A practical approach to managing the dizzy patient

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A variety of common causes can lead to dizziness and/or vertigo which are not always easy to differentiate. Here we review these causes with a focus on making a positive diagnosis

Dizziness accounts for over 20 per cent of GP visits in the UK and is associated with significant morbidity.[1] Although in the acute setting the most frequent diagnostic errors concern differentiating central from peripheral causes of vertigo,[2] even when a peripheral cause of vertigo is identified, it is often incorrectly labelled as "viral labyrinthitis" to encompass most inner ear disorders of presumed benign origin. A good long-term clinical outcome in these patients depends on a correct initial diagnosis. Here we will review the common causes of dizziness with a focus on making a positive diagnosis and initiating appropriate treatment.

What does the patient mean by "dizziness"?

Vertigo is simply the illusion of movement, but patients often use the terms "vertigo" and "dizziness" to describe a variety of subjective sensations. If the patient finds it difficult to explain their sensation, offer words like "merry-go-round", "rocking like on a boat", "unsteadiness" or "light-headedness". The duration and time course of vertigo may not be helpful because the subjective recall of time is inaccurate, particularly when episodes are brief (seconds or minutes).

Common causes of acute vertigo

Table 1 summarises the commonest causes of vertigo in acute and chronic settings. When making a diagnosis in such patients, it may be helpful to have in mind these common diagnoses and determine into which the patient best fits, rather than attempting to formulate a large differential based on the history and examination findings.

SPL

Caloric reflex test of the vestibulo-ocular reflex

Benign paroxysmal positional vertigo

Benign paroxysmal positional vertigo (BPPV) is the commonest cause of dizziness among the general population, with an incidence of 64 per 100,000 population per year.[3] Although it is commoner in the elderly,[4] it can affect people of any age but in children migraine or malignancy should be excluded first. BPPV is characterised by brief episodes of vertigo and imbalance associated with nystagmus. Nausea may occur but vomiting is rare. While a history of recurrent episodes of vertigo triggered by movement (looking up, bending down, or turning over in bed) suggests BPPV, the diagnosis can only be confirmed with the Dix–Hallpike manoeuvre or its modified form. BPPV is caused

Diagnosis	Clinical features	Examination	Investigation	Treatment
Peripheral				
Benign paroxysmal positional vertigo (BPPV)	Sudden onset brief attacks of spinning vertigo, and imbalance triggered by changes in head position (bending down, looking up, or turning over in bed)	Positive Hallpike manoeuvre with vertigo, and nystagmus (torsional and upbeat for posterior canal BPPV)	Nil	Particle repositioning manoeuvre (Epley or Semont)
Vestibular neuritis	Vertigo, nausea/vomiting and imbalance developing over minutes to hours Symptoms are worse on movement Constant oscillopsia initially (hours)	Unidirectional horizontal (+ torsional) spontaneous nystagmus. Abnormal head impulse test, when turning the head towards the side of the lesion Imbalance	Nil Diagnosis can be confirmed with bithermal caloric	Bedrest and anti-emetics for maximum of 3 days. Encourage mobility early. Vestibular rehabilitation if symptoms persist
Meniere's disease	Sudden onset severe vertigo, nausea, vomiting, imbalance, hearing loss, tinnitus, and aural fullness	Spontaneous nystagmus with horizontal/torsional component. Abnormal head impulse test, impaired hearing	Audiogram +/- caloric and vestibular evoked myogenic potentials	High-dose betahistine, intratympanic dexamethasone or gentamicin
Central				
Vestibular migraine	Episodic vertigo and imbalance often associated with nausea, photophobia, phonophobia and aversion to movement. Headache may or may not be present	Normal, or there may be nystagmus (spontaneous, gaze-evoked or positional)	May require magnetic resonance imaging (MRI) scan if first presentation	Anti-migraine prophylaxis (propranolol, amitriptyline, topiramate, pizotifen *etc*)
Posterior circulation stroke	Sudden onset vertigo, headache, vomiting and imbalance	Gaze-evoked nystagmus, broken smooth pursuit, limb ataxia, gait ataxia, positional downbeat nystagmus	Computed tomography or MRI brain	Treatment of stroke
Non-vestibular				
Postural hypotension	Recurrent episodes of dizziness, lightheadedness, or imbalance. Worse in the morning. Triggered by standing from sitting, or sitting from lying	Usually normal	Postural blood pressure recordings	Adequate hydration, reducing or stopping offending medication, compression stockings, tilting head of bed. Fludrocortisone for resistant orthostatic hypotension
Anaemia	Recurrent episodes of light-headedness, often associated with palpitations. May present with blackouts	Pallor	Full blood count	Treatment of underlying cause; iron replacement; blood transfusion
Anxiety	Episodic or chronic dizziness, usually a sensation of self-motion, accompanied by autonomic symptoms, and catastrophic fears. Avoidance behaviour	Normal	Thyroid function tests, ECG	Reassurance, explanation of symptoms. Cognitive behavioural therapy. Anxiolytics or antidepressants

Table 1. Common causes of vertigo in general practice

by calcium carbonate crystals (otoconia, or canaliths) settling within the endolymphatic fluid of the semicircular canal, almost always the posterior semicircular canal. Horizontal canal BPPV accounts for only 10–20 per cent of cases, and anterior canal BPPV 5 per cent.[5] The direction of the nystagmus allows the identification of the affected semicircular canal. In a patient with posterior canal BPPV, the examiner sees upbeat and torsional nystagmus beating towards the lowermost ear during this manoeuvre (see Figure 1).

PPV can be very disabling and is easily treated, either with the traditional Epley manoeuvre or the Semont manoeuvre, which is easier to perform and equally effective.[6] As these may trigger vertigo in patients with BPPV, clinicians must spend a few moments explaining what is done and why. Note that medications have no role in BPPV management.

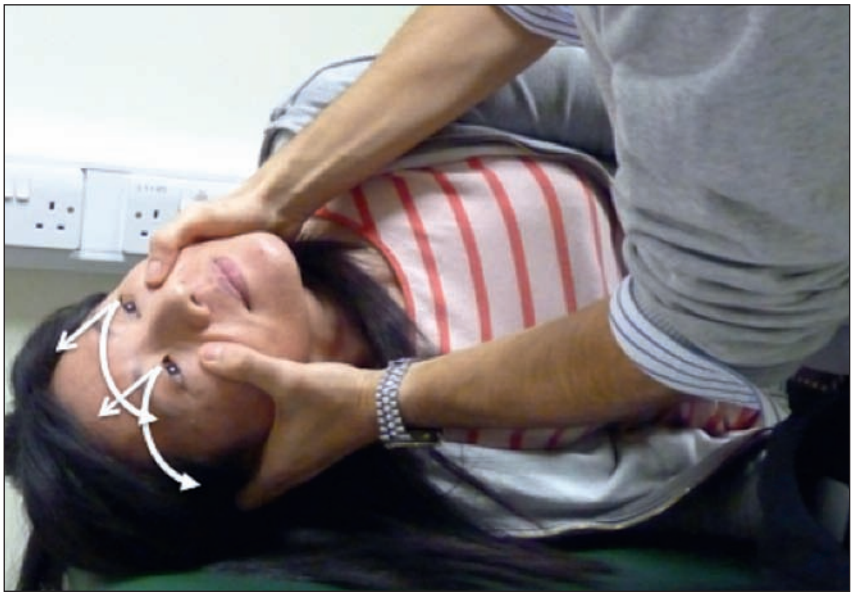

Figure 1. Upbeat and torsional (rotatory) nystagmus beating towards the lowermost ear in left-sided posterior canal BPPV

Vestibular neuritis or "labyrinthitis"

Vestibular neuritis (VN) refers to inflammation of the vestibular nerve with sparing of the cochlear nerve, but is often mistakenly referred to as "labyrinthitis" (in which both hearing and balance are affected, and is exceptionally rare). VN has an incidence of approximately 3.5 per 100,000 population.[8]

It presents with a sudden attack of rotational vertigo, nausea, vomiting, and imbalance. The vertigo and nausea typically last hours to days, during which the vertigo is constant, even when the head is held completely still. This contrasts with BPPV where vertigo is only induced by head movements. The imbalance in VN consists of "furniture-walking" type in contrast to cerebellar stroke where patients are typically unable to stand.

Patients with acute VN will have spontaneous nystagmus *ie*, looking straight ahead. The nystagmus of VN is horizontal with some rotatory (torsional) component and is unidirectional, *eg* right-beating whether looking to the left, right, up or down. The vestibulo-ocular reflex (VOR) will also be impaired on the side of the lesion and can be evaluated with the "head impulse test"[9] in the clinic.

The utility of steroids in VN remains unclear[10] and they are not used routinely in the UK. Of note, it is exceptionally rare for VN to recur in the same patient, in which case a diagnosis of BPPV or vestibular migraine should be considered. Physical activity should be encouraged as soon as the nausea settles, with bedrest and anti-emetics recommended for a maximum of three days. The norm is gradual recovery over weeks following a process of central compensation.

Vestibular migraine

A diagnosis of vestibular migraine is not widely recognised outside specialist practice but approximately half of patients with classical migraine will report dizziness and vertigo, with another 20 per cent fulfilling the criteria for migrainous vertigo (see Table 2). Patients with vestibular migraine commonly report spontaneous or positional vertigo lasting hours to days. The typical patient is a migraineur who has noticed a recent increase in headache frequency and, over the same period, developed dizzy episodes, with headache and vertigo not necessarily occurring together. Other migrainous features such as photophobia, phonophobia and nausea are often present during the vertiginous episode, in addition to increased motion sensitivity, *ie* a dislike for self- or external movement.

During an attack there may be a number of oculomotor abnormalities (in up to 60 per cent of cases), including nystagmus of a central type (*eg* vertical or pure torsional). Thus, acute brain imaging may be required on first presentation. Between attacks the examination is normal. The diagnosis is aided by a previous history of similar symptoms, or a strong personal or family history of migraine.

Lifestyle adjustments to prevent identified triggers may avoid the need for pharmacotherapy. Where attacks continue to be severe or unacceptably frequent, prophylactic medications can be considered, including beta blockers (propanolol), tricyclic antidepressants (amitriptyline), antiepileptics (valproate, topiramate, gabapentin), or antiserotonergic (pizotifen) drugs. Visually-induced dizziness (visual vertigo) is a mal-adaptive strategy that may complicate vestibular migraine. It should be treated with anti-migraine prophylactic drugs first, with the addition of vestibular rehabilitation exercises if required later.

Posterior circulation stroke

Posterior circulation stroke is characterised by the abrupt onset of vertigo (within seconds), often accompanied by occipital headache (in up to 50 per cent of cases). Other associated signs may include gait or limb ataxia, facial numb-

Definite vestibular migraine
A. Episodic vestibular symptoms of at least moderate severity
B. Current or previous history of migraine according to the 2004 criteria of the IHS
C. One of the following migrainous symptoms during two or more attacks of vertigo: migrainous headache, photophobia, phonophobia, visual aura, or other aura
D. Other causes ruled out by appropriate investigations Note: Vestibular symptoms are rotational vertigo or another illusory self- or object-motion. They may be spontaneous or positional. Vestibular symptoms are "moderate" if they interfere with but do not prohibit daily activities. "Severe" vestibular symptoms do not allow patients to continue daily activities.
Probable vestibular migraine
A. Episodic vestibular symptoms of at least moderate severity
B. One of the following: (1) current or previous history of migraine according to the 2004 criteria of the IHS; (2) migrainous symptoms during vestibular symptoms; (3) migraine precipitants of vertigo in more than 50% of attacks; food triggers, sleep irregularities or hormonal changes; or (4) response to migraine medication in more than 50% of attacks
C. Other causes ruled out by appropriate investigations

Table 2. Diagnostic criteria for vestibular migraine

ness, Horner's syndrome, hearing loss, contralateral hemiparesis and hemisensory loss, suggesting involvement of cerebellar or brainstem structures. Importantly, the head impulse test tends to be normal in posterior circulation stroke. Urgent brain imaging is indicated where a posterior circulation stroke is suspected as these patients may require thrombolysis or even surgical intervention.

Ménière's disease

Ménière's disease (MD) is overdiagnosed in general practice. Patients will present with spontaneous, episodic and disabling vertigo (lasting minutes to hours) in association with unilateral tinnitus, aural fullness and unilateral fluctuating deafness. This is usually accompanied by nausea and vomiting, with imbalance that may last several days. Examination will reveal horizontal-torsional nystagmus in the acute phase, the head impulse test may be impaired, and hearing is typically affected. Specialist investigations are required to make a diagnosis of MD, including audiography and tests of vestibular function.

Given that the pathophysiological hallmark of MD is endolymphatic hydrops, salt restriction has been used prophylactically, but this is not supported by evidence.[11] Similarly, a Cochrane review of diuretics for MD did not support their use in MD.[12] High-dose betahistine may have a prophylactic effect on the frequency of attacks of MD, at least in the first year,[13] although its effect on vestibular and audiological function is unknown. There is also weak evidence that intratympanic dexamethasone may reduce attacks of Ménière's, without significant systemic side-effects.[14]

Red flags in a patient with acute vertigo

Red flags in cases of acute dizziness include: unilateral new onset hearing loss, focal neurological signs (*eg*, facial weakness, diplopia or limb weakness), new onset headache, and a normal VOR (head impulse test). If present, the clinician should think of possible more serious causes such as posterior circulation stroke. Hearing and otoscopy are normal in VN and most other harmless causes of dizziness.

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Declaration of interests

None to declare.

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