Dizziness

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Abstract

Dizziness and vertigo are common symptoms. Because there are effective treatments for vestibular disorders, it is always important to make an accurate diagnosis. In acute vertigo, expert clinical assessment is critically important in discerning stroke from non-stroke causes because stroke-protocol brain magnetic resonance imaging results, including diffusion-weighted imaging, can be falsely negative in the first 24 hours. It follows that acute medical services must have access to clinicians expert in assessing acute vertigo. Expertise in clinical examination and the interpretation of findings requires appropriate training, but in this article we outline the basic diagnostic and therapeutic approach to patients with dizziness.

Keywords Dizziness; Ménière's disease; migraine; stroke; vertigo; vestibular disorders

Key points

- Vertigo can be a feature of, or trigger for, migraine
- Benign paroxysmal positional vertigo is a common and treatable condition
- The HINTS signs (normal horizontal head-impulse test, direction-changing nystagmus in eccentric gaze or vertical nystagmus, and skew deviation) are helpful in identifying patients with a central cause of acute vertigo
- Diffusion-weighted imaging may be negative for stroke in the first 24 hours, and should be repeated in 'HINTS'-positive patients at 48 hours
- Drugs are useful in the management of vestibular migraine

How common is dizziness?

Dizziness is a common symptom. Lifetime prevalence estimates of significant dizziness in community prevalence studies range between 17% and 30%. The exact frequency of specific vertigo diagnoses remains unclear, partly because many non-specialists are poor at accurate neurological diagnosis in the emergency setting.[1] Table 1 shows the results of a study examining neuro-otologists' diagnoses from 90 referrals seen in our emergency department over 12 months.[2] In practice, making a diagnosis is most problematic when faced with vertigo without additional focal neurological symptoms. Acute isolated vertigo is usually benign, often resulting from pathology of the vestibular organ. However, it is important to make a specific diagnosis, as life-threatening neurological conditions such as stroke can present with isolated vertigo, sometimes with a paucity of signs.

Definition and terminology

The vestibular system is optimized for detecting head motion. The system includes the peripheral sensor in the inner ear, the vestibular nerve that transmits impulses to the brainstem and cerebellum for initial processing, and thalamic circuits that relay vestibular signals to an extensive cerebral cortical network. The brainstem-mediated reflexes optimize body function during head motion, including gaze stabilization (vestibular-ocular reflex (VOR)) and balance (vestibular-spinal reflexes). Vestibular-autonomic reflexes optimize cardiovascular functioning for motion.

Patients and doctors use the term 'dizziness' (which means 'stupid' in Old English) to describe a variety of sensations, so a clarification of the patient's sensation is always needed to avoid diagnostic mistakes. Dizziness can take many forms, such as spinning like a merry-go-round (i.e. vertigo), rocking like a boat or linear motion (falling, moving upwards, fore–aft or lateral motion). Contrary to conventional teaching that only a sense of rotation indicates a likely vestibular cause, all these sensations indicate activation of the 'vestibular system' at some level, from peripheral to central.

Sometimes patients use the word 'giddiness' or 'lightheadedness', but it is still important to interrogate patients to understand exactly what they mean. Often they mean a sense of illusory self-motion or feeling unbalanced, and sometimes they feel as if they are floating. Patients with general medical conditions (anaemia, hypoglycaemia) or haemodynamic (orthostatic hypotension, presyncope) or psychological problems can also use the same words. Furthermore, sensorimotor disorders of the lower limbs (parkinsonism, gait ataxia, spinal cord syndromes) come into the differential diagnosis when patients describe their symptoms as imbalance or unsteadiness. It can be useful to ask patients if the problem is 'in their legs or in their head' and whether the sensations are as if they are 'about to faint' (pre-syncopal) as opposed to 'being on a merry-go-round' or 'on a boat' (vestibular).

Another important and useful symptom is oscillopsia. Oscillopsia is simply seeing the visual environment move when in fact

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Diagnosis of vertigo in the emergency department

Diagnosis	Vertigo cases (%)
Benign paroxysmal positional vertigo	31
Vestibular neuritis	14
History of vestibular neuritis with normal assessment	14
Vestibular migraine	12
Anxiety/panic disorder	11
Stroke	4
Presyncope	4

Table 1

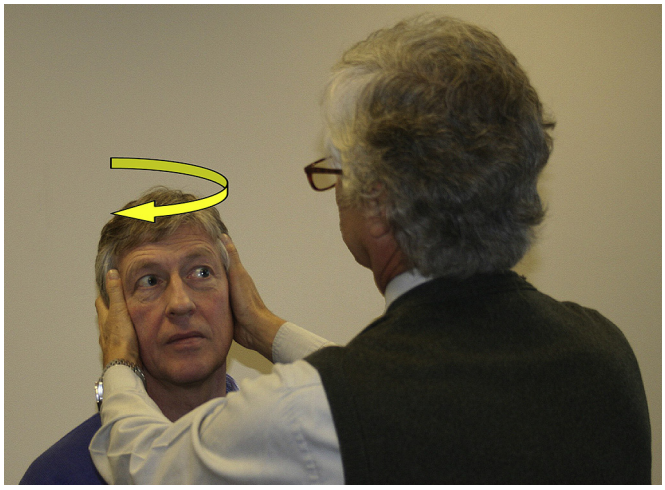

Figure 1 The head-impulse (or head-thrust) test. The examiner holds the patient's head and asks them to fixate on the examiner's nose. The examiner then delivers a discrete, low-amplitude (15–20°) but very fast head thrust to one side. If the patient's vestibular-ocular reflex (VOR) is intact, the eyes will remain fixated on the examiner's nose when the head is rotated. If the VOR is unilaterally impaired, the patient's eyes will momentarily lose their fixation on the examiner's nose when the head is rotated. The examiner should look for one or more catch-up saccades directed back towards the initial fixation point – their nose. The figure shows a right head thrust, probing right horizontal semicircular canal function. Several trials in each direction, right and left, should be carried out in a pseudo-random order.

the observable world is static. Oscillopsia arises only in two situations:

- when the patient is at rest and they see the room move – this means they have a spontaneous nystagmus
- when the patient sees the room move only when they themselves are moving – this means they have an absent VOR. Note that this includes patients with preserved peripheral function who cannot manifest a VOR because of an acute external ophthalmoplegia.

Pathophysiology

The deficits in acute peripheral vestibular loss can result in impaired functioning of the vestibular-ocular, vestibular-spinal, vestibular-autonomic and vestibular-cortical projections.

In acute peripheral vestibular loss, VOR dysfunction causes nystagmus. The nystagmus is unidirectional (i.e. the direction of the nystagmus is unaffected by changes in the direction of gaze) with the slow phase in the direction of the defunct labyrinth (i.e. fast-phase beating to the contralateral side). The nystagmus is most visible when looking in the direction of the fast phase, less so in the midline and least in the opposite direction (i.e. the intensity but not the direction of 'vestibular' nystagmus is affected by the gaze direction). Vestibular-autonomic pathway dysfunction causes symptoms such as nausea, vestibular-spinal pathway dysfunction results in imbalance, and vestibular-cortical pathway dysfunction is associated with vertigo.

Clinical approach to the patient with acute vertigo

In a patient presenting with acute vertigo, a priority is to exclude a central lesion, specifically a stroke. Stroke is life-threatening but potentially recoverable with the correct treatment (thrombolysis, clot retrieval); conversely, these treatments pose an unnecessary risk in vertiginous patients who incorrectly acquire a diagnosis of stroke.

The VOR is impaired in peripheral vestibular loss. This is demonstrable using the head-impulse test, in which the head is rapidly moved in the direction of the damaged labyrinth (or vestibular nerve). This test is the method of choice when clinically assessing the integrity of the VOR in the acute phase (Figure 1). Another key sign of 'peripheral' vestibular dysfunction is the suppression of unidirectional vestibular nystagmus when visual fixation is allowed. Conversely, without visual fixation, as in the dark, the intensity of nystagmus is increased (i.e. there is a faster slow-phase velocity). An ophthalmoscope can be used to examine the effect of loss of visual fixation on nystagmus intensity, remembering that the observed direction of retinal movement (posterior aspect of the globe) is opposite to that of the observed movement of the sclera (anterior aspect of the globe). Any clear-cut central neurological signs in the presence of a neuro-otological syndrome make lesion localization relatively easy, particularly when there is brainstem involvement. Central findings of the eye movement examination include abnormalities of smooth pursuit and saccades. However, isolated cerebellar strokes can mimic a peripheral vestibular syndrome (see below).

Examination of the external auditory meatus is relevant to exclude local pathology such as a cholesteatoma, eardrum perforation, discharge or the vesicles of Ramsay Hunt syndrome. If stroke is suspected, careful cardiovascular examination is required; an electrocardiogram may be required to exclude atrial fibrillation.

Acute idiopathic unilateral peripheral vestibulopathy

This is the most common cause of continuous vertigo lasting more than 24 hours, during which there are symptoms and signs of unilateral vestibular hypofunction. The syndrome is an updated and more accurate term for 'vestibular neuritis' or 'labyrinthitis' (a viral aetiology has been suggested). Distinguishing vestibular neuritis from stroke is essential not only to avoid missing a serious diagnosis, but also to avoid overinvestigation and inappropriate lifelong treatment for secondary stroke prevention. Typically, patients have a subacute onset of intense vertigo over hours, almost always associated with nausea