The dizzy patient
a practical approach

Dizziness is a continuum of sensations ranging from light headedness or faintness to spinning or a feeling of imbalance. A history of the duration of attacks, precipitating factors and accompanying features is vital in reaching a practical understanding of the cause.

DAVID POHL
MB BS, FRACS

Dr Pohl is Senior Neuro-Otologist, Royal Prince Alfred Hospital, and Head, Department of Otolaryngology, St George Hospital, Sydney, NSW.

Dizziness or vertigo?

Dizziness is not a disease but a symptom. The term describes perceptions of relative motion, which arise from the vestibular system or its central connections. It is up to the clinician to decide whether the descriptive term 'dizziness' used by the patient equates to real vertigo. 'Vertigo' generally describes a sensation or perception of motion of oneself or one's environment. Commonly, 'dizziness' refers to a continuum of sensations ranging from light headedness or faintness to spinning or a feeling of imbalance.

The clinician must interpret the history and gauge whether a patient is describing true vertigo or imbalance. It is important to elicit sensations of rotation (in either the horizontal or vertical plane) or feelings of imbalance with veering to one side or the other.

Classification of vertigo

The causes of vertigo are traditionally classified as peripheral or central, depending on the site of the lesion (Table 1). Determining the duration of attacks, precipitating factors and accompanying features is vital in reaching a practical understanding of the cause of vertigo in any particular patient.

Peripheral causes of vertigo

Acute vestibulopathy

Acute vestibulopathy (or acute vestibular failure) refers to the sudden loss of function in one balance organ. This may be due to viral inflammation or vascular occlusion. Acute labyrinthitis, viral labyrinthitis and vestibular neuronitis are other terms that cover this clinical picture.

Clinical features

The salient features of acute vestibulopathy are:

- the acute onset of persistent vertigo, generally lasting hours or days
- nausea and vomiting.

Generally there is a gradual improvement in these symptoms over days or months.

The auditory features of hearing loss and tinnitus are absent. Horizontal nystagmus is observed in the acute phase.

The patient generally finds it more comfortable, in the acute phase, to lie on their side with the pathological ear lowermost. This limits the sensation of vertigo.

Natural course of the disease

The vestibular loss does not recover but a central process of compensation occurs, with resetting of the vestibular input parameters – even though the acute vertigo may have settled. March testing with the eyes closed will show the patient to rotate preferentially to the pathological side. Caloric tests will show a persistent hypofunction of the labyrinth in the affected ear.

For a variable period after the acute event there may still be short episodes of acute rotational vertigo. This probably does not represent reactivation of the initial insult to the labyrinth but rather a temporary failure of compensation to the vestibular deficit.

Endolymphatic hydrops

Endolymphatic fluid volume is maintained within a physiological range by a combination of the absorption of fluid by the endolymphatic sac and osmotic gradient effects. The accumulation of endolymphatic fluid beyond the physiological range is referred to as 'endolymphatic hydrops'.

This condition is generally classified into:

- idiopathic (Ménière's disease)
- secondary (subsequent to inflammatory insults to the inner ear, e.g. mumps, rubella, syphilis).

Clinical features

There are classically four salient features of endolymphatic hydrops:

- **Episodic vertigo** accompanied by nausea and vomiting. This generally lasts for hours but may last from minutes to days. Invariably the patient is unable to walk and is confined to bed.
- **Fluctuating sensorineural deafness** with repeated attacks. Deafness increases during the acute phase and hearing improves when the vertigo subsides.
- **Crescendo tinnitus.** It is common for the tinnitus to build in intensity either prior to or during an attack. It often abates after the acute vertiginous episode ceases.
- **Sensation of aural pressure or fullness.** Again, this is most marked before or during an attack.

Ménière's disease is predominantly a clinical diagnosis. Features of both vestibular and auditory symptoms should be present when the diagnosis is being considered.

Natural course of the disease

With increasing numbers of recurrent attacks, a degree of permanent sensorineural deafness occurs. In parallel with this, there is a progressive

Table 1. Causes of vertigo

Peripheral

- Acute vestibulopathy
- Endolymphatic hydrops:
  - Idiopathic (Ménière's disease)
  - Secondary (rubella, mumps)
- Perilymphatic fistula
- Benign paroxysmal positional vertigo
- Acoustic neuroma
- Chronic suppurative otitis media (CSOM)

Central

- Multiple sclerosis
- Brainstem tumours
- Migraine
- Epilepsy
- Cerebellar infarction

IN SUMMARY

- **'Vertigo' generally describes a sensation or perception of motion of oneself or one's environment.**
- **Commonly, 'dizziness' refers to a continuum of sensations ranging from light headedness or faintness to spinning or a feeling of imbalance.**
- **Determining the duration of attacks, precipitating factors and accompanying features is vital in reaching a practical understanding of the cause of vertigo in any particular patient.**
- **When confronted with an acutely vertiginous patient in whom a diagnosis is yet to be made, the primary aim is to reduce rotational symptoms with the attendant nausea and vomiting.**
- **The longer a patient remains immobile after an acute vestibular insult, the less likely full recovery will occur.**

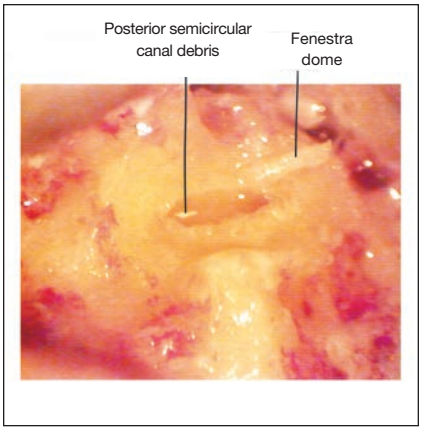

Figure 1a. Pathology of BPPV. Otoconial debris shed from the utricle deposited in the posterior semicircular canal (displayed at surgery for treatment of intractable BPPV).

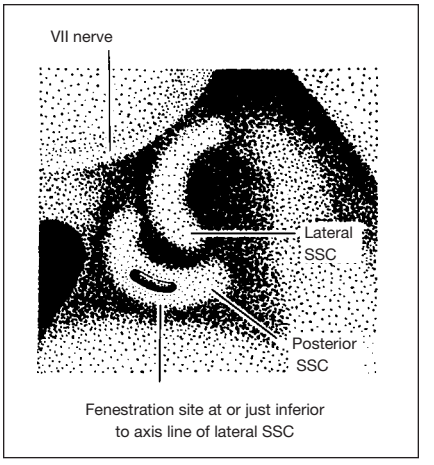

Figure 1b. Pathology of BPPV: diagrammatic representation of colour operative photograph (SCC=semicircular canal).

FIGURES 1A AND B FROM: POHL D. SURGICAL PROCEDURES FOR BENIGN POSITIONAL VERTIGO. IN: RW BALOH AND GM HALMAGYI (EDS). DISORDERS OF THE VESTIBULAR SYSTEM (CHAPTER 44). OXFORD UNIVERSITY PRESS, 1996. REPRODUCED BY PERMISSION OF OXFORD UNIVERSITY PRESS INC. (WWW.OUP.COM).

loss of vestibular function in the affected ear. As increasing vestibular loss occurs, the intensity of the vertiginous episodes tends to abate. This is referred to as 'burnt out' Ménière's disease.

Perilymphatic fistula

The integrity of the vestibular system can be affected by trauma. Such trauma can cause breaks within the oval window housing the stapes footplate, in the round window or in the semicircular canals if they are covered by congenitally thin bone.

Precipitating events

In the history, one should enquire about:

- head injury
- barotrauma (flying, diving)
- explosion
- previous stapedectomy.

Other precipitating events may include nose blowing, vomiting, straining, heavy lifting or childbirth. Persistent and protracted coughing may also cause breaks in the integrity of the vestibular system.

There is no absolutely reliable test for diagnosing perilymphatic fistula, and the diagnosis is made predominantly from the history. The following diagnostic tools are helpful:

- electrocochleography
- middle ear endoscopy
- vestibular evoked myogenic potentials.

Clinical features

Descriptive symptoms include imbalance or the sensation that one is walking on sponge or has recently disembarked from a vessel.

Tinnitus and hearing loss are generally minimal or absent.

Benign paroxysmal positional vertigo (BPPV)

Clinical features

Benign paroxysmal positional vertigo is perhaps the only condition in which dizziness is worse when lying down than when standing. Consequently, this is a hallmark symptom when enquiring about this condition.

Violent episodes of short vertigo are precipitated by lying down, sitting up or rolling over in bed. Vertigo may also be precipitated in head extension, such as when looking up at a skyscraper. In between the vertiginous paroxysms, the patient feels normal. There is no problem with walking or driving a car. Hearing loss is absent.

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Figure 2. MRI showing acoustic neuroma.

HASSO AN, LEDINGTON JA. OTOLARYNGOL CLIN NORTH AM 1988; 21(2): 219-244.

Precipitating events

Benign paroxysmal positional vertigo is most commonly idiopathic, but may follow head injury.

It is thought that the pathophysiology of the condition is secondary to otoconial debris coming loose from the utricle and finding its way into the posterior semicircular canal (Figures 1a and b). Consequently, in certain head positions the calcium carbonate crystals move along the circle of the posterior semicircular canal, causing movement of the endolymph and consequent vertigo.

Provocation of symptoms

The classical procedure used to provoke symptoms is the Hallpike test, with the head extended 30° below horizontal while the patient lies supine. The head is rotated approximately 45° so that one ear is lowermost. The eyes are observed for nystagmus.

The salient features of the Hallpike test are:

- torsional geotrophic nystagmus (rotational nystagmus beating towards the ground)
- latency – a time lag between

assuming the head dependent position and the onset of nystagmus
- fatiguability – a decrease in the severity of the nystagmus with repeated provocative head positioning
- nystagmus duration of less than 20 seconds
- reversal of the nystagmus on assuming the vertical sitting position.

Acoustic neuroma

Acoustic neuromas (see Figure 2) are schwannomas that arise from the superior vestibular nerve in the internal auditory meatus.

Clinical features

Most commonly acoustic neuromas present with unilateral (asymmetrical) sensorineural deafness.

With tumour growth, patients complain of increasing imbalance and tend to veer to the side of the lesion when walking. Rotational vertigo is rare.

Natural course of the disease

Acoustic neuromas are benign but slowly enlarging. As they enlarge, the cochlear nerve is compressed and hearing loss ensues. Further enlargement may cause cerebellar and brainstem compression.

Chronic suppurative otitis media (CSOM)

Repeated inflammatory insults secondary to suppurative ear disease can be a cause of vestibular symptoms. In all conditions so far described in this article, the drumhead is normal. In contrast, in chronic suppurative otitis media the drumhead is scarred, perforated or discharging.

Clinical features

Episodes of serous labyrinthitis usually cause a degree of imbalance rather than rotational vertigo.

A fistula in the semicircular canal may be suspected if vertigo is elicited on applying positive pressure to the external auditory canal by means of a pneumatic speculum.

Natural course of the disease

Serous labyrinthitis occurs secondary to the suppuration in the middle ear. In cholesteatoma there may be erosion of the otic capsule over the semicircular canals, causing a fistula.

Central causes of vertigo

Central causes of vertigo should be considered:
- whenever the history contains superadded neurological features (e.g. dysphagia, visual disturbance, diplopia, or sensory or motor changes of the limbs)
- if the nystagmus has a vertical component
- if positional provocative testing does not show the typical features of latency, fatiguability and reversal.

Migraine

Migraine is a common cause of vertigo. It should be suspected if there is a past history of migraine, even if the patient has been asymptomatic for many years.

Vertigo may take the place of the aura or may be a migraine equivalent, in which the headache itself is replaced by vertigo. Hearing loss is absent.

Table 2. Classification of symptoms of vertigo

Rotational symptoms are seen in:
- Endolymphatic hydrops
- Acute vestibulopathy (acute phase)
- Migraine
- Benign paroxysmal positional vertigo (BPPV)

Imbalance symptoms are seen in:
- Acute vestibulopathy (late)
- Acoustic neuroma
- Fistula
- Chronic suppurative otitis media (CSOM)

Epilepsy

Epilepsy may have an aura of vertigo. A history of amnesia should raise the suspicion of epilepsy.

Diagnosis

In a practical approach, it is worthwhile dividing dizziness into rotational symptoms and imbalance symptoms (see Table 2).

Once enquiry has been completed regarding the pattern of vertigo and any

Consultant's comment

This very practical paper gives an excellent description of the causes and management of vertigo. The important distinction is made between true vertigo or a feeling of rotation and the more common (and often more difficult to manage) symptom of dizziness – as noted, a difficult to describe feeling of lightheadedness, detachment or faintness. The latter is very nonspecific, and may be due to several causes. It is common after even minor head injuries, or other intracranial insults, or as part of the postconcussional syndrome, and may of course be a sign of postural hypotension, cardiac disturbance and so on.

Treatment of dizziness is often unrewarding but, as pointed out by Dr Pohl, the causes of vertigo and imbalance can usually be tracked down and appropriate treatment given.

Professor Nicholas W.C. Dorsch
Clinical Associate Professor
Staff Specialist Neurosurgeon
Westmead Hospital
Sydney, NSW

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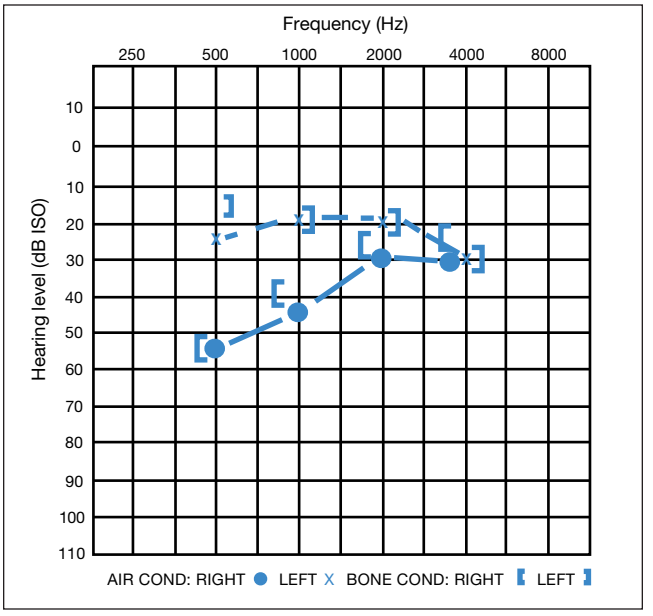
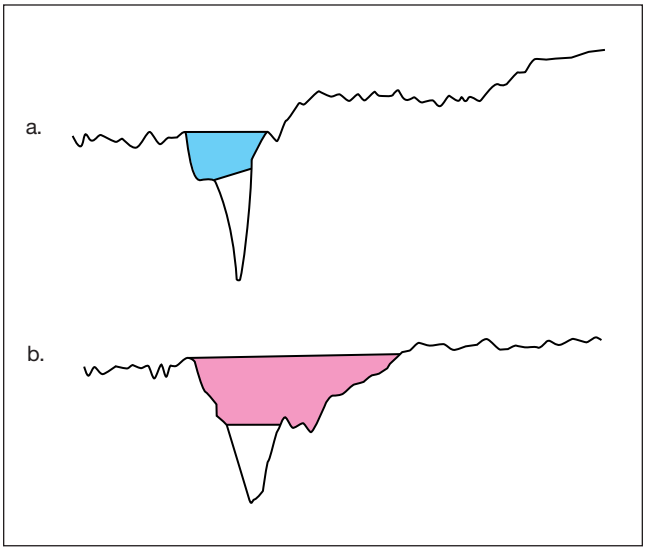


Figure 3. Audiogram showing the typical low tone sensorineural hearing loss of Ménière's disease in the right ear.



Figures 4a and b. Electrocochleography. a (top). Normal AP–SP waveform (shaded area). b (bottom). Ménière's disease: widened AP–SP waveform.

ADAPTED FROM MORRISON AW, MOFFAT DA, O'CONNOR AF. OTOLARYNGOL CLIN NORTH AM 1980; 13(4): 703-721.

associated hearing changes, specific tests help to confirm the diagnosis.

Table 3 (see next page) provides a summary of the clinical features and investigation of vertigo.

Investigation

Audiometry

Pure tone audiometry is helpful in:

- Ménière's disease (unilateral, low tone sensorineural deafness – Figure 3)
- acoustic neuroma (asymmetrical sensorineural hearing loss)
- perilymphatic fistula (high tone sensorineural deafness, or vertigo elicited by sound known as the 'Tullio phenomenon')
- chronic suppurative otitis media (conductive deafness).

Vestibular function tests

Caloric testing

Nystagmus is elicited by the irrigation of hot and cold water into the ear. The duration and intensity of the nystagmus is measured and then the ears are compared. This allows the diagnosis of canal hypofunction or canal paresis, and thus the identification of the pathological ear.

Electronystagmography (ENG)

The presence of spontaneous nystagmus in darkness (measured by ENG) indicates an uncompensated vestibular lesion. If visual fixation diminishes the degree of spontaneous nystagmus, it is likely that the lesion is peripheral.

Other specialised tests

Other specialised tests include:

- rotational chair tests
- vestibular evoked myogenic potentials, tests of perceived horizontal, head impulse tests
- electrocochleography – an electrocochleogram is the electric wave form recorded from the promontory of the middle ear in response to a sound stimulus, using a transtympanic electrode. Interpretations of the shape of this potential allow the diagnosis of Ménière's disease (Figure 4). As the sensorineural deafness progresses, the electrocochleogram frequently becomes more abnormal. During the early stages of Ménière's disease, the electrocochleogram may be normal and the diagnosis is essentially clinical.

Imaging modalities

A bone window CT scan images the otic capsule that houses the vestibular and hearing end organs. Breaks in the bony capsule, such as a fistula, may be seen on CT scanning.

The gold standard for exclusion of an acoustic neuroma is an MRI scan. This is also the investigation of choice to exclude multiple sclerosis.

Management

When confronted with an acutely vertiginous patient in whom a diagnosis is yet to be made, the primary aim is to reduce rotational symptoms with the attendant nausea and vomiting. The following drugs are appropriate in this situation:

- prochlorperazine (Stemetil, Stemzine)

Table 3. Clinical features and investigation of vertigo

Clinical features	Ménière's disease	Labyrinthitis or acute vestibulopathy	Perilymphatic fistula	BPPV	Superior SCC dehiscence	Acoustic neuroma	CSOM
Vertigo intensity and duration	++ Minutes to hours	++ Hours to days	–	++ Seconds	+ Seconds to minutes	–	–
Imbalance	–	+ late, secondary to failed compensation	+	–	–	+	+
Nausea and vomiting	++	++	–	+	–	–	–
Hearing loss	+ early ++ late	–	Mild sensorineural	–	Hyperacusis	+ unilateral	+ conductive
Symptom-free interval	+	–	–	+	+	–	+ (dry ear)
Tinnitus	++	–	±	–	–	±	±
Associated features	Fluctuating hearing loss	–	History of barotrauma	Position provoked	Triggered by sound, cough, strain, Valsalva, etc.	–	Otorrhoea
Investigation of choice	ECoG	ENG	? ECoG	Hallpike test	VEMP, bone window CT	MRI	CT

Key: BPPV = benign paroxysmal positional vertigo, CSOM = chronic suppurative otitis media, ECoG = electrocochleoography, ENG = electronystagmography, VEMP = vestibular evoked myogenic potential, SCC = semicircular canal.

- diazepam (Antenex, Ducene, Valium)
- droperidol (Droleptan Injection).

The subsequent management of vertigo depends upon the diagnosis.

Acute vestibulopathy

In the acute phase of sudden vestibular failure, labyrinthine sedatives are appropriate. Once acute vertigo has settled, optimum therapy is to withdraw the labyrinthine sedation as soon as possible. Central adaptation to a vestibular deficit may be delayed by the prolonged use of vestibular sedatives.

Labyrinthine sedatives are unable to differentiate between a diseased and nondiseased vestibular end organ; therefore, they will sedate both the good and the diseased labyrinth. This leads to problems with central compensation and the reprogramming of vestibular input. The longer a patient remains immobile after an acute vestibular insult, the less likely full recovery will occur. Elderly patients are less likely to recover fully after a vestibular insult, because of poorer co-ordination, visual impairment and impaired proprioception.

Vestibular rehabilitation is available to enhance balance by re-education of visual proprioceptive and motor cues.

Ménière's disease

Acute Ménière's disease should be treated with labyrinthine sedation. Once the diagnosis has been made, it is appropriate for diuretic treatment to be added in the acute phase. This can be either:

- 20 to 40 mg/day frusemide (Frusehexal, Frusid, Lasix, Uremide, Urex), or
- an osmotic diuretic (urea 20 g/day).

Once the acute phase has settled, interval therapy should be instituted. This includes:

- salt restriction (less than 3 to 5 g/day)
- a thiazide diuretic
- Betahistine dihydrochloride (Serc), up to 32 mg/day.

Some patients are able to predict their attacks of Ménière's disease. They experience increasing fullness in the ear, a drop in hearing or an intensification of tinnitus. In this clinical situation, immediate treatment with an osmotic diuretic is appropriate. Some patients also find it helpful to take a labyrinthine sedative at the same time. Such prodromal therapy may abort an attack or lessen its severity.

If vertigo is a continuing problem despite interval therapy, then treatment with cinnarazine (Sturgeron) or flunarazine (Sibelium) may be appropriate. Currently, it is necessary to apply for these drugs through the Special Access Scheme.

Any subsequent management of Ménière's disease is generally best performed by an ENT surgeon or a neuro-otologist. Management will be determined by the level of hearing that remains.

The treatment options include:

- intratympanic gentamicin
- endolymphatic sac surgery
- vestibular nerve section
- cochleostomy.

Perilymphatic fistula

Treatment of a suspected perilymphatic fistula involves surgical exploration of the suspected fistula site, with tissue grafting to make good the integrity of the otic capsule of the vestibular system.

Benign paroxysmal positional vertigo

Vertigo due to BPPV is intense but brief. Vestibular sedation is contraindicated. Treatment for BPPV involves the so-called 'particle repositioning manoeuvres'. The aim of these manoeuvres is to relocate otolith crystals that have found their way into the posterior semicircular canal. Treatment by predetermined head rotations allows these crystals to exit from the posterior semicircular canal back towards the utricle.

It is only appropriate to perform a particle repositioning manoeuvre if the provocative positioning test (Hallpike test) is positive at the time of examination. A positive Hallpike test indicates that a particle is present in the posterior semicircular canal and that it is mobile. The mobility of such a particle is critical to the success of a particle repositioning manoeuvre.

In intractable cases unresponsive to repeated particle repositioning manoeuvres, surgical occlusion of the posterior semicircular canal may be undertaken.

Acoustic neuroma

Management of acoustic neuroma is not directed towards symptoms of imbalance, but rather towards control of what is an expanding mass in the cerebellopontine angle. Serial MRI scans may confirm progressive enlargement of the lesion.

Stereotactic radiotherapy has been advocated for the management of these lesions, but surgical removal remains the gold standard. Surgical treatment targets total tumour removal and the preservation of facial nerve function.

Chronic suppurative otitis media

A finding of an abnormal eardrum on a background of vertigo and imbalance is grounds for referral to an ear, nose and throat surgeon for further assessment.

Guidelines for specialist referral

Referral to an ear, nose and throat surgeon or vestibular neurologist is appropriate in the following situations:

- presumed Ménière's disease unresponsive to medical therapy
- presumed viral labyrinthitis not abating after three months
- unabating vertigo following trauma
- BPPV persisting for more than 12 months despite repeated particle repositioning manoeuvres
- vertigo in the presence of suppurative ear disease
- vertigo, imbalance or drop attacks resulting in falls and injury
- acoustic neuroma.

Summary

'Vertigo' generally describes a sensation or perception of motion of oneself or one's environment. In some cultures, 'Dizziness' commonly equates to headache, light headedness, a feeling of being separate from one's environment, or simply feeling faint. It is the clinician's role to interpret the history and to gauge whether a patient is describing true vertigo or imbalance.

Once enquiry has been completed regarding the pattern of vertigo and any associated hearing changes, specific tests help to confirm the diagnosis.

When confronted with an acutely vertiginous patient in whom a diagnosis is yet to be made, the primary aim is to reduce rotational symptoms with the attendant nausea and vomiting. The subsequent management depends upon the diagnosis. MT