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Dizziness: An Evidence-Based Approach (Better Than MRI?)

A 45-year-old patient with a history of well-controlled hypertension and mildly elevated cholesterol comes to the emergency department (ED) for 8 h of continuous dizziness that began rapidly. The patient describes severe light-headedness.

An Unusual Case of Vertigo: The Usefulness of Nystagmus Examination

A 50-year-old gardener presented to our Emergency Department (ED) after the onset of a sudden objective vertigo while standing up during work, accompanied by imbalance, nausea, and vomiting. Neither tinnitus nor other aural symptoms were present, but, if asked, he also complained of a mild headache.

Neuroendocrine Tumour of the Middle Ear: A Case Report

Neuroendocrine tumour of the middle ear is a rare entity. Here we present a case of a 50-year-old male who presented with a polyp in the left external auditory canal. Surgical excision followed by immunohistochemistry confirmed it to be a neuroendocrine tumour.

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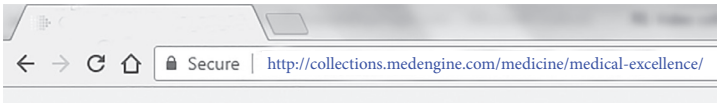

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Dizziness: An Evidence-Based Approach (Better Than MRI?)

Jonathan A. Edlow

Case Presentation

A 45-year-old patient with a history of well-controlled hypertension and mildly elevated cholesterol comes to the emergency department (ED) for 8 h of continuous dizziness that began rapidly. The patient describes severe light-headedness. There is no headache or neck pain. Vital signs and general physical examination are normal. On examining the eyes, horizontal nystagmus that beats toward the left is present. Skew deviation is absent. On head impulse testing, there is a corrective saccade when moving toward the right. The remainder of the neurological examination is normal.

Introduction

Approximately 3.5% of emergency department (ED) visits are for dizziness [1, 2]. Numerous conditions, some benign and self-limiting and others extremely serious, can present with dizziness. This is a classic emergency medicine—sorting out the large majority of patients with a given chief complaint who have a self-limiting or easily treatable condition from the smaller number that have life-, limb-, or brain-threatening problems. As of 2013, the direct ED-related costs of care for patients with dizziness in the USA were estimated to approach $4 billion [3]. In addition to economic, there is additional "cost" both in terms of patient-experienced anxiety and falls, attributed to dizziness, with their resultant morbidity.

The existing paradigm for diagnosing dizziness is based on "symptom quality" (i.e., asking the question "what do you mean 'dizzy'?"). This approach is taught in nearly all review articles and textbooks across specialties; however, newer research has shown that its scientific basis and its internal logic lack foundation.

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Currently, misdiagnosis in patients with dizziness is a problem in an environment that is paying increasing attention to diagnostic errors [4]. Misdiagnosis of patients with cerebellar stroke can have disastrous consequences [5]. This article will review the differential diagnosis of acute dizziness in adult patients, discuss newer research about the diagnosis of dizziness, and suggest a modern evidence-based approach.

The new approach emphasizes history and physical examination that will hopefully lead to emergency physicians more frequently and confidently making a specific diagnosis. When a confident diagnosis is made of a peripheral problem, time-consuming consultation, expensive imaging, and hospitalization become unnecessary. When the evaluation suggests a central problem, especially stroke, steps can be taken to diagnose and treat the offending vascular lesion and institute secondary prevention measures.

This new approach to the ED patients with dizziness should improve diagnostic accuracy and reduce length of stay and resource utilization and would be expected to improve overall patient outcomes.

Differential Diagnosis of Acute Dizziness

Numerous disorders and conditions that span multiple organ systems can present with acute dizziness. Many of these diagnoses are benign; others are life-threatening. A study from the NHAMCS patient database over a 13-year period identified 9472 patients with dizziness [2]. These data suggest that most patients have general medical (including cardiovascular) diagnoses (~50%), oto-vestibular diagnoses (~33%), and neurologic (including stroke) diagnoses (~11%) [2, 6].

Studies of large administrative databases have the limitation that the accuracy of the charted diagnosis is unknown. In the NHAMCS study, 22% of patients received a "symptom only" diagnosis (e.g., dizziness, not otherwise specified). Although assigning a diagnosis of the presenting symptom is common in emergency medicine practice, a "symptom only" diagnosis was three times more common in dizzy patients than in all other patients. In addition, even if a specific vestibular diagnosis is made,

such as benign paroxysmal positional vertigo (BPPV), the use of imaging and treatment with medications is not in accordance with best evidence [7].

In the NHAMCS study, prospectively defined "dangerous" diagnoses (various cardiovascular, cerebrovascular, toxic, metabolic, and infectious conditions in which the possibility of a poor outcome without treatment was likely) were found in 15% of patients, and this proportion increased with age [2]. The most common dangerous causes found were fluid and electrolyte disturbances (5.6%), cerebrovascular diseases (4.0%), cardiac arrhythmias (3.2%), acute coronary syndromes (1.7%), anemia (1.6%), and hypoglycemia (1.4%) [2]. Some rare causes of dizziness such as adrenal insufficiency [8], aortic dissection [9], carbon monoxide intoxication [10], pulmonary embolus [11], and thiamine deficiency [12] are treatable.

How does this study compare to others? One older single-institution study analyzed 125 patients prospectively identified over a 16-month period [13]. Forty-three percent had a diagnosis of a peripheral vestibular problem, and 30% had a "serious" diagnosis. Another larger prospective single-institution Chinese study of adult ED patients with dizziness reported results of 413 patients recruited over just 1 month [1]. A central nervous system (CNS) cause was found in 23 patients (6%).

Two retrospective studies also provide relevant data. One was done in a German ED of 475 consecutive dizzy patients who were seen by a neurologist during the index ED visit [14]. The initial diagnoses assigned by the neurologists were benign in 73% of cases and serious (mostly cerebrovascular and inflammatory CNS disease) in 27% of cases. Overall, the two most common diagnoses were BPPV (22%) and stroke (20%). In follow-up by a neurologist blinded to the ED diagnosis, 44% of diagnoses (previously made by a neurologist in the ED) were changed. Over half of these diagnostic changes were from a serious to a benign diagnosis, which errs toward patient safety but is more resource intensive than necessary. In about one patient in seven, the error was from benign to serious (five patients diagnosed with vestibular neuritis and one with vestibular migraine, all reassigned to stroke), a dangerous misdiagnosis.

The other study analyzed patients who had an ED triage diagnosis of dizziness, vertigo, or imbalance as a primary symptom, collected over a 3-year period, and identified 907 patients (only 0.8% of all ED patients over that period of time), suggesting a very targeted selection (compared to other large studies) [15]. Of the 907 patients, one in five was admitted (68% to an intensive care unit [ICU]). The most common admitting services were medicine (41% of admissions), cardiology (32%), and neurology (24%). Of the 907 patients, most had benign conditions either peripheral vestibular problems, 32%, orthostatic hypotension (13%), or migraine (4%). A full 22% could not be diagnosed. Serious neurological disease was found in 49 patients (5%) of which 37 were cerebrovascular. Finally, only two patients with serious neurological disease presented with isolated dizziness.

The incidence of important CNS disease in adult ED patients with dizziness is approximately 5%. The high-end outlier is the Royl study that reported that 27% of patients have serious CNS causes which may be skewed by the fact that the study was conducted in a neurological ED [14]. Various studies have tried to identify risk factors for ED dizzy patients with CNS causes [1, 15–19]. One ED study of dizzy patients found that abnormal gait and subtle neurological deficits on neurological examination were associated with a CNS cause [16]. Overall, the risk factors include increasing age, vascular risk factors and history of previous stroke, complaint of "instability," and focal neurological findings (Table 1).

Table 1: Risk factors for a central nervous system cause in emergency department patients with dizziness.

Risk factor	Cheung et al. [1]	Navi et al. [15]	Chase et al. [16]	Kerber et al. [19]
Age in years	6.15 for age > 65	5.7 for age > 60		
Symptom of imbalance or ataxia	11.39 for "ataxia"	5.9 for "imbalance"	9.3 for "gait instability"	
Focal neurological symptoms	11.78	5.9		
History of previous stroke	3.89			
Vascular risk factors	3.57 for diabetes			0.48 (CI crossed 1)
ABCD2 score				1.74 (scored as a continuous variable)
HINTS testing				2.82
Other neurological deficits			8.7 for "subtle" neurological finding	2.54

Not every study reported on every variable; blank cells were not reported in that study. Numbers are odds ratios (when reported)
HINTS head impulse, nystagmus, test of skew; *CI* confidence intervals

Taken as a whole, these data suggest the following conclusions:

1. Most adult patients who present to the ED with acute dizziness have general medical or cardiovascular conditions.
2. Although benign vestibular diseases are much more common than CNS causes of dizziness, when emergency physicians make these (benign) diagnoses, their use of imaging and meclizine is not in accordance with the best available evidence.
3. Of the CNS causes, acute cerebrovascular disease (ischemic stroke or transient ischemic attack [TIA]) is the most common cause, and misdiagnosis in the ED is not uncommon in these patients.

Origin of the "Symptom Quality" Approach to Diagnosing Dizziness and its Lack of Scientific Validity

A publication in 1972 led to the "symptom quality" approach to the acutely dizzy patient [20]. Over a 2-year period, the authors enrolled 125 patients. The study suffers from several shortcomings that include a low number of patients, the fact that most patients were not evaluated during the acute phase of their illness, no verification of diagnosis, no long-term follow-up, and others (Table 2). This study was the basis for the traditional approach to dizziness that starts by asking the patient, "What do you mean by 'dizzy'?"

For this "symptom quality" approach to work, two facts must be true. First, patients should be able to reliably and consistently choose one (and only one) dizziness type. Secondly, each symptom type should be tightly linked with a given differential diagnosis. Both facts are demonstrably false [21].

Patients do not choose a single dizziness type. Sensory symptoms are difficult for many patients to describe. Patients with dizziness may use words like, "dizzy," "light-headed," "spinning," "rocking," "vertigo," "giddy," "like I'm going to faint," "off-balance," "spacey," and others to describe what they feel. For this paper, I will use the word "dizziness" in a general way (incorporating all of these descriptors).

In a study published in 2007, research assistants asked a series of ED patients with dizziness a battery of questions aimed at determining "symptom quality" and timing and triggers of the dizziness [22]. Over 60% of the patients chose more than one dizziness type. The questions were then re-asked in a different sequence an average of 6 min later; more than 50% of the patients changed their primary dizziness type. The responses to timing and triggers of dizziness were much more consistent and reliable between the first and second responses.

Thus, the history should be taken just as one does with a patient with chest pain or dyspnea. One does not evaluate a patient with chest pain differently if the pain is described as "sharp" or "dull" or "discomfort" or "pressure" [21]. One does not use the descriptor of the pain in a binary way; the timing and triggers are more important in rank-ordering a differential diagnosis. We take histories of virtually all chief complaints using the concept of timing and triggers.

Another concept that physicians use regularly to construct a differential diagnosis is that of context and presence or absence of associated symptoms. One thinks very differently about a patient with chest pain associated with: (a) leg swelling and dyspnea, (b) productive cough and fever, or (c) hypotension, unilaterally diminished breath sounds, and distended neck veins. It is not simply the word that the patient uses that informs the differential diagnosis but also the timing, triggers, associated symptoms, and epidemiologic context. It should be no different with dizziness.

Finally, the differential diagnosis is not tightly linked with a given use of the descriptors. The use of the word "vertigo" was not associated with a higher incidence of stroke in a large series of ED patients with dizziness [23]. Patients with a cardiovascular cause of dizziness do endorse "vertigo" in almost 40% of cases [24]. Patients with BPPV often say they feel light-headed and not vertiginous, especially elderly patients [25]. The reality is that the differential diagnosis should *not* be based on the word but rather on the timing, triggers, associated symptoms, and the epidemiologic context.

Despite the fact that the "symptom quality" approach to dizziness is not based on strong science, it is the predominant paradigm used across specialties.

Misdiagnosis of Patients with Dizziness and Resource Utilization

Misdiagnosis of patients with dizziness is common. In the German ED study, neurologists seeing patients made diagnostic errors in 44% of patients. The authors of that study found three factors that contributed to misdiagnosis [14]. First, subsequent clinical course evolved, making the ultimate diagnosis more clear. This factor played a role in 70% of misdiagnoses. This is a regular event in emergency medicine, in which we see patients whose symptoms evolve in a variable way even over hours. The other two factors were insufficient brain imaging (mostly MRI, found to be a factor in half of cases) and failure to screen for vascular risk factors using advanced testing such as echocardiography, telemetry, or ultrasound of cervical arteries (24% of cases). There has never been a head-to-head comparison of emergency physicians versus neurologists diagnosing patients with dizziness at the same phase of care (and likely never will), but this German study clearly shows that dizziness is complicated, even to those with specialized training and focus.

Table 2: Shortcomings of the original paper on the "symptom quality" approach.

Methodological issues
Tautological hypothesis
Their methods placed patients into one of four categories of dizziness by design
Related "appropriate" questions were only asked once the dizziness category was assigned
A diagnosis of a "peripheral vestibular disorder was typically applied to a patient who complained of unmistakable rotational vertigo"
Lack of independent verification and blinding
A single individual assigned the final diagnosis; there was no independent verification of the diagnoses
The individual assigning of the diagnoses was not blinded to the data or the categories of symptom quality
Small number of subjects with 25% drop-out rate after enrollment
125 total patients were enrolled (but 25.6% were excluded)
12 (16.8%) were excluded due to "inadequate data" obtained
9 (7.2%) were excluded because of "uncertain diagnosis"
2 (1.6$) were excluded because they were "inappropriate referrals"
Selection bias
Only 125 patients were enrolled over a 2-year period
They had to be available to return on four different days for testing
They had to be fluent in English
Lack of long-term follow-up of patients
There was no long-term follow-up to verify accuracy of diagnosis
Unavoidable issues related to era in which study was performed
Lack of modern imaging
When the study was done, neither CT nor MRI was available
Lack of some diagnoses being established
Vestibular migraine (a common cause of s-EVS) had not yet been described
Posterior circulation TIA presenting as isolated dizziness was not recognized

In another study of 1091 dizzy patients in the U.S. EDs, emergency physicians documented some comment about nystagmus in 887 (80%) of whom nystagmus was documented to be present in 185 (21%) [26]. No other information beyond the presence or absence was recorded in 26% of the 185 patients, and sufficient information to be diagnostically useful was only recorded in ten patients (5.4%). Of patients given a peripheral vestibular diagnosis, the description of the nystagmus conflicted with that diagnosis. This illustrates a knowledge gap in emergency physicians' understanding of nystagmus: what to look for, how to report it, and, most importantly, how to use the findings to their advantage.

Reporting the presence or absence of nystagmus in a dizzy patient is not the key finding. In a patient with an AVS the findings of direction-fixed horizontal nystagmus versus direction-fixed vertical nystagmus versus direction-changing nystagmus have different significances (see below). A recent review illustrates how to use the physical examination in dizzy patients [27].

Multiple studies find that patients with an AVS that superficially appears to be a peripheral process in fact have posterior circulation strokes [28–30]. In one, almost 3% of patients referred to the ENT clinic for vertigo had a missed cerebellar stroke [29]. There are two major reasons that showed missed stroke is an important misdiagnosis. The first is that the underlying vascular mechanism goes untreated, leaving the patient vulnerable to another stroke, and the second is that some patients will develop posterior fossa edema that can be fatal [5]. Although lost opportunity for thrombolysis is often suggested as a third negative consequence of missing a posterior circulation stroke, many of these patients have minor deficits and are not necessarily thrombolysis candidates. Some have an NIHSS of zero [31].

Younger age and dissection as a cause were found to be risk factors for missed cerebellar stroke [32]. Posterior circulation location is a risk factor for stroke misdiagnosis in general [33–35]. To put these data into some context, only a very small proportion (0.18–0.63%) of patients who are seen in the ED diagnosed with a

benign or peripheral vestibular diagnosis return to the ED within 30 days and are hospitalized with a cerebrovascular diagnosis [36–38]. However, because dizziness is so common, this small fraction of a large number would suggest that many thousands of patients have a missed diagnosis of an acute cerebrovascular syndrome (stroke or transient ischemic attack [TIA]) each year.

The other side of the coin is that a lack of recognition of common peripheral vestibular problems (such as BPPV and vestibular neuritis) can result in undertreatment, incorrect treatment, and resource overutilization.

A recent review of misdiagnosis of patients with dizziness suggested five common pitfalls [39]. These are overreliance on a symptom quality approach to diagnosis, underuse of timing and triggers approach, lack of familiarity with key physical examination findings, overweighting traditional factors such as age and vascular risk factors to screen patients, and overreliance on CT. Although stroke is more common in older individuals, young patients do have strokes, a fact that may contribute to misdiagnosis [5, 40, 41].

A New Paradigm to Diagnose Patients with Acute Dizziness: ATTEST

A new diagnostic paradigm which is based on the timing, triggers, and context of the dizzy symptoms might reduce misdiagnosis and decrease unnecessary resource utilization. My personal experience is that it allows one to confidently make a specific diagnosis more frequently than the traditional paradigm. The basic idea is that it is the timing, triggers, evolution, and context of symptoms that should drive the workup rather than the specific words that a patient uses to describe their dizziness [6, 21]. I favor the mnemonic: ATTEST—which stands for A (*a*ssociated symptoms), TT (*t*iming and *t*riggers), ES (bedside *e*xam *s*igns), and T (additional *t*esting as needed).

This new paradigm may seem like a radically new way of approaching the dizzy patient, but this is only because the traditional "symptom quality" approach is so deeply engrained in how this subject has been taught [21]. In fact, using a timing and triggers approach is no different than taking a history in any other patient.

Using this paradigm, there are four timing and triggers categories that are important for emergency physicians (Table 3). In the traditional paradigm, a patient who endorsed "vertigo" would get an evaluation to try to diagnose peripheral vestibular versus central nervous system (CNS) causes of dizziness. This has led to confusion. Physicians tend to treat all patients with peripheral vertigo the same, whereas the two most common by far being BPPV and vestibular neuritis should be treated very differently [7]. The following sections will review the presentation, differential diagnosis, and appropriate testing to make a specific diagnosis for each of the timing and triggers categories.

Acute Vestibular Syndrome (AVS)

Spontaneous AVS is defined as the acute onset of persistent dizziness in association with nausea or vomiting, gait instability, nystagmus, and head-motion intolerance that lasts days or weeks and gradually resolves [6, 21, 42]. Patients are usually symptomatic at the time of assessment, and focused physical examination is often diagnostic. The most common cause is vestibular neuritis (dizziness only) or labyrinthitis (dizziness plus hearing loss or tinnitus) [42]. The most frequent dangerous cause is posterior circulation ischemic stroke, generally in the cerebellum or lateral brain stem [42]. A distant third most common cause is multiple sclerosis [43, 44]. Uncommon causes of an isolated AVS include cerebellar hemorrhage and a number of rare, but often treatable, autoimmune, infectious, or metabolic conditions [43, 45]. The spontaneous AVS is to be distinguished from a triggered AVS, which we will not further discuss in this paper because the cause is usually obvious, such as post-traumatic dizziness or diphenylhydantoin toxicity.

An important concept is that patients with an AVS generally experience worsening of their symptoms with head movement. These *exacerbating* features should not be mistaken for head movement *triggers* that facilitate diagnosis in EVS patients. Confusion on this point probably contributes to difficulty differentiating BPPV from vestibular neuritis [6, 7, 46]. Acute BPPV patients occasionally complain of more persistent symptoms that may be due to repeated triggering symptoms with small, inadvertent head movements or anticipatory anxiety about moving. This can usually be teased out by careful history taking. When such patients lack obvious features of vestibular neuritis or stroke, the Dix-Hallpike and supine roll test can be performed to assess for an atypical, AVS-like presentation of BPPV [47].

Vestibular neuritis is a benign, self-limited, presumed viral, or post-viral inflammatory condition affecting the vestibular nerve and causing spontaneous AVS, similar to Bell's palsy (seventh nerve) but involving the vestibular portion of the eighth nerve. Some cases are associated with inflammatory disorders (e.g., multiple sclerosis or sarcoidosis), but most are idiopathic and possibly linked to herpes simplex infections [48]. The idiopathic form is generally monophasic and resolves over days to weeks. Routine MRI with contrast is generally negative [49]. The diagnosis is usually clinical. A related condition, herpes zoster oticus (Ramsay Hunt syndrome type 2), may present with AVS, usually in conjunction with hearing loss, facial palsy, and a vesicular eruption in the ear or palate [50].

Table 3: Timing- and trigger-based "vestibular[a] syndromes " in acute dizziness[b].

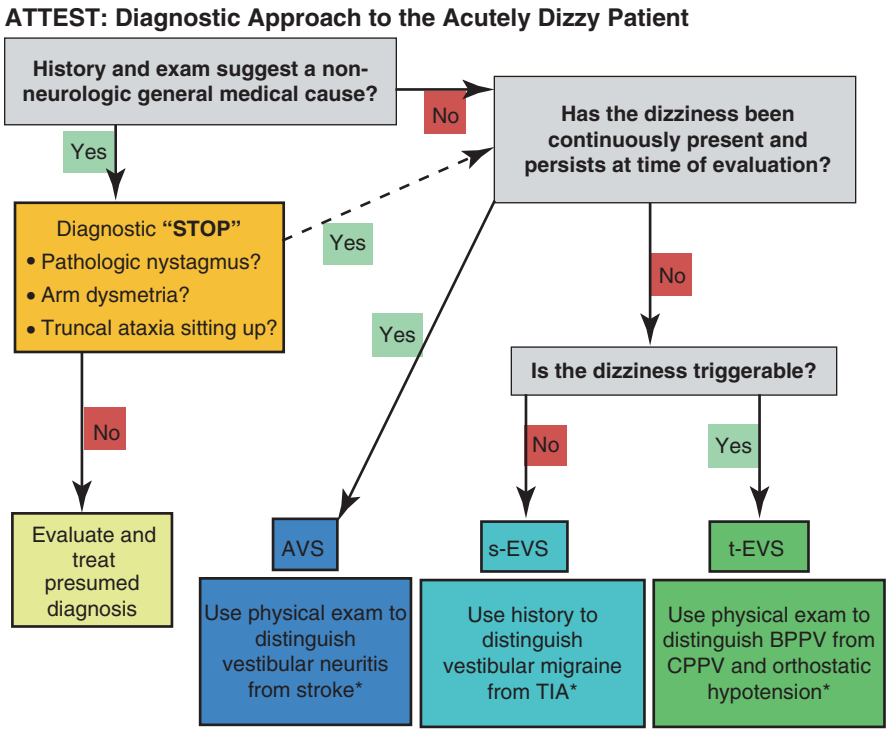
Syndrome	Description	Common causes
AVS	Rapid onset of acute dizziness that lasts days, often associated with nausea, vomiting, and head-motion intolerance	Benign: vestibular neuritis and labyrinthitis Serious: cerebellar stroke
t-EVS[c]	Episodic dizzy episodes triggered by some specific obligate event, usually head movement or standing up and usually lasts less than 1 min	Benign: BPPV Serious: orthostatic hypotension and CPPV
s-EVS	Episodic dizzy episodes that occur spontaneously are not triggered and usually last minutes to hours	Benign: vestibular migraine, Meniere's disease Serious: TIA
CVS	Chronic dizziness lasting weeks to months (or longer)	Benign: medication side effects, anxiety, and depression Serious: posterior fossa mass

AVS acute vestibular syndrome, *t-EVS* triggered vestibular syndrome, *s-EVS* spontaneous vestibular syndrome, *BPPV* benign paroxysmal positional vertigo, *CPPV* central paroxysmal positional vertigo, *TIA* transient ischemic attack, *CVS* chronic vestibular syndrome

[a]Note that the use of the word "vestibular" here connotes vestibular symptoms (dizziness or vertigo or imbalance or light-headedness), rather than that the underlying causes is necessarily vestibular

[b]This table lists the more common causes of these presenting syndromes and is not intended to be encyclopedic

[c]Dizziness is "triggered" when it is brought on from a baseline of no symptoms, as in positional vertigo due to BPPV. This must be distinguished from dizziness that is "exacerbated" from a milder baseline state; such exacerbations are common in AVS, whether peripheral (neuritis) or central (stroke)



*For each vestibular syndrome, only the most common benign and dangerous diagnoses are listed

Posterior fossa strokes may present with AVS mimicking vestibular neuritis (or labyrinthitis if auditory symptoms are present) [51]. The prevalence of cerebrovascular disease in patients presenting to the ED with dizziness is 3–6% [1, 2, 13, 23], but among AVS presentations, it is estimated at ~25% [42]. Almost all (96%) are ischemic strokes, rather than hemorrhages [42, 45]. CT sensitivity for acute ischemic stroke is low and probably worse in the posterior fossa [52–54]. Therefore, CT cannot "rule out" ischemic stroke in AVS, a fact often contributing to misdiagnosis [5, 39, 46]. Importantly, even MRI with diffusion-weighted imaging (DWI) misses 10–20% of strokes in AVS during the first 24–48 h after symptom onset, and repeat delayed imaging (3–7 days post symptom onset) may be required to confirm the presence of a new infarct [42, 55, 56].

Fortunately, the physical examination can help make the distinction between vestibular neuritis and posterior circulation stroke with greater sensitivity than early MRI [55, 56]. These two studies were done by neuro-otologists performing a targeted ocular motor exam consisting of three components—the head impulse test (HIT), testing for nystagmus, and skew

deviation or HINTS—head impulse, nystagmus, test of skew. Another study showed similar accuracy when performed by stroke neurologists [57]. Preliminary evidence suggests that "specially trained" emergency physicians can learn to use nystagmus and head impulse testing [58, 59]. My own anecdotal experience also suggests that with some training, emergency physicians can perform and interpret this examination. However, because this approach has not been fully validated when used by nonspecialists, I have added two additional components that should be a part of the basic evaluation of the acutely dizzy patient anyway—the general neurological examination and testing of gait.

I do not perform these tests in the order of the HINTS mnemonic but rather in the following order:

1. Nystagmus testing
2. Skew deviation
3. Head impulse testing (HIT)
4. General neurological exam, focusing on cranial nerves including hearing, cerebellar testing, and long-tract signs
5. Gait testing

There are two reasons for this sequence. Firstly, I like to start with the least "intrusive" parts of the examination, and, secondly, nystagmus testing is the component that helps the most, in part by its presence or absence and in part by its quality. Once any one component tests "positive" for a central cause, then the patient's disposition (admission for further neurological evaluation) is clear. Although all five components mentioned above are part of a complete examination for a patient with an AVS, causing the patient to feel worse with further intrusive testing (e.g., testing gait that provokes vomiting) after a less intrusive test is positive will not change the disposition and just result in the patient feeling worse.

Furthermore, nystagmus helps to anchor and inform the rest of the process. Essentially all patients with an AVS due to vestibular neuritis will have nystagmus if examined within the first days, so its absence should make one question the diagnosis. To be sure that nystagmus is truly absent, it should be tested with visual fixation removed. Experts state that the absence of nystagmus in a patient examined with their visual fixation removed essentially rules out a vestibular cause for the dizziness [60]. Subspecialists typically use Frenzel lenses to remove visual fixation, neither available nor common practice in emergency medicine practice. A simple solution is to take a large piece of white paper, place it close to the patient's eyes (telling them to "look through the paper"), and examine the nystagmus from the side. This technique is only needed if there is no nystagmus with the basic exam.

If nystagmus is truly absent, then this is unlikely to be a vestibular process, and therefore head impulse test is not useful and may yield false information. It is still important to perform a complete neurological exam with special attention to brainstem and cerebellar function and gait since patients with cerebellar stroke often do not have nystagmus.

The degree (or amplitude) of nystagmus can fluctuate markedly even over hours. This may represent the natural history of the underlying pathology as the CNS accommodates to the abnormal physiology from vestibular neuritis or from medications (e.g., ondansetron or a benzodiazepine) that are often appropriately used in the ED to reduce symptoms but may accelerate the rate at which the nystagmus dampens.

Nevertheless, clinical testing for nystagmus is quite simple. Have the patient look straight ahead in "neutral" or "primary" gaze and observe for eye movements. By convention, the direction of nystagmus is named by the direction of the fast component. With minimal practice this is easy to see and describe. Importantly, it is the details of the nystagmus, not simply its presence that is most diagnostically important. After observing for nystagmus in primary gaze, test for "gaze-evoked" nystagmus by having the patient look to the right and then to the left, each for several seconds, and observe for the presence of nystagmus and the direction of its fast-beating component. The patient only needs to move their eyes 20–30° off-center when testing for gaze-evoked nystagmus because many normal individuals will have a few beats of horizontal nystagmus on full end gaze. This physiologic nystagmus is generally very low amplitude and extinguishes quickly. Table 4 shows the typical findings for patients with the ocular motor examination for patients with the AVS. Direction-changing gaze-evoked nystagmus or nystagmus that is pure torsional or vertical is central in origin (in the setting of an AVS, a stroke).

Skew deviation (a vertical misalignment of the eyes due to imbalance in the gravity-sensing pathways) is not very sensitive (30%) but is very specific (98%) for a brain stem lesion [42]. For this examination, the examiner uses the "alternate cover" test. With the patient looking directly at the examiner's nose, the physician alternately covers the right eye and then the left eye and continues alternating back and forth, approximately every 2 s. In patients with skew deviation, each time the covered eye is uncovered, there will be a slight vertical correction (one side corrects upward and the other corrects downward). The amplitude of correction is small, 1–2 mm; therefore, it is key for the examiner to focus on one eye (either one), rather than following the uncovered eye. A normal response is no vertical correction, and an abnormal response should be considered a stroke in patients with an AVS.

The next component is the HIT, a test of the vestibulo-ocular reflex (VOR), only described in 1988 [61]. Standing in front of the patient, the examiner holds the patient's head by each side and instructs the patient to maintain their focus on the examiner's

Table 4: Acute vestibular syndrome oculomotor physical findings.

Oculomotor exam component	Peripheral (usually vestibular neuritis)	Central (usually posterior circulation stroke)
Nystagmus (neutral gaze and gaze to the right and left)	Dominantly horizontal and direction-fixed, beating away from the affected side	Direction-changing horizontal or dominantly vertical and/or torsional and then central[b] (often mimics peripheral)
Test of skew (alternate cover test)	Normal vertical eye alignment (i.e., no skew deviation)	Often mimics peripheral; if skew deviation is present, then central[c]
Head impulse test (HIT)	Unilaterally abnormal toward the affected side (presence of a corrective saccade)[a]	Usually bilaterally normal (no corrective saccade)

[a]Strokes in the anterior inferior cerebellar artery (AICA) 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 HIT, this is also suspicious for a central lesion if nystagmus is present (AICA stroke or Wernicke's syndrome)
[b]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 AVS patient should be considered to be central (a stroke)
[c]Skew deviation, demonstrated by the bedside alternate cover testing, is very rare in peripheral vestibular cases; its presence should be considered to be central (a stroke, often in the brainstem)

nose and to keep their head and neck loose. Then the examiner very quickly turns the patient's head about 10–20 °, using a lateral to center motion. The normal (individuals with normal vestibular function) response is that the patient's focus will stay locked on the examiner's nose. The presence of a corrective saccade (the eyes move with the head and then snap back in a fast corrective movement to the examiner's nose) is a "positive" test (abnormal VOR), which generally indicates a peripheral process, usually vestibular neuritis. The absence of a corrective saccade in AVS is consistent with a stroke.

It may seem counterintuitive that a normal finding predicts a dangerous disease. This is why the HIT is only useful in patients with the AVS and nystagmus. If an acutely dizzy patient with an AVS does not have nystagmus, it's very unlikely to be vestibular, and therefore the HIT (which is meant to distinguish neuritis from stroke) becomes far less useful and usually misleading. Similarly, if the HIT were done in a patient with dizziness from urosepsis or dehydration, the test will be negative, i.e., worrisome for a stroke.

Patients with cerebellar stroke have a negative (normal) HIT [30, 62]. This is because the circuit of the VOR does not loop through the cerebellum. On the other hand, occasional patients with posterior circulation stroke will have a falsely "positive" (abnormal) HIT, usually from a lateral brainstem infarct involving the location where the vestibular nerve enters the brainstem. These strokes are uncommon and involve the anterior inferior cerebellar artery stroke (AICA) territory or an infarction directly involving the inner ear (labyrinthine stroke) itself. In both situations, acute hearing loss usually occurs. Adding a bedside test of hearing ("HINTS plus") will help to pick up the occasional AICA stroke [63]. This last point is important because traditional teaching is that if both hearing and dizziness coexist, the problem is peripheral (in the labyrinth). However, the blood supply to the labyrinth is due to ischemia of branches of the AICA, so this co-involvement of hearing and dizziness can occur from a stroke [64–66]. The relative frequency of this occurring from a peripheral cause (true labyrinthitis) as opposed to stroke (AICA territory) is unknown.

A recent article with attached video clips reviews these physical examination findings [27]. Because HINTS testing has not been fully validated when done by nonspecialists, I recommend adding two additional components of the HINTS testing—brainstem and cerebellar testing and gait testing. Key elements include testing for pupillary function, facial motor and sensory symmetry, and dysarthria. Lateral medullary stroke (Wallenberg's syndrome), an important cause of the AVS, merits special attention. These patients have dysarthria, dysphagia, or hoarseness due to lower cranial neuropathy and may have Horner's syndrome with mild ptosis and anisocoria that may only be evident in dim light (so that the normal larger pupil fully dilates, making the difference in pupil size more apparent) [67]. Common physical examination findings are hemifacial decreased pain and temperature sensation. Routine testing of only light touch can miss this finding.

Finally, if all four initial components of the exam (nystagmus, skew deviation, HIT, and general neurological exam) are nondiagnostic, gait testing must be performed. Ideally have the patient walk unassisted, but for patients too symptomatic to walk, test for truncal ataxia by asking the patient to sit upright in the stretcher without holding onto the side rails. A patient who cannot walk or sit up unassisted is unsafe for discharge, and an AVS patient who is unable to walk is more likely to have had a stroke than vestibular neuritis [30].

Imaging is not very useful in patients with the AVS. CT is a poor test for posterior circulation stroke [52–54]. MRI, even with DWI, misses 10–20% of strokes in AVS during the first 24–48 h [42, 55, 56]. In small brainstem strokes, MRI, with DWI, can still miss upward of 50% when tested within 48 h [56].

Importantly, half of those small strokes were not due to small vessel disease, but due to vertebral artery atherosclerosis or dissection. Therefore, in patients with the AVS, the physical examination is more sensitive than MRI.

An Italian ED study (in which the emergency physicians used Frenzel lenses to test for nystagmus) exploited elements of this bedside exam and showed that it decreases both CT use and hospitalization [59]. However, another survey study found that many emergency physicians clearly do not understand or feel confident in HINTS testing and overuse CT [41]. This same study showed that emergency physicians tend to overvalue the dizziness type in making a diagnosis. Although traditional vascular risk factors underperform HINTS and neurological exam testing [19, 63], emergency physicians still value them over bedside testing [41].

Triggered Episodic Vestibular Syndrome (t-EVS)

Patients with t-EVS have short-lived episodes of dizziness lasting seconds to a few minutes, depending on the underlying etiology. There is an "obligate" trigger, meaning that each time the specific trigger occurs, the dizziness follows. Common triggers are changes in head position or body posture, especially arising from the lying or seated position to standing. Vomiting can occur and may lead patients to overestimate episode duration. Clinicians must distinguish triggers (provoke new symptoms not present at baseline) from exacerbating features (worsen preexisting baseline symptoms), since head movement will exacerbate acute vestibular dizziness of any cause. Common etiologies are BPPV and orthostatic hypotension. Dangerous causes include central (neurologic) mimics of BPPV and serious causes of orthostatic hypotension such as internal bleeding or sepsis with relative hypovolemia. Since the symptoms can be triggered, the physician should be able to re-create them at the bedside.

Benign paroxysmal positional vertigo, the most common vestibular cause of dizziness with a lifetime prevalence of 2.4% and increasing incidence with age [68], results from mobile crystalline debris in one or more semicircular canals ("canaliths"). Classical symptoms are repetitive brief, triggered episodes of rotational vertigo lasting more than a few seconds and less than a minute [69, 70]; non-vertiginous symptoms are frequent [25]. The diagnosis is confirmed by reproducing symptoms using canal-specific positional testing maneuvers (Table 5) [70–72]. Since the offending canal(s) are generally not known in advance, a sequence of multiple diagnostic maneuvers is typically performed starting with the Dix-Hallpike maneuver because this tests the posterior canal, which is by far the most common involved [60]. A detailed recent review of these exam maneuvers includes instructive video clips [27]. Despite the fact that BPPV is quite common, a majority of emergency physicians report that they do not use the Dix-Hallpike (diagnostic) or Epley (therapeutic) maneuvers in practice [41]. Once the correct canal is identified by these maneuvers, bedside treatment with canal repositioning maneuvers can follow [70].

Rarely, central paroxysmal positional vertigo (CPPV) mimics BPPV. This is usually caused by posterior fossa lesions including neoplasm, infarction, hemorrhage, and demyelination. Factors that help to distinguish BPPV from CPPV are summarized in Table 6 [73].

Orthostatic hypotension affects 16% of adults [74] and accounts for 24% of acute syncopal presentations [75]. Classical symptoms are brief presyncope on arising, but vertigo is common [24]. Orthostatic hypotension is a sustained decline in blood pressure of at least 20 mmHg systolic or 10 mmHg diastolic within 3 min of standing [76]. Recent work suggests optimal

Table 5: Positional nystagmus findings in triggered, episodic vestibular syndrome (t-EVS).

Positional tests in t-EVS	BPPV (posterior canal)	BPPV (horizontal canal)	Central
Dix-Hallpike test (diagnostic test)	Upbeat-torsional[a] 5–30 s No spontaneous reversal	None[b]	Variable direction (downbeat or horizontal; almost never upbeat) Variable duration (often >90 s) No spontaneous reversal
Supine roll test (diagnostic test)	None[b]	Pure horizontal[c] 30–90 s Spontaneous reversal typical	Variable direction (downbeat or horizontal; almost never upbeat) Variable duration (often >90 s) No spontaneous reversal

BPPV benign paroxysmal positional vertigo

[a]The nystagmus of posterior canal BPPV will have a prominent torsional component, and the 12 o'clock pole of the eye will beat toward the down-facing (tested) ear. Although the nystagmus will reverse on arising from the Dix-Hallpike position, there will be no spontaneous reversal

[b]Although the Dix-Hallpike test is fairly specific to posterior canal BPPV and the supine roll test to horizontal canal 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. The nystagmus may be considerably weaker and less evident than when using the "correct" maneuver

[c]The nystagmus of horizontal canal BPPV may beat toward the down-facing ear or away from it. The nystagmus will often crescendo and then slow down and reverse spontaneously even without moving the head. When the opposite side is tested, the nystagmus will usually beat in the opposite direction (e.g., if right-beating initially with the right ear down and then left-beating initially with the left ear down)

Table 6: Characteristics of patients with t-EVS that suggest a central mimic (CPPV) rather than typical BPPV.

1. Presence of symptoms or signs that are NOT seen in BPPV
(a) Headache
(b) Diplopia
(c) Abnormal cranial nerve or cerebellar function
2. Presence of nystagmus without dizziness
3. Atypical nystagmus characteristics
(a) Down-beating nystagmus[a]
(b) Nystagmus that beats in different directions on repeat testing
4. Poor response to therapeutic maneuvers
(a) Unable to cure patient with typical 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 also seen with central causes, it is safer for emergency physicians to consider this finding to be *always* worrisome prompting imaging, consultation, and/or referral

cutoffs should be adjusted based on baseline blood pressure [77]. However, the orthostasis can be delayed (onset >10 min), and the duration of monitoring remains controversial [78–80].

Emergency physicians are familiar with the most common causes of acute orthostasis such as medications and hypovolemia. Strong bedside predictors of moderate blood loss are postural dizziness so severe as to prevent standing or a postural pulse increment >30 beats per min, but the sensitivity of these findings is only 22% [81]. Furthermore, the benign postural orthostatic tachycardia syndrome (POTS) produces similar clinical findings [82]. The absence of tachycardia or even relative bradycardia can occur with intraperitoneal blood such as ruptured ectopic pregnancy [83].

Benign paroxysmal positional vertigo produces dizziness upon arising in 58% [68], which can mimic orthostatic hypotension [84], and often goes undiagnosed in the elderly [25, 85]. Alternatively, orthostatic hypotension may be incidental and misleading, especially in older patients taking antihypertensive medications [86]. In patients with postural symptoms, BPPV and orthostatic hypotension can usually be differentiated by considering other positional triggers such as rolling over in bed or reclining, both of which are common in BPPV but should not occur with orthostatic hypotension.

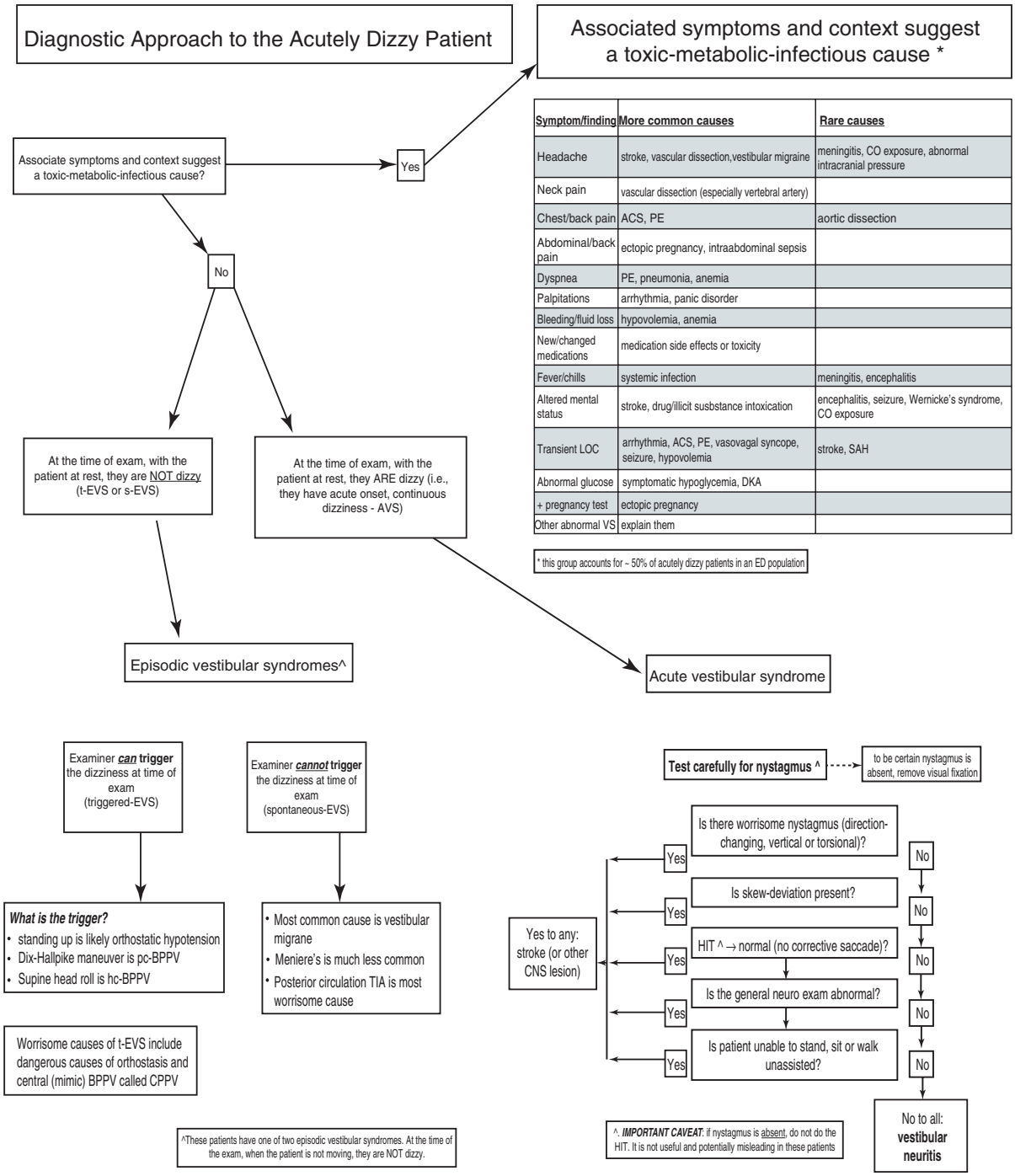
Benign paroxysmal positional vertigo notwithstanding, orthostatic dizziness and orthostatic hypotension are not always related [74, 87]. Orthostatic dizziness without systemic orthostatic hypotension has been reported with hemodynamic TIA due to vascular stenosis [88] and in patients with intracranial hypotension [89]. Neurological evaluation is probably indicated for patients with reproducible and sustained orthostatic dizziness but no demonstrable hypotension.

Spontaneous Episodic Vestibular Syndrome (s-EVS)

The s-EVS is marked by recurrent, spontaneous episodic dizziness that ranges in duration from seconds to days but usually lasts minutes to hours. Most patients are therefore asymptomatic at the time of clinical assessment, and if they are, one cannot provoke an episode at the bedside (because it is not "triggerable"), so the evaluation relies almost entirely on history. The most common benign cause is vestibular migraine [90–92] followed by Ménière's disease [91]. The most common dangerous cause is vertebrobasilar TIA [93]. Other causes of the s-EVS include reflex (e.g., vasovagal) syncope [94] and panic attacks [95]. Uncommon dangerous causes of s-EVS are cardiovascular (cardiac arrhythmia, unstable angina pectoris, pulmonary embolus), endocrine (hypoglycemia, neurohumoral neoplasms), or toxic (intermittent carbon monoxide exposure). Diagnosis is not difficult when cases are typical. Unfortunately, classical features such as frank loss of consciousness in reflex syncope [96], headache in vestibular migraine [97], and fear in panic attacks [98] are frequently absent. Atypical case presentations probably contribute to diagnostic confusion in patients with transient neurological attacks [99].

Definite vestibular migraine diagnosis requires recurrent attacks with vestibular symptoms, a history of migraine according to the International Classification of Headache Disorders, and migraine symptoms during at least half of the attacks [91]. Attack duration in vestibular migraine ranges from seconds to days [91]. Headache is often absent with the attack; when headache does occur, it may begin before, during, or after the dizziness and may differ from the patient's other "typical" migraine headaches [91, 100]. Nausea, vomiting, photophobia, phonophobia, and visual auras may accompany vestibular migraine. Hearing loss or tinnitus sometimes occurs [101], creating some overlap between vestibular migraine and Ménière's disease [102]. If present, nystagmus can be of a peripheral, central, or mixed type [100]. The diagnosis is normally made based purely on clinical history and the exclusion of alternative causes [91].

Patients with Ménière's disease classically present with episodic vertigo accompanied by unilateral tinnitus and aural fullness, often with reversible sensorineural hearing loss. Episodes typically last minutes to hours. Only one in four initially present with the complete symptom triad [103], and non-vertiginous dizziness is common [104]. Definite Ménière's disease requires at least two spontaneous episodes of vertigo lasting at least 20 min, audiometrically documented hearing loss (at least once, whether



transient or persistent), and tinnitus or aural fullness in the affected ear, with other causes excluded [91].

Reflex syncope (also called neurocardiogenic or neurally mediated syncope) includes vasovagal syncope, carotid sinus hypersensitivity, and situational syncope (e.g., micturition, defecation, cough) [105]. Those who faint usually experience prodromal symptoms; presyncopal spells without loss of consciousness substantially outnumber spells with syncope [94]. Dizziness is the most common presyncopal symptom, and it may be of any type, including vertigo [106]. Presyncopal symptoms usually last 3–30 min [107]. Diagnosis is based on clinical history, excluding dangerous mimics (especially arrhythmia), and can be confirmed by formal head-up tilt table testing [82].

The principal dangerous diagnosis for s-EVS is TIA [93]. Although for years isolated vertigo was considered to not be due to TIA, recent evidence strongly suggests that TIA can present

with dizziness, even isolated attacks of vertigo [108]. TIAs can present with isolated episodes of dizziness weeks to months or even years prior to a completed infarction [109, 110]. Dizziness is the most common symptom in basilar artery occlusion [111] and occurs without other neurological symptoms in 20% of cases [112]. Dizziness is the most common presenting symptom of vertebral artery dissection [113], which affects younger patients, mimics migraine, and is easily misdiagnosed [5]. Because 5% of TIA patients suffer a stroke within 48 h, prompt diagnosis is critical [114]. Patients with posterior circulation TIA may have an even higher stroke risk than those with anterior circulation spells [115, 116]. Rapid treatment lowers stroke risk after TIA by about 80% [117, 118].

Cardiac arrhythmias should also be considered in any patient with spontaneous EVS, particularly when syncope occurs [24]. Although some clinical features may increase or decrease the odds of a cardiac cause [105], additional testing (e.g., cardiac loop recording) is often required to confirm the final diagnosis [82].

Putting It All Together: An Overarching Algorithm

Taking a history of a dizzy patient should be no different than taking a history in nearly any other patient. The timing, triggers of the dizziness (and not the descriptor used), as well as the evolution of the symptoms, associated symptoms, and epidemiologic context inform the differential diagnosis. Bedsides, physical examination can frequently establish a specific diagnosis. This newer paradigm (see Figure on Page 11) has not yet been validated in large numbers of ED patients treated by emergency physicians, but current evidence and experience suggest that this is possible.

Conclusions

Dizziness, vertigo, and unsteadiness are extremely common complaints caused by numerous diseases that span organ systems. Diagnosis can therefore be difficult, a fact leading to over utilization of resources and misdiagnosis. The current paradigm used by most physicians is based on symptom quality, a paradigm created 40 years ago; a newer paradigm, based on timing and triggers, is more consistent with current evidence. History and physical examination is more accurate, more efficient, and more likely to result in a specific diagnosis than the traditional paradigm.

Pearls and Pitfalls

- The timing and triggers of a patient's dizziness are *much* more important than the word that a patient uses to describe their dizziness (e.g., "vertigo" versus "light-headed" versus "imbalance").
- In patients with an acute vestibular syndrome (nausea or vomiting, gait instability, nystagmus, and head-motion intolerance that lasts days or weeks and gradually improving), physical examination allows better distinction between vestibular neuritis and stroke (the two most common causes) than MRI during the first 2 days of illness.
- Patients with BPPV can be diagnosed and treated using bedside maneuvers (Dix-Hallpike and Epley maneuvers) without the need for imaging or consultation.

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An Unusual Case of Vertigo: The Usefulness of Nystagmus Examination

Carlotta Casati[1], Matteo Castelli[1], Andrea Pavellini[1], Cosimo Caviglioli[1], Rudi Pecci[2]

A 50-year-old gardener presented to our Emergency Department (ED) after the onset of a sudden objective vertigo while standing up during work, accompanied by imbalance, nausea, and vomiting. Neither tinnitus nor other aural symptoms were present, but, if asked, he also complained of a mild headache. He had no medical background of interest except for a known hypertension, left untreated, and a possible history of migraine for which he was admitted to the ED 1 year before. He denied recent viral infections or trauma. He was an active smoker (35 pack years), but denied alcohol or sympathomimetic intake; he was not taking any medication and had no allergies. Clinical examination revealed no neurological deficit, in particular neither dysmetria nor motor or sensitive deficit, and the patient's physical examination was normal except for a right beating horizontal nystagmus in primary position. Due to vomiting and overt vagal signs, upright position was impossible to evaluate. The blood pressure was found high and symmetric in both arms (160/100 mmHg), but the other vital signs were normal (HR 80 beats/min, SpO_2 99% in FiO_2 21%, temperature 36.5°C). The National Institutes of Health Stroke Scale (NIHSS) was 0 [1]. The absence of otological and aural symptoms, as well as the absence of a recurrent vertigo in the history of the patient, was not compatible with Menière disease. A vestibular migraine could be excluded too because of the lack of diagnostic criteria of migraine [2]. The patient was free from neurological signs except for nystagmus and referred imbalance. These features could be typical of an acute vestibular syndrome of peripheral origin; however, a more detailed nystagmus evaluation was needed. The STANDING, a recently developed diagnostic algorithm for the evaluation of patients with acute vertigo, was used to evaluate the patient [3] (Fig. 1).

Frenzel goggles confirmed the presence of a spontaneous, i.e., not triggered by head movements, horizontal, right beating, and unidirectional nystagmus, thus excluding a benign paroxysmal positional vertigo (BPPV). As indicated by the diagnostic algorithm, to differentiate a peripheral from a central disease, a head impulse test (HIT) was performed [4]. The HIT test was negative, strongly suggesting a central origin. Therefore, a first-level bedside eco-color Doppler of the neck vessels was obtained that showed the absence of blood flow in the right vertebral artery.

These findings prompted the execution of a CT angiography of cervical and intracranial vessels that showed a sub occlusive stenosis of the right vertebral artery at the onset, probably caused by a spontaneous dissection (Fig. 2). To confirm the diagnosis, the patient underwent a DWI sequences MRI that clearly revealed a cerebellar stroke (Fig. 3a, b). Systemic thrombolysis was not initiated, because of the delay of presentation to the ED (more than 6 h from the onset of symptoms to ED presentation), and ASA was started.

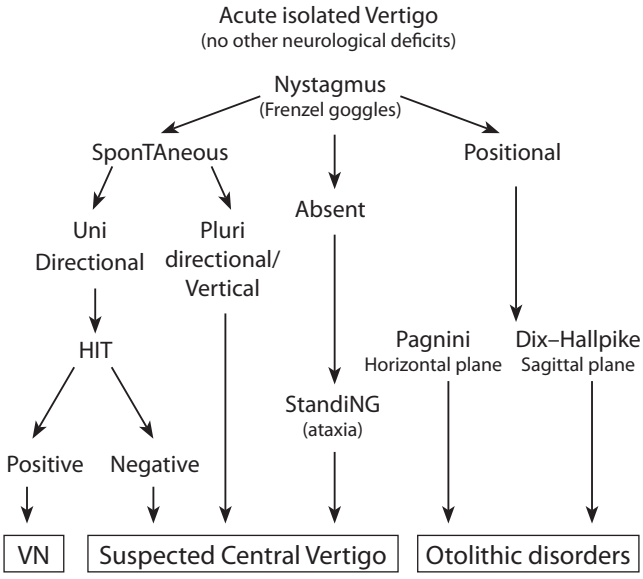

Fig. 1: Diagram of STANDING approach. *HIT* head impulse test, *VN* vestibular neuritis

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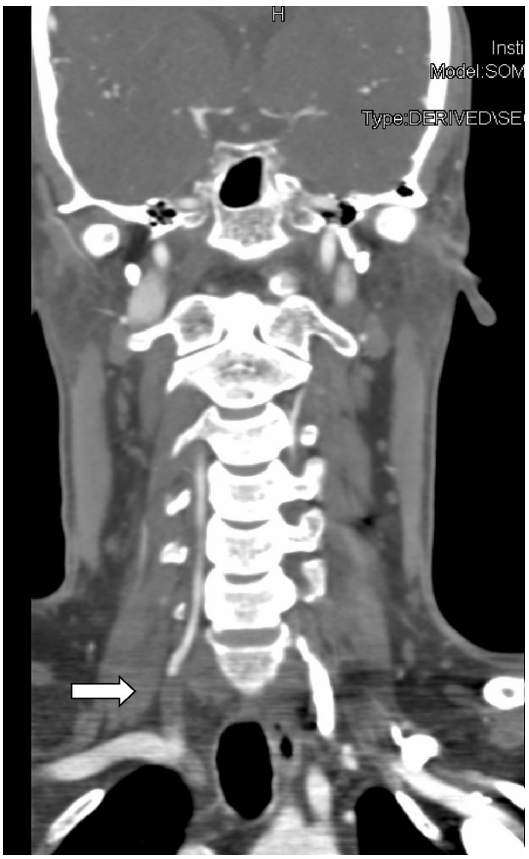

Fig. 2: CT angiography of cervical and intracranial vessels showing a sub occlusive stenosis of the right vertebral artery (*white arrow*) at the origin.

The patient was admitted to the neurologic ward. The next day the patient's neurologic state worsened, and a new onset dysmetria of the left arm, left hemi-anaesthesia of the face, left VI and VII cranial nerves palsy, and left hearing loss were detected. The patient underwent new MRI, and acute lesions in the left cerebellar peduncle were found. An anticoagulant therapy with low molecular heparin was started instead of antiplatelet agents, and after 3 days, it was switched to an oral anticoagulants therapy. The patient underwent rehabilitation program, and when he was discharged, he was able to stand and walk with aid, the left dysmetria improved, while the VI cranial nerve palsy, the left hemi-anaesthesia of the face, and the left hearing loss, as the nystagmus, were still present.

Compliance with ethical standards

Conflict of interest None.

Statement of human and animal rights All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

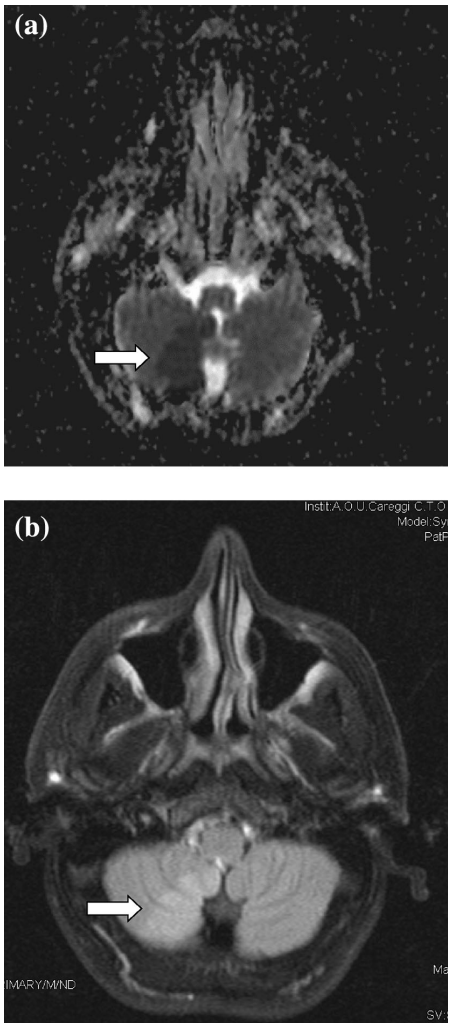

Fig. 3: (**a**) Brain DWI-MR showing a right cerebellar ischaemic lesion in the PICA territory (*white arrow*). (**b**) Brain T2 sequences MRI showing the same lesion.

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Neuroendocrine Tumour of the Middle Ear: A Case Report

Gautam Khaund[1], Nayanjyoti Sarma[1], Ronica Baruah[2]

Neuroendocrine tumour of the middle ear is a rare entity. Here we present a case of a 50-year-old male who presented with a polyp in the left external auditory canal. Surgical excision followed by immunohistochemistry confirmed it to be a neuroendocrine tumour.

Keywords: Adenoma of middle ear, Carcinoid tumour, Adenocarcinoid, Adenomatoid tumour

Case Report

Our case is a 50-year-old male, who presented with pain in the left ear since 1 week at the Out-patient Department of Nightingale Hospital, Guwahati. One examination, an aural mass in the left external auditory canal protruding to the external auditory meatus was seen. Pure tone audiometry revealed profound mixed hearing loss in the affected side. HRCT temporal bone was advised and revealed low dense component in the left mastoid antrum, aditus and middle ear extending to the external auditory canal with the ossicles completely embedded within the low dense components. Routine haematological investigations were normal and the patient was put for surgery. Modified Radical Mastoidectomy was performed. Granulation tissue at mastoid antrum, aditus and middle ear cavity and polyp at the external auditory canal was excised. The entire tissue was sent for histopathological examination. Histological examination of the excised tumor showed an epithelial neoplasm with predominantly solid and trabecular pattern embedded in fibrovascular stroma. The tumour cells were uniform, have round nuclei with salt-and-pepper chromatin, eosinophilic cytoplasm. No evidence of mitosis or necrosis was noted. An immunohistochemical evaluation showed strong positivity for Pan CK, synaptophysin and weak positivity for chromogranin. Ki67 index was low and S-100 was negative. Final diagnosis of middle ear adenoma with neuroendocrine differentiation was made (Figs. 1, 2, 3).

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Discussion

Neuroendocrine tumour are rare tumour arising from enterochromaffin or Kulchitsky cells mostly found in gastrointestinal tract, lungs and bronchi [1]. The occurrence of NET in head and neck region is rare. When affected it mostly affects the larynx followed by middle ear [2, 3]. It accounts for less than 2% of all middle ear tumours. It mostly involved the middle ear cavities engulfing the ossicles which may extend to mastoid antrum and auditory canal [4, 5]. The patient usually presents with unilateral hearing loss, tinnitus and occasional ear discharge [6]. Unlike in this case, the tumour usually remains confined to the middle ear cavity. It is usually fragmented, soft, rubbery and white to gray-tan. The tumour can be sub-divided into TC, AC, smCC, combined smCC with non-small cell carcinoma and those with neurological origins (paraganglioma) according to the 2005 WHO classification of head and neck tumours [7–10]. Immunohistochemistry shows tumour positive for Pan CK and synaptophysin. Differential diagnosis includes paraganglioma, ceruminous adenoma, metastatic adenocarcinoma and meningioma [6].

Surgical excision of the tumour through modified radical mastoidectomy is the treatment of choice [11]. Recurrences can be seen (up to 15%), especially if the ossicular chain is not removed [6]. No sufficient data exist regarding the role of chemo-radiation in middle ear adenoma. Conventional radiotherapy was used earlier either alone or in combination with surgery and chemotherapy. However, their efficacy is yet to be proved [12, 13]. In a review by Furuta et al. [14], it was proposed that radiation and chemotherapy can be performed as post-operative adjuvant therapy when a complete resection is difficult and surgery is ineffective.

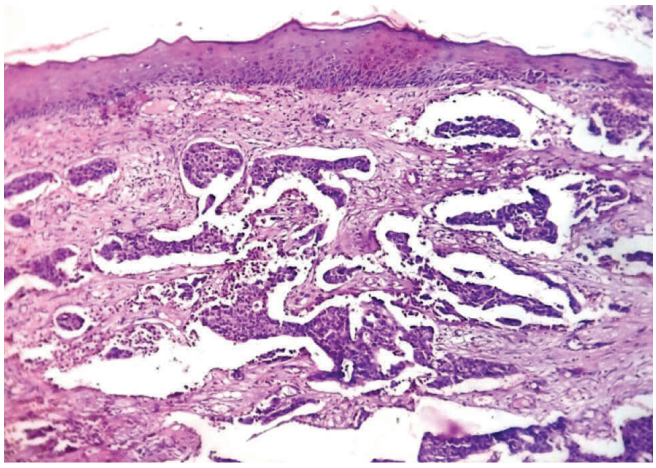

Fig. 1: H&E stained sections ×10 showing tumour cells in solid islands and trabecular arrangement.

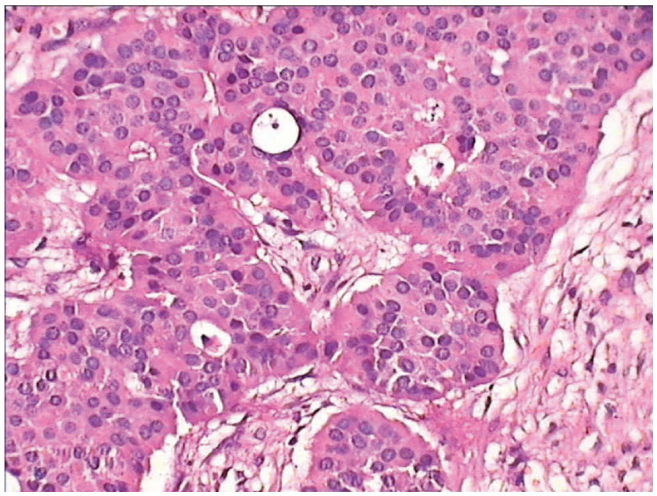

Fig. 2: H&E stained sections ×40, tumour cells showing uniform round nuclei with salt and pepper chromatin and eosinophilic cytoplasm.

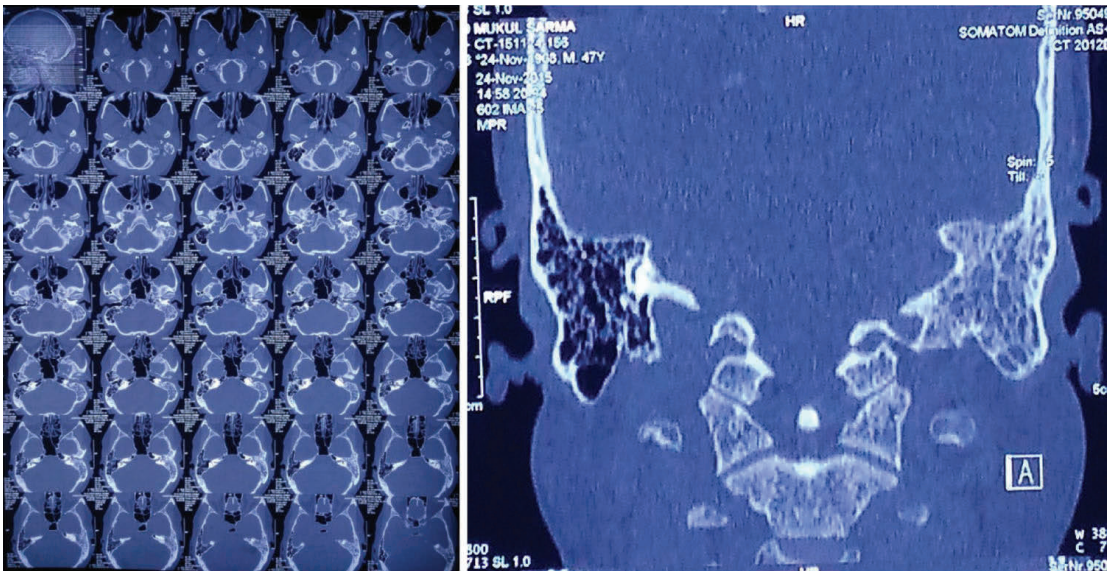

Fig. 3: HRCT temporal bone showing low dense component in the left mastoid antrum, aditus and middle ear extending to the external auditory canal.

Conclusion

Neuroendocrine tumour of the middle ear is a rare presentation. When present surgical excision is the treatment of choice. Diagnosis should be confirmed by immunohistochemistry postoperatively. The role of chemo-radiation in the treatment of neuroendocrine tumour of the middle ear is controversial.

Compliance with Ethical Standards

Conflict of interest All the authors declares that they have no conflict of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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