

An approach to vertigo in general practice

Sindhu Dommaraju, Eshini Perera

Background

Dizziness is a common and very distressing presentation in general practice. In more than half of these cases, the dizziness is due to vertigo, which is the illusion of movement of the body or its surroundings. It can have central or peripheral causes, and determining the cause can be difficult.

Objective

The aim of this article is to provide a clear framework for approaching patients who present with vertigo. A suggested approach to the assessment of vertigo is outlined.

Discussion

The causes of vertigo may be central (involving the brainstem or cerebellum) or peripheral (involving the inner ear). A careful history and physical examination can distinguish between these causes. The most common causes of vertigo seen in primary care are benign paroxysmal positional vertigo (BPPV), vestibular neuronitis (VN) and Ménière's disease. These peripheral causes of vertigo are benign, and treatment involves reassurance and management of symptoms.

Vertigo is a common, distressing presentation in general practice and constitutes approximately 54% of cases of dizziness.¹ Classically, vertigo presents as a sensation of movement of the environment around the patient.² Often patients describe a 'spinning' sensation of either their body or their surroundings. This sensation can be confused with dizziness, which is a non-specific term, so an adequate history is required to differentiate this symptom. Dizziness can be classified into four groups:

- vertigo (spinning sensation)
- disequilibrium (feeling of imbalance)
- light-headedness (sensation of giddiness)³
- presyncope (sensation of feeling faint).

Vertigo can be classified as central or peripheral on the basis of vestibular symptom pathology.⁴ Vestibular symptoms originating from pathology in the cerebellum or brain stem are classified into the central type. Conversely, symptoms arising in the inner ear or from the vestibular nerve are classified as peripheral.

Balance is controlled by the cerebellum, which receives input from the vestibular nuclei in the brain stem.⁵ This in turn receives input from the visual pathway, proprioception and inner ear. The vestibular apparatus of the inner ear consists of three semicircular canals, and two otolith organs called the utricle and the saccule.⁶ The utricle and saccule contain hair cells embedded in calcium

carbonate crystals. These cells control vertical and non-rotational movements. Receptors in the semicircular canals respond to the position of the head. When the head tilts, receptors on the ipsilateral ear are stimulated and receptors in the contralateral ear are inhibited. These send impulses to the brain stem and cerebellum to control balance. Any disruption of this pathway can lead to vertigo.⁷

History

Often, patients presenting with dizziness are unable to describe the sensation and can be vague, particularly if it is the initial presentation. It is important to differentiate vertigo from other non-rotational forms of dizziness. Once the diagnosis of vertigo is determined, this needs to be differentiated into a central or peripheral subtype.³

More serious central causes, such as cerebrovascular accidents (CVAs), tumours and multiple sclerosis (MS), need to be considered. Central causes are suspected if the patient presents with associated neurological symptoms such as weakness, dysarthria, sensory changes, ataxia or confusion. It may be difficult to distinguish between central and peripheral causes in patients who present with vertigo as their only symptom. Risk factors for vascular disease, including smoking, diabetes, obesity, hypertension and hypercholesterolaemia, need to be assessed to rule out CVAs, which

can lead to vertigo from ischemia or infarction.⁸

Peripheral pathology is associated with symptoms of nausea, vomiting and hearing loss. Vertigo can be triggered by a change in the position of the head, recent upper respiratory tract infection (URTI), stress or trauma. Patients who experience anxiety or panic attacks can have vertigo as a result of hyperventilation.⁸ Loud noises can precipitate vertigo in patients with peripheral causes in a phenomenon called Tullio phenomenon.⁹ Medications, including frusemide, salicylates and antihypertensive agents, can affect the vestibular system, which in turn causes vertigo.^{10,11} Perilymphatic fistula should be considered if there is a history of recent head injury.

Vertigo with hearing loss is seen in labyrinthitis and Ménière's disease, whereas hearing loss is not seen in benign paroxysmal positional vertigo (BPPV) and vestibular neuronitis (VN). A brief approach to assessing vertigo is outlined in Figure 1.

Physical examination

A physical examination can assist in differentiating between central and peripheral subtypes of vertigo. Examination should involve the following:

- Ear examination: an otoscopic examination should be performed to visualise the tympanic membranes for any vesicles that can be seen in a *Herpes zoster* infection or retraction pockets as seen in cholesteotoma.² Vertigo triggered by pushing on the tragus or with the Valsalva manoeuvre is seen in a perilymphatic fistula. A hearing assessment should be performed.
- Neurological examination: initially, a focused neurological examination including gait, balance and coordination needs to be performed. A gait and balance assessment (Romberg's sign and the heel-toe test),¹² and examination for cerebellar signs can exclude central causes.
- Eye examination: eyes need to be examined for nystagmus and

papilledema. Nystagmus is quick, jerky, involuntary movements of the eye. Vertical nystagmus is only seen if the cause is central.⁷ Nystagmus due to central causes may be horizontal, rotational or vertical, and does not disappear on fixing the gaze.¹³ Nystagmus in the peripheral type disappears with fixation of the gaze.

- Cardiovascular examination: pulse, blood pressure, heart rate and rhythm should be checked.¹⁴ Carotid examination to identify bruits (in the case of a CVA) is necessary. Further imaging to rule out CVA can be performed if this is clinically suspected.

A number of specialised tests are required to determine the causes of vertigo. These are highlighted in Table 1.

Causes

The most common causes of vertigo seen in primary care are BPPV, VN and Ménière's disease (Table 2).² While most of the causes of vertigo are benign, more serious causes, including CVAs,

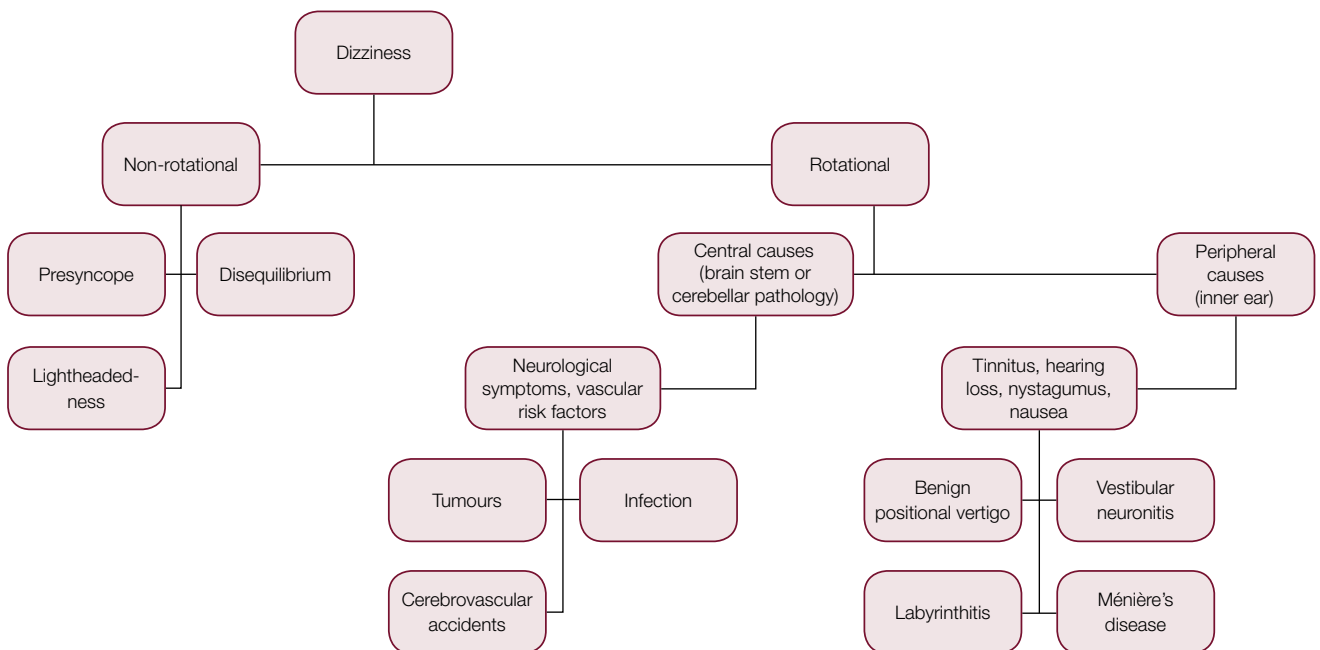


Figure 1. A brief approach to assessing vertigo

MS, tumours, psychogenic causes and perilymphatic fistula, need to be considered,² particularly in patients who are older or have risk factors for vascular diseases.

BPPV

BPPV is the most common cause of vertigo in clinical practice.¹⁵ It is caused by an accumulation of calcium crystals in the posterior semicircular canal. These crystals affect the movement of the endolymph in the semicircular canals, which causes vertigo.¹⁵ The classic symptoms of BPPV are brief episodes

of vertigo, associated with nausea and nystagmus.

The episodes of vertigo are triggered by rapid changes in the position of the head.¹ Symptoms can last for weeks and recur after remission. Nystagmus seen in BPPV has a rotational nature.¹ The Dix–Hallpike manoeuvre is used to diagnose BPPV. In contrast, the Epley manoeuvre can be used to treat BPPV (Box 1).¹ This procedure is performed in an attempt to dislodge the otoliths from the semicircular canals. It has a 77% success rate on the initial attempt and 100% on further attempts.¹⁵

Acute labyrinthitis

The inner ear is composed of the bony and the membranous labyrinth. Acute labyrinthitis is inflammation of this labyrinth. It presents with vertigo and hearing loss, preceded by a viral infection.¹⁶ Middle ear infections can spread to the inner ear and cause labyrinthitis. The duration of symptoms ranges from days to weeks. Hearing loss is the main distinguishing factor between labyrinthitis and BPPV. Typically, no treatment is required for labyrinthitis. However, if suppurative labyrinthitis is suspected, the patient should be referred to the emergency department for drainage of otitis media.¹⁷

Ménière's disease

Ménière's disease is an uncommon cause of vertigo.¹⁸ It is thought to be caused by increased fluid in the endolymph part of the cochlea, which eventually affects the semicircular canals.¹⁸ In most patients, the aetiology is unknown.¹⁸ Ménière's disease is commonly seen in women, and the incidence increases in men and women after the age of 60 years.¹⁸

Ménière's disease classically presents with episodes of vertigo lasting longer than 20 minutes, tinnitus, sensorineural hearing loss and aural fullness. Eventually, hearing loss becomes permanent. Pure tone audiometry is the most useful test to assess sensorineural hearing loss.

Table 1. Interview schedule: key topics and questions

Physical examination	Description
Dix–Hallpike manoeuvre	The patient sits at the edge of the bed and the examiner turns the patient's head 30–45 degrees to the side being tested. The patient needs to keep their eyes open and focus on a stable point, and then quickly lie supine and hyperextend the neck. Horizontal nystagmus denotes a positive test. This test can induce vertigo, so patients should be warned about this before the test is performed. ^{2,7}
Orthostatic blood pressure testing	A drop in the systolic blood pressure by more than 20 mm/Hg from a lying down to standing position is significant for a postural drop. This is seen in patients who are dehydrated or who present with autonomic dysfunction. ² Autonomic dysfunction can occur acutely and be driven by vertigo.
Head impulse test	The patient is asked to look at the examiner's nose and the examiner quickly turns the patient's head 10–20 degrees. It is abnormal if eyes move rapidly and repetitively, failing to re-fixate to the examiner's nose. A positive test indicates disrupted vestibule-ocular reflex. ⁷

Table 2. Most common differential diagnoses of vertigo

Differential diagnosis	Onset and duration of each attack of vertigo	Provoking factors	Special features	Physical exam findings
Labyrinthitis	Few seconds to minutes	Change in the head position	Tinnitus	Hearing loss present
Vestibular neuronitis	Seconds to minutes	Recent upper respiratory tract infection	Imbalance, while nystagmus is horizontal or rotational, the direction of the fast component is away from the side of the lesion	Absence of hearing loss
Benign paroxysmal positional vertigo	Seconds	Change in the head position	Positional	Positive Dix–Hallpike
Ménière's disease	Hours	Spontaneous	Hearing loss and tinnitus	Hearing assessment for sensorineural hearing loss

Box 1. Epley manoeuvre for treatment of BPPV

1. Sit the patient on the bed.
2. With the neck hyperextended and laterally rotated to 45 degrees, the patient is asked to lie supine.
3. The patient is required to maintain the supine position for one minute.
4. The head is turned to the opposite side with lateral rotation of 45 degrees, and remains in this position for one minute.
5. The head, chest and pelvis are tilted downwards to 135 degrees (almost prone), and this position is maintained for one minute.
6. Quickly sit the patient upright with their head tilted to the affected side.

This manoeuvre should not be performed in patients with neck injury, carotid stenosis and heart disease¹

There is no known cure for Ménière's disease and treatment is primarily symptomatic.¹⁹ Betahistine is the current mainstay of treatment. Surgical treatment options exist; however, most patients are adequately managed with medical treatment.

Vestibular neuronitis

VN is caused by inflammation of the vestibular nerve. This inflammation precedes a viral URTI or herpes zoster infection and is caused by immune-mediated sequelae following the viral illness.¹⁵ It is commonly seen in middle-aged adults of both sexes.⁸ VN often occurs in epidemics during outbreaks of respiratory infections.¹⁵ The main characteristic of VN is an acute onset of vertigo without hearing loss or tinnitus. Similarly to BPPV, symptoms of vertigo are aggravated by a change in the position of the head. Loss of balance is more prominent in VN, compared with other causes of vertigo, and patients may commonly present with falls.

Initially, the vertigo is severe, lasts for two to three days and is followed by gradual recovery, which may take two to six weeks.²⁰ Bed rest and antiemetics can be used in the first 24–72 hours. Patients can be reassured that symptoms will improve with time.²⁰

Investigations

Blood tests are not routinely ordered for patients presenting with vertigo. However, it is recommended that glucose levels of all patients with vertigo should

be checked.²¹ Radiological tests including computed tomography (CT), magnetic resonance imaging (MRI) or magnetic resonance angiography (MRA) are indicated if:

- the examination is not consistent with a peripheral lesion
- prominent risk factors for CVA are present
- neurological signs and symptoms are present, or
- symptoms of vertigo are accompanied by a headache.

In these cases, referral to a neurologist is recommended.^{22,23}

Treatment

Treatment is tailored to the specific causes of vertigo. Antiemetic medications such as betahistine are used for symptomatic management of acute vertigo. These medications should not be used long term. Patients should be warned about the side effects of drowsiness, dry mouth and blurred vision. Benzodiazepines are not indicated and should be avoided because of their addictive nature.^{16,24}

Lifestyle changes including salt restriction and avoiding alcohol and coffee are recommended.²⁴ Assessment and management of the patient's risk of falls is important in vertigo. Referral to an experienced physiotherapist for vestibular rehabilitation can help prevent recurrences.¹⁶ Exercises prescribed by physiotherapists involve moving the eye balls up and down and sideways in a supine or sitting position. Balance

stabilisation exercises help to regain normal activities faster.²⁴

Patients should be referred for specialist review if they have symptoms of continuous vertigo, progressive hearing loss, severe ongoing headache, cerebellar signs or if the diagnosis is not clear.⁷

Key points

- Vertigo is a common presentation in general practice.
- A careful history is required to elicit features of central or peripheral causes of vertigo.
- Serious causes including CVAs and MS need to be considered.
- Physical examination involves a neurological, cardiovascular, eye and ear examination.
- Treatment is specific to the cause of vertigo.

Authors

Sindhu Dommaraju MBBS, FRACGP, DCH, General Practitioner, University of Newcastle, Callaghan, NSW. kvdsindhu@gmail.com

Eshini Perera MBBS, BMedSci, MMed, MPH, FRACGP, Dermatology Registrar, University of Melbourne, Parkville, VIC

Competing interests: None.

Provenance and peer review: Not commissioned, externally peer reviewed.

References

1. Cranfield S, Mackenzie I, Gabbay M. Can GPs diagnose benign positional paroxysmal vertigo and does the Epley manoeuvre work in primary care? *Br J Gen Pract* 2010;60(578):698–99.
2. Labuguen RH. Initial evaluation of vertigo. *Am Fam Physician* 2006;73(2):244–51.
3. Post RE, Dickerson LM. Dizziness: A diagnostic approach. *Am Fam Physician* 2010;82(4):361–69.
4. Kuo CH, Pang L, Chang R. Vertigo – Part 1 – Assessment in general practice. *Aust Fam Physician*. 2008;37(5):341–47.
5. Purves D, Augustine GJ, Fitzpatrick D, et al. Central vestibular pathways: Eye, head, and body reflexes. *Neuroscience*. 2nd edn. Sunderland, MA: Sinauer Associates, 2001.
6. Lee SC, Abdel Razek OA, Dorfman BE, et al. Vestibular system anatomy. New York: Medscape, 2010.
7. A delicate balance: Managing vertigo in general practice. *Best Practice Journal* 2012;46(Sep):30–37.
8. Paine M. Dealing with dizziness. *Aust Prescr* 2005;28(1):94–97.
9. Colebatch JG, Day BL, Bronstein AM, et al. Vestibular hypersensitivity to clicks is characteristic of the Tullio phenomenon. *J Neurol Neurosurg Psychiatry* 1998;65(5):670–78.

10. Drozd CE. Acute vertigo: Peripheral versus central etiology. *Nurse Pract* 1999;24(4):147–48.
11. Baloh RW. The dizzy patient. *Postgrad Med* 1999;105:161–64, 167–72.
12. Baloh RW. Differentiating between peripheral and central causes of vertigo. *Otolaryngol Head Neck Surg* 1998;119(1):55–59.
13. McIntyre AK. The quick component of nystagmus. *J Physiol* 1939;97(1):8–16.
14. Karatas M. Vascular vertigo: Epidemiology and clinical syndromes. *Neurologist* 2011;17(1):1–10.
15. Mathews T. Peripheral vertigo in general practice. *Continuing Medical Education* 2006;33:267–70.
16. Swartz R, Longwell P. Treatment of vertigo. *Am Fam Physician* 2005;71(6):1115–22.
17. Boston ME. Labyrinthitis treatment and management. New York: Medscape, 2016. Available at <http://emedicine.medscape.com/article/856215-treatment> [Accessed 22 February 2016].
18. Cruz MD. Ménière's disease. A stepwise approach. *Medicine Today*. 2014;15(3):15–26.
19. Burgess A, Kundu S. Diuretics for the treatment of Ménière's disease or syndrome. *Cochrane Database of Syst Rev* 2006;(3):CD003599. Updated 2010.
20. Cooper CW. Vestibular neuronitis: A review of a common cause of vertigo in general practice. *Br J Gen Pract* 1993;43(369):164–67.
21. Herr RD, Zun L, Mathews JJ. A directed approach to the dizzy patient. *Ann Emerg Med* 1989;18:664–72.
22. Wippold FJ, 2nd, Turski PA. Vertigo and hearing loss. *Am J Neuroradiol* 2009;30(8):1623–25.
23. Gizzi M, Riley E, Molinari S. The diagnostic value of imaging the patient with dizziness. A Bayesian approach. *Arch Neurol* 1996;53(12):1299–304.
24. Sura DS, Newell S. Vertigo-diagnosis and management in primary care. *BJMP* 2010;3(4):a351.

correspondence afp@racgp.org.au