Recommended management of vestibular causes of dizziness

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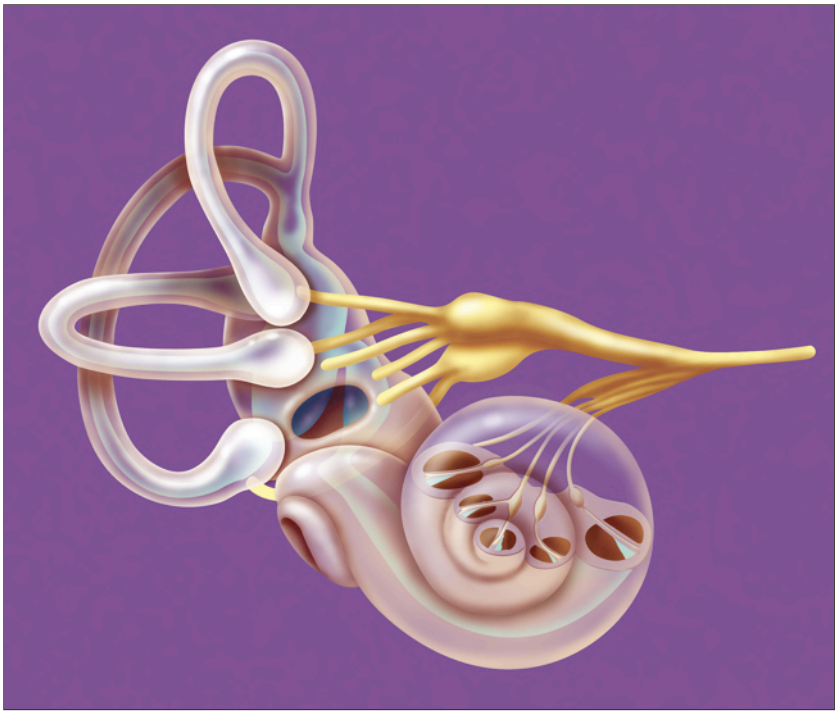

Figure 1. The inner ear showing a cross-sectional view of the cochlea

Most patients with vestibular causes of dizziness can now be treated with either drug therapy or vestibular physiotherapy. Mr Peter Rea discusses the various causes of dizziness and their appropriate management.

Dizziness or imbalance is experienced by 30 per cent of the UK population before the age of 65 and becomes the commonest reason for a visit to their GP for patients beyond 75 years of age. Recently published proposals from the Department of Health stress the importance of raising awareness of the prevalence of these disorders and the distress they can cause, while emphasising the role local balance services in the community will play in future care provision.

Medication is often useful, especially in the acute phase, but as prolonged drug therapy can slow or prevent recovery in certain vestibular conditions and many nonpharmacological treatments are available, cautious prescribing may be required.

Vestibular causes of dizziness

When assessing a patient with a vestibular disorder it is helpful clinically to consider three distinct groups of patients, as treatment and prescribing will be different for each: the acute episode, the recurrent acute episode and chronic vestibular symptoms.

The acute vestibular episode

Vestibular neuritis Acute vestibular neuritis (also referred to as vestibular neuronitis or viral labyrinthitis) is common and presents with a rapid onset of distressing rotational vertigo associated with nausea and vomiting. There are no associated neurologic or auditory symptoms. It can be terrifying and patients often believe they are having a stroke. Persistent horizontal-torsional jerk nystagmus is observed usually towards the unaffected ear (a paralytic nystagmus). The severe vertigo settles over days and most patients recover spontaneously over days or weeks. Those who fail to compensate are left with chronic vestibular symptoms.

Vestibular neuritis is believed to result most commonly from a viral inflammation of the superior division of the vestibular nerve. It can occur in clusters in the community ('epidemic labyrinthitis'). As the

nerve degenerates the end organ (the utricle) also degenerates: it is from here that the otoconia that cause benign paroxysmal positional vertigo (BPPV) are released and it is common for patients to report symptoms of vestibular neuritis months or years before the onset of BPPV.

Labyrinthitis Symptoms of labyrinthitis are those of vestibular neuritis but with additional acute hearing loss and tinnitus. Most cases are believed to be viral in origin and affect the membranous vestibular labyrinth and cochlea. Ischaemic events, particularly in the older person, may have similar results. Hearing loss is often permanent.

In contrast suppurative (bacterial) labyrinthitis is a severe invasive infection often associated with acute otitis media and sometimes meningitis. Hearing loss and vertigo are both severe.

Recurrent acute vertigo of vestibular origin

There are four classical vestibular disorders that cause recurrent vertiginous episodes, along with a host of less common conditions.

Benign paroxysmal positional vertigo

BPPV is the commonest cause of recurrent short-lasting vertigo. Over 2 per cent of people experience it at some time and its incidence increases with age. Intense unpleasant spinning lasting up to a minute is triggered by position change such as looking up, lying down, getting up from bed or rolling over in bed. It often occurs in clusters that are self-limiting. It may result from any inner ear damage, the commonest being head injury, previous vestibular neuritis and aging. Otoliths released from the injured utricle migrate to the posterior semicircular canal and their movement triggers vertigo. It is diagnosed on the Hallpike test where upbeat, torsional, geotropic nystagmus is observed.

1. Positional trigger	BPPV
2. Pressure induced	perilymph fistula, superior canal dehiscence syndrome
3. Postural trigger	postural hypotension
4. Phonophobia, photophobia	migraine
5. Psychological	hyperventilation, panic attack
6. Paroxysmia	vestibular paroxysmia
7. Periodic	perimenstrual symptoms

Table 1. Key questions in recurrent acute dizziness: the seven P's

Vestibular migraine A controversial but increasingly recognised cause of vertigo, vestibular migraine may affect 1 per cent of the population. Headaches occur in only 25-50 per cent of cases. The vertigo may last minutes, hours (usually) or even days. A family history of migraine is helpful and migraine aura diagnostic if present. Patients' balance is good between attacks but a history of motion sickness is common.

Ménière's disease has a prevalence as high as 0.2-0.5 per cent. Classical attacks are described as a build-up of unilateral tinnitus, aural fullness and hearing loss followed by severe vertigo associated with vomiting and diarrhoea lasting from 20 minutes to 10 hours. Hearing recovers after the attack to a variable degree. In practice Ménière's can present with only one or two of the classical symptoms making diagnosis challenging. Attacks occur in clusters and may be spaced over months or years. The aetiology remains obscure though some attacks seem to have an autoimmune basis.

Recurrent vestibular neuritis Recurrent vertigo attacks lasting hours or days are often very difficult to diagnose as the patient has no signs or symptoms when examined. Reactivation of a latent neurotropic virus, particularly the herpes simplex virus, is thought to be responsible for some cases. This might be similar to recurrent Bell's palsy or cold sores for example. Attacks seem to reduce in severity with time.

Other causes of recurrent dizziness There is a wide range of less common disorders. Enquiring about the seven 'P's' of recurrent vertigo may help distinguish between them (see Table 1). Vestibular paroxysmia is an interesting condition thought to arise from irritability of the vestibular nerve causing multiple very brief spins every day.

Chronic vestibular symptoms

The most common presentation in a balance clinic is of the chronically dizzy patient. Symptoms are varied and summarised in Table 2. Such symptoms usually result from inadequate central compensation to a peripheral vestibular deficit. Neurological and cardiovascular 'red flags' should be sought and referred appropriately. Examination of other systems that may be preventing recovery is helpful. In older people there may be multiple contributors, hence the term 'multisystem balance disorder' (see Figure 2).

Pharmacological treatment

Medication in vestibular disorders is usually palliative rather than curative, the exceptions being intratympanic gentamicin for Ménière's disease and antibiotics for suppurative labyrinthitis. Many drugs do, however, have a valuable role.

Treating the acute vestibular episode

A combination of vestibular suppressant and antiemetic is required for the distressing acute vestibular event.

Vestibular suppressants

Vestibular suppressants should be used only during the acute phase of the illness. Prolonged use may prevent central adaptation to a peripheral vestibular lesion and result in chronic vestibular symptoms. The benzodiazipines are among the more potent vestibular suppressants and diazepam is a good choice for an acute attack. As a gamma-aminobutyric acid (GABA; inhibitory neurotransmitter) agonist it is thought to have an inhibitory effect on the vestibular nuclei. Its anxiolytic action is an additional help.

Promethazine

Promethazine is an antihistaminic prothiazine and has significant antihistaminic and anticholinergic properties. It acts both as an effective antiemetic and mild vestibular sedative.

Cinnarizine

Cinnarizine is another useful vestibular suppressant with antihistaminic properties but also acts as a calcium-channel blocker and blocks 5-HT receptors, reducing vestibular nuclei activity. Although promoted for use in Ménière's disease it also has a role in the days following acute vestibular neuritis and in certain chronic vestibular conditions.

Dimenhydrinate

Dimenhydrinate is an antihistamine with vestibular suppressant and motion sickness suppression properties. It is not used frequently on its own in the UK, but a combination with cinnarizine (Arlevert) is a promising addition to our therapeutic options, particularly when used in the first days or weeks following an acute attack. It is popular in Europe.

cotton wool in head
walking on cotton wool
eyes not keeping up with brain when turning head
forgetfulness
fatigue
drunken feeling
mood change
neck ache
headache and facial pain

Table 2. The myriad symptoms of chronic vestibulopathy

Antiemetics

Prochlorperazine is an antidopaminergic phenothiazine that acts by blocking the chemoreceptor trigger zone centrally. It is the most commonly prescribed medication for acute vertiginous episodes in the UK. It is an effective antiemetic.

However, its prolonged use is discouraged for two reasons. Firstly, it may slow central adaptation to a vestibular injury. Many patients referred to the balance centre are taken off their prochlorperazine and it is only then that their vestibular rehabilitation physiotherapy will begin to work. Secondly, extrapyramidal side-effects are often seen with prolonged use.

Cyclizine (Valoid) is an antihistamine with anticholinergic activity helpful for acute vertigo. It also acts on central chemoreceptor trigger zones and is effective in controlling nausea, vomiting and vertigo.

Steroids The use of steroids in the management of acute vestibular neuritis is probably beneficial. A study[1] showed that a reducing dose of methylprednisolone (starting at 100mg on day 1 and reducing to 10mg by three weeks) produced a significant reduction in persistent vestibular weakness. While potential side-effects need to be discussed with patients, on balance the evidence suggests this should be considered for patients with acute vestibular neuritis. Antiviral medication, in contrast, was of no benefit.

Antibiotics If acute otitis media occurs in conjunction with vertigo antibiotics are required urgently and such patients should be referred for specialist management.

Treating recurrent acute vertigo of vestibular origin

BPPV

Medication plays no significant part in the management of BPPV as it is usually so easily treated with Epley manoeuvres (see Figure 3).

Vestibular migraine

Lifestyle and dietary alterations are often adequate in treating migraine. For those more severely affected the side-effect profile of the available drugs needs to be discussed with patients. For acute attacks analgesics and antiemetics are the mainstay of treatment, but triptans can be both helpful therapeutically and diagnostically if one is uncertain as to the cause of recurrent acute vertigo attacks.

Amitriptyline is often my first-line recommendation, or nortriptyline which is less sedating. Treatment starts at 10mg *nocte* for the first month working up in 10mg increments at monthly intervals. Some patients find it too sedating.

Propranolol is helpful, especially where anxiety is also a problem; however, cardiovascular and respiratory interactions may limit its use.

Pizotifen is an antihistamine and serotonin antagonist. It is an effective migraine prophylactic though weight gain can be a problem.

Verapamil is the most effective of the calcium-channel blockers for the management of migraine. Although less effective than the tricyclics and beta-blockers, it has the

advantage of often being well tolerated long term.

Acetazolamide, the carbonic anhydrase inhibitor, is also reported on occasion to be successful in migraine prophylaxis. It shares some features with topiramate (an anticonvulsant), which is more regularly recommended.

Ménière's disease

Lifestyle and dietary modifications are also the first-line treatment for Ménière's, in particular a low salt diet aiming for 2.5g of sodium per day. Although evidence in the literature is not strong, this is often recommended.

Betahistine is usually the first drug recommended in the UK. Its mechanism of action is unclear, but it is felt to act as a vasodilator on the stria vascularis of the inner ear, which may reduce hydrops. It has some vestibular sedative properties. It is extremely well tolerated. While many patients report little benefit, there is no doubt many feel a rapid return of symptoms if they stop it.

Bendroflumethiazide is usually the second-line therapy, especially if a low salt diet is difficult. It is thought to work by reducing the sodium load and thus reducing hydrops in the labyrinth. Again, evidence is sparse. Other diuretics may also be recommended.

Diuretics are particularly helpful for women whose symptoms are exacerbated with their menstrual period, and I recommend bendroflumethiazide for the five days before their period to reduce the fluid load in the labyrinth.

Urea is an interesting medication. It tastes absolutely foul and patients are rather reluctant to drink 'concentrated urine' despite assurances that it is entirely synthetic; 25mg dissolved in orange juice and knocked back quickly is just about bearable. It does have a place, though, for Ménière's patients: they are almost guaranteed 24 hours free from attacks after taking it as it dehydrates the labyrinth. Consider it before important exams or weddings (and there can be no greater confirmation of devotion). A test dose before the big day is recommended.

Steroids have a place in the management of both Ménière's disease and autoimmune inner ear disease, the symptoms of which can overlap. They may be given orally as prophylaxis but have gained popularity when given via a transtympanic route with a spinal needle under local anaesthetic. The treatment is very well tolerated (I have never had anyone not complete the course) and may give long periods free from attack. Dexamethasone is preferred for injection.

Gentamicin, the aminoglycoside antibiotic, has revolutionised the management of severe Ménière's disease. It is selectively vestibulotoxic and is also administered via a transtympanic route in clinic.

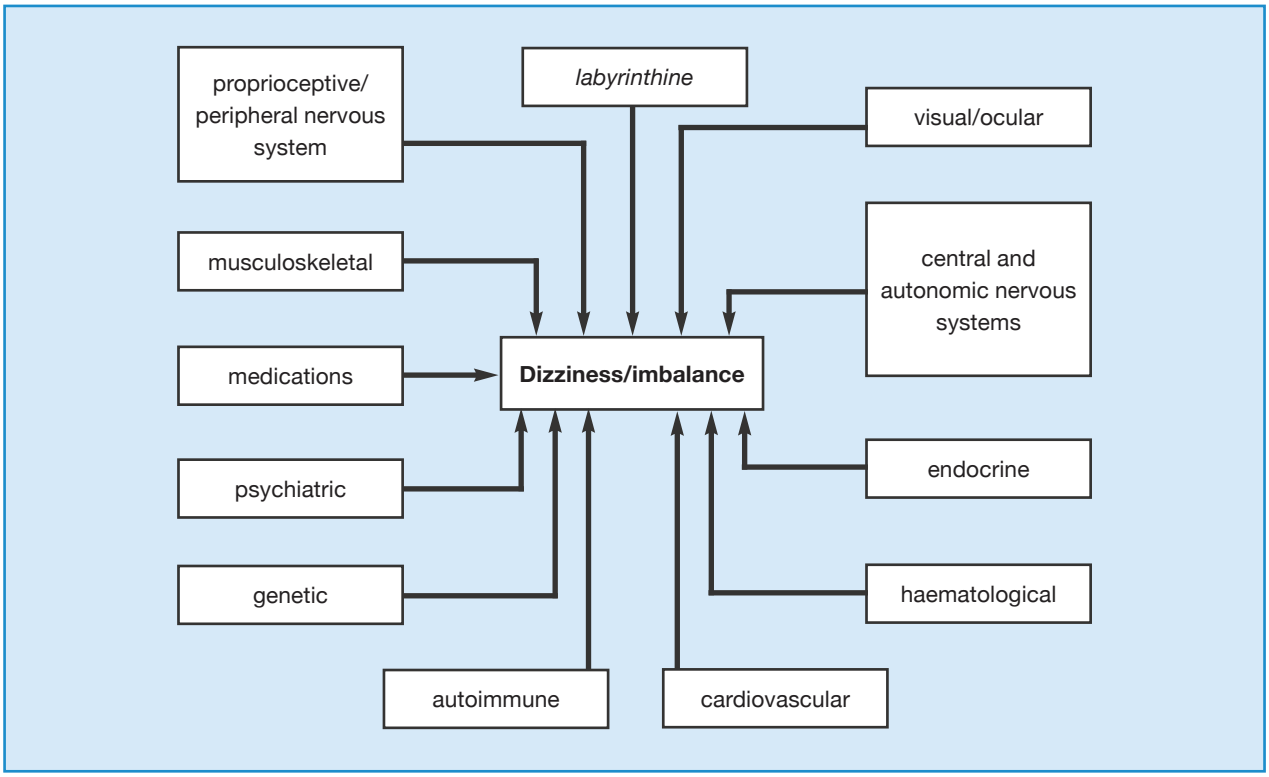

Figure 2. Systems that can affect balance

Reduction in vertigo is observed in 96 per cent of patients and permanent abolition of vertigo in 80 per cent; 5 per cent lose all the hearing in the treated ear (though this is often very poor already), and a small number experience imbalance for a while. This treatment has transformed the lives of many of my patients.

Cinnarizine or *Arlevert* is sometimes prescribed to help patients through clusters of attacks.

Recurrent vestibular neuritis

As this condition is thought to have a reactivated virus as its aetiology there is a potential advantage in prescribing a short course of prednisolone and aciclovir at the onset of symptoms. It is a treatment I use only occasionally and the evidence is in part theoretical.

Rare conditions

Carbamazepine, an anticonvulsant, is used to treat vestibular paroxysmia as it appears to stabilise the irritable vestibular nerve.

Acetazolamide is particularly interesting as it can be a very effective treatment for familial episodic ataxia type II – although perhaps more cerebellar than vestibular, it is seen in the balance centre as it shares features with migraine and Ménière's and can be a rewarding condition to diagnose and treat.

Treating chronic vestibular symptoms

Medication plays a much smaller role in chronic vestibular symptoms, in part for fear of preventing central adaptation.

Prochlorperazine has no significant place in the long-term management of chronic vestibular deficits.

Hyoscine hydrobromide is effective in preventing motion sickness, though a recent Cochrane review was unable to compare it to other motion sickness medications such as antihistamines. There is no good evidence to support its use in established motion sickness syndromes.

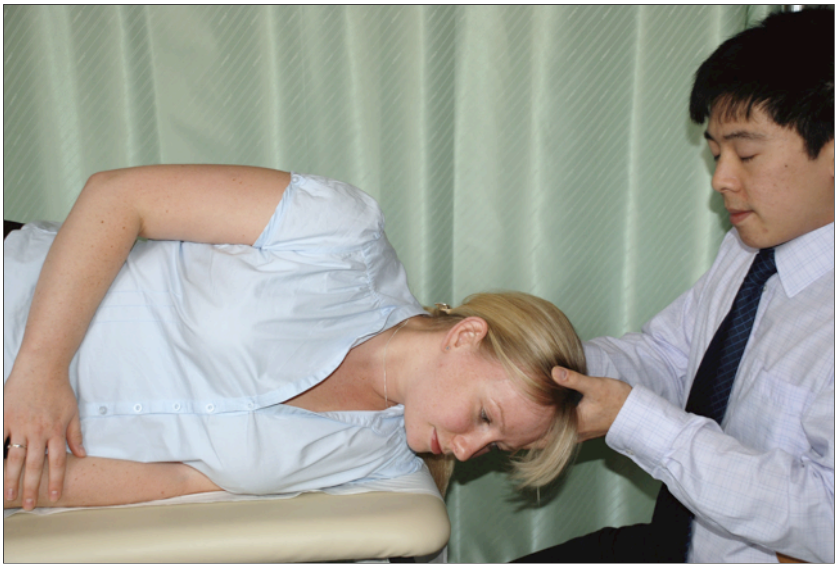

Figure 3. The Epley manoeuvre is used to treat benign paroxysmal positional vertigo. The head is held in specific positions on the opposite side to that which elicits symptoms.

Cinnarizine or *Arlevert* is sometimes used to help patients through exacerbations of their chronic symptoms.

Many patients with chronic vestibulopathies have a significant emotional reaction to their illness – indeed this is almost universal. If anxiety and panic disorder are prominent and psychological interventions are unsuccessful, I sometimes use a short course of the benzodiazepine clonazepam in very low dose (this is off licence, however, and careful explanation is required), which also helps settle their sleep pattern and anxiety as well as being a vestibular sedative. Lorazepam or diazepam might be considered too.

SSRIs such as citalopram are often helpful but as an ENT surgeon I do not feel it appropriate to prescribe these myself.

Conclusion

A wide variety of drug classes have been discussed that may help patients with vestibular disorders and all have their place. In many cases, however, vestibular physiotherapy or particle repositioning manouvres will be the mainstay of treatment. In selected cases surgery can be curative. With this combination of therapies now available, most patients with vestibular disorders can be successfully managed.

Further reading

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Reference

1. Strupp M, *et al. NEJM* 2004;351 (4):354-61.

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