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Vertigo

Part 1 – Assessment in general practice

Background
Vertigo is a common and diagnostic challenge faced by clinicians.

Objective
This article discusses the assessment of patients with vertigo.

Discussion
The clinical assessment aims to: establish the presence of true vertigo, differentiate between vertigo of central or peripheral origins, and to evaluate the need for urgent investigations and referrals. Peripheral causes of vertigo are more common, but central causes such as transient ischaemic attack or stroke should always be considered and ruled out appropriately. Presence of syncope excludes the peripheral causes of vertigo. Vertigo in the elderly population is likely to be multifactorial and warrants careful evaluation. Online videos of the head impulse test and the Dix-Hallpike manoeuvre are valuable as these tests are of great diagnostic value. Audiological testing and neuroimaging can provide further information to guide patient management.

The word vertigo is derived from the Latin 'vertere' meaning to turn and '-igo' meaning a condition. Medically, vertigo refers to a specific symptom describing a false sense of motion, usually spinning or rotatory, in the surroundings or within oneself despite the absence of physical movement. In clinical practice, the term 'vertigo' is not usually volunteered by patients. Instead, a nonspecific complaint of 'feeling dizzy' is commonly used and clinicians are therefore faced with the challenge of deciphering the actual meaning of such a complaint.

The need for medically meaningful interpretations of 'dizziness' has resulted in a classification system with four different subtypes.[1] Features and clinical implications of these subtypes are summarised in *Table 1*. Out of the four subtypes of 'dizziness', vertigo accounts for around 32% of all cases[2,3] and up to 56.4% in the elderly population.[4]

Causes of vertigo

The labyrinth is an inner ear neurosensory organ made up of two components: semicircular canals (for balance) and cochlear (for hearing) (*Figure 1*). Typically, vertigo is caused by an imbalance of sensory inputs into the two vestibular nuclei from overactivity or underactivity of either or both sides of the labyrinth.[5] The brain interprets such input differences as a sensation of movement. However, any disturbances to the labyrinth, visual-vestibular interaction centres in the brain stem and cerebellum, and sensory pathways to or from the thalamus, can result in vertigo. Conventionally, causes of vertigo are separated into central or peripheral origins as shown in *Table 2*. Such classification serves to guide further investigations and management of the patient. In the general practice setting, the three most common causes of vertigo (accounting for 93% of all patient presentations)[6] are:

- benign paroxysmal positional vertigo (BPPV)
- acute peripheral vestibulopathy (vestibular neuritis or labyrinthitis), and
- Meniere disease.

Central causes of vertigo, although not as common, are generally more serious and should always be considered. In the elderly

Table 1. Subtypes of dizziness and their clinical signficance[1]

	Vertigo	Presyncope	Disequilibrium	Light headedness
Description	Illusion of movement, usually rotatory of self or surrounding	Sensation of impending loss of consciousness	Postural unsteadiness, imbalance	Also called 'dizziness', 'giddiness' or 'wooziness'. No clear definition
Clinical significance	A wide range of possible causes requiring further assessment	Reduction of total cerebral blood flow, usually of cardiovascular origin Presence of syncope exclude peripheral causes of dizziness	Neurological disorder, musculoskeletal weakness or visual impairment	This term is now used interchangeably with presyncope

Table 2. Classification for causes of vertigo[8]

	Peripheral vertigo (labyrinth, vestibular nerves)	Central vertigo (central nervous system)	Other
Causes	**Common**		
	Acute vestibulopathy: vestibular neuritis or labyrinthitis	Transient ischaemic attack or stroke, especially if vertebrobasilar system affected	Psychogenic vertigo
			Medication induced vertigo
	Benign positional paroxysmal vertigo		Cervical vertigo
	Meniere disease	Migrainenous vertigo	
		Multiple sclerosis	
	Rare		
	Perilymphatic fistula	Cerebellopontine angle tumour	
	Cholesteatoma erosion		
	Herpes zoster oticus		
	Otosclerosis		

population, the causes of vertigo may be multifactorial and can be more difficult to assess.

Clinical assessment

The clinical assessment is aimed at determining if the patient has true vertigo, whether the vertigo is of central or peripheral origin, and to rule out life threatening conditions such as cerebellar haemorrhage. An algorithm to guide initial history taking is summarised in *Figure 2*.

History

Whether the patient has vertigo or not can be checked by asking the simple question, 'When you have dizzy spells, do you just feel light headed or do you see the world spinning around you?'

Features of an attack such as duration, onset, frequency, severity and aggravating factors should be explored. Typically, an attack of central vertigo has a longer duration but is milder in severity. Associated features such as nausea and vomiting, headache, hearing loss, ear discharge, ear fullness and tinnitus should be elicited. The presence of cardiovascular risk factors increases the likelihood of cerebrovascular ischaemia causing vertigo, especially in an elderly person with a spontaneous and mild attack. History of recent upper respiratory tract or ear infection suggests the possibility of

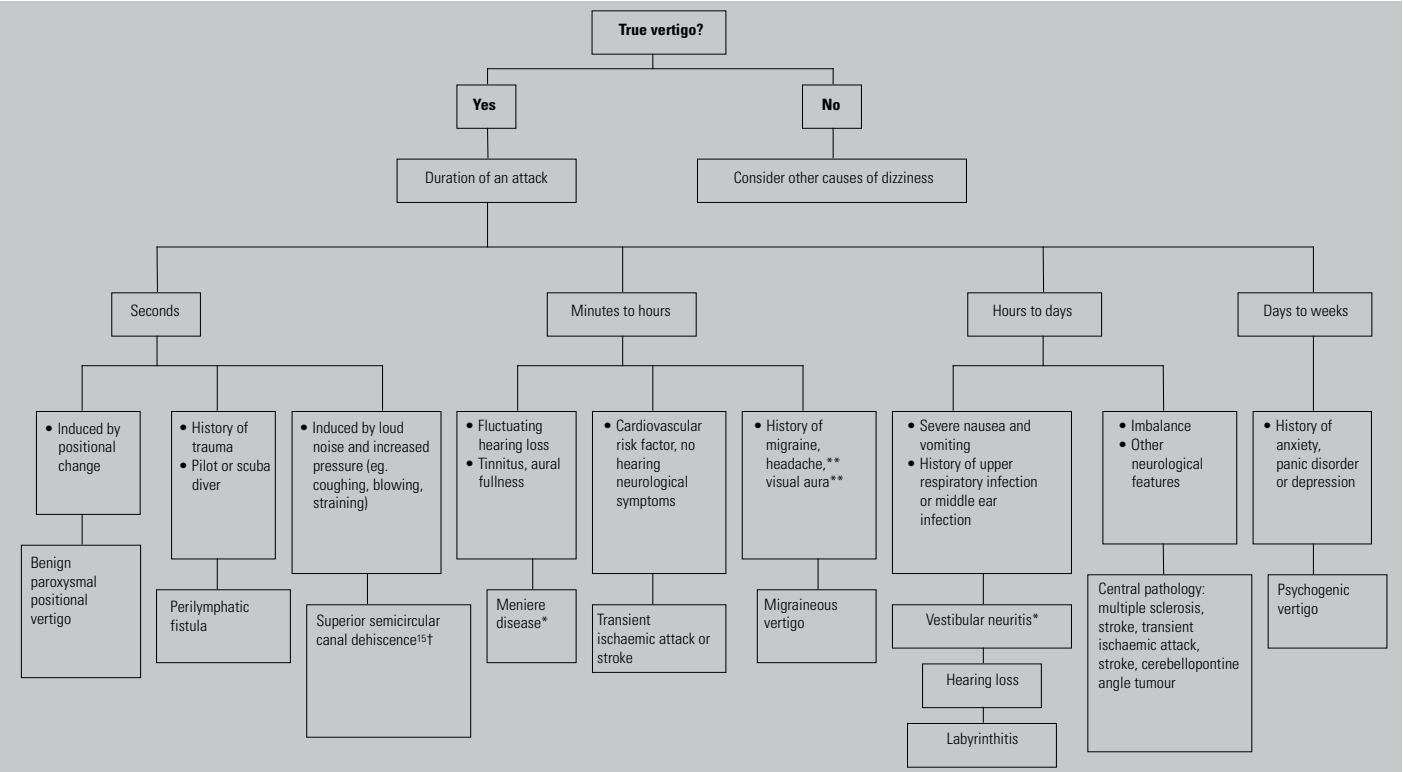
Figure 1. Structure of the ear

vestibular neuritis or labyrinthitis. Past history of head trauma or ear surgery makes perilymphatic fistula more likely. Medication such as aminoglycoside, frusemide, antidepressants, alcohol and antipsychotics can all cause vertigo. Patients with migraine or Meniere disease might have a strong family history. Anxiety disorders and depression can also manifest as dizziness and vertigo.

Examination

A detailed examination of the patient starts with a general inspection looking for patterns of facial asymmetry suggesting either peripheral

Figure 2. Algorithm to guide initial history taking



* In late stages of Meniere disease and late vestibular neuritis, the duration of an attack can be reduced to seconds but more frequent[8]

** Typical headache and aura can be absent

† Recently recognised disease entity caused by congenital breakage in the bony labyrinth capsule. The breakage leads to hypersensitivity of vestibular labyrinth to changes in pressure and sound causing vertigo. There are only about 100 reported cases

facial nerve involvement or a cerebrovascular event. The vesicles of herpes zoster on the external ear might also be visible. Otoscopic examination may reveal signs of inflammation associated with acute vestibulopathy, scarring of the eardrum from chronic suppurative otitis media, or an erosive cholesteatoma. The Hennebert sign is positive when the symptom of vertigo is reproduced by applying external pressure on the tragus. The positive sign suggests the presence of a perilymphatic fistula.

Careful observation for features of nystagmus such as spontaneity, direction, and associated changes with eye movements, convey valuable diagnostic information. The direction of the nystagmus is determined by the 'fast phase' of the eye movement. Horizontal and torsional nystagmus, which beats to a unilateral direction regardless of whether the eyes are gazing to the left or right, suggests the vertigo is of peripheral origin.[7] Conversely, if the direction of nystagmus changes when the eyes are gazing toward a different direction, a central cause of vertigo is more likely.[7,8]

Vertical nystagmus also implies central and brainstem involvement. In peripheral vestibulopathy, central adaptation can lead to a reduction of the magnitude of nystagmus within 24–48 hours. One way to observe for attenuated nystagmus during this period is to use an ophthalmoscope to examine one eye while blocking or closing the other eye to remove the external visual fixation. While keeping the head still, observations for subtle nystagmus-like movements of the optic disc or optic blood vessels can be made.[9]

Clinical tests

Four clinical tests are useful tools for evaluating vestibular function:

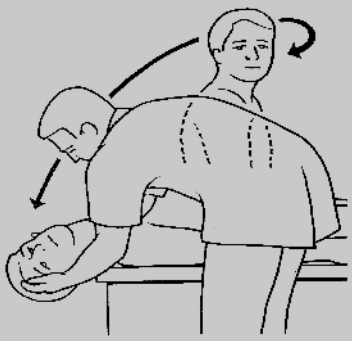
- the head impulse test
- the Romberg test
- the Fukuda-Unterberger test, and
- the Dix-Hallpike manoeuvre.

Suitability of these tests should be considered, especially in patients with severe nausea and vomiting during the acute phase of illness.

Head impulse test

The head impulse test is both sensitive and specific to detect unilateral hypofunction of the peripheral vestibular system, which is commonly due to acute vestibulopathy.[10] Usually, a functional vestibular system can detect small changes in the head position and rapidly adjust eye movements so the centre of vision remains on a target. In patients with acute vestibulopathy, when the head is turned toward the affected side there will be a delay in vestibular adjustment. Such a delay will manifest as a brief and fixed gaze toward the affected side followed by a corrective saccadic eye movement back to the centre.

Figure 3. Dix-Hallpike manoeuvre



1. Sit patient on examination couch and explain procedure
2. Reassure the patient that, although they may feel dizzy, they will not be allowed to fall
3. Turn the patient's head 45 degrees to one side
4. Lie patient supine with their head over the end of the examination bed, supporting their head with a hand on each side of head. Maintain the 45 degree head turn as you lie the patient down
5. Inspect the eyes for nystagmus, and ask the patient if they feel dizzy
6. Hold this position for at least 30 seconds, and for 1 minute if there is no reponse
7. The result is positive if the patient develops symptoms (vertigo) and nystagmus
8. Repeat on the opposite side

One important role of the head impulse test is to differentiate between cerebellar infarction and acute vestibular neuritis. In patients with acute vertigo but a normal head impulse test, acute vestibulopathy is ruled out and cerebrovascular causes of vertigo such as ischaemia or infarction should be considered.[5]

An excellent video of the head impulse test can be found at the *Journal of Neurology, Neurosurgery, and Psychiatry* website (see *Resources*). Note: the head impulse test is usually performed with the clinician sitting face-to-face with the patient and holding the patient's head from the front. Due care should be taken when performing this test on patients with neck pathology as the manoeuvre requires a rapid repositioning of the head.

Romberg test

A Romberg test assesses the integrity of peripheral proprioception, cerebellar and vestibular functions. A Romberg test is positive when the patient can maintain their balance with both feet placed close together with visual input, but not when the eyes are closed.

Fukuda-Unterberger test

In the Fukuda-Unterberger test, the patient is asked to march on the spot with their eyes closed. The test is positive when the patient deviates from the midline; usually toward the side with a relatively lower vestibular activity.

Dix-Hallpike manoeuvre

The Dix-Hallpike manoeuvre should be performed if the history is suggestive of BPPV or if the nystagmus is inducible. The manoeuvre is easy to perform and has a positive predictive value of 83.3% and a negative predictive value of 52%[11] (*Figure 3*). A short video demonstrating the Dix-Hallpike manoeuvre as the first part of the Epley manoeuvre can be found at the *Australian Prescriber* website (see *Resources*). Explanations of the Dix-Hallpike manoeuvre and constant reassurance during the process can help reduce patient discomfort and anxiety. It is also important to wait for at least 30 seconds to observe for nystagmus or symptoms of vertigo before testing the other side.

If symptoms are more suggestive of central vertigo, a thorough neurological examination should be performed. Signs of cerebellar dysfunctions such as dysdiadochokinesia, dysmetria, dysarthria and ataxia should also be sought. Cardiovascular examination and testing for postural hypotension can also provide useful clues.

Investigations

A thorough history and examination can usually reveal the underlying causes of vertigo in the majority of patients. Investigations without a proper clinical reasoning are unlikely to help in reaching a diagnosis. For example, routine blood tests are not recommended as they usually fail to identify an underlying cause of vertigo.[12]

Audiological testing can check for the presence of hearing loss and quantify it. Bilateral low frequency sensorineural or conductive hearing loss is typical of Meniere disease. Caloric testing evaluates the vestibular labyrinth function, however this test should only be done in a specialist centre and the results interpreted by a clinician with expertise in the field.[5]

Neuroimaging is an important investigative tool if there is a concern of a central pathology. Clinical features that warrant urgent neuroimaging are summarised in *Table 3*. Magnetic resonance imaging is the preferred imaging modality when conditions such as multiple sclerosis, vascular infarction or cerebropontine tumour are suspected. Computerised tomography is superior to detect any petrous bone abnormality or cerebellar haemorrhage, and as a follow up tool for trauma induced vertigo.[13]

Conclusion

Dizziness and vertigo can present a diagnostic challenge because of confusion in the nomenclature and a vast number of diagnostic possibilities. During the initial assessment, the role of the clinician is identifying benign and treatable underlying causes and to rule

Table 3. Warning clinical features warranting neuroimaging[14]

• Very sudden onset (seconds) of vertigo that persists and not provoked by position
• Association with new onset of (occipital) headache
• Association with deafness but no typical Meniere history
• Acute vertigo with normal head impulse test
• Associated with central neurological signs such as severe gait and truncal ataxia

out serious conditions. This can usually be achieved by using a systemic approach with careful history, physical examination and appropriate investigations.

Resources

- Journal of Neurology, Neurosurgery, and Psychiatry
  http://jnnp.bmj.com/content/vol0/issue2007/images/data/jnnp.2006.109512/DC1/78101113webonlymedia.mpg
- Australian Prescriber
  www.australianprescriber.com/upload/issue_files/2804_epley.mov.

Conflict of interest: none declared.

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