Dealing with dizziness

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Summary

Dizziness is a term used to describe a range of symptoms, but is rarely a sign of a serious disorder. Usually the patient describes vertigo, however they may be referring to a presyncopal sensation or disequilibrium. Clinical evaluation will determine whether the dizziness is vestibular or non-vestibular in origin and then whether the dizziness is peripheral or central in origin. The common causes of vertigo are benign positional vertigo, Ménière's syndrome, vestibular neuritis and migraine.

Key words: Ménière's syndrome, vertigo, vestibular disorders.

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Introduction

Dizziness is a common symptom in general, specialist and hospital practice. Although it is rarely a sign of a life-threatening or serious disorder, it may be incapacitating and result in lost productivity and reduced quality of life. Usually patients complaining of dizziness are referring to vertigo – a sensation of self or environmental motion. Other possibilities exist, for example a light-headed, faint or presyncopal sensation, and sometimes the patient will be actually referring to a gait imbalance. Taking a thorough history will usually clarify the nature of the complaint. Occasionally a slight vestibular disturbance or a slight impairment of cerebral perfusion will produce a rather vague symptom that even the most eloquent of patients will have difficulty describing and will therefore present quite a diagnostic challenge.

Clinical assessment – history

A framework of questions in the clinician's mind will serve as a guide in analysing the patient's complaint.

Is the dizziness of vestibular origin or non-vestibular origin?

Dizziness of vestibular origin (vertigo) is usually provoked or aggravated by movement and improved or relieved by rest (sitting or lying down). Its many causes (Table 1) may be central or peripheral.

The dizziness of benign positional vertigo may be provoked by specific positional manoeuvres while dizziness which is unaltered between resting and moving about is usually non-vestibular in origin. Dizziness which precedes a blackout or near blackout will usually indicate a syncopal/presyncopal disorder which requires a different approach.

Drugs which can cause dizziness include antihypertensives, anticonvulsants, antidepressants and sedatives. The dizziness occurs either through postural hypotension or direct effects on the central or peripheral vestibular system.

If the patient has vertigo, is it peripheral or central in origin?

Although recurrent isolated vertigo is usually peripheral and benign, this is not always the case. Central vestibular disorders include brainstem lesions and multiple sclerosis. Both the clinician and the patient want to rule out a serious cause, but localising the source of vertigo may be difficult.

The occurrence of associated symptoms may help localise the origin of the vertigo. For example, symptoms such as deafness, tinnitus and aural fullness or pressure might suggest a labyrinthine cause, whereas symptoms such as headache, diplopia, facial/limb paraesthesiae, weakness or clumsiness, dysarthria or dysphagia might suggest a neurological cause.

The temporal profile of vertigo is important in analysing the patient's symptoms. Vertigo lasts less than one minute in benign

Table 1
Causes of vertigo

Common	Less common
Benign paroxysmal positional vertigo	Vertebro-basilar transient ischaemic attack/stroke
Vestibular neuritis/labyrinthitis (acute unilateral peripheral vestibulopathy)	Multiple sclerosis
Ménière's syndrome	Posterior fossa tumours
Migraine	Arnold-Chiari malformation
Psychogenic	Autoimmune inner ear disease
Idiopathic	Perilymph fistula/superior semicircular canal dehiscence
	Invasive middle/inner ear disease (e.g. otomastoiditis, tumours, cholesteatoma)
	Bilateral peripheral vestibulopathy (if asymmetric)

positional vertigo, minutes in transient ischaemic attacks, minutes to hours in migraine and Ménière's syndrome and for more than 24 hours in vestibular neuronitis or posterior circulation stroke.

The patient's medical history may reveal clues to potential causes of vertigo. A history of migraine-like headaches may suggest migraine-associated vertigo, while a recent head injury may suggest a variety of different mechanisms of vertigo. The patient's cardiovascular risk profile may raise the possibility of vascular vertigo.

Is the vertigo spontaneous or provoked?

The circumstances that provoke the symptom of vertigo may provide important clues to possible causes. For example, a change of head position may provoke benign positional vertigo, noise-induced vertigo (Tullio phenomenon) occurs in superior semicircular canal dehiscence, and pressure-induced vertigo (valsalva, straining, exercise) suggests a perilymph fistula. Hyperventilation may provoke or aggravate psychogenic dizziness, however it may also provoke vertigo from brainstem demyelination or an Arnold-Chiari malformation.

Clinical assessment – examination

A patient with dizziness requires a thorough neurological, otological and cardiovascular examination, including supine and upright blood pressure measurements, particularly if the cause of the dizziness is not apparent from the history. Clearly an abnormality in any of these systems will direct any further investigation and may lead to the diagnosis. For example, an examination for signs of infection may find vesicles near the external auditory canal which would suggest herpes zoster as a cause of dizziness. Frequently, however, many patients with recurrent isolated dizziness will not display any obvious abnormality, particularly if they consult between symptomatic periods.

Nystagmus

If the patient has nystagmus, specific note should be made of the trajectory. Purely vertical or torsional nystagmus suggests a central lesion, whereas a mixed horizontal/torsional nystagmus suggests peripheral vestibular nystagmus.

Note whether the nystagmus is direction changing or direction fixed. Peripheral vestibular nystagmus produces a nystagmus which beats in the same direction regardless of the eye position. Nystagmus which changes direction with different eye positions (for example, beating to the right on right gaze and then beating to the left on left gaze) may indicate a central neurological lesion. The nystagmus of peripheral vestibular disorders tends to be attenuated by visual fixation and so nystagmus may not be detected in the conventional manner in these patients.

Acute severe vestibular insults such as vestibular neuritis may produce nystagmus, which is obvious in the acute phase, but in many cases this rapidly attenuates over 24–48 hours. The nystagmus may then only be apparent with special techniques. One such technique involves using the ophthalmoscope to observe the nystagmus (movement of the optic disc and retinal vessels) while occluding the other eye to remove visual fixation.

Vestibulo-ocular reflex

Assessment of the vestibulo-ocular reflex is useful in confirming a peripheral origin of vertigo. It is also useful in determining which labyrinth is abnormal. The vestibulo-ocular reflex can be assessed by performing the head-impulse or head-thrust test. This test involves asking the patient to fixate on the examiner's nose. The examiner then rapidly rotates the patient's head to either side (after excluding any significant neck problem). A 10–20° movement is usually sufficient. In healthy people the eyes remain fixed on the examiner's nose regardless of the head position.

In a patient with a labyrinthine lesion, the eyes will move with the head when turned to the side of the lesion and then after a short delay the visual system will trigger a quick corrective eye movement back to the examiner's nose. This quick corrective eye movement is the abnormality sought when undertaking the test. The test will also be abnormal when the head is thrusted to either side in a patient with bilateral peripheral vestibular disease, for example gentamicin vestibulotoxicity.

The head-impulse test is also useful in the differential diagnosis of cerebellar infarcts and vestibular neuritis. The test is positive in vestibular neuritis, but negative with a cerebellar infarct.

Visual acuity

Testing the dynamic binocular visual acuity is an additional method of determining whether there is bilateral peripheral vestibular disease. In healthy people the dynamic binocular visual acuity is similar to the static visual acuity. If they can read the 6/6 line on the Snellen chart then they will still be able to read the same line while their head is moving to and fro at approximately two cycles per second. In patients with bilateral peripheral vestibular impairment the visual acuity often drops several lines from the static to dynamic condition, for example from 6/6 to 6/36 or 6/60. Normal dynamic visual acuity does not rule out a unilateral vestibular lesion.

Hallpike manoeuvre

The Hallpike positional manoeuvre is a particularly important part of the clinical examination of the dizzy patient as it can confirm the presence of benign positional vertigo which is one of the commonest causes of vertigo. The manoeuvre should therefore be performed if there is any hint of positional vertigo and in all patients where there is no obvious cause for their symptoms. This is important as benign positional vertigo can be cured with a simple physical positioning manoeuvre and the patient will be very grateful. The manoeuvre is usually simple to

perform. The patient sits upright with the head rotated 30–45° laterally. The patient is then rapidly moved into a supine position on the examination couch with the head hanging over the end of the couch or a pillow placed behind the shoulders. The patient's head is supported either by the examiner or by the couch if a pillow is placed behind the shoulders. The examiner then observes the patient for nystagmus and asks about vertigo. Then the test is repeated with the patient's head turned to the opposite side.

Typically a patient with benign positional vertigo will develop, after a short latency of up to several seconds, a torsional/vertical nystagmus with fast phases directed towards the lower ear accompanied by vertigo. This means the lesion is on the side of the lower ear. Occasionally atypical forms of positional nystagmus will be observed indicating one of the less common variants of peripheral benign positional vertigo or a central form of positional nystagmus.

Further investigation

Some patients presenting with vertigo may require further investigation, however comprehensive audio-vestibular testing does not replace a thorough clinical assessment. Audio-vestibular investigations may be useful for confirming and characterising suspected vestibular dysfunction. They help to distinguish peripheral from central vestibular disorders, localise the side of peripheral vestibular dysfunction and evaluate the state of adaptation following an insult to the vestibular system. The principal components of quantitative vestibular investigation include electronystagmography, caloric testing and rotational chair testing. An audiogram may be helpful in suggesting a labyrinthine cause of the vertigo, for example the low frequency sensorineural hearing loss seen in Ménière's syndrome.

Imaging of brain, audio-vestibular nerves and labyrinth may be needed, particularly if there are neurological signs or asymmetric sensorineural hearing loss. However, in patients with recurrent isolated vertigo and normal hearing, there is a low yield from imaging studies particularly in regard to cerebellopontine angle tumours or masses.[1]

Treatment of vertigo

Basic symptomatic treatment

There is no singularly effective pharmacological treatment to abolish vertigo (see box). Nevertheless a patient can be supported through an acute episode of vertigo with short-term use of vestibular sedatives such as benzodiazepines, antihistamines or prochlorperazine. Antiemetics and bed rest are also useful. The evidence of efficacy of the vestibular sedatives is based on experimental laboratory animal research which shows reduced vestibular neural activity in response to these drugs. Clinical observation of this efficacy is anecdotal and there are no controlled trials.

Drugs used to manage vertigo

Vestibular sedatives

Antihistamines
- Diphenhydramine
- Promethazine

Benzodiazepines
- Diazepam
- Lorazepam
- Clonazepam

Butyrophenones
- Droperidol
- Anticholinergics
- Hyoscine hydrobromide

Antiemetics
- Prochlorperazine
- Metoclopramide

Patients with chronic or recurring vertigo are commonly treated with betahistine and/or prochlorperazine although there is no evidence of the drugs' long-term efficacy. These patients will often do well with vestibular adaptation exercises or a formal vestibular rehabilitation program to help manage their symptoms. The drugs prescribed for acute vertigo should be used sparingly as they may impair the vestibular adaptation process.

Selective serotonin reuptake inhibitors such as sertraline may have a role in the management of vertigo, however, they have not been extensively studied.

Treatment of specific causes

The most effective treatment of vertigo is treatment of the specific cause if it can be identified (Table 2).

Benign paroxysmal positional vertigo

Benign positional vertigo can be treated with simple physical manoeuvres, although recurrences are frequent. The particle repositioning manoeuvres, which include the Epley[2] and Semont manoeuvres, may provide complete symptomatic relief in the majority of patients by moving debris from the semicircular canals to the utricle (see video*). The particle repositioning manoeuvres for benign positional vertigo are 80–90% effective following a single manoeuvre compared to a spontaneous recovery rate of approximately 50% over six weeks. The Brandt-Daroff positional exercises are a possible alternative treatment.

Migraine

Migraine is an important diagnostic consideration in patients presenting with dizziness, whether they have an accompanying headache or not. Although there is no definitive diagnostic test for migraine, there may be a suggestive clinical profile. The response to migraine therapy may strengthen the diagnostic impression.

Table 2

Specific treatment of vertigo

Benign paroxysmal positional vertigo	Otolithic particle repositioning manoeuvre (e.g. Epley[2] or Semont manoeuvres), Brandt-Darrof exercises
Vestibular neuritis/labyrinthitis	Bed rest, vestibular sedatives, antiemetics in first 24–72 hours Vestibular adaptation exercises/rehabilitation in recovery phase
Ménière's syndrome	Vestibular sedative, antiemetic for acute episodes Low salt diet +/– diuretic for maintenance treatment Intra-tympanic gentamicin or surgery for severe refractory cases
Migraine	Aspirin, non-steroidal anti-inflammatory drugs, 'triptans', antiemetics in acute episodes Migraine preventive treatment e.g. pizotifen, propranolol, verapamil, amitriptyline, valproate
Psychogenic	Treat associated anxiety/depression/panic disorder Reassurance

Ménière's syndrome

Ménière's syndrome may cause severely disabling and distressing vertigo. Typically patients present with a combination of vertigo, fluctuating low frequency hearing loss/tinnitus and an aural pressure sensation. These symptoms may not necessarily occur simultaneously. Fortunately the majority of patients will respond to conservative medical management including a low salt diet and possibly diuretics. Vestibular sedatives and antiemetics may be useful for prolonged acute attacks. Short-term corticosteroids may be useful in refractory cases with relatively preserved hearing. More aggressive therapies such as intra-tympanic gentamicin and/or surgery are reserved for refractory cases unresponsive to other measures and where there is significant hearing loss. Vestibular rehabilitation therapy may be useful in patients whose symptoms are stable.

Vascular disease

An acute vestibular syndrome resembling vestibular neuritis and recurrent brief isolated vertigo may occur as manifestations of cerebrovascular disease. Increased diagnostic suspicion is required in older patients with associated vascular risk factors. It is, however, unusual for posterior circulation transient ischaemic attacks to manifest as recurrent isolated vertigo for more than a few months.

* See the internet version of this article at www.australianprescriber.com for
- figure showing Epley manoeuvre
- short video (1 min) showing Epley manoeuvre.

See also **Patient Support Organisation: Ménière's Support Group** page 107

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Further reading

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Conflict of interest: none declared

Self-test questions

The following statements are either true or false (answers on page 107)

5. There is no effective treatment for benign paroxysmal positional vertigo.
6. A bilateral peripheral vestibular impairment can reduce a patient's dynamic visual acuity.