

Dizziness in the emergency department: an update on diagnosis

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Summary

This review aims to assist emergency physicians in finding the underlying aetiology when a patient presents with dizziness to the emergency department. After reading this review, the emergency physician will be able to consider the most relevant differential diagnoses and have an idea about dangerous aetiologies that require immediate action. The emergency physician will also know what diagnostic steps need to be taken at what time, such as the three-component HINTS Test (Head Impulse, Nystagmus, and Test-of-Skew), which helps with distinguishing central from peripheral causes of the acute vestibular syndrome. Furthermore, episodic vestibular syndromes and chronic vestibular syndromes are discussed in detail. The five most frequent categories of dizziness are vasovagal syncope / orthostatic hypotension (22.3%), vestibular causes (19.9%), fluid and electrolyte disorders (17.5%), circulatory/pulmonary causes (14.8%) and central vascular causes (6.4%).

Given that it would neither be economical nor practical to send all patients to specialists from the start, we present general guidelines for the diagnostic workup of patients presenting with dizziness to the emergency department. This review will focus on epidemiology, aetiologies, differential diagnoses and diagnostics. Treatment is described in a separate article.

Key words: dizziness, emergency, vertigo, vestibular, HINTS

Epidemiology

Dizziness and vertigo are responsible for approximately 5% of unselected presentations to emergency departments or outpatient clinics [1–3]. Among all major symptoms, dizziness is regarded as the third most common in general medical practice [4, 5], with a 30% lifetime prevalence [6]. Dizziness and vertigo, on their own or in association with presyncope or a feeling of unsteadiness, account for

approximately 10 million outpatient visits per year in the United States [5, 7], with approximately one fourth of these patients admitted to the emergency department [3, 5]. This does not include the diseases where another cardinal symptom is the main problem and dizziness is only an accompanying symptom [8, 9]. Although patients rarely need to be referred to the emergency department, the underlying pathology can be serious. According to a population-based study in the United States, 35% of vestibular strokes are missed at initial evaluation [10]. Stroke has been reported to be present in 0.7 to 3.2% of emergency department patients with dizziness [11]. Misdiagnosis has a serious impact on treatment, quality of life and patient outcome.

Aetiology

The aetiologies underlying dizziness can be diverse. On the basis of a publication from a nationally representative sample comprising 3083 patients with dizziness [3], we have divided the 22 single aetiologies of dizziness into a total of 12 different categories. Some of these categories had already been proposed in the original publication [3], others we derived by subsuming different subcategories into one category. For example, we formed the umbrella category “central vascular causes” for all transient ischaemic attacks and strokes, no matter whether the strokes were due to ischaemia or to the various types of bleeding that were associated with stroke symptoms. On the basis of this classification, the majority of cases of dizziness can be attributed to vasovagal syncope or orthostatic hypotension (22.3%), followed by vestibular causes (19.9%), fluid and electrolyte disorders (17.5%), circulatory/pulmonary causes (14.8%), central vascular causes (6.4%), anaemia (5.4%), psychosomatic causes (4.2%), hypoglycaemia (3.6%), migraine (3.2%), intoxication or substance withdrawal (2.4%), inflammatory lesions in multiple sclerosis (0.3%), and aortic dissection (0.06%). Up to 50% of stroke patients with persistent acute vertigo cannot be correctly

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diagnosed with neurological workup because they lack other focal neurological symptoms [5].

A large body of recent literature suggests a change of paradigm in how to assess patients with vertigo or dizziness, by avoiding questions about the type of dizziness. Instead, a range of types of evidence, including history, clinical examination, laboratory results, an electrocardiogram and, if necessary, brain imaging, should be combined [12, 13]. Dizziness may not be the main problem. Rather, it may be a co-symptom of a more severe pathology. In this case, the cardinal symptom might be something else, such as chest pain, neck pain, tachy- or bradycardia or dyspnoea. In such cases, the underlying pathology might be myocardial infarction, pulmonary embolism, spontaneous pneumothorax, dissection of the aorta or extracranial arteries, or cardiac rhythm disorders. It is worth keeping all these underlying aetiologies in mind.

Definition of dizziness

The definitions of vertigo and dizziness are often vague and inconsistently used [14]. A categorisation was proposed by an international committee for the classification of vestibular disorders [15]. According to this classification [15], vertigo is a feeling of self-motion even if there is no self-motion, or a feeling of distorted self-motion even though self-motion is normal. In contrast, dizziness is a feeling of disturbed orientation in space with a normal sense of motion [15]. In this review, as with other classifications [14, 15], we use dizziness as an umbrella term, and vertigo is subsumed under this umbrella term.

History-taking in the emergency department

Clinicians focus on disease history by asking about the type of dizziness and the severity of symptoms. History-taking should be combined with an initial focus on the patient's vital parameters to form an immediate clinical impression. It should focus on the following points.

1. Is the patient conscious and are respiration and circulation stable? As dizziness could be a sign of hypotension or hypoxia, it is of tremendous importance to spot the unstable patient and initiate life-saving procedures. Impaired consciousness could be a sign of low blood glucose or intoxication, a severe metabolic disorder or an acute stroke, either embolic or caused by intracerebral bleeding. In addition to a rapid clinical examination, assessment of the patient's level of consciousness and the respiratory rate, and questions about the environment (history of intoxication) are important first steps.
2. Did the patient have a trauma or a thunderclap headache or is the patient's consciousness level reduced without any reasonable explanation? In a patient with dizziness after trauma, the head and neck should be assessed and stabilised rapidly to avoid further damage.
3. Does the dizzy sensation have triggers, when does it occur, how long does it last, and does it recur? These are all necessary questions, because according to recent studies, asking only about the type of dizziness (rotational vertigo, sway vertigo) is associated with inadequate bedside diagnosis [10, 12, 16]. For that rea-

son, clinical decision rules based on available medical history and clinical examination were suggested [17]. Table 1 provides an example of important questions in this process. Taking into account timing, duration and recurrence, as well as making a short clinical examination to determine whether the patient has a nystagmus, can help to better understand the dizzy sensation of the individual patient [18–20]. This process ultimately has therapeutic consequences. Triggers such as positional changes when getting up or turning the head, stress reactions, or arm movements should be investigated [18–20]. If positional changes trigger sensations of dizziness, this is referred to as a positional vestibular syndrome. Based on timing and triggers of vestibular symptoms, these may be classified as acute, episodic or chronic vestibular syndromes. An acute vestibular syndrome has an acute onset and typically lasts days to weeks, whereas an episodic vestibular syndrome lasts seconds to hours and may occur only once, but generally occurs repeatedly. In an episodic vestibular syndrome, symptoms completely resolve intermittently, whereas a chronic vestibular syndrome has persisting symptoms and lasts months or longer [18].

4. Is the ear affected? The medical history should include questions about upper respiratory tract infections, hearing loss and tinnitus, as well as ear infections, because of the close anatomical connection of the inner ear and the eighth cranial nerve [19]. A painful ear could be caused by otitis media [19] or externa, both possibly leading to the sensation of dizziness, in particular in the setting of prior ear surgery. Tinnitus, a perception of sound in the absence of an external source of sound, may occur on its own or in combination with other signs and symptoms, for example, as a part of Menière disease (episodes of dizziness, hearing loss, tinnitus), which has a peripheral aetiology [19]. One should also ask about medications that are known to be associated with hearing loss or dizziness, such as aminoglycosides [19].
5. Does the patient have thromboembolic risk factors such as diabetes, hypertension, smoking or high cholesterol [19, 20]?
6. Does the patient have a history of migraines? In migraine with aura the patient may experience visual phenomena, but no double vision. It is necessary to ask about concomitant double vision, because this may indicate lesions of the upper brain stem or telencephalic mass lesions with subsequent brain stem compression [5, 19, 20].
7. Does the patient report black stool or other signs of bleeding [5, 20, 23]?
8. Did the ambulance crew or the person responsible for the triage process in the emergency department note a pathological score in the Cincinnati Prehospital Stroke Scale [24]? In the Cincinnati Prehospital Stroke Scale, the clinician looks for (1) facial asymmetry, (2) lowering and pronation of one arm when holding out the arms straight for 10 seconds while having the eyes closed, and (3) dysarthria or aphasia. If at least one of the three signs is present, there is a 72% probability of stroke; if all three symptoms are present, there is an 85% probability of stroke.

9. Does the patient take medications or drugs typically associated with dizziness [19, 21, 22]?

General physical examination and electrocardiogram

In the case of dizziness, a physical examination including the immediate assessment of vital signs is mandatory. We suggest an assessment of the airways, breathing, circulation, disability (e.g., assessing orientation, neurological status) and environment (e.g., a body check to look for trauma signs, anisokoria, pupillary light reflex, etc.) [5, 19, 20]. The examination should focus on volume status, since hypovolaemia can accompany dizziness. It should include a Schellong test and a bedside test for peripheral vestibular aetiologies, such as the Dix-Hallpike manoeuvre. A murmur on heart auscultation may suggest an aortic valve stenosis, whereas auscultation of the carotid and the subclavian arteries can suggest a carotid or a subclavian artery stenosis. A carotid stenosis, however, typically does not cause dizziness (only if other significant stenoses are present). Pulsus paradoxus may indicate another heart-related problem or severe asthma, although in this case dizziness is unlikely to be the cardinal symptom. If anaemia is suspected, a rectal examination can detect gastrointestinal bleeding. Subsequently, an electrocardiogram (to detect cardiac rhythm disorders) should be recorded.

Neurological and otological examination

A detailed neurological examination includes examination of the cranial nerves, looking for Horner's syndrome, defects of the visual field and cerebellar signs (ataxia, gait, dysmetria, abnormal Romberg test, etc.) [5, 19, 20], and testing sensation in the upper and lower extremities. Furthermore, an orienting otological examination is necessary. This consists of inspection of the ear canal, the ear drum and the mastoid, in addition to a bedside hearing test. The mastoid should be inspected for the signs of mastoiditis (redness, warm skin, swelling and pain on palpation), which requires urgent referral to an ear, nose and throat specialist, or the involvement of neurology. In the absence of mastoiditis, concurrent ear pain and dizziness may suggest a peripheral pathology [19].

Bedside eye movement tests

Visual acuity and the visual field should be tested before the examination of eye movements. The examiner focuses on to-and-fro eye movements (nystagmus) with and without Frenzel goggles (see fig. 1B) at different gaze positions. Frenzel goggles have magnifying glasses which allow easier examination and remove any visual fixation. Nystagmus can occur either spontaneously at rest or after stimulation (e.g., positional nystagmus). Other oculomotor examinations such as tests of smooth pursuit, saccades or convergence give additional information. Table 2 provides

Table 1: Relevant questions for medical history-taking [5, 18–20].

Symptoms to look for		Clinical associations
Vestibular syndrome		The combination of dizziness, nausea and vomiting, gait disturbance and motion intolerance is defined as acute vestibular syndrome (peripheral or central aetiologies)
Triage questions Timing	Prodromal dizziness ("Do you suffer from intermittent, yet completely resolving episodes of dizziness along with other symptoms such as brief difficulties speaking, seeing clearly, moving a limb or do you feel one side of your body less compared to the other side of the body?")	Prodromal, intermittent episodes of dizziness due to hypoperfusion can precede an infratentorial stroke [5]
	Onset ("When did it start?")	Acute (an abrupt start, from one second to the next) or subacute (a slow start before culminating within minutes to hours). Abrupt points towards a cerebrovascular cause
	Duration of symptoms ("For how long does it persist / how long did it last?")	Seconds, minutes, hours, days or weeks, permanent or episodic. Important for triage (acute, episodic or chronic vestibular syndrome)
	Recurrence ("Do the symptoms come and go?")	Recurrence can happen in both peripheral and central aetiologies, such as benign paroxysmal positional vertigo and transient ischaemic attacks.
Headache? Cervical pain?		– Thunderclap headache (subarachnoid headache)? – Sudden, severe craniocervical pain (dissection of vertebral artery)? – Prior diagnosis of migraine, cluster headache, tension headaches?
Double vision and other neurological signs		Consistent with a central origin, such as in a transient ischaemic attack, stroke or brain tumour.
Upper respiratory infection? Ear pain?		A complicated acute otitis media must be ruled out.
Hearing loss or tinnitus (uni- or bilateral)		Can be due to infection, Menière's disease, ototoxic antibiotics (e.g., aminoglycosides) and labyrinthine infarction.
Triggers and risk factors		
Triggers?		– Positional changes – Arm movements – Valsalva manoeuvre – Stress – Hyperventilation
Vascular risk factors?		Thromboembolic events in family history, hypertension, diabetes, cardiac arrhythmia, smoking, high cholesterol, rheumatic disease, age >50 years.
Intoxication?		In particular alcohol, and amphetamine- and opiate-based drugs.
Medication? Any new medications? Any recent dose adjustments of existing medications?		Dizziness as a very frequent side effect (>10%): ototoxic antibiotics, diuretics, opioid-based analgesics, anti-epileptic drugs, immunosuppressants, antidepressants, antipsychotic drugs, anti-HIV-drugs. Dizziness as a frequent side effect (1–10%): antiarrhythmics, benzodiazepines, lithium, antihypertensive treatment, medications treating erectile dysfunction, antifungal medication, antimalarial drugs, anti-Parkinson treatment [19, 21, 22].
Recent trauma?		Risk factors for vertebral artery dissection, cerebral bleeding. Associated with post-traumatic benign peripheral positional vertigo or labyrinthine concussion.

an overview of likely origins of dizziness. It by no means aims at providing a complete list. Overlaps are still possible, such as symptoms typical for peripheral aetiology associated with central origins and *vice versa*.

Laboratory analyses

Blood count and blood chemistry analyses including blood glucose may provide diagnostic hints of treatable underlying aetiologies such as hyponatraemia, hypoglycaemia, intoxication or anaemia [5, 19, 20]. Dizziness can also be associated with abnormalities in blood gas analysis (acidosis or alkalosis, high lactate levels), elevated inflammatory parameters and pathological urinalysis such as urinary tract infections, which often have detrimental effects on the central nervous system, especially in the elderly). We also suggest screening for opiates, cocaine, amphetamines and benzodiazepines in dedicated patients. Although these make no difference to the immediate sensation of dizziness, a positive screen may result in offering the patient professional help so that he or she can avoid these symptoms in the future.

Diagnostic neuroimaging

Cranial computed tomography (CT) has a low sensitivity of 16% for detecting ischaemic stroke [25] and therefore contributes little to the detection of the origin of dizziness [3, 26, 27]. If there is the possibility to perform cranial magnetic resonance imaging (MRI) in the emergency department, one should consider that an acute diffusion-weighted MRI shows 20% false negative results (compared with delayed MRI after 3–10 days) and has a sensitivity of 80% for detecting a stroke [5]. Patients with small strokes had 53% false negative MRI results if the initial MRI was performed between 6 and 48 hours after onset of dizziness [28]. Canadian CT head rules [29] assist in deciding when a CT scan is required in head trauma, provided the patient had an initial loss of consciousness and a score of 15 to 13 on the Glasgow Coma Scale at presentation (i.e., minor head injury. This must not be confused with moderate to severe brain injury (a score lower than 13 on the Glasgow Coma Scale), where a CT is always required. If quantitative and qualitative consciousness is normal and if there is no history of trauma, neuroimaging may be deferred and the patient’s detailed medical history may be taken first, followed by a careful clinical examination [5, 19, 20]. In indi-

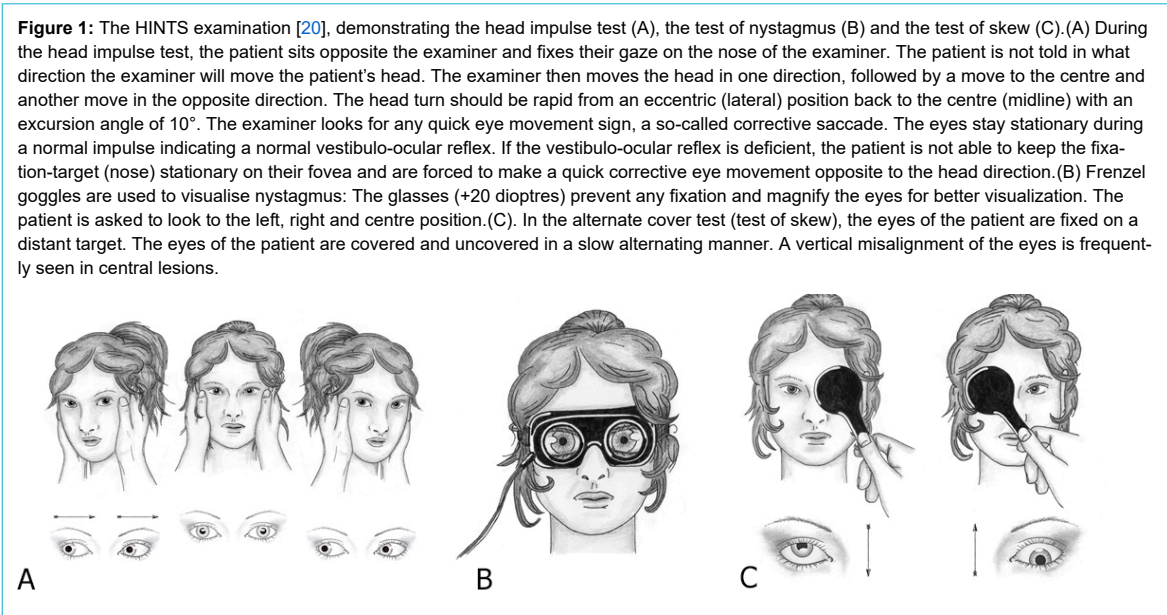


Table 2: Origins of dizziness: central versus peripheral origin. (A table with overlapping content in German will appear in our article on “Rotational vertigo - practical guidelines for General Practitioners” in the journal *Hausarzt Praxis*, which has given us permission to use this English version here.)

Central	Peripheral
Thunderclap headache	Dizziness elicited by positional changes
Sudden, severe craniocervical pain	Pathological head impulse test
Double vision	Pathological Dix-Hallpike manoeuvre
Other neurological deficits	Glasgow Coma Scale score of 15
Recent head trauma	Autophony, hyperacusis, and tinnitus
	Painful ear or ear pressure
Direction-changing nystagmus	Stress and hyperventilation
Vertical and rotational nystagmus no proof, but highly suggestive for central aetiology	Nausea/vomiting during head-movements
Pathological score on the HINTS examination	Oscillopsia during walking/head-shaking
Skew deviation	
Horner’s syndrome	
Reduced Glasgow Coma Scale score	
Visual field defects	

vidual circumstances, such as when a significant cranial injury is suspected for some reason, even though the patient has a score of 13 to 15 on the Glasgow Coma Scale at presentation, experienced emergency physicians should evaluate the patient. The emergency physicians should then decide on a case-by-case basis whether a CT is nevertheless needed, rather than applying the Canadian CT head rule to every patient.

A CT scan is indicated in patients with a suspected haemorrhage (thunderclap headache or lethargy with a reduced score on the Glasgow Coma Scale, head trauma, hemiparesis). It should be kept in mind, however, that haemorrhage accounts for only 4% of patients with acute vestibular syndrome [5]. In the case of subarachnoid bleeding, acute vestibular syndrome is unlikely to be the predominant symptom, because severe headache (thunderclap headache) is the cardinal symptom [19].

An MRI scan has the advantage that it not only permits strokes to be recognised, but also enables the diagnosis of inflammatory, degenerative or neoplastic lesions, especially if combined with detailed history-taking, clinical examination and laboratory tests [30–37].

Differential diagnoses of vestibular syndromes

Acute vestibular syndrome

An acute vestibular syndrome is a well-described syndrome of acute onset, continuous dizziness lasting days to weeks, in combination with nausea, vomiting or gait dysfunction [2, 3, 5]. Aetiologies include benign causes such as vestibular neuritis and central aetiologies including lesions affecting the vestibular nuclei or the tracts leading from the brainstem to the cerebellum, thalamus or vestibular cortex [30], mainly caused by stroke, tumours and demyelinating diseases.

The advantage of relying on clinical decision rules in acute vestibular syndrome

Recent prospective studies support the use of the clinical bedside test “HINTS” for the triage of patients with an acute vestibular syndrome [20, 31]. The HINTS examination consists of a head-impulse test to analyse the vestibulo-ocular reflex, putting Frenzel goggles on the patient and looking for nystagmus, and looking for skew deviation in the alternate cover test [5, 19, 20]. HINTS stands for horizontal Head Impulse test, Nystagmus and Test of Skew [20]. An explanation of this diagnostic test is provided in figure 1. If carried out by a skilled examiner, the results will distinguish central from peripheral origins of dizziness with high specificity and even higher sensitivity than early MRI [5, 19, 20]. The clinical procedures of HINTS [32–34] outperform even acute diffusion-weighted MRIs (within 48 hours of dizziness onset) in diagnosing isolated vestibular stroke. The presence of a normal head impulse test and a direction-changing nystagmus in eccentric gaze or a pathological skew deviation showed a 100% sensitivity and a 96% specificity for stroke [20]. According to a systematic review [5], there is evidence that the HINTS test, especially the combined HINTS-hearing loss battery (where one-sided hearing loss on the side of a pathological head impulse test points towards a labyrinthine or pontine infarction [5, 17]), performs favourably compared with repeated MRI in acute vestibular syndrome [17, 28]. In the

future, emergency department triage of patients with acute vestibular syndrome may predominantly rely on clinical decision rules such as the HINTS battery [17, 20, 28], which has a negative likelihood ratio of 0.02 (reduces post-test probability for vestibular stroke 50-fold) instead of relying on an acute MRI (within 6–48 hours after the onset of dizziness), which has a negative likelihood ratio of 0.21 (five-fold reduced post-test probability for vestibular stroke).

The discrimination between a central and a peripheral vestibular deficit can be further improved by use of new developments of the HINTS battery, such as performing the head impulse test [32] with portable video oculography devices that contain a high-speed infrared camera to measure eye movements and a gyroscope for head movements [26, 35–37].

Additional clinical examinations in acute vestibular syndrome

To add more diagnostic power to HINTS, additional clinical tests for the examination of cranial nerves should be added. These include a clinical examination for hearing loss (HINTS plus) [17], double vision, pathological findings in tests of the cranial nerves, Horner’s syndrome, cerebellar signs (dysarthria, ataxia) and dissociated loss of pain and temperature sensitivity [5, 19, 20]. Upbeat nystagmus, downbeat nystagmus, or a direction-changing nystagmus with gaze movement all make a central origin likely [5, 19, 20]. A sudden/acute onset of dizziness is often of central origin [5, 19, 20]. Suspected acute stroke, subarachnoid haemorrhage or brainstem encephalitis all require urgent intervention [5]. In ischaemic stroke, thrombolysis should be administered only within certain time limits. These time intervals are subject to constant re-evaluation and new evidence suggests that patients with stroke can benefit from intravenous thrombolysis administered up to 6 hours after symptom onset [38]. In some extreme cases where the patient is highly symptomatic and the benefits of intra-arterial thrombolysis are expected to exceed the risks, some hospitals administer intra-arterial thrombolysis even up to 12 hours after symptom onset, but there is no generally validated standard for this. If the patient history suggests stroke and the HINTS examination suggests a central deficit, cranial CT is still needed to exclude bleeding before starting thrombolysis.

It is necessary to consider that not every patient with acute vestibular syndrome was included in the previous analyses. HINTS included patients with at least one risk factor for stroke and excluded patients with recurrent dizziness [17, 20]. As with an MRI scan, which can miss small ischaemic areas not only within the first 48 hours but also beyond this period, HINTS cannot detect every stroke. HINTS is predominantly useful in patients who exhibit nystagmus.

The following clinical findings make a peripheral origin more likely: a normal neurological examination, an abnormal head impulse test (though it does not exclude a stroke, especially if associated with hearing loss), or a mixed horizontal and torsional spontaneous nystagmus. This nystagmus is typically unidirectional and the intensity increases under Frenzel goggles or when the patient looks in the direction of the fast phase of nystagmus. An increase of nystagmus with Frenzel goggles indicates intact suppression of nystagmus under fixation [5, 19, 20, 39]. A subacute

onset (a slow start and maximum intensity after minutes to hours) is often characteristic of a peripheral origin [5, 19, 20]. If the emergency physician cannot treat the dizziness successfully, ear, nose and throat specialists should be contacted. Figure 2 shows an example of clinical decision rules in the emergency department incorporating the HINTS examination and other steps in the decision process.

Episodic vestibular syndrome

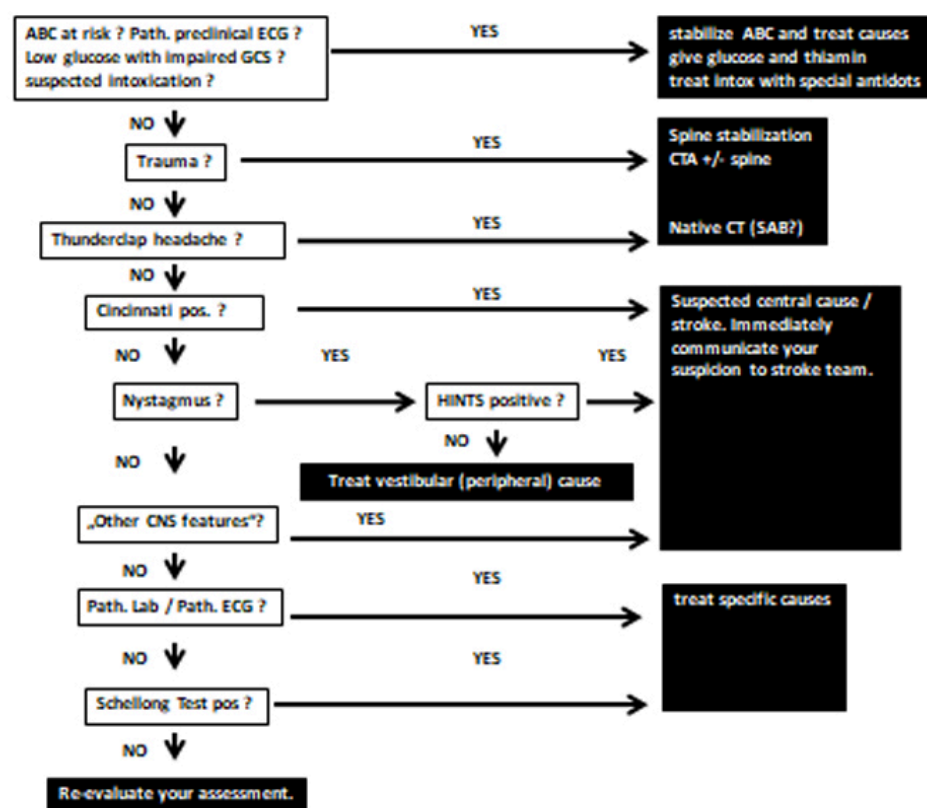
An episodic vestibular syndrome is a clinical syndrome of transient dizziness lasting seconds to hours [18]. According to a classification that was established in a specialised outpatient clinic for dizziness, the most frequent peripheral aetiologies for episodic vestibular syndromes include benign peripheral positional vertigo, Menière's disease, neurovascular compression and perilymphatic fistula [30]. Overall, peripheral aetiologies are more frequent than central aetiologies [30]. Specific questions about timing and triggers point to possible differential diagnoses; for example, symptoms occurring only during arm movements might indicate a subclavian steal syndrome, symptoms occurring when changing body position might indicate benign paroxysmal positional vertigo, symptoms associated with an upright posture might point to orthostatic dizziness [5, 19, 20, 40]. Sensations lasting for seconds are typical of benign paroxysmal positional vertigo or a vasovagal reaction, sensations lasting for minutes could result from hypoglycaemia (if treated symptoms will disappear), a panic reaction or a transient ischaemic attack [19, 20]. In contrast,

the most frequent central aetiologies of episodic vestibular syndromes are vestibular migraine [41], followed by brainstem lesions or cerebellar lesions [30]. Central causes are identified when dizziness is combined with oculomotor disorders such as upbeat or downbeat nystagmus or episodic ataxia type 2 [30, 42, 43]. Similarly, there are episodic arrhythmic conditions, such as atrial fibrillation, that cause episodic sensations of dizziness [3]. More details on episodic vestibular syndromes and their treatment are to be found in our accompanying article on treatment of dizziness [44].

Chronic vestibular syndrome

This syndrome consists of chronic dizziness or unsteadiness lasting months to years [18]. The central nervous system can, to some extent, automatically compensate – the so-called central compensatory processes, which are a complex interplay of neurological pathways [6, 30]. To an unpredictable extent, central compensatory processes may not work, or they may be hindered by the patient taking anti-vertigo medication for too long [6, 30]. Conditions such as a poorly compensated unilateral vestibulopathy, chronic bilateral vestibulopathy, cerebellar degeneration, posterior cranial fossa neoplasms and chronic psychological or behavioural disorders may present as chronic vestibular syndrome. There are syndromes such as a persistent postural-perceptual dizziness (also known as phobic positional vertigo [45]) that cannot be classified as either of peripheral or of central origin. Nevertheless, patients who present with persistent postural-perceptual dizziness constitute a

Figure 2: A proposed combination of HINTS (Head impulse test, Nystagmus, Skew Deviation) and other clinical decision rules in the emergency department. ABCDE = airway, breathing, circulation, disability, environment; GCS = Glasgow Coma Scale; CTA = CT angiography; SAB = subarachnoid bleeding; CNS = central nervous system; ECG = electrocardiogram



large group: they accounted for about 15% of patients who presented to the dizziness outpatient clinic of the German Dizziness Center between 1988 and 2012 [30]. Another group includes cervical vertigo, for example due to injuries to the cervical spine. It often is the result of vertebrobasilar insufficiency [46]. Other examples are Arnold-Chiari malformation [30], which is characterised by chronic postural instability. Children who are born with Arnold-Chiari malformation have a malformation of the skull, so that the tonsils and the posterior cerebellum extend into the cervical spine.

Conclusions

Classifications seem helpful for differential diagnosis and for guiding physicians towards a uniform diagnostic approach to patients presenting with dizziness. First, it is necessary to apply clinical decision rules and ask about specific symptoms while keeping in mind life-threatening differential diagnoses such as myocardial infarction or pulmonary embolism. Similarly, it is necessary to consider stroke as a differential diagnosis of acute vestibular syndrome. Second, it is important to understand the different aetiologies. Therefore, we defined three diagnostic groups: acute, episodic and chronic vestibular syndromes. In particular, we have given examples of how to differentiate between central and peripheral origins of acute vestibular syndrome by applying the HINTS examination and other clinical decision rules. Consequently, we recommend quick and effective triage of the dizzy patient in the emergency department. Episodic and chronic vestibular syndromes require a sequence of action different from acute vestibular syndrome, because there is typically more time to make the correct diagnosis. In acute vestibular syndrome of central origin, symptoms are typically new to the patient. As a result, there is the danger that the patient has suffered a stroke. In such a case, the HINTS examination can improve diagnostic accuracy. Like the Cincinnati pre-hospital stroke scale [24], HINTS [20] can be applied very rapidly and even in the prehospital setting. If the history and HINTS suggest a stroke in the preclinical or clinical setting, rapid action is needed to ensure that thrombolysis is not delayed. Reasons for introducing HINTS as an important diagnostic step in patients with acute vestibular syndrome are its rapidity and ability to recognise or to rule out stroke. It requires training of the emergency physician or the primary care provider who carries out the HINTS examination. The current limitation is that there are no studies to show how much training is needed, and there is no comparison between emergency physicians, ambulance crews or general practitioners and neurologists and ear-nose-throat specialists. In the future, this bedside test has the potential to become an important standard for emergency physicians. HINTS, however, should not be overestimated. Nor should any other clinical decision rule be overestimated, because there is always the risk of a rare case that is not explained by evidence from previous studies. This applies to patients where mild imbalance alone or in combination with a horizontal nystagmus suggest a peripheral deficit, but a central aetiology is present [47–49], or where double vision suggests a central pathology [19], but a peripheral aetiology is present [50]. We would therefore like to emphasise that the clinician can never be ab-

solutely certain, even with highly sensitive and highly specific clinical decision rules such as HINTS. It is wise for any clinician to be self-critical, and to constantly re-evaluate the diagnosis. The presented algorithm on how to apply the clinical decision rules is just one example. There are other possibilities. Depending on the individual circumstances, the clinician will have to remain flexible and might need to alter the algorithm.

In summary, dizziness is frequent and decision making difficult owing to a lack of agreement between the disciplines involved. In the emergency department, it is for the physician to decide what steps can be carried out by the emergency physician and when to consult specialists. It is necessary to combine different steps in the triage process and to continuously re-evaluate the initial hypothesis once new diagnostic information is obtained. To date, HINTS plays an important role in this triage process. Conclusions based on the current evidence are certainly limited owing to a lack of prospective, randomised comparative studies with specific research questions. We hope that this review will ultimately stimulate future studies. These are strongly recommended because of the frequency of dizziness.

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Competing interests

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