand and walk unaided. Apart from obvious disposition issues, many of these patients have a stroke that causes this finding.

*Approximate numbers based on pooled data from multiple studies in some cases.

[†]For AVS patients with nystagmus, the combined sensitivity of the first 3 elements (HINTS) approaches 100% when done by specialists.

strokes was 4 times higher than stroke misdiagnosis overall and that patients who received a misdiagnosis had greater disability and higher 12-month mortality.²⁸

Prospective data showing worse outcomes in dizzy ED patients who received a misdiagnosis and had posterior circulation strokes compared with patients receiving a correct diagnosis are lacking. Nonetheless, significant harm likely exists because untreated patients are at risk for edema, stroke extension, and second strokes because of nontreatment of the causative vascular pathology.²⁹ Further harm from not implementing evidence-based treatment for benign causes of dizziness also exists.

DISTINGUISHING VESTIBULAR NEURITIS FROM STROKE

No single element of the history distinguishes neuritis from stroke, but the presence of an abrupt-onset dizziness, headache or neck pain, vascular risk factors, and other neurologic symptoms such as diplopia suggest a stroke diagnosis.^{30–32} Although transient ischemic attacks precede some strokes, 25% of patients with vestibular neuritis also have transient episodes of dizziness within the previous week.³³ Traditionally, simultaneous dizziness and hearing loss has been ascribed to peripheral causes (labyrinthitis). However,

Table 2. Nystagmus and skew deviation interpretation in patients with acute vestibular syndrome.*

Finding	Significance	Comments
No nystagmus	Normal finding	Essentially rules out vestibular neuritis but is consistent with a cerebellar stroke. Rarely, patients with BPPV will endorse continuous dizziness and not have nystagmus at rest.
Spontaneous horizontal nystagmus in primary gaze	Does not distinguish between central and peripheral causes	Observed more commonly with peripheral causes of AVS, but is not diagnostic.
Gaze-evoked horizontal nystagmus that beats in only one direction	Does not distinguish between central and peripheral causes	Suggests a peripheral cause of AVS, but is not diagnostic. In neuritis, there is often a slight torsional component.
Direction-changing gaze evoked horizontal nystagmus (see explanation below)	Central	This is always central but can be a benign central cause (eg, acute alcohol intoxication, anticonvulsant use).
Pure vertical nystagmus	Central	In the ED, this should always be considered a central finding.
Torsional nystagmus	Central	Torsional nystagmus is the expected finding in posterior canal BPPV, but these patients do not present with AVS (see text) but rather a triggered episodic vestibular syndrome. There is often a slight torsional component in neuritis.
Skew deviation (see below)	Normally absent; its presence means a central cause	Not very sensitive, but if present, this should be considered to be a central cause of the AVS.

BPPV, Benign paroxysmal positional vertigo.

*(1) Nystagmus is nearly always present in vestibular neuritis but is observed in only 50% of cerebellar stroke patients and variably in other brainstem strokes. Therefore, it is the quality of the nystagmus that is diagnostically important, not the mere presence or absence. Absence of nystagmus essentially excludes a diagnosis of acute vestibular neuritis, or “labyrinthitis.” (2) To test for nystagmus, ask the patient simply to open his or her eyes and look directly forward to determine whether there is nystagmus in primary gaze. Then have the patient look to the right and then the left. If the fast component of the nystagmus changes direction (ie, is right beating on rightward gaze and left beating on leftward gaze), this is the central finding. In vestibular neuritis, the nystagmus is primarily horizontal, with a slight torsional component. (3) Test skew deviation by using the alternate cover test. With the patient's eyes focused on a target (the examiner's nose), the examiner alternately covers and then uncovers each eye, every 2 to 3 seconds. It is important to focus on just one eye (it does not matter which one) to see the small-amplitude vertical corrections that occur when one eye is uncovered (one eye will go up and the other down, so either one will have a vertical correction, which is why either eye can be observed).

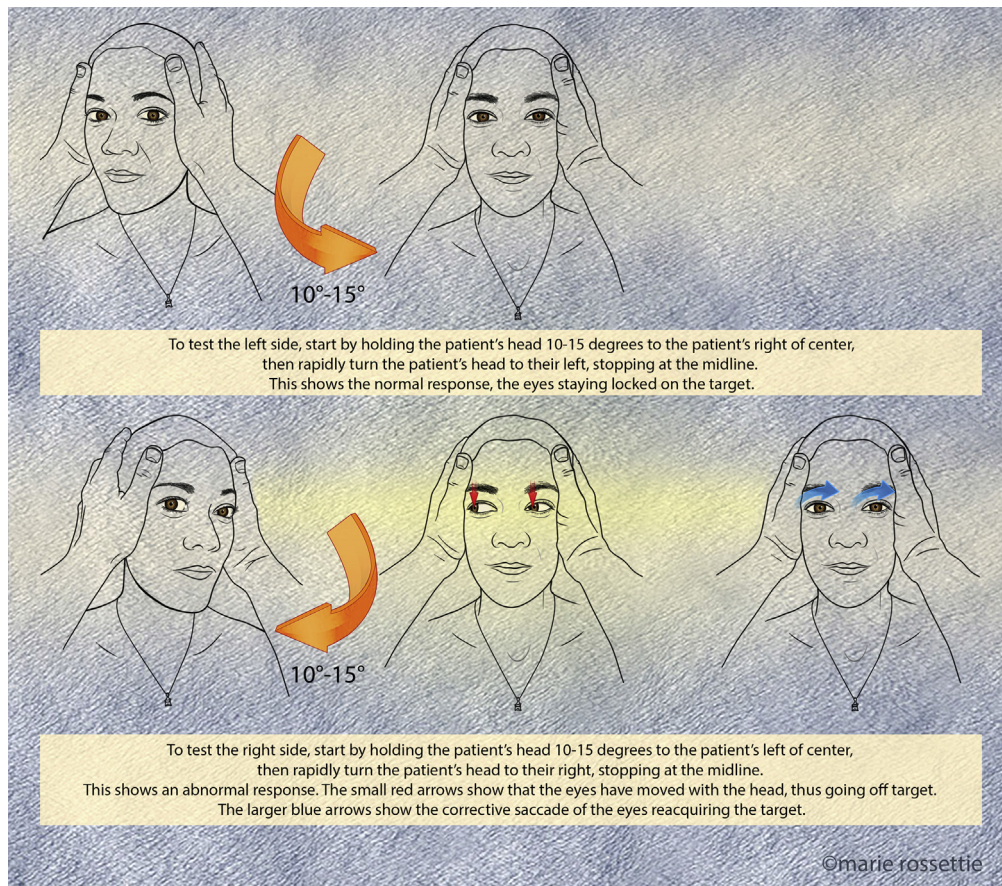


Figure 4. The head impulse test (HIT) result is positive (abnormal) in nearly all patients with a peripheral cause of AVS and negative (normal) in 85% to 90% of patients with a central cause of AVS. It cannot therefore be used as a stand-alone test. It has been validated only in patients with an AVS with nystagmus. The HIT tests the vestibulo-ocular reflex, which coordinates the eyes and stabilizes the image of a moving target. The afferent limb of the reflex loops from the peripheral vestibular labyrinth through the vestibular nerve to the vestibular nuclei in the brainstem, which in turn connects with the cranial nerve nuclei (3, 4, and 6) that move the eyes. The efferent limb for the horizontal HIT is the third and sixth cranial nerves that control horizontal eye movements. Because the reflex does not loop through the cerebellum, patients with cerebellar strokes have a normal HIT result. Occasionally, patients with strokes that affect the vestibular nucleus or the eighth nerve root entry zone in the brainstem have a positive HIT result. These strokes involve the AICA. The other less common explanation is that the stroke is of the labyrinth, a stroke involving the labyrinthine artery (a branch of the AICA). A key point is that the HIT should only be applied to patients with the AVS who have nystagmus; otherwise, it may give misleading results.

anterior inferior cerebellar artery territory strokes and labyrinthine infarcts are central processes that also affect both balance and hearing. The relative proportions of central versus peripheral causes of simultaneous dizziness and hearing loss are unknown.³¹

Fortunately, physical examination can accurately distinguish neuritis from stroke.³⁴ The presence or absence of nystagmus is key. In patients with nystagmus, 3 ocular motor tests (Head Impulse Test, Nystagmus, and Test of Skew, referred to as HINTS) can distinguish peripheral from central causes, at least when conducted by neuro-otologists.³⁵ Data suggest that stroke neurologists and specially trained emergency physicians using Frenzel lenses can achieve similar accuracy.^{36,37}

However, the elements of this examination are not traditionally taught to emergency physicians, and to my knowledge no published data exist about accuracy of emergency physicians performing HINTS under routine conditions. My own experience suggests that this is a fixable knowledge gap. With time and practice, physicians can learn to perform and interpret these tests. In acute vestibular syndrome patients without nystagmus, the head impulse test has not been validated. Therefore, in this group, other elements of the examination—focused neurologic examination and gait testing—are more important.

For a combination of all these reasons, I ask 5 sequential questions that incorporate HINTS but add 2 additional components in an attempt to maximize patient safety

Table 3. Important components of the focused examination for posterior circulation stroke in patients presenting with acute vestibular syndrome.

Examination Component	Significance	Comments
Hearing by finger rub in each ear	Can be central or peripheral	The classic teaching that dizziness plus decreased hearing is nearly always peripheral is wrong. Infarcts of the labyrinth or eighth nerve root entry zone (AICA distribution) will also cause this combination of findings.
Extraocular movements	If diplopia is present, this should be considered central.	This finding suggests that nuclei of 3 cranial nerves (3, 4, or 6) or their connections are involved, suggesting a brainstem localization.
Ptosis	Suggests a lateral medullary infarct	Part of Horner's syndrome
Anisocoria	Suggests a lateral medullary infarct	Best observed in a dark room to accentuate the difference in pupillary size. Part of Horner's syndrome.
Facial weakness	Suggests a lesion in the internal auditory canal or brainstem	Standard seventh nerve testing
Decreased facial pain and temperature sensation	Suggests a lateral medullary infarct	Light touch is preserved, so one must test pain or temperature.
Hoarseness (listening to the patient speak)	Suggests a lateral medullary infarct	Be careful about administering oral medications in this setting.
Limb ataxia (finger to nose and heel to shin)	Cerebellar stroke	In the dizzy patient, these findings should be tested but may be absent in some patients with cerebellar strokes.
Truncal ataxia	Cerebellar or brainstem stroke	Test the ability of the patient to maintain the seated position unassisted in the stretcher without holding onto the guard rails for support.
Gait ataxia	Cerebellar or brainstem stroke	Test the ability of the patient to stand and walk unassisted. Patients with neuritis may have some unsteadiness but usually can stand and walk, whereas many patients with stroke cannot.

(Figure 3). If the answer to any question is yes, the cause is presumed central and treated as stroke. If the answer to all 5 questions is no, the cause is presumed peripheral and treated as vestibular neuritis. All 5 answers must be no because individually, none of the 5 findings is sufficiently sensitive to exclude stroke (Table 1).

Although the HINTS acronym suggests otherwise, test nystagmus first³⁴ because HINTS has been validated only in patients with nystagmus and also because nystagmus is the least intrusive part of the examination for the patient, head movement being unnecessary.

First, is there a central pattern of nystagmus? Emergency physicians' understanding and documentation of nystagmus is incomplete.³⁸⁻⁴⁰ Its mere presence or absence is not as diagnostically useful as its characteristics. Understanding simple "rules" allows one to make rapid and confident diagnoses (Table 2). Start by observing the patient's eyes when he or she looks straight ahead and then have him or her look to the right, left, up, and down. Nystagmus is named for the direction of the fast phase. Nystagmus whose fast component changes direction with gaze (ie, it beats to the right on rightward gaze and to the left on leftward gaze) or nystagmus that is primarily torsional or vertical is central.³⁴

Second, is skew deviation present? One performs the alternate cover test (described in Table 2) and looks for small vertical corrections off the target by the uncovered eye.³⁴ This test is not sensitive but is very specific for central causes.

Third, is the head impulse test result negative? The test was described in 1988 and many physicians are unfamiliar with it.⁴¹ A key point is that a positive (abnormal) test result suggests a peripheral cause and a negative (normal) test result is worrisome for a central cause. If one were to perform a head impulse test on a patient with dizziness from sepsis or dehydration, the result would be negative (ie, worrisome for a stroke, which of course those patients are not having). For this reason, the head impulse test is meaningful only in acute vestibular syndrome patients with nystagmus. This is why questions 4 and 5 are important to help to distinguish peripheral from central causes of acute vestibular syndrome patients without nystagmus.

To conduct the head impulse test (Figure 4), stand in front of the patient, hold both sides of the head and loosen up the neck, having the patient relax, and fix his or her gaze on your nose.³⁴ Then very rapidly but very minimally turn the head to either side. A 10- to 15-degree arc is all that is required to elicit the sign. Test each side several times, changing directions randomly so that the patient cannot predict which side will be tested. The normal result is that the patient's eyes stay locked on the target. A positive result is that the patient's eyes move with the head and then snap back to target in a single corrective saccade usually visible to the naked eye.

In acute vestibular syndrome patients without nystagmus, 2 additional questions are important. First, are there any central nervous system signs on the focused neurologic examination? Beyond the basic neurologic examination, specifically test the cranial nerves for hearing,

anisocoria, phonation, and facial loss of pain or temperature sensation and cerebellar function for limb ataxia (Table 3).

Finally, is the patient unable to sit or walk unassisted? Although patients with vestibular neuritis may have difficulty walking, they usually can walk. Severe gait instability would be a reason to admit any patient for safety purposes, but in the setting of an acute vestibular syndrome, it strongly suggests a stroke cause.^{23,32,42}

IMAGING

Physical examination trumps imaging. Computed tomography (CT) has extremely poor sensitivity for ischemic posterior circulation stroke.⁴³⁻⁴⁷ Because brain hemorrhage is a rare cause of an isolated acute vestibular syndrome, without other worrisome symptoms or signs,⁴⁸ CT is an illogical test, despite physicians' beliefs otherwise.⁴⁹ In a retrospective cohort study, dizzy ED patients who had a negative CT result returned to the ED with a stroke within 30 days more than twice as often as patients who did not have a CT scan, suggesting that physician gestalt was good but the wrong test was used to exclude stroke.⁵⁰

Although magnetic resonance imaging (MRI) with diffusion-weighted imaging is markedly superior to CT, 3 studies have found that MRI–diffusion-weighted imaging conducted within 48 hours of the stroke is falsely negative in 12% to 18% of acute vestibular syndrome patients (the criterion standard being delayed MRI),^{35,51,52} and for small strokes, MRI is falsely negative in 53% of patients.⁵² A recent meta-analysis of negative diffusion-weighted imaging stroke results reported an overall rate of 6.8%, but posterior circulation strokes were 5 times more likely to be diffusion-weighted imaging negative compared with anterior circulation strokes.⁵³ Indiscriminate CT angiography also has very low utility (1.3%).⁵⁴

CAUTIONS

Ideally, one would not perform positional testing (eg, Dix-Hallpike's test) in patients with an acute vestibular syndrome, but occasionally patients with benign paroxysmal positional vertigo report persistent dizziness, superficially resembling an acute vestibular syndrome. They usually endorse severe worsening intermittently and do not have spontaneous nystagmus at rest. In this situation, positional testing to diagnose benign paroxysmal positional vertigo, if the result is positive, will reveal the true diagnosis and avoid a lengthy evaluation.⁵⁵

Some patients with vestibular neuritis have such prominent nystagmus that it makes the head impulse test interpretation difficult. Parenteral benzodiazepines often reduce symptoms and dampen the nystagmus within 30 to 60 minutes, facilitating head impulse test interpretation.

In the first 48 hours, physical examination outperforms MRI. If the physical examination suggests stroke, do not use a negative MRI result to exclude stroke.

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