

Problem based review: the patient with dizziness on the AMU

A Kennedy & N Cooper

Abstract

Unsteadiness, balance disturbance, and 'dizziness' are common presenting complaints on the Acute Medical Unit. This article outlines key components in the evaluation of these problems and describes the most common clinical patterns of dizziness. A case history is used to highlight some key learning points.

Keywords

Intra-abdominal; Abscess; Elderly

Key points

- Unsteadiness and dizziness are common symptoms and can be disabling
- There are three main 'types' of dizziness: light-headedness, vertigo and disequilibrium
- The key question when faced with a patient with a single prolonged episode of vertigo is whether the underlying cause is peripheral (i.e. inner ear) or central
- After history, examination of the eye movements is key in the diagnosis of vertigo
- After benign positional vertigo, by far the most common cause of recurrent attacks of spontaneous vertigo is migraine; headache is not required to make this diagnosis
- Unsteadiness and dizziness are treatable

Introduction

Evaluating a patient who has unsteadiness/dizziness is often challenging. Half of all women and a third of men have had problems with dizziness by the age of 80 years. Vertigo accounts for 0.5% of GP consultations.¹ Dizziness can be disabling and lead to falls in older people (falls are the commonest cause of accidental death in the over 75s).

Case history

A 70-year-old woman had recurrent admissions to the Acute Medical Unit with disabling vertigo, vomiting and ataxia, which was now occurring around once a fortnight. The attacks had been present for around five years, but had become more frequent and severe in the last 6 months. Each attack was the same: relatively sudden onset vertigo with nausea, vomiting and ataxia, lasting around a day. There was no headache and never any focal neurological symptoms. At the age of 21 she had suffered 'a burst mastoid', had surgery, and had been under ENT follow-up ever since, with no known complications. She did not suffer from tinnitus, and did not have any ear discharge or pain.

On examination there was no spontaneous or gaze evoked nystagmus. Eye movements, including pursuit, saccades and the head-thrust test, were normal. Apart from reduced hearing in the left ear, the rest of the neurological examination was normal including a Hallpike manoeuvre on both sides. A CT head, including views of the petrous temporal bones, was normal (apart from signs of previous surgery). An MRI and MR angiogram of the brain was also normal.

What are the key anatomical structures involved in dizziness?

The body has continual sensory input from different modalities that provide us with spatial orientation and good balance. These are:

- Vision
- The vestibular system (i.e. the inner ear and its connections)
- Proprioception / sensation
- The brain (central processing system)

The so-called 'multi-factorial dizziness of the elderly' results from problems in several of these modalities in combination.

The vestibular system consists of the inner ear and its connections (peripheral) and the brain (central). Figure 1 illustrates the anatomy of the peripheral vestibular system.

Why are eye movements important in the assessment of dizziness?

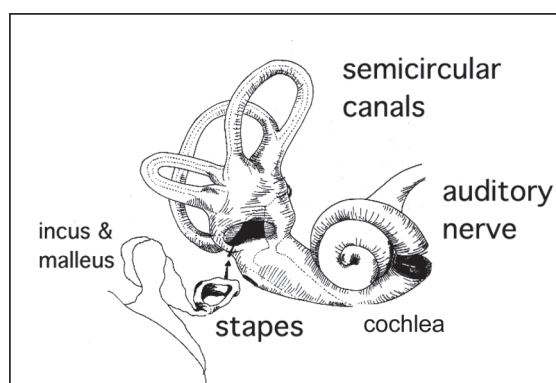
The vestibulo-ocular reflex (VOR) stabilises the eyes in space during head movements. At its most basic the reflex consists of a three neuron arc involving the vestibular nerve, an inter-neuron, and a motor neuron to the eye muscles. The reflex is co-ordinated by both of the vestibular nuclei. With head movement there is an asymmetry between the nuclei, and this can also occur with disease. For example, a complete loss of vestibular function on one side will mean the ipsilateral semicircular canals and otoliths are inactivated. The person will sense this as an asymmetrical input from the vestibular

Adrian Kennedy
MBBS BSc MSc MRCP

Nicola Cooper
MBChB FAcadMed
FRCPE

Correspondence:
Nicola Cooper
Consultant Physician
Tauranga Hospital, Private
Bag 12024
Bay of Plenty District
Health Board
New Zealand
Email: nacoooper@doctors.
org.uk

Problem based review: the patient with dizziness on the AMU



Each inner ear contains three semicircular canals (SCCs) and two otolith organs. The SCCs combined detect angular acceleration in any direction (via activation of hair cells that stimulate branches of the vestibular nerve), while the otoliths detect linear motion and orientation with respect to gravity.

The otolith organs (utricle and saccule) contain otoconia ('ear stones') that are constantly being formed and absorbed. An imbalance in this process is probably important in the pathophysiology of benign positional vertigo.

The cochlea is the organ of hearing.

Figure 1. Peripheral vestibular anatomy.

nuclei (equating to head movement when the head is actually still) and experience vertigo. Nystagmus is a result of the brain attempting corrective eye movements.

Smooth pursuit is a mechanism that allows fixation and clear vision of slowly moving objects (for example, when watching a slow game of tennis). In humans it is mediated largely by the cerebral cortex and cerebellum. Saccades are fast eye movements that allow fixation on different objects (for example, when startled or in response to commands). Control of saccadic eye movements is in the brainstem.

As a rule therefore:

- Peripheral vestibular disorders produce an abnormal VOR but spare other eye movements
- Central disorders may produce abnormal pursuit and saccades with or without VOR problems

The 'head thrust test' is useful in the evaluation of a single prolonged episode of vertigo as a test of VOR. The patient is asked to fix their eyes on the examiner, while their head is turned rapidly about 30 degrees first to the left and then to the right. In a normal person, the eyes move quickly and smoothly to remain fixed on the examiner. Jerky catch-up movements are seen when turning the head in one direction when there is a peripheral vestibular disorder on that side. The head thrust test is usually normal in central causes of vertigo (note however that the *full* history and examination are required to make a diagnosis).

What should the doctor look for when assessing eye movements?

- **Spontaneous nystagmus.** This is when the patient is asked to fixate on a stationary object straight ahead (e.g. a pen) and nystagmus is present.
- **Gaze-evoked nystagmus.** This is when the pen is moved 30 degrees to the left, right,

up and down, and nystagmus is seen only in certain positions. NB the pen should be at an average fixation distance of 30cm away and only moved 30 degrees as many normal people will have nystagmus at extremes of gaze. Stay a few seconds in each position.

- **Pursuit.** The patient is asked to follow a slowly moving target (e.g. a pen) with their eyes. Normal pursuit is smooth, but central disorders or *old age* lead to broken/jerky eye movements.
- **Saccades.** The patient is provided with two fixation targets (e.g. two fingers) and is asked to quickly look at one and then the other. Normal saccades are quick and accurate. Jerky catch-up movements are abnormal and indicate a central lesion (e.g. cerebrovascular disease).
- **Positional nystagmus.** It is important to look for positional nystagmus with the Hallpike manoeuvre. This is because benign positional vertigo is so common and *frequently co-exists with other vestibular disorders.*

Spontaneous nystagmus that is horizontal (or horizontal with a minor torsional component) and unidirectional is typical of an acute peripheral vestibular disorder (e.g. vestibular neuronitis). *Any other form of nystagmus in an upright patient is central* (the only caveat being that over time, the nystagmus of an acute peripheral vestibular disorder improves, becoming only gaze-evoked before resolving completely).

Patients with no neurological symptoms/signs and normal eye movements are very unlikely to have a central disorder. On the other hand, patients with neurological symptoms/signs *at any time during their acute illness* or abnormal eye movements are likely to have a central disorder. Therefore the examination (and understanding) of eye movements is key!

How can different types of dizziness be differentiated?

The first step in differentiating different types of dizziness is *pattern recognition* as different causes of dizziness have very distinct patterns of presentation. Fig 2 outlines the most common causes of dizziness encountered on an Acute Medical Unit. The next section describes the typical pattern in each. This will mainly focus on different types of vertigo, as this is what most doctors find difficult to assess. Tables are provided for drugs that cause dizziness/unsteadiness, and also summarize how to take a history and examine a patient with unsteadiness/dizziness.

What are the key elements in the history?

There are three main patterns of dizziness: lightheaded, vertigo (which means a sensation of movement – in any direction – not just 'spinning') and disequilibrium.

Lightheadedness (or pre-syncope) can be postural (in which case a low blood pressure is usually the problem) or unrelated to posture (in which case

Problem based review: the patient with dizziness on the AMU

Table 1. Some drugs that cause dizziness/unsteadiness

Anti-hypertensives	Alpha blockers Calcium channel blockers ACE inhibitors Diuretics Beta blockers
Alcohol	
Anti-epileptic medication	Carbamazepine Lamotrigine Valproate
Antidepressants	SSRIs
Other	Statins Sedatives Drugs with anti-cholinergic side effects Opioid analgesics
Chemotherapy	Carboplatin Cisplatin
Ototoxic drugs	Vancomycin Gentamicin

one should consider cardiac and endocrine causes, drugs and psychological causes). Be aware of postural orthostatic tachycardia syndrome (POTS) which causes severe lightheadness in the upright position with no change in blood pressure but a significant increase in heart rate on standing.²

Disequilibrium is the word used to describe symptoms of a sense of imbalance, 'vague' dizziness, or a feeling of being 'fuzzy all the time', particularly when mobilising. It can be hard for patients to describe. A complete neurological examination is important in these cases. However, the most common causes of disequilibrium encountered in practice are:

- An uncompensated vestibular disorder
- Migraine
- Multi-factorial dizziness of the elderly
- Neurological conditions e.g. sensory peripheral neuropathy

- Bilateral vestibular failure (e.g. from gentamicin ototoxicity) – rare

Uncompensated vestibular disorders are common! They occur after a person has a severe attack of vertigo – of any cause. In the subsequent days and weeks the symptoms slowly improve and most people make a full recovery. However, a significant proportion of people never fully recover and are left with disequilibrium. A typical patient will describe feeling 'off balance or as if I am walking on a ship' or 'dizzy all the time' with brief vertigo on turning his head. Often patients develop a stiff posture to limit head movement and secondary neck pain. On further questioning, many find visually stimulating environments make their symptoms worse, e.g. supermarkets, busy traffic, checkerboard patterns. The most important question for the doctor to ask is: 'Have you ever had a bad attack of vertigo?' These types of symptoms can also be caused by migraine. However, it is vital that a complete history is obtained and neurological examination performed to exclude other causes. Vestibular physiotherapy is effective in improving symptoms due to an uncompensated vestibular disorder³ and most neuro-physiotherapists are familiar with the assessment and treatment of such patients.

What are the main causes of 'vertigo'?

There are five major causes of vertigo *alone* (i.e. without any focal neurological symptoms or signs at any time):

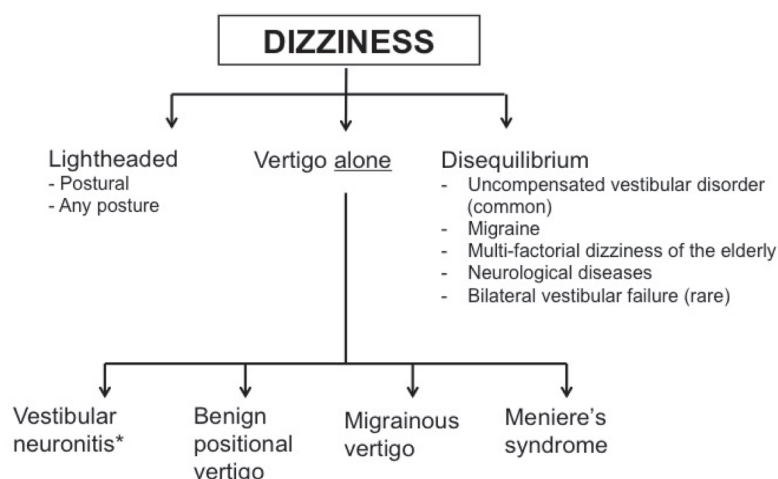
A single episode of prolonged vertigo alone

- Vestibular neuronitis
- Stroke

Recurrent attacks of vertigo alone

- Benign positional vertigo (very common)
- Migrainous vertigo (common)
- Meniere's syndrome (rare)

Note that 'vertebrobasilar insufficiency' and cervical spondylosis are *not* causes of recurrent vertigo.



* Causes a single episode of prolonged vertigo, whereas the others all cause recurrent 'attacks' of vertigo.

Figure 2. Patterns of dizziness.

Problem based review: the patient with dizziness on the AMU

Table 2. Clinical criterion for suspecting a central cause of a single prolonged attack of vertigo

Clinical criterion	Suspected diagnosis
Older or vascular risk factors	Ischaemia of the labyrinth or root entry zone of the 8th nerve
Abrupt hearing loss	Labyrinthine infarction (AICA occlusion); concomitant brainstem /cerebellar infarction
Previous unexplained neurological symptoms for days / weeks in a younger patient	Undiagnosed MS
Central oculo-motor findings: saccadic pursuit, pure torsional or upbeat /downbeat nystagmus, gaze evoked nystagmus, central positional nystagmus	Involvement of central vestibular structures in brainstem / cerebellum
Cranial nerve abnormalities	Brainstem lesion
Long tract symptoms or signs: hemi-sensory loss, weakness/ extensor plantars, Horner's syndrome	Brainstem lesion
Cerebellar symptoms or signs: truncal or limb ataxia, dysarthria	Cerebellar lesion

Abrupt hearing loss can also be a feature of basilar migraine, but should be considered a 'red flag' for a possible cerebrovascular cause of vertigo.

1. Vestibular neuronitis (the proper term for 'viral labyrinthitis')

Viral infection is the most probable cause of vestibular neuronitis. Patients present with vertigo, ataxia and nausea, usually on rising in the morning. However, up to a quarter of patients will experience a more gradual onset of symptoms throughout the course of a day. Most are still be able to mobilize independently but are reluctant to do so.

An acute episode of vestibular neuronitis usually lasts several days but patients can be left with non-specific dizziness for many weeks. Vestibular suppressants (e.g. prochlorperazine) are used during the acute attack. Vestibular rehabilitation exercises can be given to patients to use later.

If seen acutely, patients will have no other neurological signs apart from those of acute unilateral vestibular failure: *spontaneous* nystagmus towards the healthy ear, a positive head thrust test (a defective vestibulo-ocular reflex when thrusting the head towards the affected side), unsteadiness and veering towards the affected side on walking. The diagnosis of vestibular neuronitis can therefore be made clinically. If you see a patient with vertigo acutely (i.e. on the day of onset) and they do not fit this pattern, the diagnosis is unlikely to be vestibular neuronitis.

2. Stroke

Recurrent attacks of vertigo without any other focal neurological symptoms or signs are very unlikely to be TIAs – there are nearly always accompanying focal neurological symptoms/signs e.g. diplopia, motor weakness, speech or sensory disturbance. It is however relatively common for older people with vascular risk factors to present with a single prolonged episode of acute unsteadiness/vertigo alone, with no other apparent cause (after a detailed assessment) apart from a vascular one. They do not have the signs of vestibular neuronitis on examination, cannot mobilize independently due to ataxia and often

have gaze-evoked nystagmus. Fewer than 10% of patients with vertigo presenting to the Emergency Department have a central cause but these are important not to miss. Certain brainstem and midline cerebellar lesions can produce isolated vertigo (with associated ataxia and sometimes nausea/vomiting). Even diffusion weighted MRI has a significant false negative rate in detecting clinical strokes⁴ so often the diagnosis has to be clinical. Table 2 summarizes the clinical criterion for suspecting a central cause in a single prolonged attack of vertigo. Referral to a stroke specialist is recommended in these cases.

The next section covers causes of recurrent attacks of vertigo (although it is possible that patients may be presenting with their first ever episode, in which case diagnosis can be difficult and often only achievable over time).

3. Benign positional vertigo (BPV)

This is the most common cause of recurrent vertigo. The lifetime prevalence of BPV in one study was 2.4% and this increases with age.⁵ Posterior BPV is thought to arise from calcium debris (loose otoconia) in the posterior semi-circular canal. It can occur spontaneously (e.g. in older people), after minor head trauma, or following a vestibular insult (e.g. vestibular neuronitis). Patients describe *several seconds to minutes* of vertigo when they do certain things like stooping, looking up, lying down or turning over in bed. The Hallpike manoeuvre reproduces nystagmus that is typical of posterior BPV: rotational, delayed and fatiguable. The neurological history, examination and other eye movements will be normal. Around 10% of patients have the condition in both ears, and 10% have horizontal (rather than the more common posterior) canal BPV. While BPV tends to spontaneously resolve in most cases, it is easily treated at the bedside with the Epley manoeuvre (for posterior canal BPV that is – for information on horizontal canal BPV see Further Resources).

Problem based review: the patient with dizziness on the AMU

4. Migrainous vertigo

Migraine is very common. Around 25 million people are affected by migraine in the USA, for example.⁶ Of these, approximately one third are affected by episodic vertigo. This suggests around 3% of the US population are affected by migrainous vertigo – compared to only 0.2% who have Meniere's disease. While vertigo may accompany a headache or other classical migrainous features (e.g. photo/phonophobia), patients commonly present with recurrent attacks of vertigo alone. In most cases, there is a personal or family history of migraine. Symptoms tend to be variable and can last from several minutes to several weeks. This is in contrast to Meniere's which is characterized by progressive hearing loss and tinnitus with attacks lasting 20 minutes to several hours. In between attacks, the clinical examination is normal in migrainous vertigo.

Migrainous vertigo is *not* the same as vertebrobasilar migraine which is a distinct form of migraine involving posterior circulation symptoms in addition to vertigo.

Proposed criteria for the diagnosis of migrainous vertigo are:⁷

1. Episodic vestibular symptoms (rotational vertigo, other illusory self-motion, positional vertigo, head motion discomfort – a sensation of imbalance or vertigo that is provoked by head motion)
2. Migraine according to the criteria of the International Headache Society⁸
3. At least one of the following migrainous symptoms during at least two vertiginous attacks –
 - Migrainous headache
 - Photophobia
 - Phonophobia
 - Visual or other auras
4. Other causes ruled out by appropriate investigations.

The International Headache Society criteria for the diagnosis of migraine without aura (the most common form of migraine) remind us that migraine cannot be diagnosed with confidence after a single attack, and this is particularly true for older people. More details about 'probable migraine', 'probable migrainous vertigo' and 'benign recurrent vertigo' (which is probably migraine-related but does not meet any diagnostic criteria) can be found in the Further Resources by Bronstein and Lempert.

Attacks of migrainous vertigo are treated in the same way as migraine, and some patients who have symptoms similar to an uncompensated vestibular disorder derive additional benefit from a programme of vestibular physiotherapy.

5. Meniere's syndrome

Meniere's syndrome is thought to be caused by 'endolymphatic hydrops' – too much fluid in the

endolymphatic space. Meniere's Disease is the idiopathic form. It is thought that the sudden rupture of the membranes separating endolymph and perilymph leads to vestibular dysfunction on that side, which recovers fairly rapidly. Hence, typical symptoms of Meniere's are attacks of vertigo with hearing loss, 'rushing' tinnitus and aural fullness. Attacks last anywhere between 20 minutes to several hours. Patients are unable to walk. Vomiting is common and diarrhoea can also occur.

At first there is fluctuating sensorineural hearing loss (especially in the low frequency range) then there is progressive hearing loss with associated tinnitus. Over time, 40% of patients have bilateral disease.⁹ This is the only vertiginous condition in which there is evidence for the use of betahistidine, along with thiazide diuretics (thought to reduce the pressure in the endolymphatic space). These patients should be referred to an ENT specialist for further evaluation as surgical treatments can also be effective.

Acoustic neuromas can present with similar symptoms, but usually cause only mild vestibular symptoms, rarely episodic vertigo, and tend to affect high frequency hearing. However, many ENT specialists request an MRI scan in patients with features of Meniere's to exclude acoustic neuroma.

No other type of dizziness benefits from *long term* vestibular suppressants which impair vestibular rehabilitation (leading to the disequilibrium of an uncompensated vestibular disorder) and can cause unpleasant side effects in older people (e.g. Parkinsonism).

What are the psychosocial aspects of dizziness?

There is an interplay between psychiatric disorders and dizziness. As much as 30-50% of complex dizziness disorders are not completely explained by an organic diagnosis alone.¹⁰ In a *small minority* of cases there is a pure psychiatric basis to symptoms. Most commonly, organic dizziness also has a psychiatric element to it – dizziness can be disabling and is associated with anxiety.

Table 3. How to take a history in unsteadiness/dizziness

- It is most useful simply to ask patients to tell the story of their dizziness and to listen without interrupting. Remember that having more than one type of dizziness is not unusual – if this appears to be the case, then take a separate history for each type of dizziness.
- Decide if the patient is describing lightheadedness, vertigo or disequilibrium (this can sometimes be difficult, so the bigger picture/other features sometimes help)
 - If lightheaded is it postural or unrelated to posture?
 - If vertigo, is it a single prolonged attack or recurrent?
 - If disequilibrium, ask about a previous attack of severe vertigo and migraine
 - Associated symptoms (neurological, migrainous, hearing/ears)
 - Past medical history including migraine
 - Medication
 - In recurrent dizziness, ask about triggers and whether the patient back to normal in between attacks.

Problem based review: the patient with dizziness on the AMU

Table 4. Summary of how to examine a patient with unsteadiness/dizziness

Patients with no neurological symptoms/signs and normal eye movements are very unlikely to have a central disorder. Patients with neurological symptoms/signs at any time during their acute illness or abnormal eye movements – apart from those typical of acute vestibular neuritis – are likely to have a central disorder. This examination is geared mainly towards patients with *vertigo* or *disequilibrium*.

- Lying and standing blood pressure/pulse
- Watch the patient walk
- Complete neurological examination (ie cranial nerves and limbs)
- Eye movements – in all patients look for:
 - Spontaneous nystagmus
 - Gaze-evoked nystagmus
 - Pursuit
 - Saccades
 - Positional nystagmus
- Head thrust test if dealing with a single episode of prolonged vertigo
- Depending on the history, an audiological examination may be required

After the onset of an acute unsteadiness/dizziness disorder there is a process of adaptation and hopefully resolution in symptoms. Anxiety and other psychiatric diagnoses impair this process, mainly by avoidance behaviour. This is important to recognize and treat in its own right if the patient is to improve. Vestibular physiotherapy and sometimes cognitive behavioural therapy are useful treatments for certain people. Cognitive behavioral therapy provides the patient with an explanatory model for their symptoms, taking into account medical and psychological factors. It can also provide techniques to deal with dizziness and other symptoms (e.g. hyperventilation).

Case history: what happened next

The frequency of attacks, lack of focal neurological symptoms, and the fact that they had been occurring for 5 years meant that TIAs were very unlikely. There was no tinnitus or progressive hearing loss to support a diagnosis of Meniere's, and again the attacks were too frequent. As migraine is by far the most common cause of recurrent attacks of spontaneous vertigo, the patient was started on metoprolol to treat migraine. She had no personal history, but there was a family history of migraine. Following this, the attacks ceased. She is currently being followed up in a dizzy clinic and is doing well.

References

1. Furman JM, Cass SP. Vestibular disorders. A case-study approach 2nd Ed. Oxford University Press, Oxford, 2003.
2. Heaps T, Cooper N. A case of postural orthostatic tachycardia syndrome on the AMU. *Acute Medicine* 2009; **8(1)**: 29–31.
3. Strupp M, Arbusov V, Maag KP *et al*. Vestibular exercises improve central vestibulo-spinal compensation after vestibular neuritis. *Neurology* 1998; **51**: 838–44.
4. Doubal FN, Dennis MS, Wardlaw JM. Characteristics of patients with minor ischaemic strokes and negative MRI: a cross-sectional study. *J Neurol Neurosurg Psychiatry* 2011; **82**: 540–2.
5. Von Brevern M, Radtke A, Lezius F *et al*. Epidemiology of benign paroxysmal positional vertigo: a population based study. *J Neurol Neurosurg Psychiatry*. 2007; **78(7)**: 710.
6. Stewart WF, Shechter A, Rasmussen BK. Migraine prevalence. A review of population-based studies. *Neurology*. 1994; **44(6)**: S17–23.
7. Bronstein A and Lempert T. Migrainous vertigo. In: Dizziness. A practical approach to diagnosis and management. Cambridge University Press, Cambridge, 2007.
8. International Headache Society. The International Classification of Headache Disorders 2nd Edition. *Cephalalgia* 2004; **24** (1).
9. Kitahara M. Bilateral aspects of Menière's disease. Menière's disease with bilateral fluctuant hearing loss. *Acta Otolaryngol Suppl*. 1991; **485**: 74–7.
10. Psychotherapy in Dizziness G Schmid; P Henningsen; M Dieterich; H Sattel; C Lahmann. *J Neurol Neurosurg Psychiatry*. 2011; **82(6)**: 601–6.

Further Resources

1. Bronstein A and Lempert T. Dizziness. A practical approach to diagnosis and management. Cambridge University Press, Cambridge, 2007. Highly recommended, and comes with a DVD demonstrating examination of the eyes and positional manoeuvres.
2. Furman JM and Cass SP. Vestibular disorders. A case-study approach 2nd Ed. Oxford University Press, 2003. Covers common and more unusual cases of dizziness with discussion.
3. Cooper N, Forrest K and Mulley G. Falls. In: ABC of Geriatric Medicine. Wiley-Blackwell, Oxford, 2009. Contains a useful illustration of how to perform the Epley Manoeuvre.
4. Hearing and balance disorders. Achieving excellence in diagnosis and management. Report of a Working Party. Royal College of Physicians, London, 2007. www.rcplondon.ac.uk Includes guidelines on referral to a specialist for a balance disorder.