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Misdiagnosing the Dizzy Patient: Common Pitfalls in Clinical Practice

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Abstract

This chapter highlights five pitfalls in the approach to the diagnosis of common vestibular disorders. These include (1) overreliance on dizziness symptom type to drive diagnostic inquiry, (2) underuse and misuse of timing and triggers to categorize patients, (3) underuse, misuse, and misconceptions linked to hallmark eye exam findings, (4) overweighting age, vascular risk factors, and neuro exam to screen for stroke, and (5) overuse and overreliance on head CT to 'rule out' neurologic causes. For each, we discuss the evidence base describing the pitfall's frequency, its likely causes, and potential alternative strategies that might be used to improve diagnostic accuracy or mitigate harms.

Introduction

As a general rule, peripheral vestibular disorders are not correctly diagnosed or managed in the ED, with misdiagnosis rates estimated in the range of 74–81%.[1, 2] Common disorders such as benign paroxysmal positional vertigo (BPPV) and vestibular neuritis are frequently confused for one another[1] and for more serious central causes such as stroke.[2] Management is non-evidence-based and suboptimal.[3 4]

Identifying peripheral vestibular disorders should be a priority for several reasons. First, these disorders are very common – BPPV alone has an estimated lifetime prevalence around 2%.[5] Second, evidence-based treatments exist. The most common peripheral vestibular disorder, BPPV, has systematic reviews and clinical guideline statements to support the use of the Dix-Hallpike test and the highly effective canalith repositioning maneuver.[6, 7] Physical therapy strategies are also supported by systematic reviews for the treatment of acute unilateral vestibulopathy (e.g., vestibular neuritis).[8] Optimal identification of peripheral vestibular disorders could enable more efficient care since additional laboratory or imaging tests are not necessary and are generally not even warranted in the management of these disorders.[6] Lastly, physicians who can accurately identify peripheral vestibular

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disorders are probably better equipped to identify dangerous central vestibular disorders because the probability of a central disorder substantially increases when peripheral vestibular disorders are ruled out.

Identifying central vestibular disorders is also important. Vascular causes – stroke or transient ischemic attack (TIA) – are the most common central disorders that present with acute dizziness[9] and evidence-based treatments exist for both acute and chronic management to improve functional recovery and reduce the risk for future stroke events.[10] Clinical practice research suggests that identifying stroke-dizziness presentations may be suboptimal. One population-based study found that 16 out of 46 (35%) acute stroke/TIA-dizziness cases did not receive a stroke or TIA diagnosis from the treating emergency department (ED) provider.[11] Dizziness was also found to be the presenting symptom most closely linked with subsequent stroke presentations in the ED, a finding that suggests the initial dizziness presentations may have actually been misdiagnosed as non-vascular events.[12] In another population-based study, transient ischemic attacks (TIAs) presenting posterior circulation symptoms such as isolated dizziness and vertigo were initially misdiagnosed in 90% of cases (n=9/10).[13]

Harms may result from missed opportunities to apply timely therapies for true underlying disorders, including both peripheral and central diseases. For example, BPPV patients not treated within 24 hours of the ED visit have more than double the recurrence risk (46% vs. 20%, p=0.002)[14] and unrecognized BPPV confers 6.5-fold greater odds of falling.[15] When stroke diagnosis is delayed, missed opportunities for thrombolysis,[16] early surgery for complications such as malignant posterior fossa edema,[17] and early prevention of subsequent vertebrobasilar infarction can result in permanent disability or death.

In this chapter, we explore some of the likely reasons why misdiagnosis is frequent. We highlight five diagnostic pitfalls (Table 1) often encountered in clinical practice, and make recommendations for how to avoid these known traps when assessing the acutely dizzy patient.

Pitfall 1. Overreliance of the type of dizziness to guide diagnostic inquiry

The traditional approach to the evaluation of patients who present with dizziness symptoms has been to heavily weight defining the type of symptom when assessing the most likely etiology.[18, 19] The recommended first question for the patient is generally, “What do you mean by dizzy?” In this traditional paradigm, the dizziness is classified into one of four specific ‘types’: (1) vertigo, the illusion of spinning or other false motion; (2) presyncope, a feeling of impending faint or loss of consciousness; (3) disequilibrium or loss of balance without head sensation; and (4) other ill-defined symptoms such as lightheadedness, wooziness, or giddiness.[18] The teaching suggests that once a patient is categorized within a type of dizziness, the likely causes are as follows: vertigo is felt to indicate a vestibular disorder, presyncope indicates a cardiovascular disorder, disequilibrium indicates a neurologic disorder, and non-specific dizziness indicates a psychiatric or metabolic disorder.[19]

This symptom ‘type’ approach has been frequently endorsed in medical literature.[20] Studies confirm that frontline physicians consider the type of dizziness to be very important. A survey of over 400 emergency department (ED) physicians revealed consensus that the approach to dizziness based on the type of symptom is the dominant method presented in the medical literature and teaching, and roughly 90% personally endorse this approach.[20] When asked to rank the relative importance of several symptom attributes to diagnostic assessment of a patient with dizziness in the ED, most (64%) ranked symptom type first, roughly 5-fold more often than the next nearest attribute. Most (69%) respondents agreed that they do not pursue cardiovascular causes if the patient reports vertigo, nor vestibular causes if the patient reports presyncope, indicating that they make clinical decisions based on the type of symptom, including when not to pursue diagnoses.[20]

The problem with the type of dizziness being used as the principal factor in the diagnostic process is that it is neither a reliable symptom attribute reported by patients nor a valid discriminator among different causes of dizziness.[21, 22] Low reliability of patient reports of the type of dizziness was demonstrated by research that queried patients presenting to the ED regarding dizziness symptoms that included a test-retest paradigm.[21] Of more than 300 dizziness patients interviewed, more than half changed their choice of the “best” descriptor for their dizziness when asked the same question 5–10 minutes after their initial selection. Furthermore, patients’ open-ended descriptions of dizziness were often vague, circular, or hard to understand, making them difficult to categorize using the traditional paradigm. Examples of responses to the question “What do you mean by ‘dizzy’?” included the following: “Yes, like your head is becoming empty”, and “I think the general meaning would be the point where that woozy feeling; now I don’t know how you want to describe the adjective for that; I guess woozy at that point.”

Categorizing patients based on the type of dizziness is also problematic because patients typically use more than one type of dizziness to describe their symptoms. Patients allowed to choose from a list of 6 types of dizziness to describe their symptoms selected an average of 2.6 types, with nearly 80% selecting more than 1 type and 50% selecting 3 or more types.[21] Roughly 75% of those who selected a descriptor *other than vertigo* answered directed questions about false spinning or motion that indicated they were, in fact, experiencing vertigo. It is difficult to imagine, therefore, how the type of dizziness could possibly inform the differential diagnosis for the underlying cause.

Regarding the validity of the type of dizziness as a discriminator among causes, systematic reviews have found no published studies that support the predictive validity of the symptom type.[22, 23] Small studies have shown no association between dizziness type and final clinical diagnoses (e.g., type of dizziness fails to adequately differentiate central from peripheral causes[24] or psychiatric from vestibular causes[25]).

Reliability and validity of information gathered are the cornerstones of diagnostic evaluation. Factors shown to not be adequately reliable or valid should be downgraded rather than emphasized when formulating a case and planning the evaluation and management. In this case, the ‘type’ or ‘quality’ of dizziness symptoms should be given little or no diagnostic weight.

Pitfall 2. Underuse and misuse of timing and triggers to categorize patients for diagnosis

When frontline providers typically over-emphasize symptom types, they also usually under-emphasize or misuse timing and triggers in dizziness,[26] which are aspects that are more reliably reported by patients and thus can be used to more effectively categorize clinical presentations.[21] In contrast to the low reliability of patient descriptions of the type of dizziness, patients have been shown to be reliable in describing the duration and triggers of the dizziness symptoms.[21] Regarding the timing of symptoms (episode duration), 81% of subjects report consistent responses on being asked the same question within minutes of the initial question. Similarly, a very high proportion of patients (91–100%) provide consistent responses regarding questions about symptom triggers (i.e., postural/positional, head motion, rolling in bed).

The reliability of these features is important because common causes of dizziness such as BPPV and vestibular neuritis have hallmark features in terms of their timing and triggers.[27, 28] The timing of vestibular symptoms (including episode, relapse, and illness duration; symptom onset; and frequency) has been studied fairly extensively in individual disease populations.[25, 29–31] The much higher reliability of timing and triggers compared with dizziness type, coupled with important differences in timing and trigger patterns among common causes of dizziness, should increase the diagnostic utility of this information.

There is, however, an important pitfall related to misuse of dizziness trigger information: the tendency of physicians to over-emphasize positional aspects of the dizziness symptoms and to presume a benign disorder, typically BPPV, when symptom intensity is associated with positional changes.[32] The problem hinges on the critical (but often unrecognized) distinction between symptoms that are positionally *triggered* from those that are positionally *exacerbated*.[28] Positionally triggered dizziness or vertigo is most often due to BPPV, while positionally exacerbated dizziness could be of any cause. This is because virtually every cause of persistent dizziness (benign or dangerous, peripheral or central) is exacerbated by positional changes and at least partially relieved by remaining completely still. Despite this, 80% of physicians mistakenly endorsed the idea that, in patients with persistent dizziness, head motion causing an exacerbation of symptoms is an indicator of a benign condition.[20] This endorsement is likely because these providers assume a diagnosis of BPPV whenever the patient reports that head motion contributes to the symptoms, without distinguishing triggers (in true episodic dizziness, from an asymptomatic baseline) from exacerbating features (in continuous, ongoing dizziness). BPPV is specifically characterized by very discrete, brief, and high-intensity attacks of dizziness triggered by certain head movements (e.g., lying down, rolling over in bed, or looking up).[6] A patient with vestibular neuritis or stroke may report minimal or sometimes no symptoms when lying still, particularly if is a mild case or if hours or days have passed since the onset, but the return of continuous symptoms during normal head movements usually makes the distinction clear. The overlap of a “positional component” among various causes of dizziness is why it is critical to correctly identify the characteristic BPPV nystagmus with the Dix-Hallpike test (see Pitfall 3).[6]

Pitfall 3. Underuse, misuse, and misconceptions linked to hallmark eye exam findings

In the acute setting, the eye movement examination is often the essential element to discriminating among causes of dizziness because the patterns of eye movements are hallmark features of the various disorders (Table 2).[27, 33] Emphasizing the examination is important because the history of present illness can overlap among the various causes of dizziness, particularly on the first day of symptoms. Although timing and triggers are reliably reported by patients,[21] a patient with severe BPPV symptoms may be difficult to distinguish from a patient with a mild case of vestibular neuritis or stroke simply based on the timing and triggers, or even using additional details about the clinical scenario. It is common, particularly in the acute setting, for patients with BPPV to report continuous (i.e., inter-ictal) low-level dizziness symptoms such as mild unsteadiness or mild nausea[34, 35] and they may be restricting head movement such that very few actual paroxysmal attacks have occurred. Vestibular neuritis patients may first notice their symptoms on awakening and sitting up from bed—a description that closely mimics a typical positional trigger history seen in BPPV. Further, patients with mild vestibular neuritis may report feeling near normal at rest and substantial worsening of symptoms with movement. Frontline clinicians seeing the patient in the first 24–48 hours will not have the advantage of knowing how the symptoms later evolve. The progression over days to weeks often makes distinguishing these disorders more straightforward—BPPV as numerous brief paroxysmal attacks triggered by typical head movements vs. vestibular neuritis as a single prolonged bout with dizziness symptoms aggravated/alleviated in relation to activity.

Fortunately, common disorders causing dizziness all have hallmark findings on eye movement examination (Table 2). For example, BPPV can be confidently diagnosed in a matter of minutes when there is no spontaneous or gaze-evoked nystagmus and the Dix-Hallpike test triggers transient upbeat-torsional nystagmus—even if the patient reports a continuous dizziness sensation that might suggest otherwise. This BPPV pattern of nystagmus has been well defined based on the anatomy and physiology relevant to BPPV.[33] Similarly, a central cause of dizziness can be presumed whenever a central pattern of nystagmus (e.g., dominantly vertical or torsional spontaneous nystagmus; or gaze-evoked bi-directional) is identified. Spontaneous unidirectional nystagmus is typically caused by vestibular neuritis, though additional exam elements are needed in this situation to more confidently distinguish this peripheral vestibular disorder from a central vestibular disorder such as stroke (See Chapters 2, 10, & 11).[36]

Although these features enable a rapid diagnostic assessment, frontline physicians frequently underuse or misinterpret the bedside eye movement examination.[1, 4] BPPV and vestibular neuritis should be diagnosed, particularly in the acute setting, by confirming the presence of a characteristic pattern of nystagmus.[6 37] A population-based study of ED dizziness presentations found that a nystagmus assessment was documented in most of the visits (81.3%, 887 of 1,091 visits).[1] However, when nystagmus was documented as present, only infrequently were there additional details such as the direction of the nystagmus, its presence in primary gaze vs. lateral gaze, or the findings with positional testing. Because of the sparse nystagmus details documented in the medical record, neuro-otology raters were only able to draw inferences about the suspected localization or etiology in 5.4% of cases documented

as having nystagmus present. The use of the Dix-Hallpike test was rarely documented even when the treating physician recorded a diagnosis of BPPV.[4, 5] For visits receiving a peripheral vestibular diagnosis (i.e., BPPV or vestibular neuritis) from the treating physician, most of the nystagmus descriptions (81%) were incompatible with the diagnosis rendered by the physician.[1] The most common reason that reported nystagmus findings were against the diagnosis was documentation of nystagmus being absent, since BPPV and neuritis both require a specific nystagmus type to be present. However, even when nystagmus was documented to be present, most of the descriptions were also against the diagnosis rendered.

Pitfall 4. Overweighting age, vascular risk factors, and general neurologic exam to screen for stroke

Since hallmark eye exam findings are underused in the clinical evaluation, frontline doctors probably overweight vascular risk factors and the general neurologic exam when considering the possibility of acute stroke. Indeed, older age, vascular risk factors, and abnormal neurologic exam findings have been shown to be indicators of patients at higher risk of stroke.[11] The demographic factors increase the pre-test probability of stroke in acute dizziness presentations whereas abnormalities on the general neurologic exam (e.g., dysarthria, dysmetria) suggest the dizziness stems from a central lesion. Nevertheless, a patient with central eye movement abnormalities has a substantial probability of stroke even if there are no vascular risk factors or general neurologic abnormalities.[36] Based on data from a series of 190 cases of acute continuous dizziness, providers relying on the $ABCD^2$ vascular risk score (an aggregate score based on traditional vascular risk factors and components of the general neurologic exam) to identify stroke would miss nearly 40% of the stroke cases while also overcalling stroke in nearly 40% of peripheral vestibular cases.[36] Relying on vascular risk factor stratification is particularly likely to miss younger stroke patients and research suggests that younger patients with dizziness may be more at risk for stroke misdiagnosis than are older patients.[12, 36]

The presence of other neurological symptoms or signs is a strong predictor for a central nervous system cause, typically stroke.[22, 38] However, the converse is not necessarily true. Isolated dizziness and vertigo presentations are the most common initial manifestation of posterior circulation (vertebrobasilar) ischemia,[13] and roughly 80% of strokes with the principal symptom of acute continuous dizziness do not have abnormalities on the general neurologic examination.[39] When stroke patients have an NIH stroke scale score of zero, it is often a posterior circulation stroke presenting dizziness, vertigo, or nausea as the chief symptom.[40] Thus, the absence of typical neurologic symptoms or signs suggesting central nervous system involvement should not be relied upon to exclude stroke in acute dizziness.

Pitfall 5. Overuse and overreliance on head CT to ‘rule out’ neurologic causes

Neuroimaging can be useful in patients with acute dizziness or vertigo to diagnose (or ‘rule out’) central nervous system causes, particularly stroke, but current practice patterns do not match best evidence. In particular, there is a heavy overreliance on CT of the brain,[9, 41–43] despite ample evidence of its lack of utility in this clinical scenario.

The use of neuroimaging, particularly CT, has increased substantially among ED visits for dizziness over time.[9, 41] In 1995, only about 10% of ED visits received a head CT and this increased to 25% by 2004.[9] Despite this increase in neuroimaging utilization, there was no corresponding increase over time in the proportion of visits receiving a neurologic diagnosis.[9] Contemporaneous local-area practice variation in use of head CT by different hospitals (from 21 to 33%) does not influence the probability of identifying strokes.[43] Although CT is not recommended for diagnosis of BPPV,[6] visits diagnosed with BPPV do not have less frequent use of head CT compared to visits that receive other diagnoses.[3, 4]

More recent estimates indicate that roughly 50% of ED dizziness patients now undergo a head CT.[44, 45] At this frequency of use, head CT is essentially being used as a 'rule out' test similar to use of cardiac enzyme biomarkers to 'rule out' heart attack in chest pain. Unfortunately, head CT is not useful to 'rule out' stroke in acute dizziness because CT has a very low sensitivity (<40%) for acute ischemic stroke,[46] the most common central cause of acute dizziness presentations.[9] In fact, for posterior fossa ischemic strokes presenting dizziness, the sensitivity of CT may be even lower, estimated in one recent study to be just 7%.[47] Based on the low sensitivity, a negative head CT—the typical result in acute dizziness presentations—does not meaningfully alter the probability of acute stroke. Beyond its significant limitations as a diagnostic test, the use of head CT in dizziness presentations also has other potential serious downsides including an association with longer length of stay in the ED,[41] unnecessary radiation exposure, false reassurance for patients, and additional costs (hundreds of millions of dollars per year in the US).[45]

It is sometimes suggested that CTs are necessary to 'rule out' brain hemorrhage. It is certainly valid to obtain acute brain CT looking for hemorrhage if a dizzy patient is hemiparetic, lethargic, or neurologically deteriorating, but screening for intracranial hemorrhage in ED patients with dizziness as the dominant symptom is highly unlikely to yield a positive result.[42] It is also reasonable to perform a CT to exclude brain hemorrhage if a patient is being considered for thrombolytic therapy. However, neither scenario is common, so these cannot explain the frequent use of head CT in dizziness. Instead, ingrained misconceptions,[20] medicolegal fears, patient requests, or other factors may drive neuroimaging test overuse in clinical practice.

Magnetic resonance imaging with diffusion-weighted sequences (MRI-DWI) is a much more accurate test for ischemic stroke[46] and other central causes of dizziness. It is favored over CT in clinical practice guidelines (http://www.guideline.gov/content.aspx?id=47674), and should generally be the test of choice when neuroimaging is required. However, physicians should also be cautious not to overuse MRI or over-interpret negative MRI-DWI results. MRI can substantially extend length of stay at the acute visit, and in some centers its use may require the patient to be hospitalized. Perhaps more importantly, MRI can miss acute infarction, particularly with lesions in the posterior fossa (the typical location for strokes causing dizziness or vertigo) and when it is performed within 48 hours of symptom onset.[36, 48] Recent studies suggest that in patients presenting acute, continuous dizziness, MRI-DWI misses ~10–20% of all strokes, including ~50% of those strokes less than one centimeter in diameter.[22, 49]

Conclusions

Opportunities exist to improve the diagnosis and management of patients with acute dizziness in routine care settings. The traditional approach to dizziness has been to determine a specific type of dizziness and to then weight this attribute heavily when formulating the case and planning further evaluation and management,[19, 20] but this approach is not an evidence-based practice. Instead, providers should be focused on timing and triggers, rather than type.[28] Observational research of routine care has found that providers are frequently and increasingly using a low-value test—head CT—in typical dizziness presentations.[4, 9] At the same time, providers are underusing or misusing higher-value bedside eye movement exams.[1, 4, 6] To optimize the accuracy and efficiency of diagnosis in acute dizziness and vertigo, providers should de-emphasize the type of dizziness, patient demographics, and the routine use of CT (or MRI) neuroimaging, and instead emphasize the timing and triggers for dizziness symptoms and leverage bedside eye movement assessments to identify opportunities to effectively and efficiently diagnose and treat common peripheral vestibular disorders and simultaneously to determine whether MRI neuroimaging is indicated to search for dangerous central causes.

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Table 1.

Summary of common pitfalls in current approaches to diagnosing acute dizziness

Pitfalls	Additional Information
1. Overreliance on type of dizziness to guide diagnostic inquiry	• Patients' descriptions of symptom type are not reliable. • Types of dizziness are not valid discriminators. • The type of dizziness should be de-emphasized when making diagnostic and management decisions.
2. Underuse and misuse of timing and triggers to categorize patients for diagnosis	• Patients' report of timing and triggers are reliable. • Major causes of dizziness have characteristic timing and triggers, so these attributes should be emphasized. • Care should be taken to distinguish 'triggers' from 'exacerbating' features, which have very different implications for diagnosis.
3. Underuse, misuse, and misconceptions linked to hallmark eye exam findings	• Major causes of dizziness have hallmark eye movement examination findings that are virtually pathognomonic. • Frontline providers and neurologists should be better trained in the use of these hallmark examination findings.
4. Overweighting age, vascular risk factors, and neuro exam to screen for stroke	• Although older patients with vascular risk factors are more likely to have stroke as a cause for dizziness or vertigo, young patients with stroke are far more likely to be missed, with potentially devastating consequences. • Patients with central patterns of eye movements are still at a very high risk of acute stroke even when there are no vascular risk factors or general neurologic abnormalities.
5. Overuse and overreliance on head CT to 'rule out' neurologic causes	• Head CT is commonly and increasingly used in acute presentations of dizziness. • Head CT is a very insensitive test for acute ischemic stroke, which is the most common central cause of acute dizziness, so its use should be severely curtailed. • If neuroimaging is required, MRI-DWI is the test of choice.

Table 2.

Common eye movement patterns, their nystagmus characteristics, and typical causes.

Pattern Types	Nystagmus Characteristics	Typical Cause(s)
Peripheral Vestibular Patterns	Upbeat-torsional nystagmus triggered by the Dix-Hallpike test and that is transient (lasting <30 seconds)	BPPV (posterior canal)[a]
	Dominantly horizontal spontaneous nystagmus (i.e., present in primary gaze during routine gaze testing) that is uni-directional (i.e., never changes direction with gaze shifts or head shaking) but increases in velocity with gaze in the direction of the nystagmus fast phase and decreases with gaze in the opposite direction	Vestibular neuritis (less commonly caused by a central lesion typically associated with other findings[b])
Central Vestibular Patterns	Dominantly vertical (upbeat or downbeat) or torsional spontaneous nystagmus (i.e., present in primary gaze during routine gaze testing) Dominantly horizontal gaze-evoked direction changing nystagmus (i.e., persistent left-beat on left gaze and persistent right-beat on right gaze) Non-fatiguing positional down-beat nystagmus triggered by a positional test such as the Dix-Hallpike test	Stroke, multiple sclerosis, Chiari malformation, other structural central disorders, medication side effects (e.g., anti-epileptic medications)
Physiological Patterns	Few beats of end-gaze nystagmus (non-sustained left beat nystagmus on far left gaze with symmetric right-beat nystagmus on far right gaze)	Normal variant

BPPV = benign paroxysmal positional vertigo

[a]The second most common variant of BPPV is the horizontal canal variant which is characterized by transient horizontal nystagmus (rather than upbeat-torsional nystagmus) triggered by a head turn to either side while lying supine (e.g., head turn to right triggers right-beat horizontal nystagmus, head turn to left triggers left-beat horizontal nystagmus).

[b]See Chapters 2, 10, and 11 for information regarding findings suggesting a central vestibular lesion in the presence of uni-directional spontaneous nystagmus.