Vertigo: clinical assessment and diagnosis

Dizziness is a common presenting complaint to emergency departments and medical admission units; the key is to differentiate between central and peripheral causes of vertigo. This review focuses on bedside assessment of vertigo and, by looking at patterns of symptoms and signs, provides guidance as to when to suspect a central cause.

Vertigo is an illusion of rotation caused by an asymmetric disorder of the peripheral labyrinths of the inner ear or the central connections including the vestibular nerve, nuclei and the cerebellum. The patient feels as if either he/she or the environment is rotating or, less commonly, tilting. It is associated with nausea, vomiting and unsteadiness. It is always temporary, always made worse by movement of the head and almost never causes loss of consciousness unless this is secondary to a fall or other complication (Halmagyi, 2005).

Table 1. Key features in history

Vertigo	Acute onset or more gradual
	Preceding or precipitating factors
	Hearing loss
Auditory symptoms	Tinnitus
	Ear discharge or pain
	Sense of fullness or blockage in the ear
Headache	Before, during or after the vertigo
Associated neurology	Diplopia
	Other visual disturbance
	Dysarthria or dysphagia
	Paraesthesia or muscle weakness
Medication	Aminoglycosides
	Anticonvulsants
Risk factors for cerebrovascular accident or transient ischaemic attack	Previous angina or myocardial infarction
	Diabetes mellitus
	Hypertension
	Smoking
	Atrial fibrillation
Past history	Head injury
	Chronic ear infections
	Migraine

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To correctly recognize vertigo it is helpful to know what it is not. It is not a sensation of faintness or impending loss of consciousness, often called pre-syncope, which is most often caused by an inadequate supply of blood to the brain. It is also not impaired balance or gait in the absence of any abnormal head sensation which is often the result of impaired motor control. It is not vague lightheadedness, heavyheadedness, wooziness or a hallucination of movement whereby the patient believes there is movement where there is none. Having established that the prominent symptom is indeed vertigo the following section details what to focus on in the history and examination.

Bedside assessment of acute vertigo

Most causes of vertigo can be elucidated with a thorough history and examination. Only rarely are further tests required. It is important to distinguish peripheral vestibular lesions from central lesions. *Tables 1* and *2* outline the key features which need to be assessed in the history and examination respectively.

Specific tests can be performed at the bedside and contribute greatly to making a diagnosis.

Table 2. Key features on examination

Eye movements	Range of movement
	Diplopia
	Gaze evoked or spontaneous nystagmus
Nystagmus	Rotatory, horizontal, vertical or mixed
	Direction of gaze in which most pronounced
	Does it change direction on changing direction of gaze
Ears	Tympanic membrane for any perforation
Hearing	Whispered voice at arm's length with other ear occluded
	Weber's and Rinne's test to determine whether sensorineural or conductive deafness
Other neurology	Cranial and peripheral nerves
Gait and balance	Romberg's test
	Ability to stand and walk unaided

Head impulse test

This was developed by Halmagyi and Curthoys (1988). The patient sits upright with gaze fixed on a target roughly 3 m away and is instructed to keep looking at the target while the examiner turns the patient's head (*Figure 1*). Starting with the head slightly turned to one side the examiner turns the head rapidly in a horizontal direction to the other side to about 20°, watching the eyes for corrective eye movements (saccades). The test is then repeated turning the head in the opposite direction. In normal people there are no saccades, indicating that their gaze is fixed on the target. If there are corrective saccades after a head thrust in one direction it indicates that there is a peripheral vestibular lesion affecting that side.

Positional test

This was developed by Dix and Hallpike (1952). With the patient sitting upright on the bed the head is turned 45˚ to one side (*Figure 2*). The patient is then laid back quickly with the head turned until it is extended over the end of the bed with one ear downwards, and examined for nystagmus. If present the latency (time taken for the nystagmus to start after the manoeuvre), the direction of nystagmus in relation to the downward ear, and whether the nystagmus fatigues over time (wait for 60 seconds) should be noted. After this the patient should sit up and be examined for further nystagmus, again noting its features. The procedure is repeated on the same side to see whether there is any reduction in the nystagmus (and associated vertigo and nausea) on repeat testing. If there is no nystagmus the patient should sit up, be examined for nystagmus again and then the whole test should be repeated on the opposite side.

The classic result of the Hallpike test, seen in benign paroxysmal positional vertigo, is nystagmus after a latency of 2–6 seconds which is mixed rotatory and horizontal (seen as down towards the lower ear), associated with vertigo and nausea which wanes as the nystagmus reduces over the subsequent 30 seconds. A similar but milder result is seen on sitting the patient up. The nystagmus is less pronounced (fatigues) on repeat testing.

Blocking visual fixation of nystagmus

An ophthalmoscope can be used to block visual fixation and allow easier detection of a spontaneous nystagmus (Traccis et al, 2004). The patient is asked to cover one eye with their hand while the fundus of the other eye is viewed with the ophthalmoscope. A common result is for initially unapparent nystagmus to become obvious or for known nystagmus to increase in amplitude. This is associated with a peripheral vestibular lesion. It is important to note that because the optic nerve head is behind the centre of rotation of the eye the nystagmus is in the opposite direction to what is observed normally. Nystagmus that is not reduced by fixation, is vertical in direction, changes direction on changing direction of gaze or is non-fatiguable suggests a central cause.

Common peripheral causes of vertigo

Benign paroxysmal positional vertigo

Benign paroxysmal positional vertigo is the single most common cause of vertigo according to Furman and Cass (1999) with an incidence of 64/100 000/year; it is more common in the elderly with a mean age of onset of 51 years (Froehling et al, 1991). It is generally thought to be caused by the movement of otolith particles within the semicircular canals of the inner ear. This predominantly affects the posterior canal giving rise to the classical symptoms but it can also affect the horizontal and anterior canals (Fife, 1998). Benign paroxysmal positional vertigo is usually idiopathic but can also follow

Figure 1. Head impulse test. From Halmagyi (2005).

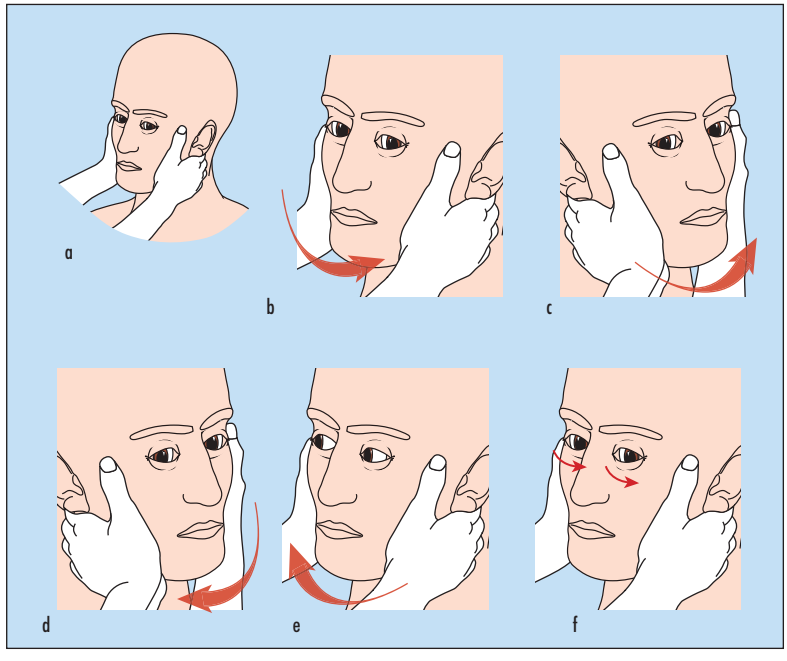


Figure 2. Dix–Hallpike test.

head injury, chronic ear infections, surgery and previous vestibular neuronitis, although the symptoms of benign paroxysmal positional vertigo may not occur till years after the episode of neuronitis (Baloh et al, 1987).

Posterior canal benign paroxysmal positional vertigo usually presents with episodic vertigo lasting for 10–20 seconds and is precipitated by rotational movement of the head, classically turning in bed, getting up from bed or extending the head to look upwards (Furman and Cass, 1999). It is associated with nausea and vomiting but no auditory symptoms. Although the acute vertigo only lasts for seconds, following repeat episodes patients can get non-specific dizziness or lightheadedness which can last for some hours. Standard examination can be normal but may reveal nystagmus which is mixed rotatory and horizontal and is reduced with visual fixation (i.e. enhanced by blocking visual fixation). Diagnosis is via the Hallpike positional test which gives the classical findings outlined above. Most patients spontaneously recover over several weeks although this can be facilitated by conducting special repositioning manoeuvres. The modified Epley manoeuvre takes 2–3 minutes, can be performed in the emergency department, and has been shown to be effective at decreasing and resolving symptoms (Froehling et al, 2000). *Figure 3* shows the Epley particle repositioning manoeuvre for left posterior semicircular canal benign paroxysmal positional vertigo.

Ménière's disease

Ménière's disease affects 15–40/100 000/year and is the result of endolymphatic hypertension which is essentially a dilatation of the membranous labyrinth within the semi-circular canals of the inner ear with an increased volume of endolymph (Andrews and Honrubia, 1996). This can be either idiopathic or secondary to viral or bacterial infections of the ear or metabolic disorders.

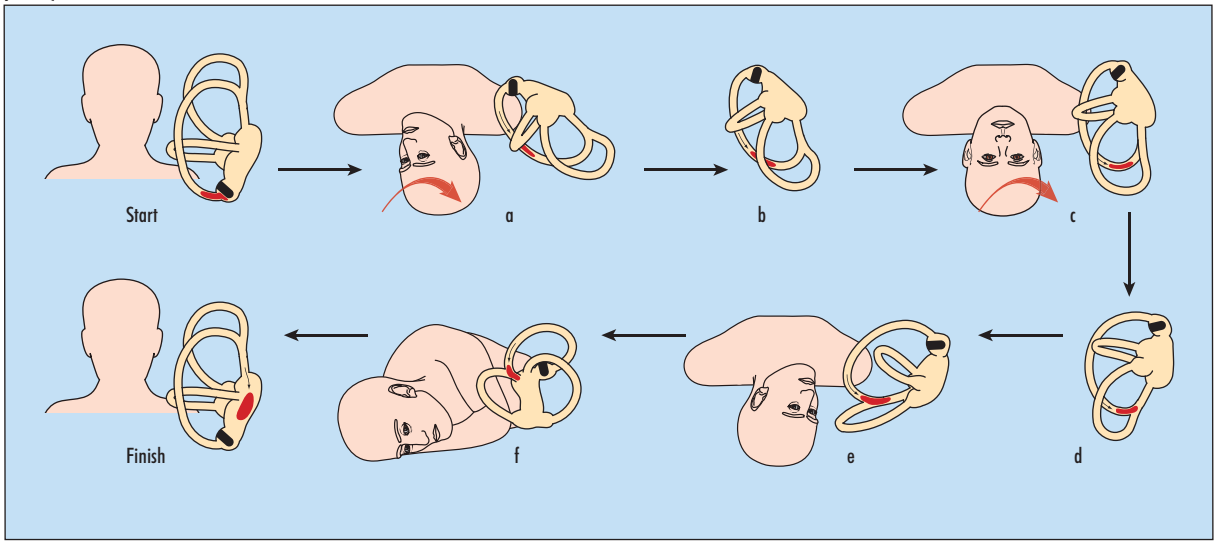
Ménière's disease presents with spontaneous, well-defined, prolonged episodes of vertigo that can last from 20 minutes to 24 hours, the vertigo rapidly increasing in intensity over the first few minutes. It is frequently associated with nausea and vomiting but there are no neurological symptoms. There is, however, associated deafness and tinnitus which can take a few days to resolve after the vertigo settles, and with recurrent episodes there may be progressive deafness. There is no specific test for Ménière's disease but examination during an episode may reveal sensorineural deafness. There is always nystagmus which is typically horizontal and beating away from the affected ear although it can beat towards the affected ear during the recovery period.

Ménière's disease can follow a widely different course for each patient. This ranges from symptoms over a few months only, to episodic symptoms throughout life, to progressive episodes which lead to a chronic state of hearing loss, tinnitus and decreased balance. Treatment is aimed at maintaining remission with initially a low salt diet and diuretics which block sodium reabsorption (Andrews and Honrubia, 1996). Surgery can be effective at stopping the attacks of vertigo but cannot restore any hearing loss.

Vestibular neuronitis

This has also been called labyrinthitis, vestibular neuritis, neurolabyrinthitis and acute unilateral peripheral vestibulopathy. It is thought to be caused by inflammation of

Figure 3. The Epley particle repositioning manoeuvre for left posterior semicircular canal benign paroxysmal positional vertigo. The patient is rapidly reclined into the left Dix–Hallpike position (a) and remains in that position until both the vertigo and nystagmus have well and truly disappeared and the otoconial particles have settled into the lowest portion of the posterior semicircular canal duct. The patient's head is slowly turned by 90° into the right Dix–Hallpike position (b–e) so that the particles are guided into the common crus. Then the patient slowly rolls onto the right shoulder and the head is turned another 90° so that the particles fall back into the vestibule. From Halmagyi and Creamer (2000).



the vestibular nerve presumably secondary to viral infection as it often follows a viral infection (Baloh, 2003). In keeping with this it tends to occur in epidemics, can affect members of the same family and tends to be more common in spring.

It presents with spontaneous vertigo developing over a period of hours and remains severe for a few days and takes weeks to subside. It is associated with nausea, vomiting and decreased balance with the patient falling towards the affected ear although he/she can stand and walk unaided. There are no auditory or neurological symptoms. On examination there is nystagmus which is typically horizontal with the slow phase towards the affected ear. It is always unidirectional (i.e. beats in the same direction even if the head is turned) and always decreased by fixation. This means that blocking visual fixation as outlined above will make it more readily apparent.

The head impulse test is invariably positive but the rest of the neurological examination is normal (Traccis et al, 2004). Attacks terminate through vestibular compensation so the treatment is aimed at controlling symptoms during the acute severe phase. Medications include vestibular sedatives such as promethazine coupled with an anti-emetic such as prochlorperazine. Once the acute phase is over it is important that vestibular sedatives are not used as the vertigo and symptoms are important for successful restoration of peripheral vestibular function and central compensation. Treatment at this point focuses on vestibular rehabilitation exercises which aim to improve ocular stability and balance.

Migraine

Selby and Lance (1960) found vertigo to be a common symptom, reported by 27–33% of all patients with migraine. It has been previously known as part of the aura (along with visual loss, diplopia and dysarthria) for 'basilar migraine' where it precedes the typical hemicranial headache. However, vertigo is increasingly being recognized as a migrainous symptom in its own right. In these cases the vertigo often lacks the temporal relationship to the headache, lasts much longer than a typical aura and often improves with treatments used for migraine (Reploeg and Goebel, 2002).

Migrainous vertigo can be positional or spontaneous. It can last from seconds to days but most commonly it lasts a few hours. As with a typical migraine it can be precipitated by alcohol, lack of sleep or emotional stress and it may be associated with nausea, visual auras, photophobia and phonophobia. Hearing loss and tinnitus are not prominent. It can improve with rest and in most cases the individual will have suffered from a migraine headache in the past (Neuhauser and Lempert, 2004). Examination findings include poor balance, nystagmus and normal hearing. The nystagmus can be of different types and so is not helpful in making the diagnosis.

Central causes of vertigo

Vertebrobasilar circulation infarct and haemorrhage

Blood supply to the peripheral vestibular labyrinth, vestibular nerve, brainstem vestibular nuclei and cerebellum comes from the vertebrobasilar system. Interruption to flow in any of the branches of the vertebrobasilar system can lead to vertigo. The vertigo tends to be of sudden onset, severe and prolonged with nausea, vomiting and instability such that the patient struggles to stand unaided, even with eyes open. Most patients have risk factors for cerebrovascular disease and there are often other neurological symptoms such as headache, visual disturbance, diplopia, dysarthria, weakness or numbness. Sometimes symptoms can be very subtle and careful history and examination is needed, particularly when the vertigo is not positional and there are no auditory symptoms (Huang and Yu, 1985).

Owing to the proximity of structures in the brainstem and posterior fossa, it is unusual to sustain an infarction without associated neurological signs on examination. Amarenco (1991) found these included dysarthria, dysphagia, focal sensory or motor deficits, visual field deficits, cortical blindness or significant ataxia. The nystagmus can be of any type but it is commonly pure horizontal or vertical nystagmus which is bidirectional (i.e. changes direction on changing the direction of gaze), non-fatiguable and not reduced by fixation. Brainstem strokes may lead to abnormal eye signs: pupillary reflexes, corneal reflex and oculocephalic ('doll's eyes') response. The Hallpike positional manoeuvre may reveal nystagmus which is not classical; this would include cases where there is no latency or fatiguability, and the direction of nystagmus is not the classical horizontal towards the downward ear. Halmagyi and Curthoys (1988) found that a negative head impulse test also raises suspicion of a central cause.

It is important to diagnose vertebrobasilar circulation infarcts as they are often caused by cardiogenic embolism and may need long-term anticoagulation. Cerebellar infarctions in particular carry a significant risk of severe complications including cerebellar oedema and swelling which can lead to increased pressure in the posterior fossa, brainstem compression and obstructive hydrocephalus (Amarenco, 1991). Up to a third of cerebellar infarcts require urgent neurosurgical decompression. A less common but important cause of cerebellar ischaemia, particularly in younger patients, is vertebral artery dissection, sometimes occurring after minimal neck trauma or manipulation (Bison and Artico, 2004).

Vertebrobasilar insufficiency

This commonly presents with recurrent episodes of acute onset spontaneous vertigo which for most patients lasts from seconds to minutes. It is more common in the elderly and there are often risk factors for cerebrovascular disease. It is most commonly associated with visual symp-

toms including diplopia and visual field defects, but it can also be associated with incoordination, drop attacks and weakness (Grad and Baloh, 1989). Examination between attacks is usually normal.

Intracranial tumours

It is unusual for intracranial tumours to present with vertigo as most are slow growing which allows time for central compensation. A more common presentation is hearing loss or other neurological symptoms or signs. However, there have been reports (Dunniway and Welling, 1993) of intracranial tumours presenting as vertigo in patients with histories suggestive of benign paroxysmal positional vertigo. In most cases there were symptoms or signs on careful assessment which would have made the diagnosis of benign paroxysmal positional vertigo unlikely. These included auditory symptoms like hearing loss, tinnitus and auditory fullness, and atypical findings on the positional test. Posterior fossa tumours involving the fourth ventricle or Chiari malformations are likely to be undetectable on computed tomography scan and to require a magnetic resonance image for diagnosis.

Brainstem multiple sclerosis can present with acute vertigo and nystagmus although there is usually a history of other neurological events and it is rare to get vertigo without other neurological symptoms or signs.

Conclusions

Most cases of acute vertigo are the result of one of the common peripheral vestibular causes. Suspicion of a central cause depends on recognizing when the signs and symptoms presented by the patient do not match the features of any of the peripheral disorders with reasonable accuracy.

The diagnostic yield of a computed tomography scan in an emergency setting is very low, being unable to detect many posterior fossa lesions as well as demyelination. The investigation of choice for a central cause is magnetic resonance imaging or, if vertebrobasilar insufficiency or vertebral artery dissection is suspected, magnetic resonance angiography. If magnetic resonance imaging is unavailable it may be safe to observe the patient if there is only mild concern, as in those with a peripheral cause the nystagmus will improve significantly over 48 hours (Baloh, 1998). **BJHM**

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Conflict of interest: none.

Amarenco P (1991) The spectrum of cerebellar infarctions. *Neurology* **41**: 973–9
Andrews JC, Honrubia V (1996) Meniere's disease. In: Baloh RW, Halmagyi GM, eds. *Disorders of the Vestibular System.* Oxford University Press, New York: 300–19
Baloh RW (1998) Differentiation between peripheral and central causes of vertigo. *Otolaryngol Head Neck Surg* **119**: 55–9
Baloh RW (2003) Vestibular neuritis (clinical practice). *N Engl J Med* **348**: 1027–32
Baloh RW, Honrubia V, Jacobsen K (1987) Benign positional vertigo: clinical and oculographic features in 240 cases. *Neurology* **37**: 371–8
Bison E, Artico R (2004) Vertebral artery dissection as a rare cause of vertigo: case report. *Acta Otorhinolaryngol Ital* **24**(5): 284–7
Dix MR, Hallpike CS (1952) The pathology, symptomatology and diagnosis of certain common disorders of the vestibular system. *Proc R Soc Med* **45**: 341–54
Dunniway HM, Welling DB (1993) Intrancranial tumours mimicking benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg* **109**: 853–60
Fife TD (1998) Recognition and management of horizontal canal benign positional vertigo. *Am J Otol* **19**: 345–51
Froehling DA, Silverstein MD, Mohr DN et al (1991) Benign positional vertigo: incidence and prognosis in a population based study in Olmsted county, Minnesota. *Mayo Clinic Proc* **66**: 596–601
Froehling DA, Bowen JM, Mohr DN et al (2000) The canalith repositioning procedure for the treatment of benign paroxysmal positional vertigo: a randomised controlled trial. *Mayo Clinic Proc* **75**: 695–700
Furman JM, Cass SP (1999) Benign paroxysmal postional vertigo. *N Engl J Med* **341**: 1590–6
Grad A, Baloh RW (1989) Vertigo of vascular origin. Clinical and electronystagmographic features in 84 cases. *Arch Neurol* **46**: 281–4
Halmagyi GM (2005) Diagnosis and management of vertigo. *Clin Med* **5**: 159–65
Halmagyi GM, Curthoys IS (1988) A clinical sign of canal paresis. *Arch Neurol* **45**: 737–9
Halmagyi GM, Creamer PD (2000) Assessment and treatment of dizziness. *J Neurol Neurosurg Psychiatry* **68**: 129–36
Huang CY, Yu YL (1985) Small cerebellar strokes may mimic labyrinthine lesions. *J Neurol Neurosurg Psychiatry* **48**: 263–5
Neuhauser H, Lempert T (2004) Vertigo and dizziness related to migraine: a diagnostic challenge. *Cephalalgia* **24**: 83–91
Reploeg MD, Goebel JA (2002) Migraine associated dizziness: patient characteristics and management options. *Otol Neurotol* **23**: 364–71
Selby G, Lance J (1960) Observations on 500 cases of migraine and allied vascular headache. *J Neurol Neurosurg Psychiatry* **23**: 23–32
Traccis S, Zoroddu GF, Zecca MT, Cau T, Solinas MA, Masuri R (2004) Evaluating patients with vertigo: bedside examination. *Neurol Sci* **24**: S16–S19

KEY POINTS

- It is important to establish whether the presenting complaint truly is vertigo.
- A number of useful bedside tests can be easily learned.
- Associated neurological symptoms, severe imbalance and persistent, severe vertigo are features suggestive of a central cause.
- Any neurological signs, nystagmus of a central type, an abnormal response to the Hallpike test, a negative head impulse test or an inability to stand up even with eyes open are suggestive of a central cause.
- Magnetic resonance imaging is the modality of choice for suspected central causes.