Clinical examination and management of the dizzy patient

Dizziness is the commonest reason for a GP appointment over the age of 75 years and many dizzy patients will end up in hospital. This article introduces the range of vestibular disorders commonly seen in hospital practice with a symptom-based approach and discusses the range of treatments that might be considered.

Dizziness is a common and debilitating complaint. It accounts for more than 20% of primary care consultations (Yardley et al, 1998), many of which are then referred for further management in secondary or tertiary care settings. The term itself is non-specific and may be used to indicate true vertigo, light-headedness, imbalance or syncope. Outside primary care patients may be seen in outpatient clinics by otologists, neurologists, care of the elderly teams, cardiologists or syncope clinics among others, and a considerable number present to accident and emergency departments and acute medical takes where they can prove to be problematic both from a diagnostic and management standpoint. Deciphering between (rare) life-threatening disorders and (common) benign disorders can be challenging (Norrving et al, 1995; Bertholon et al, 2002; von Campe et al, 2003; Lee and Cho, 2004; Lee et al, 2006). This is especially true in the acute setting, as tests ordered to screen for life-threatening disorders can be insensitive (Savitz et al, 2007).

As with all clinical presentations the key to an accurate diagnosis lies in obtaining a comprehensive history. However, this can be difficult in these patients as the symptoms described can be wide ranging and vague. Determining if they are experiencing true vertigo, which is defined as the illusion of movement while remaining still, or not can be extremely difficult (*Table 1*). The issue is further complicated as very often there is an interweaving of physical and psychological issues which can be difficult to separate (Newman-Toker et al, 2007). A detailed history is therefore imperative to pick out other non-otological reasons for the dizziness such as cardiovascular, endocrine or neurological causes. This article presents the most common vestibular causes of vertigo and dizziness together with their management strategies.

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The balance system

Human balance relies on the complex integration of information arising from the vestibular, visual and proprioceptive sensory inputs at the level of the brainstem and mid-brain with modulation from higher centres. As a result, any lesion affecting the structure or function of the sensory inputs, the CNS structures or the effector pathways is likely to result in a balance disorder. The labyrinthine apparatus is contained within the left and right temporal bones and comprises five components (*Figure 1*). These include three semicircular canals which are labelled in relation to their anatomical position, namely the lateral, anterior (also referred to as the superior canal) and posterior canals. The semicircular canals in each ear are anatomically arranged to be orthogonal to each other and therefore can

Table 1. Myriad symptoms of dizziness

Vertigo	Cotton wool in head
	Walking on cotton wool
	Eyes not keeping up with head
	I am moving, world is still
	Room spinning
	Falling into a hole
	Eyes flickering
	Merry-go-around
Non-vertigo	Tiredness and fatigue
	Nausea
	Headache
	Disconnected from the world
	Swaying from side to side
	Disorientated
	Lightheadedness

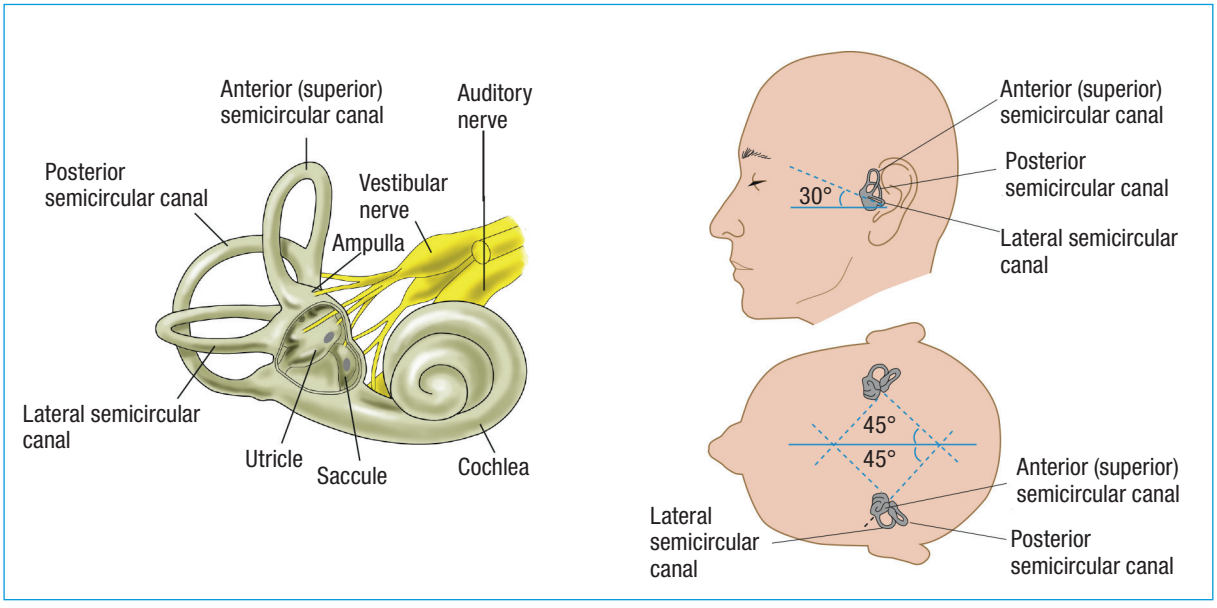


Figure 1. Anatomy of the cochlear-vestibulo apparatus and its relationship to the ear.

detect angular accelerations of the head in any direction. The canals use a push–pull mechanism so that when the head moves one ear is excited while the other is inhibited. The canals are paired as follows:

- Right lateral and left lateral canal
- Right anterior and left posterior canal
- Left anterior and right posterior canal.

The remaining two sub-organs known as the saccule and utricle are responsible for the detection of linear accelerations as well static head tilt. The saccule detects movements in the vertical plane whereas the utricle responds to linear accelerations in the forward–back and side-to-side planes.

When any of the vestibular subcomponents is stimulated a change in neural activity occurs which generates a compensatory eye movement via the vestibular ocular reflex. The purpose of the vestibular ocular reflex is to ensure that image stability is maintained during head movements. If there is damage to the vestibular apparatus then a change in the baseline firing rate of the nerve occurs. This is interpreted by the vestibular nuclei as a head movement even though the head has remained stationary. As a consequence, involuntary eye movements occur which are clinically noted as nystagmus. The mismatch between the visual and vestibular systems often results in the commonly described symptoms of nausea and vomiting.

Categorization of symptoms

Dizzy patients can find it difficult to articulate their symptoms but with experience key patterns and features can be extracted. To help formulate an understanding of the pathophysiology it is vital that a clear timeline is obtained with the characteristics and severity of the symptoms. The temporal pattern of the symptoms can be broadly split up as:

1. Acute onset vertigo
2. Paroxysmal attacks of vertigo
3. Chronic continuous symptoms of dizziness.

Identifying the length of continuous vertigo with the first attack is often the key to getting the diagnosis. For example benign paroxysmal positional vertigo lasts from seconds to minutes, vestibular neuronitis lasts hours to days, Ménière's disease can vary from a few minutes to 12 hours while vestibular migraine can be fleeting or last for hours and in extreme cases can continue for days.

Acute onset vertigo

Vestibular neuronitis (a very common diagnosis in the balance clinic)

Vestibular neuronitis should be considered when patients present with acute onset vertigo with nausea or vomiting without hearing loss or other neurological deficit. The cause of vestibular neuronitis is thought to be virally-mediated inflammation of the vestibular portion of cranial nerve eight. Usually it is the superior division that is affected, causing a partial dysfunction of the vestibular apparatus including the lateral semicircular canal while sparing cochlear function. Symptoms include a rapid onset of rotational vertigo with associated nausea and occasional vomiting. The vertigo and nausea typically last hours to days, but these do usually settle and only in a minority of cases do they develop in chronic disorders.

Vestibular sedatives are the mainstay of acute management with benzodiazepines or prochlorperazine most commonly used. There is strong evidence for the use of oral steroids at the onset of an acute attack of vestibular neuronitis (Strupp et al, 2004). However, vestibular suppressants should not be continued for more than a few days as this can lead to incomplete central compensation and potentially the development of symptoms of chronic subjective dizziness.

> The underlying cause of benign paroxysmal positional vertigo is migration of calcium carbonate otoconia from the utricle to one of the semicircular canals.

Labyrinthitis (uncommon)
This is often the default diagnosis that most clinicians use when referring to vestibular dysfunction-related vertigo. Since labyrinthitis refers to inflammation of the whole labyrinth hearing should also be affected. Therefore in the absence of an obvious source of infection in the middle ear this diagnosis cannot be made in the acute setting without a vascular cause being first excluded. However, if the patient presents many weeks after the event then once a hearing loss has been confirmed labyrinthitis is a reasonable diagnosis to consider. The distinction is important as the severity of the dizziness and hearing loss is often much greater than with other causes. Acute management is similar to that of vestibular neuronitis.

Posterior circulation stroke (a rare diagnosis in the balance clinic)
Patients presenting with acute onset of vertigo with nausea or vomiting, and gait unsteadiness together with a headache should raise immediate suspicion of a posterior circulation stroke. Other associated red flag symptoms include gait or limb ataxia, paraesthesia, Horner's syndrome, hearing loss or hemi-sensory loss as these signs all suggest involvement of cerebellar or brainstem structures. In such cases an immediate neurological opinion should be sought in order to exclude the possibility of a vascular event. While the current gold standard is a magnetic resonance imaging scan, a combined clinical examination method know as HINTS (head-impulse-nystagmus-test-of-skew) may provide enhanced sensitivity and specificity to detect posterior fossa infarcts within the first 24 hours (Kattah et al, 2009).

Table 2. The 10 Ps of paroxysmal dizziness

1	Positional	Benign paroxysmal positional vertigo, Arnold–Chiari malformation, vertebral basilar insufficiency
2	Postural	Postural hypotension
3	Phobia (phono-/photo-)	Migrainous vertigo, Cogan syndrome
4	Pressure induced	Perilymph fistula, superior canal dehiscence
5	Peripheral sensation	Peripheral neuropathy (diabetes, alcohol, diet)
6	Psychological	Anxiety, hyperventilation
7	Preference (visual)	Visual vertigo
8	Pills and polypharmacy	For example, antihypertensives
9	Poor hearing	Labyrinthitis, Ménière's disease, middle ear
10	Petit mal	Epilepsy

Paroxysmal attacks of vertigo

There are many otological and non-otological causes of paroxysmal vertigo. Details of the more common causes are provided below with a broader overview in *Table 2*.

Benign paroxysmal positional vertigo (very common)
The most common cause of acute onset vertigo is benign paroxysmal positional vertigo (Froehling et al, 1991) and is even more common in the elderly (von Brevern et al, 2007). It typically manifests as a short-lived attack of vertigo (usually less than a minute) associated with nausea brought on by a sudden change in head position (lying down in bed, rolling over, looking up). Once the nystagmus has built to a maximum (crescendo phase), it settles once the head is kept still and then abates (decrescendo phase). However, nystagmus can be regenerated with further head movements and as a result patients may interpret this as continuous vertigo, which can confuse the clinician and is often overlooked especially in the emergency room.

The underlying cause of benign paroxysmal positional vertigo is migration of calcium carbonate otoconia from the utricle to one of the semicircular canals. Once there, the otoconia lie dormant until a sudden head movement in the plane of the canal causes displacement of the crystals and sets up a canal-appropriate nystagmus via the vestibular ocular reflex. These eye movements settle once the debris comes to a standstill.

Although the otoconia debris can be placed in any of the three canals, by far the most common site is the posterior semicircular canal which accounts for more than 90% of presentations. The definitive diagnosis of benign paroxysmal positional vertigo of the posterior canal is made using the Dix–Hallpike test which involves lying the patient in a supine position with his/her head turned to one side by 45°. The technique is described in more detail later. During this test torsional upbeat nystagmus is generated which confirms that site of lesion very accurately. Once benign paroxysmal positional vertigo has been diagnosed, one can immediately perform an appropriate particle repositioning manoeuvre to move the otoconia back into the utricular space. This is usually done using the modified Epley manoeuvre although alternatives such as the Semont manoeuvre also are available (Teixeira and Machado, 2006).

Vestibular migraine (very common)
Despite being one of the most common causes of episodic vertigo, vestibular migraine remains under-diagnosed (Reploeg and Goebel, 2002). The criteria required for vestibular migraine are shown in *Table 3*. Vertiginous symptoms vary from a few minutes to hours or days. There are often prodromal alterations in the sensory modalities including taste, smell, vision and audition. It is worth noting that headache is absent in over 10% of migraine attacks. There may be abnormal eye movements during an

Table 3. Diagnostic criteria required for a diagnosis of vestibular migraine

Definite vestibular migraine	
1 Episodic vestibular symptoms of at least moderate severity	
2 Current or previous history of migraines	
3 One of the following symptoms during two or more vertiginous attacks	Phonophobia
	Photophobia
	Aura including visual

attack, which may be exacerbated by head positions. To the unfamiliar this can mimic benign paroxysmal positional vertigo although the lack of crescendo–decrescendo nystagmus is usually a giveaway. It is important to enquire about family history, travel sickness as a child, diet and motion intolerance as these are usually present in migraine patients. Dietary and lifestyle changes help many patients, with caffeine withdrawal making the biggest difference. If conservative management is unsuccessful then the usual range of migraine prophylactics can be used. The authors have found low dose amitriptyline (10 mg at night) to be most helpful for these patients.

Ménière's disease (rare, 1 in 2000 population)

This condition is overdiagnosed in primary care. Ménière's disease is considered if three of the following symptoms are present: sensorineural hearing loss (usually low frequency), tinnitus (described as roaring), episodic vertigo or aural fullness. The formal guidelines for the diagnosis of Ménière's disease have been revised and are summarized in *Table 4* (Lopez-Escamez et al, 2015). Although endolymphatic-hydrops is used synonymously with Ménière's disease, and all patients with Ménière's disease have hydrops, it does not follow that if a patient has endolymphatic-hydrops that he/she has Ménière's disease. The underlying aetiology in most cases remains uncertain (Foster and Breeze, 2013). Ménière's disease can have a devastating effect on patients as the vertiginous symptoms can come on with minimal warning and last hours. Attacks often occur in clusters and there can be long symptom-free spells. Ménière's disease is often associated with benign paroxysmal positional vertigo, migraine, vestibulopathy and stress. In balance medicine multiple diagnoses often coexist. Treatment protocols have changed dramatically. Betahistine is now seen as relatively ineffective, while intra-tympanic therapies with steroids or gentamicin have revolutionized treatment. Specialist referral is required.

Recurrent vestibular neuronitis (uncommon) or central vestibular decompensation (common)

Following an attack on the peripheral vestibular system, such as is the case in vestibular neuronitis, the central neurological pathways compensate for the deficit and attempt to restore balance between the two ears.

Table 4. Diagnostic criteria required for a diagnosis of Ménière's disease

Definite Ménière's disease
1 Two or more episodes of vertigo 20 minutes and 12 hours
2 Low to medium frequency sensorineural hearing loss. Thresholds must be at least 30 dB worse in the affected ear at two contiguous frequencies below 2000 Hz
3 Fluctuating aural symptoms (hearing, tinnitus and/or fullness) in the affected ears. Must occur within 24 hours of the vertigo episode
4 Not better accounted for by another vestibular diagnosis
based on Lopez-Escamez et al (2015)

However, during periods of stress or physical ailment the compensation process can deconstruct and the tonic imbalance re-manifest, thereby recreating vertiginous symptoms which can be mistaken for recurrent neuronitis. That said, while recurrent vestibular neuronitis is uncommon it can occur, just like recurrent Bell's palsy. There is emerging evidence that antivirals might have a role in cases of recurrent vestibular neuronitis.

Central positional dizziness (rare)

Central positional vertigo arises from a lesion of the cerebellum or the brainstem. As with the acute dizziness presentations, the key to distinguishing a CNS disorder from a peripheral vestibular disorder is the pattern of nystagmus. The most common pattern of central positional nystagmus is pure down-beating nystagmus that lasts as long as the position is held. This is classically associated with Arnold–Chiari malformation but many posterior fossa lesions can generate these signs and symptoms. Pure torsional nystagmus is another common type of central positional nystagmus.

Chronic continuous symptoms

Unilateral vestibular loss

Although the great majority of patients with an acute unilateral vestibular loss compensate in days or weeks, those attending specialist clinics, or doing the rounds of multiple clinics seeking a diagnosis, have not. Explanations may be maladaptation of central balance compensation becoming reliant on visual stimuli for example. Contributions from poor vision, peripheral neuropathy, arthritis, anxiety and aging among many others should be considered. A tailored programme of vestibular rehabilitation physiotherapy will lead to significant improvement in 85% of cases.

Bilateral vestibular loss

Although it is possible to have sequential vestibular events resulting in a bilateral loss in practice this remains fairly rare unless following a disease process such as Ménière's disease, which in 30% of cases can become bilateral.

> In addition to the otoneurological examination a complete cardiovascular and neurological examination is also essential to exclude non-otological causes of imbalance.

Most commonly the cause is head injury or iatrogenic damage (the most common drugs being intravenous ototoxic medications such as aminoglycoside antibiotics). In the case of chemical vestibulotoxicity, both end organs are destroyed simultaneously and therefore acute onset of vertigo is usually absent. Instead a severe ataxia and imbalance ensues. Interestingly in many cases the hearing is spared as gentamicin is preferentially vestibulotoxic rather than ototoxic.

Persistent postural-perceptual dizziness

Left untreated any of the previous conditions can result in chronic subjective dizziness (now termed persistent postural-perceptual dizziness) (Bittar and von Söhsten Lins, 2015) in which patients feel disassociated with their surroundings and are constantly left with a feeling of imbalance and disequilibrium. As a result of the dysfunctional vestibular system, central systems become increasingly reliant on the visual pathways. This becomes problematic when the visual surround becomes complex such as in busy supermarkets – a phenomenon known as visual preference. Treatment starts with vestibular physiotherapy, but persistent postural-perceptual dizziness may need psychological support as well as pharmacological interventions as augmentation therapies to help with the central maladaptation. The authors would usually leave this for more resistant cases after months of physiotherapy.

Clinical examination

Introduction

In addition to the otoneurological examination a complete cardiovascular and neurological examination is also essential to exclude non-otological causes of imbalance. This will be guided by the patient's symptoms but may include lying and standing blood pressure looking for a postural drop, reflexes, muscle tone, plantar responses, peripheral sensation (looking for a peripheral neuropathy), speech, and fundoscopy (looking for papilloedema and possible optic axis deviation seen in acute otolith dysfunction).

Neurotological examination

The neurotological examination is summarized. A more comprehensive review of neurotological examination of a dizzy patient can be found elsewhere (Tusa, 2007).

Aural examination

The external auditory meatus and the tympanic membrane must be examined to exclude peripheral causes of imbalance such as perforation, infection, otitis media, cholesteatoma and otosclerosis (Schwartze sign). Otitis media with effusion is a common cause of imbalance in children but is much rarer in adults. It is also useful to observe for nystagmus while applying positive and negative pressure to the tragus (Hennebert sign), during Valsalva or with a loud noise (Tullio phenomenon). Positive responses are found in patients with perilymph fistula, hypermobile stapes, superior-canal dehiscence and occasionally in Ménière's disease.

Assessment of hearing

Rinne and Weber tuning fork tests distinguish conductive from sensorineural hearing loss. However, in most balance patients a pure-tone audiogram and tympanometry are needed as abnormalities can help identify certain pathologies. Examples include the low frequency sloping sensorineural hearing loss associated with Ménière's disease, the characteristic improved bone-conduction thresholds, air-bone gap and type A (normal) tympanogram seen in superior canal dehiscence syndrome, or a unilateral sensorineural hearing loss seen in labyrinthitis or rarely an acoustic neuroma.

Romberg test

The patient is asked to first stand with his/her feet together, eyes open and arms folded across the chest for 30 seconds and then closed for 30 seconds. The test is described as positive if the patient is stable with eyes open but there is increased body sway with the eyes closed. This is usually ascribed to a reduced proprioceptive input, but an acute vestibular deficit also results in a positive test. A sharpened Romberg test can also be used whereby the patient stands heel-to-toe. This variant is sensitive to the same pathologies as the normal Romberg test but additionally can detect chronic vestibular deficits. A 'wooden soldier' fall straight backwards is often non-organic. Romberg is a relatively non-specific test.

Unterberger test

The patient is asked to march on the spot fifty times with his/her arms outstretched and eyes closed. In the absence of auditory cues and musculoskeletal factors the test can highlight a unilateral vestibular deficit, as the patient is more likely to rotate in the direction of the loss. However, while the test can be helpful in guiding the clinician, there is a large inter- and intra-subject variability and therefore the results must be interpreted in conjunction with all other available information.

Gait

This is a 5 m walk at normal speed, first with eyes open and then eyes closed. The examiner walks alongside the patient for safety reasons and looks out for deviation towards the side of the lesion. As with the Unterberger test the sensitivity and specificity for vestibular disorders is low and therefore results should not be interpreted in isolation. Observe for the shuffling gait of Parkinson's disease, the broad based or ataxic gait of central balance disorders, the

erratic gait of non-organic imbalance, a slapping gait from foot drop or joint position sensory deficits (listen as well as look at the patient walking down the corridor), or the rigid gait of spasticity. Orthopaedic abnormalities should also be observed.

Tandem gait

Here the patient is asked to walk heel-to-toes in a straight line for five steps. With the eyes open this primarily tests cerebellar function as the visual inputs compensate for chronic vestibular and proprioceptive deficits. However, with eyes closed the test assesses vestibular integrity assuming that the visual and proprioceptive inputs are intact. It is also a relatively non-specific test.

Clinical assessments of eye movements

This is essential. The examiner should first ascertain visual acuity of both eyes. Next cover tests should be used to determine strabismus and any latent nystagmus that may be present. The full range of gaze should be examined in both horizontal and vertical planes to 30° from the midline and conjugate eye movements assessed. Care needs to be taken not move the eyes beyond 30° as physiological nystagmus may result. Look for saccadic eye movements and abnormalities of gaze-evoked nystagmus. Consider using Frenzel's glasses to remove optic fixation or better use infra-red video goggles.

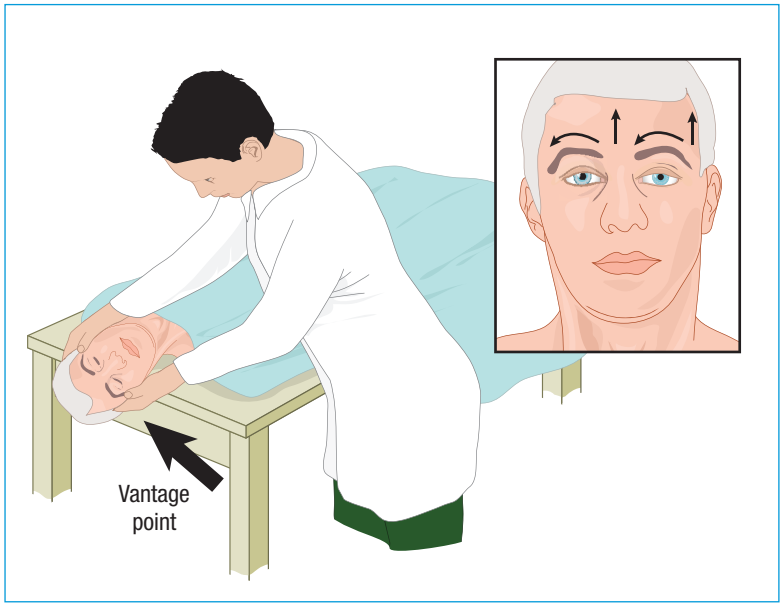
Spontaneous nystagmus: peripheral *vs* central

Selective lesions in the peripheral and central vestibular pathways result in spontaneous nystagmus because of the unopposed neural activity in the intact pathway, e.g. the nystagmus observed immediately following vestibular neuronitis. The nystagmus is graded using Alexander's law, which states that in patients with spontaneous nystagmus as a result of an acute vestibular asymmetry, the slow phase velocity, or drift, of nystagmus is lower when the subject looks toward the side of the slow component of nystagmus compared with the fast phase direction. That is to say that if the nystagmus is only present when the eyes are deviated in the direction of the fast phase it is first-degree, if the nystagmus is also present in primary gaze it is second-degree, and if it is also present with the eyes deviated in the direction of the slow phase it is known as third-degree. Furthermore, the nystagmus should become more pronounced with the removal of optic fixation (as with Frenzel's glasses). If the pattern of nystagmus follows Alexander's law then it is likely to be generated by dysfunction of the peripheral apparatus whereas nystagmus that changes direction with gaze is central in origin until proven otherwise.

Halmagyi head thrust

This is a test for semicircular canal paresis in which the patient is asked to fixate at a distant visual target while the examiner makes a small angle high velocity head thrust in the plane of the canals being tested (it is best to quickly rotate the head back towards the neutral midline position). While these are usually the lateral canals the manoeuvre can be conducted in the right-anterior and left-posterior canals or the left-anterior and right-posterior geometries also. In the presence of unilateral weakness, movements towards the lesion result in the generation of a catch-up saccade, as the vestibular system is unable to provide the corrective vestibular ocular reflex gain. In bilateral weakness these catch-up saccades are seen on both left and right head thrusts.

Figure 2. Schematic showing the Dix–Hallpike position and resulting nystagmus seen in posterior canal benign paroxysmal positional vertigo.



Dix–Hallpike manoeuvre (posterior canal benign paroxysmal positional vertigo)

There are a number of different manoeuvres that can be used to test for benign paroxysmal positional vertigo depending upon the canal that is affected. The Dix–Hallpike test is used to diagnose posterior canal benign paroxysmal positional vertigo, which is much the most common manifestation (*Figure 2*). The patient's head is turned 45° in the horizontal plane while he/she is in the sitting position. The patient then quickly lies down with his/her head hanging over the edge of the bed by approximately 30°. This brings the posterior canal of the downside ear into the vertical plane allowing gravity to act fully. The patient is required to keep his/her eyes open throughout this procedure and the examiner checks for nystagmus.

The most commonly observed eye movement is an upbeating torsional geotropic nystagmus, which results from excitation of the posterior semicircular canal. It is important to wait at least 30 seconds before sitting the patient up as the onset of nystagmus can be delayed in canalithiasis. This is the most common form of benign paroxysmal positional vertigo whereby the otoconia are free floating in the canal as opposed to cupulolithiasis

KEY POINTS

- Dizziness is widespread and patients can present in a variety of settings. Key to an accurate diagnosis is a comprehensive history followed by a thorough clinical examination.
- While most causes of acute vertigo are benign, clinicians need to remain vigilant for central pathologies that can be life threatening.
- The most common cause of vertigo is benign paroxysmal positional vertigo and this can be diagnosed by history and examination alone.
- Early diagnosis of vestibular disorders is crucial and therefore onward referrals should be made promptly. If left untreated symptoms of chronic imbalance can develop, which are very difficult to treat and have a considerable impact on quality of life.

where the otoconia debris become attached the sensory epithelium of the cupula. In canalithiasis there is a delay in the onset of nystagmus which then fatigues. This is in contrast to cupulolithiasis where the nystagmus develops as soon as the head is brought into a gravity dependent position and generally does not fatigue. However, in both forms of benign paroxysmal positional vertigo the patient is usually symptomatic whereas in central positional disorders there perception of vertigo is generally absent. Once the patient is ready, the test is then repeated on the other side.

The treatment of posterior canal benign paroxysmal positional vertigo involves repositioning the otoconia back into the utricular space. This can be done in a number of ways, the most common of which is the modified Epley manoeuvre (Teixeira and Machado, 2006). While the technique is relatively straightforward to perform it is imperative that it is done correctly and it is advisable that specialist training is gained before attempting the technique.

Conclusions

The management of the dizzy patient is critically dependent upon first establishing the correct diagnosis. This can be challenging given that many conditions may present with what appears to be the same symptom: dizziness. A brief introduction to the management of each condition has been given along with the description of each disorder. Patients in the balance clinic often have multiple pathologies. These should be sought and examples have been given. Management of the dizzy patient may need input from more than one specialist if there is diagnostic doubt. However, it is recommended that each region has a named balance centre where dizzy patients without red flag signs may be sent and where expertise can limit the number of appointments. In such centres multidisciplinary teams including otologists, physicians, psychologists, audiologists and physiotherapists can work together to streamline services and shorten the patient journey. **BJHM**

Conflict of interest: none.

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