Dizziness in older adults

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Abstract
Dizziness is a very common symptom in older adults. Its prevalence increases with age, with approximately one-third of elderly people experiencing the symptom. It may be caused by true vertigo, of peripheral or central cause, pre-syncope, disequilibrium or a combination of these. A detailed history and examination are essential in distinguishing common causes. Providing there is no evidence of central vertigo or of a cardiac cause, further investigation is rarely helpful in diagnosis or management. Management of the patient with dizziness should be holistic; it should not only focus on treatment of the underlying cause but also include other elements such as medication and functional review, and the input of allied health professionals as appropriate.

Keywords benign positional paroxysmal vertigo; dizziness; elderly; Hallpike test; Ménière's disease; migrainous vertigo; neurology; nystagmus; postural hypotension; vertigo; vestibular neuronitis

Dizziness is a common problem in older people, with approximately 30% of community-dwelling elderly people reporting the symptom.[1,2] Its prevalence increases with age, with 54% of the over-90s experiencing dizziness in the preceding 6 months.[2] In one community study, the 1-year prevalence of elderly people presenting to their family doctor with dizziness was 8.3%[3] and the symptom is also frequently encountered amongst older acute medical referrals or hospital in-patients, as a primary or associated symptom.

What challenges do dizzy patients present?

- *Dizziness* is a non-specific word. When patients use dizziness to describe their symptoms they may be referring to true vertigo, or light-headedness, pre-syncopal symptoms or a sensation of disequilibrium. The clinician must obtain a history to clarify what the patient means.
- The differential diagnosis of the patient with *dizziness* is extensive.
- In older patients presenting with *dizziness* the cause will often be multi-factorial.
- Health professionals often hold the erroneous belief that treatment is ineffectual.

Clinical approach to the dizzy patient

History

A good history is paramount in assessing a patient with dizziness and can identify the diagnosis in around 70% of patients.[4–6] The initial challenge is to determine what the patient means when they say that they are 'dizzy', as the precise sensation experienced will give clues as to the underlying cause(s).

- *Vertigo* refers to an illusion of movement. The patient may feel as if objects in their surroundings are moving or that they are moving in relation to their environment.
- *Pre-syncope* refers to a feeling of light-headedness, sometimes associated with nausea or sweating and 'clamminess'. Positive responses to questions such as 'does it feel as if you are about to faint?' and 'does it feel similar to how you feel when you stand up too quickly?' suggest pre-syncope.
- *Disequilibrium* refers to a feeling of unsteadiness or 'veering' to one side, primarily when walking. It is typically worsened when vision is simultaneously impaired, for example in the dark or if the patient is asked to close their eyes.

If the symptom described can be classified into one of these three broad sensations, the causes shown in Figure 1 can be considered.

Precipitating and aggravating factors may provide important clues to the underlying cause.

Vertigo that is provoked by head movements such as turning over in bed or looking upwards is suggestive of benign paroxysmal positional vertigo (BPPV). An antecedent upper respiratory tract infection suggests acute vestibular neuronitis or, less commonly, viral labyrinthitis. Symptoms associated with Ménière's disease and perilymphatic fistulas can be provoked by loud noise. Perilymphatic fistulas can also be aggravated by sneezing or coughing. Stress can trigger psychogenic vertigo. Acute vestibular neuronitis, stroke and Ménière's disease may present spontaneously without any precipitating cause.

Pre-syncope preceded by a sudden change in posture may be due to postural hypotension; if preceded by more prolonged standing it may be due to a malignant vasovagal syndrome.

The time course is useful in differentiating causation. The presence or absence of associated features can also aid in distinguishing the underlying cause. Table 1 demonstrates this.

As with all common presentations in the elderly, a drug history is essential. Common drugs that cause vestibulotoxicity resulting in vertigo include gentamicin, non-steroidal anti-inflammatory agents, furosemide and quinine. Drugs that commonly induce postural hypotension include antihypertensive agents, tricyclic antidepressants, diuretics and levodopa.

Examination

A complete examination should be performed (Figure 2) in all patients presenting with dizziness, focussing on the following:

- *Erect and supine blood pressures*. If the patient's symptoms do not occur in association with a fall in blood pressure, it should not be assumed that the observed postural hypotension is the cause of recurrent symptoms.
- *Pulse*. Sustained or paroxysmal tachy- and bradyarrhythmias can cause pre-syncopal symptoms.

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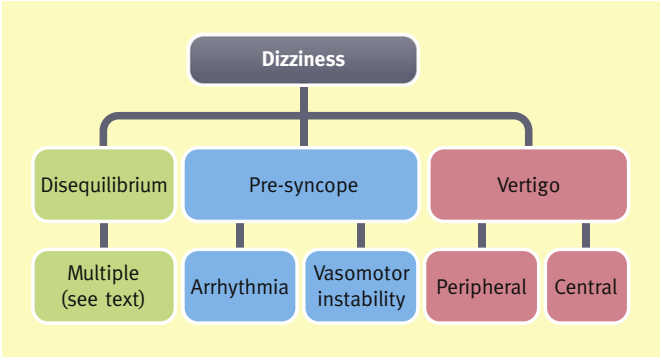

Figure 1

- *Nystagmus* can help in differentiating between central and peripheral causes of vertigo. In peripheral vertigo, nystagmus is horizontal and unidirectional with the fast phase away from the lesion. Visual fixation inhibits the nystagmus. Tinnitus and deafness may be present. In central vertigo, nystagmus can be in any direction. Vertical or purely torsional nystagmus are classically associated with central lesions, where this is often also associated focal neurological signs.
- *Neurological examination* including cranial nerves. This will identify focal neurology that would suggest a central cause of vertigo such as stroke or multiple sclerosis. It will also identify factors that may contribute to disequilibrium such as peripheral neuropathy and reduced visual acuity.
- *Examination of gait* will identify features contributing to disequilibrium, such as a wide-based gait, and may provide evidence of focal neurological disease.
- *Bedside hearing tests*. Hearing can be assessed simply at the bedside by gently whispering into each ear and asking the patient to repeat what was said. Weber's and Rinne's tests are used to differentiate between conductive and sensorineural hearing loss.
- *Provocation tests*. In patients who do not have vertigo and nystagmus at rest the Dix–Hallpike manoeuvre can be useful (Figure 3). It has a sensitivity of 50–88% in patients with BPPV.[7] In patients with BPPV there is a latent period of between a few seconds to 20 seconds before the onset of the nystagmus and it will last less than 1 minute. It will be unidirectional and will usually be horizontal or rotational. The nystagmus is fatigable with repetition. In central disorders there will not be a latent period before the onset of nystagmus and it will last more than 1 minute. There is

Type of dizziness		Associated symptoms	Episode duration	Possible aetiology
Vertigo	Central	Headache	Several minutes–1 h	Posterior circulation transient iscahemic attack
		Vomiting		
		Double vision	Several hours	Migraine
		Staggering gait	Days	Posterior circulation stroke
		Clumsiness		Multiple sclerosis
		Dysarthria		Migraine
		Numbness of the face or body		
	Peripheral	Hearing loss	Few secs	Acute vestibular neuronitis
		Tinnitus	Few seconds–few minutes	Benign paroxysmal positional vertigo
		Feeling of fullness in the ear		Perilymphatic fistula
		Nausea and vomiting	Several minutes–1 h	Perilymphatic fistula
			Several hours	Acoustic neuroma
				Ménière's disease
				Perilymphatic fistula
Pre-syncope		Sweating	Few secs–few minutes	Orthostatic hypotension
		Blurred or tunnel vision		Situational syncope (e.g. post-micturition, post-cough)
		Palpitations		
		Breathlessness		
		Fatigue		Vasovagal – mediated by emotional distress
				Arrhythmia
Disequilibrium		Numbness of feet	Weeks–months	Cerebellar disease
		Impaired vision		Parkinson's disease
		Gait disturbance		Gait disorders
				Peripheral neuropathy
				Reduced visual acuity
Other			Weeks–months	Psychogenic

Table 1

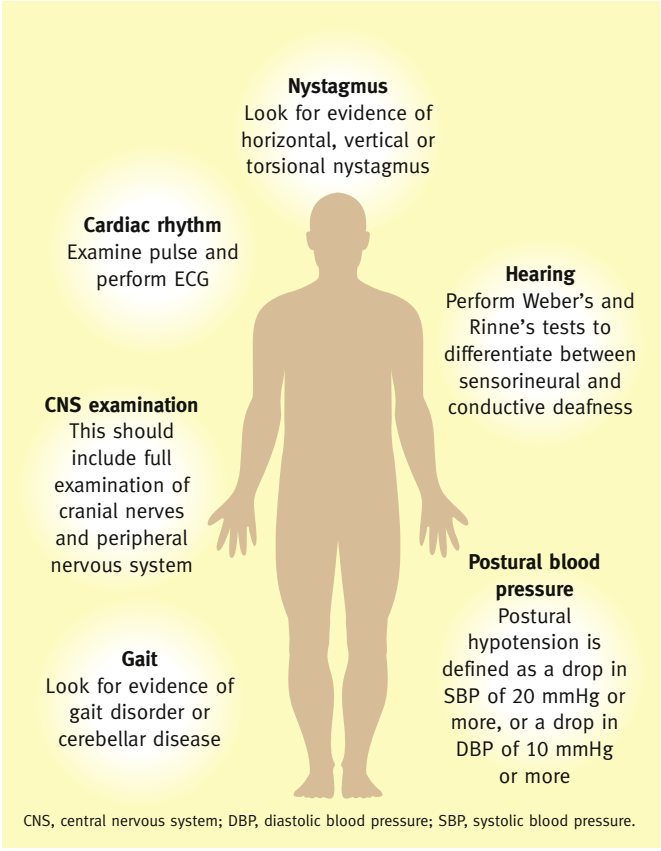

Figure 2

no fatigability. The intensity of the vertigo will usually be less than with peripheral disorders.

Further investigations

Routine investigations for all patients presenting with dizziness are rarely helpful and the cause of dizziness can usually be elicited by a thorough history and examination.[8]

If neurological examination reveals previously unrecognized focal neurological signs suggestive of central nervous system disease, neuroimaging is indicated. If an arrhythmia is suspected, perhaps because of clearly paroxysmal episodes of pre-syncope without clear precipitant, then further cardiac investigations such as Holter monitoring are warranted.

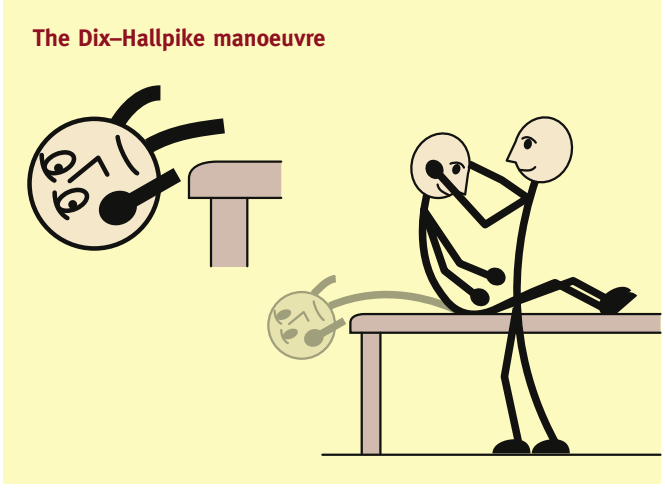

Figure 3

Management

Management depends on the underlying cause. Any contributing drugs should be stopped. It is useful to refer the patient to an optician if they have reduced visual acuity. Assessment by a physiotherapist is useful.

Postural hypotension

The first priority should be the withdrawal of any offending medications. Patients should be taught to stand up slowly and in stages. Thigh-high thrombo-embolic deterrent (TED) stockings can be useful. However, many elderly patients have difficulties in getting them on and off and they are not suitable for patients with peripheral vascular disease. Fludrocortisone, a synthetic mineralocorticoid, can be used in patients who do not respond to the above measures.[9] Midodrine, a peripheral selective α_1-adrenergic agonist, is another agent that can be used but its use is off-licence in the UK.[10,11]

BPPV

Particle-repositioning exercises such as the Epley manoeuvre are the mainstay of treatment in BPPV. In a meta-analysis, 74% of patients who had the Epley manoeuvre had clinical resolution of their symptoms within 1 month, compared to only 33% of controls.[12] Brandt–Daroff exercises can be performed at home and may be useful for patients with recurrent episodes, although the response rates for these exercises are less than those obtained with the Epley manoeuvre.[13]

Ménière's disease: a low-salt intake is recommended, and nicotine and caffeine, which may result in vasoconstriction and impaired microvascular flow within the labyrinthine system, should also be restricted. There is little evidence for any of the drug therapies commonly used in Ménière's;[14] two systematic reviews concluded that there was insufficient evidence to determine whether either betahistine or diuretics, both believed to act by reducing the volume of fluid in the inner ear, had any effect on Ménière's.[15,16] Vestibular rehabilitation may be useful for patients with residual balance problems after their vertigo has settled. For the minority of patients who do not respond to lifestyle interventions and medical management, interventional treatment such as intratympanic gentamicin or vestibular neurectomy can be used.[17,18]

Migrainous vertigo

The treatment of acute episodes should include standard treatments for acute episodes of migraine, including analgesia, anti-emetics, and $5HT_1$-receptor agonists such as sumatriptan. Known migraine triggers should be avoided and prophylactic medications (for example tricyclic antidepressants or β blockers) may also help patients with recurrent episodes of migrainous vertigo.[19]

Vestibular neuronitis

Symptomatic treatment in the form of anti-emetics and vestibular sedatives (e.g. prochlorperazine and cyclizine) are often required in the first few days of an acute episode.[20] These treatments

should not be used for more than a few days as they delay compensation mechanisms and, therefore, long-term recovery. A recent review found that, compared to placebo, corticosteroids improved the rate of complete caloric recovery after 1 month but not at 12 months. However, corticosteroids did not appear to improve symptoms of vertigo at 24 h compared with placebo and there is currently insufficient evidence to support the routine use of corticosteroids in acute vestibular neuritis.[21]

Central causes of vertigo

These are generally best managed in a specialist environment by either a neurologist or a neurosurgeon.

Summary

Dizziness in the elderly is a common symptom and can have many different causes. A systematic approach to the history and examination will reveal the underlying diagnosis in the majority of cases, allowing for management to be targeted appropriately. ◆

REFERENCES

1 Colledge NR, Wilson JA, MacIntyre CCA, et al. The prevalence and characteristics of dizziness in an elderly community. *Age Ageing* 1994; **23:** 117–20.

2 Rupprecht R, Lang E, Schoffski O, et al. Dizziness in an older community dwelling population: multifactorial syndrome. *J Nutr Health Ageing* 2009; **13:** 278–82.

3 Maarsingh OR, Dros J, Schellevis FG, van Weert HC, Bindels PJ, Horst HE. Dizziness reported by elderly patients in family practice: prevalence, incidence, and clinical characteristics. *BMC Fam Pract* 2010; **11:** 2.

4 Tinetti ME, Williams CS, Gill TM. Dizziness among older adults: a possible geriatric syndrome. *Ann Intern Med* 2000; **132:** 337.

5 Kroenke K, Lucas CA, Rosenberg ML, et al. Causes of persistent dizziness. A prospective study of 100 patients in ambulatory care. *Ann Intern Med* 1992; **117:** 898–904.

6 Sloane PD, Baloh RW. Persistent dizziness in geriatric patients. *J Am Geriatr Soc* 1989; **37:** 1031–8.

7 Hoffman RM, Einstadter D, Kroenke K. Evaluating dizziness. *Am J Med* 1999; **107:** 468–78.

8 Colledge NR, Barr-Hamilton RM, Lewis SJ, et al. Evaluation of investigations to diagnose the cause of dizziness in elderly people: a community based controlled study. *Br Med J* 1996; **313:** 788–92.

9 Maile S, et al. Orthostatic hypotension: evaluation and treatment. *Cardiovasc Hematol Disord Drug Targets* 2007; **7:** 63–70.

10 Ward CR, et al. Midodrine: a role in the management of neurocardiogenic syncope. *Heart* 1998; **79:** 45–9.

11 Low PA, et al. Efficacy of midodrine vs placebo in neurogenic orthostatic hypotension. A randomised, double-blind multicentre study. Midodrine Study Group. *J Am Med Assoc* 1997; **277:** 1046–51.

12 Prim-Espada MP, De Diego-Sastre JI, Perez-Fernandez E. Meta-analysis on the efficacy of Epley's manoeuvre in benign paroxysmal positional vertigo. *Neurologia* 2010; **25:** 295–9.

13 Soto VA, Bartual MJ, Santos PS, et al. Benign paroxysmal vertigo: a comparative prospective study of the efficacy of Brandt and Daroff exercises, Semont and Epley maneuver. *Rev Laryngol Otol Rhinol* 2001; **122:** 179–83.

14 Coelho DH, Lalwani AK. Medical management of Ménière's disease. *Laryngoscope* 2008; **118:** 1099–108.

15 Thirlwall AS, Kundu S. Diuretics for Ménière's disease or syndrome. *Cochrane Database Syst Rev* 2006. CD003599.

16 James AL, Burton MJ. Betahistine for Menière's disease or syndrome. *Cochrane Database Syst Rev* 2001. CD001873.

17 Pullens B, van Benthem PP. Intratympanic gentamicin for Meniere's disease or syndrome. *Cochrane Database Syst Rev* 2011. CD008234.

18 Colletti V, et al. Vestibular neurectomy and microvascular decompression of the cochlear nerve in Meniere's disease. *Skull Base Surg* 1994; **4:** 65–71.

19 Reploeg MD, Goebel JA. Migraine-associated dizziness: patient characteristics and management options. *Otol Neurotol* 2002; **23:** 364.

20 Hamid M. Medical management of common peripheral vestibular diseases. *Curr Opin Otolaryngol Head Neck Surg* 2010; **18:** 407–12.

21 Fishman JM, Burgess C, Waddell A. Corticosteroids for the treatment of idiopathic acute vestibular dysfunction (vestibular neuritis). *Cochrane Database Syst Rev* 2011. CD008607.

