Diagnosing Stroke in Acute Dizziness and Vertigo
Pitfalls and Pearls

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Public Health Concern

Dizziness and vertigo are responsible for an estimated 4.4 million emergency department (ED) visits in the United States each year and account for 4% of chief symptoms in the ED.[1] Strokes are the underlying cause of ≈3% to 5% of such visits (130 000–220 000).[2] These visits are associated with a high cost, estimated now to exceed $10 billion per year in the United States.[3] This results from neuroimaging obtained in roughly half the patients[1] and admissions for nearly 20%.[4] ED physicians worldwide rank vertigo a top priority for developing better diagnostic tools.[5] An evidence-based, cost-effective approach to diagnosing acute dizziness and vertigo is needed.

Diagnostic Errors

Improving diagnosis is recognized by the National Academy of Medicine as a public health priority.[6] Nearly 10% of strokes are misdiagnosed at first medical contact.[7] Of the ≈130 000 to 220 000 patients with stroke presenting with vertigo or dizziness to the ED, it is estimated that perhaps 45 000 to 75 000 are initially missed,[3] with misdiagnosis disproportionately affecting the young (age<50), women, and minorities.[8,9] A population-based cohort study found that patients discharged from the ED said to have benign dizziness are at 50-fold increased risk of a stroke hospitalization in the 7 days post-discharge relative to propensity score-matched controls.[10] Another population-based registry showed that 90% of isolated posterior circulation transient ischemic attacks (TIAs), half presenting isolated vertigo symptoms, were not recognized at first medical contact.[11] Overall, dizziness and vertigo are the symptoms most tightly linked to missed stroke.[7,9]

Given their low sensitivity (7%–16%),[12] computed tomography (CT) scans are of little use for identifying acute ischemic strokes,[13] particularly in the posterior fossa.[12] Despite this, nearly 50% of US ED patient presenting dizziness are imaged by CT, and <3% by magnetic resonance imaging (MRI).[1] Even MRI with diffusion-weighted imaging (MRI-DWI), misses ≈15% to 20%[12] of acute posterior fossa infarctions <24 to 48 hours from symptom onset.[12] One study suggests perhaps even a higher fraction (≈50%, n=8/15) are missed by MRI-DWI when these strokes are small (<1 cm in diameter)[14]; but small does not mean benign, because half of these patients had large-artery stenosis or dissection, increasing recurrent stroke risk.[15] Obtaining MRI for every patient presenting with ED dizziness in the United States would be time consuming and cost >$1 billion annually, making it unrealistic.[3]

Harms related to missed stroke accrue from missed opportunities for acute thrombolysis,[8] early secondary prevention,[16,17] and surgical treatment of stroke complications from malignant edema.[18] Small studies have suggested a high risk of permanent morbidity (33%, n=5/15) and mortality (40%, n=6/15) among those whose cerebellar strokes were initially missed,[19] much higher than what is reported in large cohorts with cerebellar infarction (5%, n=15/282).[20] Although these may be overestimates, it is clear that delayed stroke diagnosis increases morbidity and mortality substantially.[7] One estimate suggests that with strokes presenting dizziness and vertigo, 15 000 to 25 000[3] suffer serious and potentially preventable harms from the initial misdiagnosis.[3]

These numbers indicate that our current diagnostic practices in the ED are largely ineffective[21] and there is substantial room for improvement.[22,23] In addition to problems with stroke, many acutely dizzy patients with peripheral vestibular causes for their symptoms are overtested, misdiagnosed, and undertreated.[21] The costs of unnecessary imaging and hospital admissions for these patients waste an estimated $1 billion each year.[3] Thus, accurate and efficient diagnosis for these patients will likely save lives and reduce costs through prompt and appropriate treatments.[4]

Causes of Diagnostic Errors

When evaluating dizziness, physicians seek to differentiate inner ear conditions from stroke or other central causes.[24]

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Vestibular strokes are often missed because clinical findings mimic benign ear disorders. Because ≥95% of ED patients with dizziness do not have stroke, detecting these cerebrovascular cases presents an enormous challenge. Neuroimaging seems like a natural solution, but, as noted above, CT is ineffective and MRI is imperfect and too costly to apply to all ED dizziness. This means that accurate bedside diagnosis is at a premium.

The traditional approach to evaluating these patients places heavy emphasis on defining the type of dizziness[25]—vertigo (vestibular) versus presyncope (cardiovascular) versus disequilibrium (neurological) versus nonspecific (psychiatric/metabolic).[26] This approach, however, has been largely debunked.[21] A recent study by Kerber et al[27] adds further confirmation against the type approach using data from a large national survey. On average, people with likely peripheral vestibular disorders complained of 3 different types of dizziness, and experienced vertigo as the primary type in just 25%.[27] Frequent use of this faulty diagnostic paradigm[26] combined with inadequate knowledge of bedside eye movement-based diagnosis, overreliance on vascular risk factors, and false reassurance by negative CT imaging to rule out stroke[28] likely explains why missed strokes are more common than one would hope.[21]

In this article, we describe an approach to bedside diagnosis derived from current best evidence that relies first on classifying acute vertigo or dizziness by timing (episodic versus continuous) and trigger (positional versus not), rather than type, and then uses that classification to hone in on specific, targeted bedside eye movement examinations to differentiate peripheral versus central causes.[2]

Approach to Cerebrovascular Diagnosis

Symptom Definitions

Expert international consensus definitions for vestibular symptoms have been developed as part of the International Classification of Vestibular Disorders.[29] These define dizziness as the sensation of disturbed or impaired spatial orientation without a false or distorted sense of motion and vertigo as the sensation of self-motion when no self-motion is occurring or the sensation of distorted self-motion during an otherwise normal head movement.[29] In this article, we use these terms mostly interchangeably, because there is little diagnostic value in distinguishing between them.[21]

Timing, Triggers, and Targeted Examination

The TiTrATE acronym stands for timing, triggers, and targeted examinations.[2] Timing refers to key aspects of onset, duration, and evolution of the dizziness. Triggers refer to actions, movements, or situations that provoke the onset of dizziness in patients who have intermittent symptoms. In the acute setting, a history based on timing and triggers in dizziness results in 4 possible syndromes in patients presenting in the ED with recent intermittent or continuous dizziness: triggered episodic vestibular syndrome (t-EVS), spontaneous EVS (s-EVS), traumatic/toxic acute vestibular syndrome (t-AVS), and spontaneous AVS (s-AVS).[2] Most TIAs present with s-EVS and most strokes or hemorrhages present with s-AVS but there are exceptions (Table 1). To get to the correct cerebrovascular diagnosis, therefore, it is necessary for the practitioner to understand all 4 syndromes (Figure 1).

Episodic Vestibular Syndrome

The EVS is defined as a clinical syndrome of transient vertigo, dizziness, or unsteadiness lasting seconds to hours, occasionally days, and generally including features suggestive of temporary, short-lived vestibular system dysfunction (eg, nausea, nystagmus, and sudden falls).[34] There are triggered and spontaneous forms.

Triggered EVS

Attacks usually last seconds to minutes. The most common triggers are head motion or change in body position (eg, arising from a seated or lying position). Clinicians must distinguish triggers from exacerbating features, because movement of the head typically exacerbates any dizziness of vestibular cause whether benign versus dangerous, central versus peripheral, or acute versus chronic.

In the ED, benign paroxysmal positional vertigo (BPPV) is likely the second most common cause of t-EVS after orthostatic hypotension, accounting for ≈5% to 10%[35] of acute dizziness cases.[36] The diagnosis is confirmed by canal-specific positional testing maneuvers and identifying a canal-specific nystagmus.[37] BPPV of the posterior canal is the most common

Table 1. Cerebrovascular Causes Linked to 4 Acute Dizziness/Vertigo Syndromes

Syndrome	TIA	Ischemic Stroke	Hemorrhage
t-EVS (brief, repetitive)	Rotational vertebral artery syndrome[30]	CPPV from small ischemic strokes near the fourth ventricle[31]	CPPV from small hemorrhages near the fourth ventricle[32]
s-EVS (<24 h)*	PICA–isolated vertigo; AICA–vertigo±tinnitus or hearing loss*	Small ischemic strokes presenting transient symptoms[49]	Subarachnoid hemorrhages mimicking TIA[33]
t-AVS (>24 h)	Overlap syndrome with trauma and vertebral artery dissection/TIA	Overlap syndrome with trauma and vertebral artery dissection/stroke	Overlap syndrome with trauma and traumatic hemorrhage (subdural, subarachnoid, etc)
s-AVS (>24 h)*	Not yet reported (would be difficult to differentiate from vestibular migraine)	PICA–isolated vertigo; AICA–vertigo±tinnitus or hearing loss*	Small to medium-sized cerebellar hemorrhages

AICA indicates anterior inferior cerebellar artery; PICA, posterior inferior cerebellar artery; s-AVS, spontaneous acute vestibular syndrome; s-EVS, spontaneous episodic vestibular syndrome; t-AVS, traumatic/toxic acute vestibular syndrome; and t-EVS, triggered episodic vestibular syndrome.

*Common cerebrovascular presentations; the other combinations are rare.

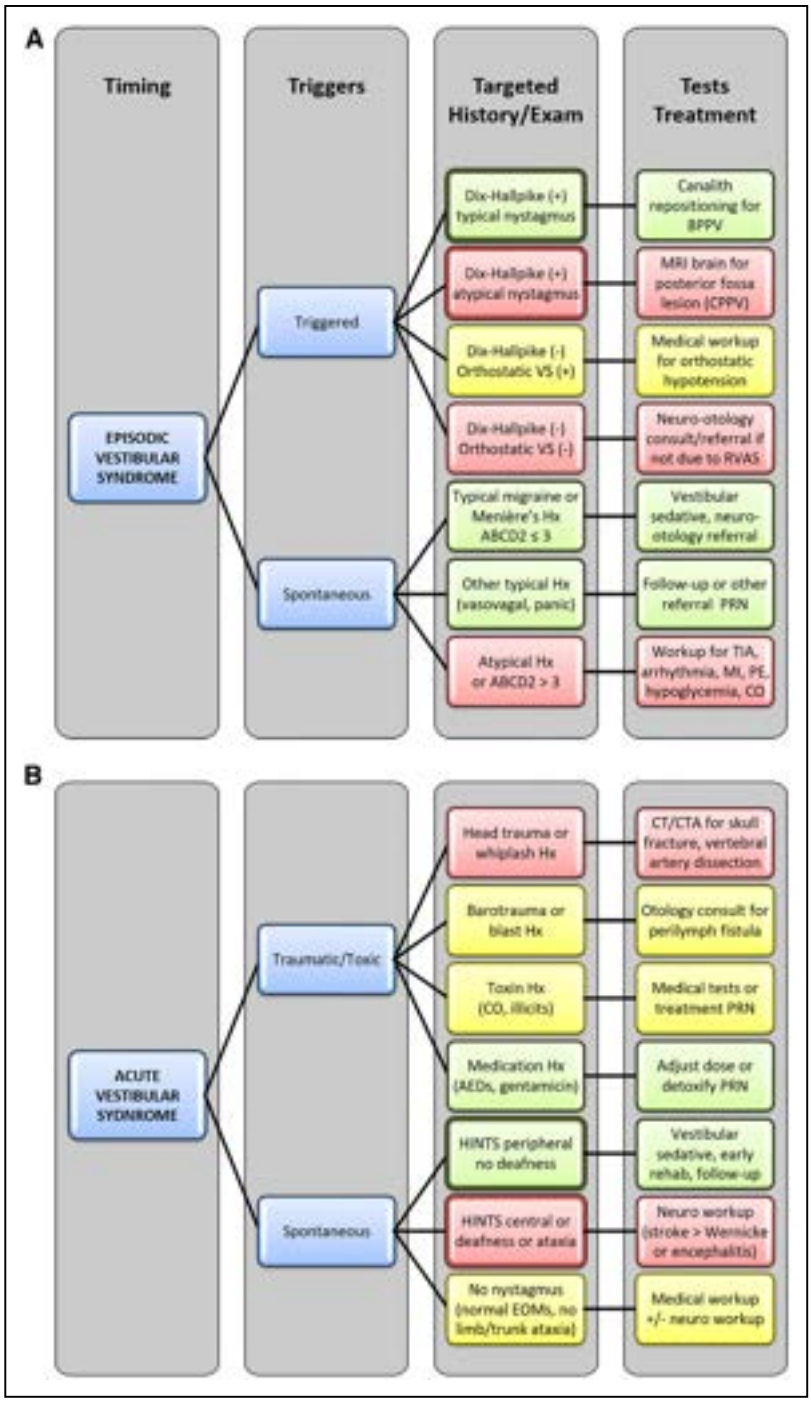

Figure 1. Timing, triggers, and target examination (TiTrATE) approach to diagnosing dizziness and vertigo, highlighting cerebrovascular causes (red). Adapted from Newman-Toker et al[2] with permission. Copyright ©2017, Elsevier. AED indicates antiepileptic drug; CO, carbon monoxide; EOM, extraocular movement; Hx, history; MI, myocardial infarction; PE, pulmonary embolus; PRN, pro re nata (as needed); and VS, vital signs.

and produces a transient, crescendo–decrescendo upbeat-torsional nystagmus that lasts less than a minute. The second most common is BPPV affecting the horizontal canal, which produces a transient, usually brisk horizontal nystagmus that lasts <90 seconds. Patients with atypical nystagmus forms (eg, persistent positional downbeat or horizontal nystagmus; no latency between the head reaching the target position during positional testing and nystagmus onset) may have mimics known as central paroxysmal positional vertigo. Central paroxysmal positional vertigo may result from benign central causes, such as alcohol intoxication or vestibular migraine, but other cases are caused by posterior fossa structural lesions.[38,39]

Although t-EVS is only uncommonly because of cerebrovascular disease, there are 2 distinct forms. Small strokes or hemorrhages near the fourth ventricle sometimes cause central paroxysmal positional vertigo.[31,32,39] The nystagmus is usually horizontal, so differs from the most common (posterior canal) form of BPPV. The other triggered cerebrovascular syndrome is the rare condition known as rotational vertebral artery syndrome.[30,40] Here, far lateral rotation of the neck leads to mechanical occlusion of 1 or both vertebral arteries,[40] causing temporary symptoms of vertigo and nystagmus when the offending position is maintained.[30] This occurs even with the patient upright, so is rarely confused for central paroxysmal positional vertigo.

Spontaneous EVS

The majority of spells in this category last minutes to hours. Although there may be predisposing factors such as dehydration, sleeplessness, or specific foods, there are no obligate, immediate triggers. Because episodes are usually resolved at the time of assessment, examination is often normal and evaluation relies almost entirely on history taking. If symptoms are still present when the patient is being assessed, eye examination techniques for s-AVS, described below, can distinguish central from peripheral forms.[41]

Menière disease is sometimes considered the prototype cause of s-EVS but vestibular migraine is the most common. Other benign causes include vasovagal presyncope and panic attacks. Principal dangerous causes are cerebrovascular (vertebrobasilar TIA), cardiorespiratory (especially cardiac arrhythmia), and endocrine (especially hypoglycemia). Cardiac arrhythmias should be considered in any patient with s-EVS, even if the presenting symptom is true spinning vertigo.[42,43] On rare occasions, subarachnoid hemorrhage may present as s-EVS.[33]

Multiple studies show that dizziness and vertigo, even when isolated, are the most common premonitory vertebrobasilar TIA symptoms in the days to weeks preceding posterior circulation stroke.[11,44] In a large population-based study, 51% (n=23/45) of premonitory vertebrobasilar TIAs presented with isolated vertigo, and 52% of these lasted longer than 1 hour, making them highly atypical for modern definitions of TIA.[11,45] When TIAs affect the anterior inferior cerebellar artery vascular territory, which supplies blood to the inner ear in most individuals,[46] the symptoms may include recurrent vertigo with auditory symptoms that can mimic Menière disease.[23,47] If seen during an attack, a patient with anterior inferior cerebellar artery territory TIA may demonstrate unilateral sensorineural hearing loss and peripheral-type nystagmus from labyrinthine ischemia, so diagnostic caution is advised when hearing loss is present.[48] Some s-EVS cases may show small strokes by MRI-DWI.[49] Dizziness is also the most common presenting symptom of vertebral artery dissection (58%),[50] which affects younger patients, mimics migraine, and is easily misdiagnosed.[19] Vertebral artery dissection can be linked to minor neck injuries sustained during exertional sports, abnormal head postures, or chiropractic neck manipulation, but fewer than half of patients with dissection have such a history.[50] Although sudden, severe, or sustained headache or neck pain probably points to a vascular pathology,[51] photophobia or phonophobia probably points to migraine.[52]

Acute Vestibular Syndrome

The AVS is defined as a clinical syndrome of acute-onset, continuous vertigo, dizziness, or unsteadiness lasting days to weeks, and generally including features suggestive of new, ongoing vestibular system dysfunction (eg, vomiting, nystagmus, and postural instability).[34] It is important to note that patients experience worsening of AVS symptoms with any head motion during the attack. These exacerbating features must be distinguished from head movements that trigger dizziness.[51] The way to differentiate the two is that a patient with t-EVS is largely asymptomatic at rest and specific head motions induce transient dizziness, whereas a patient with s-AVS is dizzy at rest and feels worse with any head motion.

Traumatic/Toxic AVS

In patients with t-AVS, the key exposures are usually obvious, so there is less need for the examination to drive diagnostic decision-making. The most common causes are blunt head injury and drug intoxication, particularly with medications such as anticonvulsants or aminoglycoside antibiotics.[2] Carbon monoxide poisoning should also be considered.[53] Most patients experience a single, acute attack resolving gradually over days to weeks once the exposure has stopped. The principal cerebrovascular concern in such patients is an overlap syndrome in which a primary pathology (eg, direct labyrinthine concussion from blunt head injury or alcohol intoxication with a fall) may mask a secondary pathology (eg, vertebral artery dissection causing cerebellar infarction).

Spontaneous AVS

As with patients with t-AVS, those with s-AVS are usually still symptomatic at the time of assessment. Because no obvious exposures are present in s-AVS (and exposures such as recent viral syndrome are nonspecific), the bedside eye examination is the primary tool for differentiating between central and peripheral causes. The most common s-AVS cause is vestibular neuritis. Approximately 10% to 20% of patients with s-AVS have stroke[54,55] (typically in the brain stem or cerebellum, 95% ischemic[55]) as a cause. Sometimes these strokes occur after a series of spontaneous relatively short-lived dizziness episodes in the preceding weeks or months that likely reflect premonitory TIAs.[55] Other dangerous causes include thiamine deficiency and listeria encephalitis.[2]

Strong evidence indicates that a 3-part bedside ocular motor examination battery (HINTS—Head Impulse, Nystagmus Type, Skew deviation) plus acute hearing loss by finger rub (HINTS plus) rules out stroke more accurately than early MRI (Table 2).[14,55–59]

Although it is possible for ED providers to learn these techniques,[59a] only a minority use them and even fewer are confident in these bedside skills.[60] Skills also vary among neurologists.[61] Widespread dissemination of these approaches may require the use of portable video-oculography devices to assist in training and feedback to providers.[23]

Details of how to perform these tests and associated videos are found elsewhere (online-only Data Supplement). Of note, HINTS only applies to patients with s-AVS (or s-EVS while the patient remains acutely symptomatic[41]), including spontaneous or gaze-evoked nystagmus; HINTS should not be relied on in patients who have other syndromes, particularly t-EVS, where normal head impulse test results would erroneously imply stroke in the majority with BPPV. Pitfalls and pearls in the diagnosis of stroke in acute dizziness and vertigo are described in Table 3.

Cerebellar hemorrhages rarely present with isolated dizziness or without clear central neurological deficits (eg,

Table 2. Pretest and Post-Test Probabilities of Stroke Using Different Tests to Rule Out Stroke in the Spontaneous Acute Vestibular Syndrome

	Post-Test Probability of Stroke Following a Negative Test Obtained in First 24 h			
Pretest probability of stroke (vascular risk profile)	General neuro examination (Sn, 19%[56]; Sp, 95%; NLR, 0.85)	CT brain (Sn, 16%[13]; Sp, 98%[13]; NLR, 0.86)	MRI-DWI brain (Sn, 80%[55]; Sp, 96%[13]; NLR, 0.21)	HINTS+Battery (Sn, 99%[58]; Sp, 97%[58]; NLR, 0.01)
10% (low)	8.7%	8.7%	2.3%	0.1%
25% (average[55])	22.2%	22.3%	6.5%	0.3%
50% (high)	46.1%	46.2%	17.2%	0.8%
75% (very high)	71.9%	72.1%	38.5%	2.4%

CT indicates computed tomography; HINTS, head impulse, nystagmus, test of skew, plus hearing; MRI-DWI, magnetic resonance imaging with diffusion-weighted imaging; NLR, negative likelihood ratio; Sn, sensitivity; and Sp, specificity.

Adapted from Newman-Toker et al[23] with permission. Copyright ©2015, Georg Thieme Verlag KG.

dysarthria),[65,70] and just 5% of cerebrovascular cases presenting s-AVS are hemorrhages, rather than ischemic strokes.[55] Although noncontrast CT identifies acute intracranial hemorrhage with high sensitivity, the low pretest probability of hemorrhage combined with low sensitivity for acute posterior fossa ischemia makes CT a poor initial choice for screening patients with isolated dizziness, absent a specific brain hemorrhage risk (eg, known history of metastatic melanoma).

Neuroimaging

The general rule in acute dizziness and vertigo is that if you need neuroimaging, it should be by MRI, rather than CT.[12] There are specific exceptions, including the need to definitively exclude hemorrhage before thrombolysis or confirm a suspected vertebral artery dissection by CT angiography. The optimal timing of neuroimaging by MRI is also complex, because the risk of false negatives in the first 48 hours

Table 3. Ten Pitfalls and Pearls in the Diagnosis of Stroke in Acute Dizziness and Vertigo

Pitfall	Pearl	Notes
True vertigo implies an inner ear disorder.	Focus on timing and triggers, rather than type.	Cerebrovascular disorders frequently present with true vertigo symptoms.[62,63]
Worse with head movement implies peripheral.	Differentiate triggers from exacerbating factors.	Acute dizziness/vertigo is usually exacerbated by head movement, whether peripheral or central.[51]
Auditory symptoms imply a peripheral cause.	Beware auditory symptoms of vascular cause.	Lateral pontine and inner ear strokes often cause tinnitus or hearing loss.[46,48,58]
Diagnose vestibular migraine when headaches accompany dizziness.	Inquire about headache characteristics and associated symptoms.	Sudden, severe, or sustained pain in the head or neck may indicate aneurysm, dissection, or other vascular pathology;[51] photophobia may point to migraine.[52]
Isolated vertigo is not a TIA symptom.	Some TIA definitions do not recognize certain transient vertebrobasilar neurological symptoms (including isolated vertigo) as TIAs.	Isolated vertigo is the most common vertebrobasilar warning symptom before stroke[11,44]; it is rarely diagnosed correctly as a vascular symptom at first contact.[7,11]
Strokes causing dizziness or vertigo will have limb ataxia or other focal signs.	Focus on eye exams: VOR by head impulse test, nystagmus, eye alignment.	Fewer than 20% of stroke patients presenting with AVS have focal neurological signs.[55,56] NIH stroke scales of 0 occur with posterior circulation strokes.[64]
Young patients have migraine rather than stroke.	Do not overfocus on age and vascular risk factors. Consider vertebral artery dissection in young patients.	Vertebral artery dissection mimics migraine closely[50]; young patients 18–44 with stroke are 7-fold more likely to be misdiagnosed than patients over age 75.[9]
CT is needed to rule out cerebellar hemorrhage in patients with isolated acute dizziness or vertigo.	Intracerebral hemorrhage rarely mimics benign dizziness or vertigo presentations.	Only 2.2% (n=13/595) of intracerebral hemorrhages presented with dizziness or vertigo and only 0.2% (n=1/595) presented with isolated dizziness.[65]
CT is useful to search for acute posterior fossa stroke.	Recognize the limitations of imaging, especially CT.	Although some retrospective studies[66,67] suggest CT may be up to 42% sensitive, prospective studies suggest the sensitivity is no higher than 16%.[13,68]
A negative MRI-DWI rules out posterior fossa stroke.	Recognize the limitations of imaging, even MRI-DWI.	MRI-DWI in the first 24 h misses 15% to 20% of posterior fossa infarctions.[12] MRI-DWI sensitivity for brain stem stroke is maximal 72–100 h after infarction.[69] Labyrinthine strokes are not visible.

AVS indicates acute vestibular syndrome; MRI-DWI, magnetic resonance imaging with diffusion-weighted images; NIH, National Institutes of Health; TIA, transient ischemic attack; and VOR, vestibulo-ocular reflex.

is nontrivial; in some cases, it may be necessary to obtain a repeat MRI if eye findings are suspicious and the initial MRI does not reveal a causal central lesion.[14] Although MRI protocols with thinner slices and smaller gaps may improve sensitivity, early false negatives likely relate to the time course of DWI signal intensity change, rather than slice and gap parameters.[12,69] In addition to magnetic resonance angiography, special MRI sequences such as black blood T1, fat-suppressed axial images through the neck may be necessary to identify intramural hematoma in vertebral artery dissection.[12,71] A suggested algorithm for choosing the most appropriate neuroimaging is provided in Figure 2.

Summary

Diagnosing cerebrovascular causes of acute dizziness and vertigo is both important and difficult. It is not routinely done well in current clinical ED practice, where misdiagnosis is frequent, patient harms are significant, and costs are high.[3] This stems from a focus on dizziness type and overreliance on negative neurological examinations and CT results. Current best evidence suggests an alternative approach emphasizing dizziness timing and triggers, focused ocular motor examinations, and MRI, as needed. This timing, triggers, and targeted examination approach is commonly used by subspecialists in vestibular neurology but is not yet common practice among emergency physicians or neurologists. Because most patients in some settings will never be seen by a neurologist,[72] and routine MRI for these patients is untenable, alternative strategies to disseminate this approach may be required. Preliminary studies have found accurate diagnosis using a portable video-oculography device that measures eye movements quantitatively.[73,74] This approach has the potential to be broadly scalable in the form of an eye ECG that helps diagnose stroke in acute dizziness via device-based decision support with telemedicine backup.[23] An ongoing NIH [NIDCD] phase II clinical trial of this approach (AVERT; URL: http://www.clinicaltrials.gov. Unique identifier: NCT02483429) seeks to develop this general approach going forward.

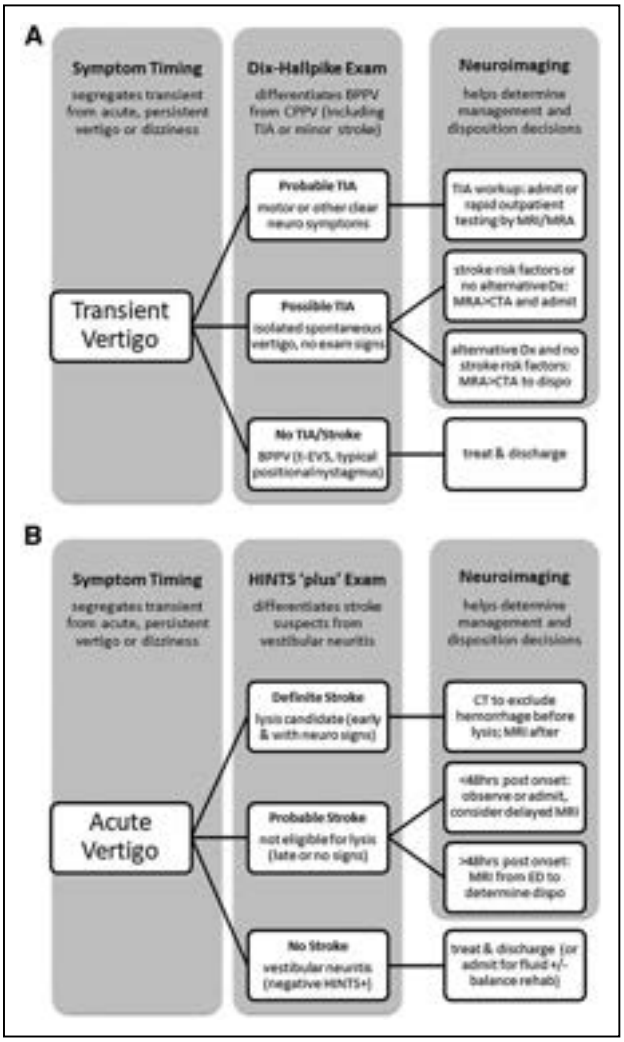

Figure 2. Suggested imaging strategy for stroke in patients with new (**A**) transient or (**B**) acute, continuous dizziness, or vertigo. Adapted from Newman-Toker et al[12] with permission. Copyright ©2017, Elsevier. BPPV indicates benign paroxysmal positional vertigo; CPPV, central paroxysmal positional vertigo; CTA, computed tomography angiography; Dx, diagnosis; ED, Emergency Department; HINTS, head impulse, nystagmus, test of skew; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; and TIA, transient ischemic attack.

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