Abstract

Whilst dizziness, vertigo and imbalance may be due to systemic disease, they commonly originate from the vestibular system. The presence of vertigo, in particular, implies vestibular involvement. This article will address the diagnosis and management of vestibular dysfunction in adults, highlighting the three commonest causes which can be diagnosed and treated in a primary care setting, and stressing the “red flags” that should trigger referral to more specialist services.

Keywords

Vertigo, Imbalance, Migraine, BPPV, Peripheral vestibular impairment.

Introduction

Dizziness is one of the commonest symptoms to present in General Practice. It has been estimated that 30% of the UK population will have experienced some dizziness or imbalance by the age of 65\(^1\). In a random sample survey of four North London GP practices, 25% of 2064 respondents suffered intrusive dizziness\(^2\). The treatment of these symptoms is patchy: all too often patients are told to “learn to live with it”, are given the “wrong” vestibular exercises or, worst of all, extended courses of inappropriate vestibular sedatives which may further impair balance.

In January 2009, The UK Department of Health launched its “Good Practice Guide” to the provision of adult balance services\(^3\). The proposals aimed to improve primary care provision for this group of patients, increase primary care practitioners’ awareness of balance disorders and the scope to treat them, improve the rate and appropriateness of referrals and eliminate unnecessary hospital attendances and investigations. The aims of this article could be summarised in the same way. The three commonest conditions presenting to the author’s specialist balance clinic, making up roughly 75% of his caseload, could readily be diagnosed and treated in Primary Care. Practitioners also need to be aware of those (mainly elderly) at risk of falling and triage them into the appropriate falls clinic, and be alert for those with potentially more serious disease (“red flags”).

Dizziness, as a symptom, can arise from vestibular, cardiovascular, neurological or metabolic disorders. The key question, therefore, is what exactly the patient means by “dizzy”. Vertigo, although often rotational, is more correctly defined as an illusion of movement. Whilst vertigo implies some involvement of the vestibular system and its absence makes vestibular disease less likely, these distinctions are not absolute. Thus vertigo is a common symptom of pre-syncope (due to transient ischaemia of the vestibular system). Furthermore, vestibular disorders (especially bilateral or central) may sometimes present with imbalance but little or no actual vertigo. Vestibular disorders themselves may be roughly divided into peripheral (inner ear, vestibular nerve – generally benign) and central (brainstem, cerebellum, higher cortical centres – potentially more worrying).

Our understanding of the pathology underlying many vestibular disorders is patchy and our ability to treat it necessarily limited. Fortunately, effective symptomatic treatments are available and, on that basis, it is useful to divide vertiginous symptoms into three main categories:

1. **Spontaneous.** Vertigo arises without provocation and often without warning and is not triggered by any particular activity.

2. **Movement-provoked.** Vertigo (generally transient) is triggered by any movement but the patient feels relatively well as long as movement is avoided.

3. **Positional.** Vertigo is triggered by movement to or from specific positions.

A. **Spontaneous Vertigo**

The diagnosis and management of acute spontaneous vertigo depends upon whether it is an isolated event or one of a series of recurrent attacks, bearing in mind the possibility that an isolated attack could be the first in a series.
i) Single episode

Acute vestibular events (typically with sustained vertigo, lasting several hours) are often unthinkingly labeled as “labyrinthitis” and the underlying pathology assumed to be viral. Apart from the fact that, in the absence of hearing symptoms, the term “vestibular neuritis” is to be preferred, there are problems with this. Because the neural and vascular supply to the inner ear follow an identical distribution, it is not possible on the basis of clinical signs to distinguish between viral and ischaemic aetiologies and it is therefore always worth enquiring about vascular risk factors. The inclusive term “Acute Peripheral Vestibulopathy” has much to recommend it.

More seriously, whilst the vast majority of cases of acute vertigo are peripheral in origin, it is vital not to miss the occasional case of brainstem or cerebellar stroke. Five percent of new cases of Multiple Sclerosis are said to present with acute vertigo. The “red flag” features prompting the need for urgent referral are outlined in Box 1.

Additionally, patients with acute hearing loss (especially if unilateral) need referral to rule out middle ear or VIII nerve pathology, though it should be noted that Vestibular Schwannomas and other cerebello-pontine angle lesions very rarely present with acute vertigo.

Examination of the acutely vertiginous patient

There are three key examinations which can readily be performed in primary care and which will help to distinguish peripheral from central pathology:

1. Examine for nystagmus.
   i) Spontaneous and Gaze-evoked (30 degrees to left and right). Vestibular nystagmus has a “sawtooth” pattern: a slow drift (the pathological phase) and rapid, corrective jerk (saccade). The direction of nystagmus is defined (rather confusingly) by the direction of the fast (physiological) phase. Nystagmus will generally be most pronounced when the patient looks in the direction of the fast phase (Alexander’s Law). Nystagmus of peripheral origin does not change direction with direction of gaze and is never vertical or purely torsional (though it may have a torsional component).

   ii) Re-examine in the absence of optic fixation: peripheral nystagmus is always enhanced. This can be done with an ophthalmoscope (as long as it has a bright halogen light source) with the patient covering the contra-lateral eye. This is more effective than the 20 diopter illuminated “Frenzel’s glasses” used in some ENT clinics. On fundoscopy, the movement of the retinal vessels can be observed and will be more marked when the contra-lateral eye is covered that when the patient is able to fixate. (As the back of the eye is being observed, note that the direction of the nystagmus will be opposite to that which would be noted by looking at the front of the eye.)

2. The Head-Thrust test (Figure 1) may be positive in peripheral cases, especially acutely. (See also Barraclough and Bronstein (2009) for an excellent account. This test is also known as the Halmagyi Head-Thrust or Head Impulse test.) The examiner jerks the patient’s head, very rapidly, some 20 degrees to the side whilst the patient tries to maintain fixation on the examiner’s nose. A patient with intact vestibular function can do this. In the presence of moderate to severe loss of function on the side to which the head is thrust, the patient’s eyes move with the head and then
require a corrective saccade in order to re-fixate on the examiner. The test has a high specificity: if positive (a corrective saccade needed), one can be confident that the vestibular lesion is peripheral.

3. **The Hallpike positional test** (Figure 2) may reveal a central pattern of nystagmus (see below and Boxes 3 and 4).

### Treatment of acute vertigo

Vestibular sedatives and anti-emetics are useful but in the acute phase only; prolonged use (greater than one week) may delay full recovery. Vestibular exercises (see below) should be introduced at an early stage, in the event of persistent symptoms, to enhance central compensation. Consider treating vascular risk factors.

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**Figure 2:** Right Hallpike test (photos 1 & 2) and Epley Manoeuvre (photos 3 – 6)
Dizziness, Vertigo and Imbalance

ii) Multiple episodes

Whilst it is possible to suffer more than one attack of Vestibular Neuritis, it would be unusual to have more than three in a year or more than a dozen in total. Alternative diagnoses should be sought.

Vestibular Migraine is the commonest cause of recurrent attacks of acute, spontaneous vertigo: far commoner than Menière’s disease (with which it is often confused) and constituting roughly a quarter of all new cases seen in the author’s balance clinics. The diagnosis is by exclusion of other causes and by response to treatment. The presentation is very variable and the distinction from Menière’s (see Table 1) can be difficult in the early stages. Headache is an inconsistent feature, occurring in under 50% of cases and, even in those cases, not with all attacks. Whilst balance generally returns to normal between attacks, persistent motion sensitivity is common.

Menière’s Disease attacks are associated with unilateral aural fullness, hearing loss (which classically fluctuates and may return to normal, at least in the early stages) and tinnitus, generally low-tone. Attacks of Menière’s (and of migraine) commonly occur in clusters with periods of (often prolonged) remission. Attacks can occur at any time and in any place: one of their most distressing features. (In contrast, vestibular migraine attacks in some patients are present, characteristically, on waking.) To confuse the picture further, some patients may present with both vestibular migraine and Menière’s Disease, probably due to a genetic overlap. In some cases one may trigger the other: an episode of acute vertigo may trigger a migraine headache in susceptible individuals. Diagnostic criteria exist for both conditions6,7.

Other, rare causes of recurrent attacks of acute spontaneous vertigo include posterior circulation TIAs (consider especially if there are other posterior circulation symptoms or signs such as facial numbness, dysarthria, diplopia etc), hyperventilation syndrome/panic attacks (which can cause recurrent attacks of ‘constant dizziness’), autoimmune inner ear disease (vertigo with bilateral progressive hearing loss especially in the presence other autoimmune disorders) and, very rarely, otosyphilis or vestibular epilepsy. Uncommon structural anomalies such as wide vestibular aqueduct, dehiscent superior semicircular canal or neuro-vascular compression can give rise to recurrent vertigo. These are specialist diagnoses and therefore fall outside the scope of this article.

Prophylactic treatment of recurrent attacks of acute vertigo

Eliminate migraine triggers. If attacks are frequent enough to warrant it, beta-blockers (propranolol), amitriptyline or pizotifen may be effective in migraine. A low salt diet, betahistine and a thiazide diuretic may help Menière’s. Patients with suspected Menière’s should be referred for specialist investigation. All patients with unilateral hearing loss require an MRI to exclude a vestibular Schwannoma, though these very rarely present with attacks of acute vertigo.

B. Positional Vertigo

Positional vertigo is defined as vertigo that is provoked by movement to or from certain positions.

Benign Paroxysmal Positional Vertigo (BPPV) (Box 2) is by far the commonest cause of positional vertigo (constituting a further quarter of all new cases seen in the author’s balance clinics) but the rarer, central forms must always be considered. Very rare in childhood and adolescence, BPPV becomes progressively commoner with age and may account for a third of all dizziness in the over-80’s8. Positional vertigo in children should be considered neurological until proved otherwise and must

<table>
<thead>
<tr>
<th>Feature</th>
<th>Migraine</th>
<th>Menière’s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nature of vertigo</td>
<td>Acute rotational</td>
<td>Acute rotational</td>
</tr>
<tr>
<td>Duration of attacks</td>
<td>20 sec–10 days</td>
<td>&gt;20 mins; &lt;24 hours</td>
</tr>
<tr>
<td>Hearing loss</td>
<td>Rare</td>
<td>Unilateral – fluctuating/progressive</td>
</tr>
<tr>
<td>Tinnitus</td>
<td>Bilateral or “in head”</td>
<td>Unilateral, increases with attacks</td>
</tr>
<tr>
<td>Aural fullness</td>
<td>Rare (bilateral if present)</td>
<td>Common. Unilateral</td>
</tr>
<tr>
<td>Headache with attacks</td>
<td>Inconsistent relationship</td>
<td>Around 50% of cases</td>
</tr>
<tr>
<td>Nystagmus during attack</td>
<td>Usually mild: may be peripheral or central (often positional)</td>
<td>Severe, classically irritative (beats towards affected ear)</td>
</tr>
<tr>
<td>Photophobia/phonophobia</td>
<td>Common</td>
<td>Absent (hearing may be distorted)</td>
</tr>
<tr>
<td>Imbalance between attacks</td>
<td>Mild. May be travel sick.</td>
<td>Commoner as disease progresses</td>
</tr>
<tr>
<td>Examination between attacks</td>
<td>Usually normal</td>
<td>Usually normal</td>
</tr>
<tr>
<td>Past/family history of migraine</td>
<td>Common – desirable in order to make diagnosis</td>
<td>Fairly common</td>
</tr>
<tr>
<td>Triggers</td>
<td>Stress, Diet, Hormonal</td>
<td>Stress</td>
</tr>
</tbody>
</table>

Table 1: Features that help distinguish Vestibular Migraine from Menière’s Disease
Dizziness, Vertigo and Imbalance

Classical provoking movements:
- Lying flat
- Sitting up from lying flat
- Turning over in bed (especially to affected side)
- Looking up (extending neck), eg hanging washing, reclining in a chair
- Bending down (flexing neck), especially to side, eg looking under sink

Vertigo:
- Usually rotational.
- May be “falling through bottom of bed”. On getting up, “being thrown back onto bed”.

Duration:
- Usually 5 – 20 seconds. Always* under 1 minute.
- May notice “latent period” (before onset of symptoms) of up to 40 secs.

Accompanying symptoms:
- No neurological or audiological symptoms.
- But BPPV may complicate other inner ear disease.

Pattern of attacks:
- Bouts: days, weeks or months at a time, with symptom free periods.
- OR, may occur every day for years on end.

Constant unsteadiness rather than vertigo may be the main symptom, especially in the elderly.

Box 2: Symptoms of Benign Paroxysmal Positional Vertigo

- Sit patient on centre of couch, legs dangling.
- Turn head 45º to (L)
- Lie patient rapidly onto (R) side: head position is same as in Hallpike test.
- Maintain position until vertigo/nystagmus stops.

- Patients ends up 45º nose down and is likely to be vertiginous.
- Maintain position for 2 minutes.
- Sit patient up again and incline head 30º forwards. Patient may be dizzy again and may need firm support to prevent falling off couch!

- throw patient VERY RAPIDLY onto (L) side WITHOUT turning their head: it MUST still face 45º to (L).

Figure 3: Right side-lying test and Semont Liberatory Manoeuvre
Dizziness, Vertigo and Imbalance

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Not be confused with Benign Paroxysmal Vertigo of Childhood, which is probably a migraine equivalent.

The vertigo is caused by calcium carbonate (presumed otoconial, dislodged from the macula in the utricle) debris in one of the semicircular canals (SCCs), usually the posterior. The Hallpike test is the best method for identifying BPPV (Box 3 and Figure 2, photos 1 and 2) and is obligatory in all patients with dizziness.

**Box 3: Features of posterior Semi-circular canal Benign Paroxysmal Positional Vertigo on Hallpike test**

### A. Vestibular causes (other than posterior semi-circular canal canalithiasis. These are difficult diagnoses and should be referred to a specialist.)

- **Horizontal canal BPPV**: positional nystagmus is horizontal, beating to the undermost ear (geotropic).
- **Anterior canal BPPV**: (rare) positional nystagmus is downbeat and torsional.
- **Cupulolithiasis**: (BPPV caused by debris adherent to the cupula instead of free-floating) positional nystagmus is sustained from 1-3 minutes. The nystagmus of **Horizontal SCC cupulolithiasis** is sustained and beats to the uppermost ear (apogeotropic).
- **Central (cerebellar) disorders**: positional nystagmus may be sustained, horizontal, purely vertical (usually downbeat) or purely torsional and may be asymptomatic.
- **Migrainous vertigo** may mimic the symptoms of BPPV but positional nystagmus, if present, is likely to have central features and should not easily be confused with that of posterior SCC BPPV.

### B. Non-vestibular causes

- **Orthopnoea** may cause dizziness on lying flat, with breathlessness but NO nystagmus.
- **Cervicogenic vertigo** is overdiagnosed and may not exist at all. Suspect other causes of positional and movement induced vertigo, especially BPPV.
- **Vertebrabasilar ischaemia** is grossly over-diagnosed. Many of these patients actually have BPPV. Only consider if there are other posterior circulation symptoms/signs (facial numbness, dysarthria, diplopia etc).
- **Postural hypotension** will cause dizziness on sitting up but not on lying. There is NO accompanying nystagmus.

**Box 4: Rarer causes of positional vertigo**

C. **Movement-provoked Vertigo**

The balance organs of the inner ear are sensitive to movement, specifically to acceleration. The peripheral vestibular system works on a “push-pull” basis: any movement which stimulates one side results in an equivalent inhibition of the other. If one side is impaired, the brain is unable to interpret the fact that it is no longer receiving complementary input from the two sides and the result is a transient sense of vertigo and imbalance, often described by patients as a feeling of drunkeness or perpetual seasickness, provoked or exacerbated by head movement (which they often try to avoid).

The diagnosis is of **incompletely or poorly compensated** (or decompensated, see below) **unilateral peripheral vestibular impairment** and is commonly the long-term consequence of an episode of vestibular neuritis. This group constitutes a further quarter of all new cases seen in the author’s balance clinics.

Patients with persistent (unilateral or asymmetrical) peripheral vestibular impairment recover normal balance (given time: generally weeks, sometimes months) because the central vestibular system compensates, as long as they exercise and do not avoid the movements that provoke their symptoms. Unfortunately, the tendency of many patients to avoid symptoms by avoiding movement may delay compensation and recovery, which are also impeded by prolonged use of vestibular sedatives, by anxiety, panic etc. if present.
attacks, depression and any associated visual and proprioceptive abnormalities. (Figure 4.) Ask about a past history of acute vertigo. This may have occurred years previously: a period of stress or inactivity may provoke the recurrence of symptoms through central decompensation.

Many patients with chronic movement-related imbalance become anxious and lose confidence in their balance. As a standard physical examination will generally be normal, they are all too often dismissed as “mad”, or as suffering from “medically unexplained symptoms”. Alternatively (and almost as bad), their symptoms may be blamed on cervical spondylosis. In these cases, nystagmus may be apparent on the absence of optic fixation (use an ophthalmoscope: see above) and may be enhanced or provoked by a period of passive head-shaking (20 brisk shakes over an arc of around 45 degrees). The Head Thrust test is less likely to be positive than in cases of acute vestibular failure but is still worth trying.

**Bilateral vestibular hypofunction/failure** causes imbalance and oscillopsia (an illusion that the world moves as the patient moves, eg bobs up and down as the patient walks) rather than vertigo. Balance is much worse in the dark. Reduced dynamic visual acuity (blurred vision on head movement) may be noticed and can be tested for using a standard Snellen chart whilst the patient’s head is shaken at around 2 Hz. In severe cases, even reading may be difficult. Causes include meningitis, ototoxic drugs, autoimmune disease, head trauma and bilateral “burnt-out” Menière’s. Many cases are idiopathic. A good screening test which can be carried out anywhere is to spin the patient ten times rapidly on a rotating office chair: a patient with intact vestibular function should be quite vertiginous when the spinning stops, and should have readily apparent nystagmus, even with fixation. If the patient is not dizzy (when spun in either direction), a diagnosis of bilateral vestibular hypofunction is probable.

“**Visual vertigo**” may be triggered by movement in the visual surroundings (eg: crowds, traffic, busy supermarket aisles or shopping centres, “panning” scenes on TV or cinema), by looking at repetitive patterns (eg: striped shirts, patterned floors and fences) or by flickering lights and computer screens. It is caused by “visuo-vestibular conflict”, visual dependence or visual substitution often (but by no means invariably) following an acute vestibular event. Physiologically related to height vertigo, it may have a prominent phobic element, exacerbated by avoidance behaviour.

**Central (brainstem / cerebellar) disorders** may present as movement-related imbalance. Look for central, especially downbeat or bidirectional, spontaneous or positional nystagmus. Imbalance is usually more prominent than vertigo. Progressive imbalance in the absence of vertigo is a particularly worrying symptom which may merit imaging even in the absence of specifically central signs.

**Multisensory Disequilibrium of ageing** is a very common cause of imbalance and non-specific dizziness in the elderly. Relevant factors include visual impairment, peripheral neuropathy, arthritis and cerebrovascular disease as well as vestibular impairment.

**Treatment of Movement-provoked Vertigo**

Stop all vestibular sedatives. Anti-vertiginous medication is a major cause of imbalance.
Prochlorperazine is the worst, with the additional risk of inducing Parkinsonism with medium to long term use. A thorough explanation (and reassurance) is the key to successful intervention in cases where anxiety has become a significant factor. Treat with vestibular rehabilitation exercises. Gaze stabilization exercises (involving repetitive head movement whilst the eyes maintain fixation on a target), acting on the vestibulo-ocular reflex, are probably better tolerated and more effective than traditional Cawthorne–Cooksey exercises which are designed to provoke symptoms and are more akin to aversion therapy. In some patients, especially those with long-term symptoms, Cognitive Behavioural Therapy or other psychological interventions may be beneficial.

Conclusion

Table 2 illustrates the practical value of the division of symptoms of vertigo and imbalance into the three categories of spontaneous, positional and movement-provoked, as the treatment depends more on this vertigo type than on the underlying pathology. Patients with Ménière’s disease, for example, may present at different times with each different type of vertigo and it is important not to dismiss their new vertigo as “a recurrence of the Ménière’s” (giving them further betahistine) without looking into the exact pattern of their new symptoms.

Vestibular migraine, BPPV and unilateral peripheral vestibular dysfunction each account for about 25% of the patients seen in the author’s balance clinics. The majority of these cases could, potentially, have been treated successfully in Primary Care. Increasing awareness of these common disorders, and of how to diagnose and treat them, should revolutionize the experience of patients with vertigo and imbalance, whilst awareness of “red flags” warranting early referral for specialist opinion should prevent delayed diagnosis of those with more sinister symptoms. Specialist opinion should always be sought if the primary care physician is concerned about lack of progress or poor response to treatment.

References


**Further reading**

Bronstein AM and Lempert T; *Dizziness – A practical approach to Diagnosis and Management*. 2007, Cambridge University Press

(Concise, comprehensive and inexpensive. Includes CD-ROM illustrating examination techniques and types of nystagmus.)