

Dizziness

Louisa Murdin

Rosalyn Davies

Abstract

Dizziness is common, and approximately 1% of the population consult a GP each year for this symptom. Vertigo is more specific, and suggests a vestibular disorder. Visual and proprioceptive inputs are also important in maintaining balance. Features in the history help localize the problem. Physical examination includes assessment of hearing, eye movements, including the Halmagyi test and Hallpike positional testing and postural blood pressure. Investigations, including caloric testing or electronystagmography, may support the diagnosis. Patients benefit from an accurate diagnosis and explanation of their condition. Vestibular sedatives can be used to manage symptoms acutely but prolonged use should be avoided as these drugs delay compensation. Benign paroxysmal positional vertigo (BPPV) can be treated with particle repositioning manoeuvres. Ménière's disease can usually be managed with a low-salt diet and diuretics, while patients with migrainous vertigo can be successfully treated with migraine prophylactic drugs. Many of these disorders remit spontaneously, but in some cases symptoms are progressive. It is appropriate to refer the patient to an audiovestibular physician or other specialist with an interest if the diagnosis is unclear, compensation fails to occur within 3 months following an acute episode, or the dizziness is associated with hearing loss, tinnitus or neurological symptoms.

Keywords benign positional paroxysmal vertigo; dizziness; electronystagmography; Hallpike test; Ménière's disease; migraine; neurology; peripheral vestibular dysfunction; vertigo; vestibular rehabilitation

Dizziness is used to describe various symptoms ranging from violent spinning vertigo to vague symptoms of light-headedness, muzziness or disorientation. The term 'vertigo' is more specific and is defined medically as an illusion of movement, which may occur horizontally, vertically or obliquely. Whereas dizziness can occur due to a variety of causes, vertigo is commonly due to

Louisa Murdin MSc BMBCh MRCP is a Clinical Research Fellow in Neuro-otology at the National Hospital for Neurology and Neurosurgery, which is part of University College London Hospitals NHS Trust, London, UK. Her research interests include the relationship between migraine and vertigo. Competing interests: none declared.

Rosalyn Davies PhD FRCP is Consultant Physician in Neuro-otology at the National Hospital for Neurology and Neurosurgery, and Honorary Senior Lecturer, Institute of Neurology, London, UK. Her research interests include vestibular rehabilitation of intractable dizziness, migrainous vertigo and Tullio phenomenon. Competing interests: none declared.

disturbance in the vestibular system. The causes of dizziness and vertigo are listed in [Table 1](#).

Dizziness is common, more so in the elderly, and affects women more than men. Estimates suggest that one-third of the population have experienced an episode of vertigo by the age of 70 years. One large population study in the UK has shown that 5 out of every 1000 people consult their general practitioner each year for vertigo, and 10 out of every 1000 for dizziness.¹ Benign positional paroxysmal vertigo (BPPV) and migrainous vertigo are both common causes of episodic vertigo, with lifetime prevalences of 2.5%² and 0.98%³ respectively.

Anatomy and physiology of balance

A complex sensorimotor system is in place to maintain balance in humans ([Figure 1](#)). There are two primary vestibular effector pathways. Firstly, there are projections from the vestibular nuclei to the oculomotor nuclei and on to the muscles of eye movement (the vestibulo-ocular reflex, VOR). This system stabilizes gaze in space during head movements, enabling activities such as reading a street sign while walking. Secondly, there are pathways to the neck, trunk and lower limb antigravity muscles via the spinal reflex arc (vestibulo-spinal reflexes). The purpose of this system is to contribute to control of posture and movement.

The sensory epithelium of the vestibular end-organ comprises the maculae of the utricle and saccule, and the cristae in the ampullae of the three semicircular canals. The semicircular canals and macular hair cells are orientated such that head movements in virtually any plane can be encoded.

The sensory mismatch hypothesis and vestibular compensation

Sensory afferent information is integrated and stored in a data centre thought to be located in the reticular formation of the brainstem. New sensory input is constantly compared with this databank and, under normal circumstances, there is a good match between visual, proprioceptive and vestibular inputs. Symptoms of vertigo result from a mismatch between the sensory information generated by these inputs, which can occur in dysfunction of a sensory system. For example, violent vertigo is a cardinal feature in the initial phase of acute vestibular neuritis. Compensation then occurs because of the inherent plasticity and sensory substitution within the central integrating system, and the symptoms subside. The unpleasant autonomic responses (nausea, vomiting and anxiety) that accompany vertigo are mediated by the vestibulo-autonomic pathways ([Figure 1](#)). The term 'multisensory dizziness' is used to describe the condition in which multiple sensory deficits are contributing to symptoms and inhibiting compensation. For example, an elderly patient with impaired vision (e.g. cataracts), impaired proprioception (e.g. diabetic neuropathy), and impaired vestibular input (e.g. labyrinthine concussion as a result of a minor head injury).

Diagnosis

History

Because the symptoms of dizziness and imbalance caused by vestibular upset can be similar whether the problem arises from

Some causes of dizziness grouped into otological, neurological and medical disorders

Classification	Aetiology
Otological	
Middle ear disease	Perilymph fistula due to trauma or surgery Glomus tympanicum tumour
Unilateral peripheral vestibular dysfunction	Vestibular neuritis, Ramsay–Hunt syndrome, Lyme disease Trauma, e.g. head injury, barotrauma Ischaemia, e.g. atheroma, emboli or vasculitis Basal meningitis, e.g. tuberculosis
Benign paroxysmal positional vertigo (BPPV)	Head injury, vestibular neuritis, migraine, idiopathic
Bilateral vestibular failure	Ototoxicity, e.g. due to aminoglycosides, cisplatin Autoimmune disease Genetic disease, e.g. Usher's syndrome
Ménière's syndrome	Ménière's disease Secondary endolymphatic hydrops
Benign recurrent vertigo	Presumed migrainous phenomenon
Neurological	
VIIIth nerve	Vestibular schwannoma Neurofibromatosis type II
Cranio-cervical junction	Arnold–Chiari malformation
Brainstem/cerebellum	Multiple sclerosis Spinocerebellar atrophy types 1–9, 19, 21, 22, 26 Episodic ataxia types 1 and 2 Friedreich's ataxia Posterior circulation ischaemic disease Space-occupying lesions, e.g. meningioma, brainstem glioma Inflammatory disorders, e.g. Behçet's, lupus, sarcoid Multisystem atrophy, e.g. olivopontocerebellar atrophy
Cortex	Vestibular epilepsy
Generalized	Raised intracranial pressure Drugs, e.g. anticonvulsants, alcohol, antidepressants, tranquillizers Migraine, e.g. basilar migraine, migrainous vertigo
General medical	
Orthostatic hypotension	Prolonged bed rest, hypotensive drugs, autonomic neuropathy, Addisonian states
Vasovagal episodes	Stress, prolonged standing, hot stuffy environments
Low cardiac output	Cardiac arrhythmia, carotid sinus hypersensitivity, aortic stenosis, hypertrophic obstructive cardiomyopathy
Hyperventilation	Panic attack
Hypoglycaemia	Liver disease, diabetic treatment complication
Anaemia	From any cause
Physiological extremes (visual-vestibular conflict)	Height vertigo, motion sickness

Table 1

the labyrinth, VIIIth nerve or the central vestibular pathways, differential diagnosis can be difficult. Various features in the history distinguish peripheral (otological) causes from the central (neurological) causes (Table 2).

Peripheral causes of vertigo are more likely to begin with an acute episode of vertigo associated with nausea and/or vomiting, whereas central dizziness is more likely to be insidious in onset and give more severe symptoms of imbalance. Exceptions to these generalizations include an acute relapse of multiple sclerosis, which can present as peripheral type vertigo, and posterior inferior cerebellar artery infarction, which can present with acute

vertigo with associated neurological features. Bilateral peripheral vestibular hypofunction can present with little dizziness but, instead, symptoms of oscillopsia (bobbing of vision on walking) and ataxia.

Decompensation syndromes following vestibular neuritis may present with various forms of dizziness. Dizziness can vary widely in duration from minutes to weeks. It is typically triggered by movement (e.g. turning the head rapidly to one side, bending forwards) or visual triggers (e.g. travelling on a moving escalator, being surrounded in a crowded shopping mall or supermarket). This latter group of symptoms is termed 'visual vertigo'.⁴

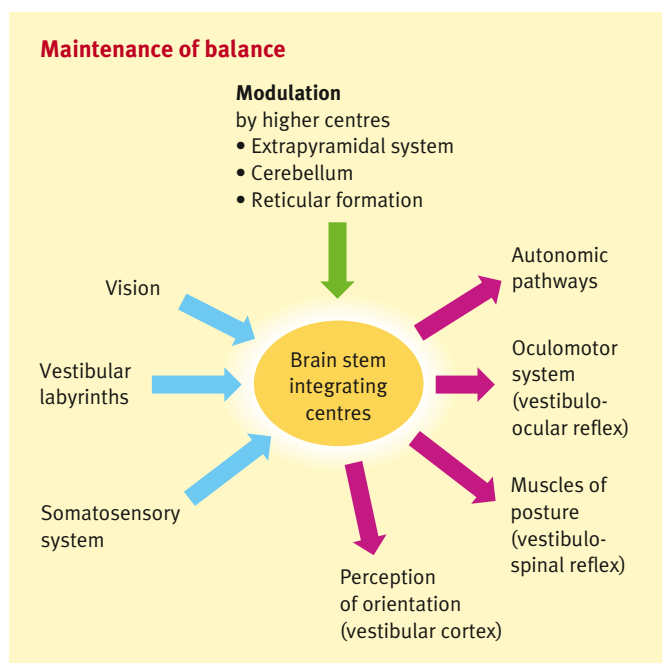


Figure 1

Ménière's disease is a well-known but relatively uncommon cause of episodic vertigo, in which attacks are associated with hearing loss, tinnitus and aural fullness. Aural symptoms are usually unilateral in the initial stages of the disease, but bilateral involvement can occur in a minority of cases. In addition to Ménière's disease, which is by definition idiopathic, Ménière's syndrome can arise as a secondary endolymphatic hydrops in a hearing-damaged ear.

The strong association of migraine with balance disorders is not entirely explained. Vertigo can occur as an aura in the basilar form of migraine. More commonly, episodic vestibular symptoms occur in association with migraine headaches or with other migrainous symptoms (e.g. aura, photophobia, phonophobia) or in response to migraine triggers.⁵ The term 'migrainous vertigo' can be applied to these cases. Although common, migrainous vertigo is known to be under-diagnosed.³

Benign paroxysmal positional vertigo (BPPV) causes severe spinning vertigo lasting less than 1 minute, triggered by specific head movements. Classically, symptoms are provoked by lying back, extending the neck or rolling over in bed. It is thought to result from accumulation of otolith debris, which has become displaced from the utricle and settled as a bolus of crystals in the posterior semicircular canal. This is the canal most effectively activated by the typical triggering head movements described above.

Examination

A full neuro-otological examination comprises otological and vestibular assessment, and a general medical and neurological examination (Table 3). In most cases of dizziness, no abnormality is identified unless the patient is experiencing an acute vestibular episode when examined, or there is associated neurological pathology.

Investigations

Pure tone audiometry is useful to identify conductive or sensorineural hearing loss. Other auditory site of lesion tests are available in specialist centres.

Distinguishing central from peripheral causes of dizziness

Dizziness – localizing features

- Inner ear – hearing loss, tinnitus, aural fullness, otalgia
- VIIIth nerve – poor speech discrimination, associated facial weakness (if VIIth also involved)
- Cerebellopontine angle – impaired facial sensation, clumsiness, dysarthria, incoordination
- Brainstem – hemisensory loss, peri-oral paraesthesiae, hemiparesis, cranial nerve palsies, dysarthria, dysphagia, memory disturbances, loss of consciousness
- Cerebellum – incoordination, clumsiness, dysarthria
- Cortex – loss of consciousness, olfactory or gustatory hallucinations

Duration of dizziness – possible causes

- Few minutes – Benign paroxysmal positional vertigo (<1 minute), vestibular epilepsy, vestibular paroxysmia
- Several minutes to <2 hours – benign recurrent vertigo, vestibular aura of migraine
- 2–24 hours – Ménière's disease, transient ischaemic attack in the posterior circulation, migrainous vertigo
- >24 hours – vestibular neuritis, relapse of brainstem multiple sclerosis, bilateral vestibular failure

NB Poorly compensated peripheral vestibular dysfunction may present as dizziness of any duration.

Table 2

The caloric test and rotational chair testing both evaluate the vestibulo-ocular reflex. In the caloric test, each external auditory canal is irrigated sequentially with water at temperatures of 30 and 44 °C. The nystagmus generated by each irrigation is quantified to indicate absence or asymmetry of vestibular function.

Posturography measures body sway. It has limited sensitivity and specificity for detecting vestibular lesions, but is used to plan rehabilitation strategies.

Imaging is useful in suspected structural lesions. For example, MRI can detect vestibular schwannoma, and high-resolution CT scans may be used for detecting superior semicircular canal dehiscence in patients with sound-induced dizziness (Tullio phenomenon).

Management

Patients benefit from a thorough assessment, explanation of their symptoms and reassurance as appropriate. The avoidance behaviour seen in patients with protracted dizziness may require desensitization programmes, constructed by an appropriately trained therapist. Medical management of dizziness depends on the diagnosis.

Acute vestibular episode

The symptoms of an acute vestibular episode can be treated with various anti-emetics or vestibular sedatives (e.g. prochlorperazine, promethazine, cyclizine, cinnarizine) during the first days of the illness. Buccal prochlorperazine causes less drowsiness

Neuro-otological examination

Otological assessment

- Otoscopy – looking for evidence of middle ear pathology
- Rinne and Weber tuning-fork tests – to identify conductive and asymmetrical sensorineural hearing loss

Vestibular assessment

- Eye movement examination – nystagmus (spontaneous or gaze-evoked)
- Hallpike head positioning test for positional nystagmus – useful in the diagnosis of BPPV (Figure 2). The nystagmus of posterior canal BPPV is torsional, with the upper pole beating towards the undermost ear (geotropic). It occurs after a delay of at least 2 seconds, when the head-hanging position is maintained. It adapts (i.e. disappears) in less than a minute, may reverse on returning to the upright position, and fatigues on repetition of the Hallpike manoeuvre.
- Halmagyi head thrust test – to identify the laterality of an abnormal vestibulo-ocular reflex.⁶
- Romberg test and gait assessment – to identify sensory ataxia and uncompensated vestibular lesions.

Neurological and medical assessment

- Blood pressure after lying for 10 minutes, immediately on standing and for at least 3 minutes afterwards
- Cardiovascular examination for arrhythmia, bruits, murmurs
- Cranial nerves
- Cerebellar tests

Table 3

and sedation, and is slightly more effective than the oral preparation. It is important to wean patients off these medications quickly because they delay the compensation process necessary for recovery and can have severe side effects, such as tardive dyskinesia after prolonged use of prochlorperazine. In most cases, drugs can be stopped within a week. There is some evidence that steroid treatment (i.v. methylprednisolone 100 mg, reducing over 3 weeks) may be useful in the treatment of severe acute vestibular neuritis.⁷ Treatment for motion sickness should

The Hallpike manoeuvre

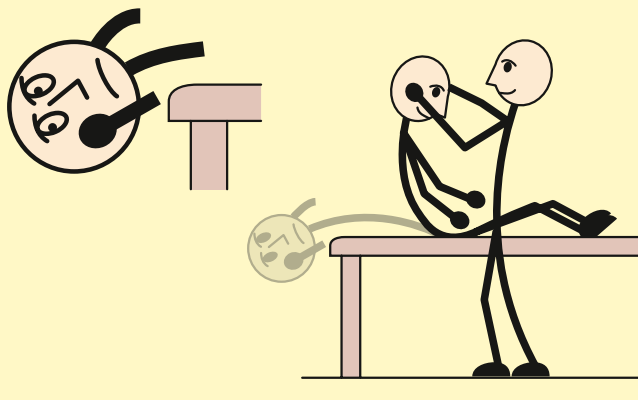


Figure 2

be prophylactic. Hyoscine (scopolamine) patches are available and are known to be more effective than placebo.⁸

Ménière's disease

Ménière's disease is managed by a salt-restricted diet (1–2 g/day) and thiazide diuretics, and regular medication with betahistine may be useful. Balance exercises may be required as vestibular function is progressively lost in the affected ear. There is some evidence that severe intractable vertigo that is unresponsive to medical therapy can be treated by intra-tympanic aminoglycosides,⁹ dexamethasone or, as a last resort in selected cases, vestibular neurectomy.

Migrainous vertigo

Acute episodes of migraine should be treated symptomatically with analgesics, anti-emetics and 5-HT_{1B/D} antagonists, such as sumatriptan or rizatriptan, if required. The effect of migraine drugs on vertigo is not yet clear, but prophylaxis with propranolol or amitriptyline may be beneficial according to one retrospective study.¹⁰

Peripheral vestibular dysfunction

When dizziness continues to recur after an acute peripheral vestibular episode, the patient is described as 'poorly compensated'. Central compensation is expedited by vestibular rehabilitation exercises such as the Cawthorne–Cooksey programme or individually tailored exercise regimens, both of which are known to be effective.¹¹ The use of rating scales by therapists to design customized programmes has improved outcome, as has intervention within 6 months of the acute vestibular episode.¹² For patients with visual vertigo, rehabilitation using optokinetic stimulation with full-field stimuli or immersive visual environmental 'virtual reality' displays can be used in a graded manner.¹³

Benign paroxysmal positional vertigo

Particle repositioning manoeuvres are extremely successful in the treatment of BPPV; cure is achieved on the first attempt in 70% of the patients. The Epley or Semont manoeuvres are commonly used for posterior canal BPPV. Specialist expertise is required to confirm the diagnosis, as positional nystagmus can also be caused by central pathology, and to screen for significant cervical pathology, which can be a contra-indication to the repositioning manoeuvre. Recurrent BPPV can be treated with Brandt–Daroff exercises, which can be performed at home by the patient.

Central vestibular disorders

Management is generally best supervised by a neurologist or neurosurgeon. Central vestibular disorders, causing symptomatic eye movement abnormalities that are unlikely to remit, can be managed with low dose clonazepam, baclofen or gabapentin.¹⁴ Some patients with gait ataxia may benefit from gait and posture advice from a physiotherapist.

Prognosis

BPPV may remit spontaneously, but about 50% of treated patients may experience recurrences. The course of Ménière's disease is variable and a majority will develop progressive hearing loss. The second ear is eventually involved in 50% of cases,

although the vertigo 'burns out' in about 70% of patients over the years. Following acute vestibular neuritis, chronic imbalance may develop in 20% of subjects, and vestibular rehabilitation may reduce disability. Chronic dizziness has far-reaching consequences on the psychological, professional and social life of the individual, and may lead to long-term morbidity. It is appropriate to refer the patient to an audiological physician or an ENT surgeon with an interest in balance disorders if compensation fails to occur within 3 months following an acute episode of dizziness, or the dizziness is associated with hearing loss, tinnitus or neurological symptoms. ♦

REFERENCES

- 1 Davis A, Smith P, Ferguson M, Stephens D, Gianopoulos I. Acceptability, benefit and costs of early screening for hearing disability: a study of potential screening tests and models. *Health Technol Assess* 2007; **11**(Suppl 42): 5–53.
- 2 von Brevern M, Radtke A, Lezius F, et al. Epidemiology of benign paroxysmal positional vertigo: a population based study. *J Neurol Neurosurg Psychiatry* 2007; **78**: 710–15.
- 3 Neuhauser HK, Radtke A, von Brevern M, et al. Migrainous vertigo: prevalence and impact on quality of life. *Neurology* 2006; **67**: 1028–33.
- 4 Guerraz M, Yardley L, Bertholon P, et al. Visual vertigo: symptom assessment, spatial orientation and postural control. *Brain* 2001; **124**: 1646–56.
- 5 Furman JM, Marcus DA, Balaban CD. Migrainous vertigo: development of a pathogenetic model and structured diagnostic interview. *Curr Opin Neurol* 2003; **16**: 5–13.
- 6 Halmagyi GM, Curthoys IS. A clinical sign of canal paresis. *Arch Neurol* 1988; **45**: 737–39.
- 7 Strupp M, Zingler VC, Arbusow V, et al. Methylprednisolone, valacyclovir, or the combination for vestibular neuritis. *N Engl J Med* 2004; **351**: 354–61.
- 8 Spinks AB, Wasiak J, Villanueva EV, Bernath V. Scopolamine (hyoscine) for preventing and treating motion sickness. *Cochrane Database Syst Rev* 2007(3) CD002851.
- 9 Kisilevsky V, Einarson TR, Kozar E, Koren G, Rutka JA, Cohen-Kerem R. Intratympanic gentamicin for Menière's disease: a meta-analysis. *Laryngoscope* 2004; **114**: 2085–91.
- 10 Maione A. Migraine-related vertigo: diagnostic criteria and prophylactic treatment. *Laryngoscope* 2006; **116**: 1782–86.
- 11 Hillier SL, Hollohan V. Vestibular rehabilitation for unilateral peripheral vestibular dysfunction. *Cochrane Database Syst Rev* 2007(4) CD005397.
- 12 Bamiou DE, Davies RA, McKee M, Luxon LM. Symptoms, disability and handicap in unilateral peripheral vestibular disorders. Effects of early presentation and initiation of balance exercises. *Scand Audiol* 2000; **29**: 238–44.
- 13 Pavlou M, Lingeswaran A, Davies RA, Gresty MA, Bronstein AM. Simulator based rehabilitation in refractory dizziness. *J Neurol* 2004; **251**: 983–95.
- 14 Strupp M, Brandt T. Pharmacological advances in the treatment of neuro-otological and eye movement disorders. *Curr Opin Neurol* 2006; **19**: 33–40.

FURTHER READING

- Baloh RW, Honrubia V. Clinical neurophysiology of the vestibular system. Oxford: Oxford University Press, 2001.
- Bronstein AM, Lempert T. Dizziness: a practical approach to diagnosis and management. Cambridge: Cambridge University Press, 2007.
- Davies R. Bedside neuro-otological examination and interpretation of commonly used investigations. *J Neurol Neurosurg Psychiatry* 2004; **75**(Suppl 4): 32–44.
- Luxon LM, ed. Textbook of audiological medicine. Clinical aspects of hearing and balance. London: Martin-Dunitz, 2003.

Practice points

- The history is the key to the correct diagnosis and thus management of dizziness
- In acute vestibular episodes, avoid overprescription of vestibular sedatives because they delay cerebral compensation
- Benign paroxysmal positional vertigo (BPPV) is a common and treatable cause of dizziness in the general population
- Migrainous vertigo is increasingly recognized as a common cause of dizziness