

A systematic review of vertigo in primary care

Karena Hanley, Tom O' Dowd and Niall Considine

SUMMARY

The symptom of vertigo is usually managed in primary care without further referral. This review examines the evidence on which general practitioners can base clinical diagnosis and management of this relatively common complaint. Research in this area has in the main been from secondary and tertiary centres and has been of variable quality. Indications are that the conditions that present in general practice are most likely to be benign positional vertigo, acute vestibular neuronitis, and Ménière's disease; however, vascular incidents and neurological causes, such as multiple sclerosis, must be kept in mind. An important practice point is that vestibular sedatives are not recommended on a prolonged basis for any type of vertigo. There is a need for basic epidemiological and clinical management research of vertigo in general practice.

Keywords: vertigo; vestibular disorders; diagnosis; disease management.

Introduction

DIZZINESS is a common complaint in general practice¹ and has been described as 'confusing' and 'discouraging' by GPs.² A subgroup of those with dizziness complain of vertigo which is defined as an illusion or hallucination of movement, usually rotation, either of oneself or the environment.^{3,4} Vertiginous syndromes account for 10.7 consultations per 1000 person years in general practice morbidity statistics.⁵ Our literature review indicates that it is possible to classify the types of vertigo that present in general practice.

Such classification has advantages for the patient in that a better explanation is likely to allay patient anxiety and psychological sequelae, which are common in chronic vestibular disorders.⁶ More treatment options are available for recurrent vertigo with growing use of vestibular rehabilitation⁷⁻⁹ and specific 'particle repositioning' therapies.¹⁰⁻¹³

Method

Ovid and Silverplatter Medline and the Cochrane databases were searched using the keyword 'vertigo' with the MeSH terms of 'classification', 'prevention and control', 'epidemiology', 'diagnosis' and 'management'. The key word 'dizziness' was also searched in combination with 'general practice' or 'family medicine' or 'primary care'. No limit on year or language of publication was used. From over a thousand references, 200 abstracts were read and 59 articles were retrieved on the basis of applicability to vertigo in general practice. The citations of all papers obtained were examined for further articles of interest. Thirty-five were original articles with methodologies as follows: four were case control studies,¹⁴⁻¹⁷ four were prospective cohort studies,^{2,4,18,19} and eight were prospective surveys.^{1,19-25} Eight were retrospective surveys,^{13,26-32} and eight were case series.¹⁰⁻¹² There were three placebo-controlled trials of treatment, of which two were randomised^{9,38} and one was a crossover trial.³⁹ Seventeen review articles relating to the subject were retrieved, most of which were clinical reviews. Only one was a systematic review⁴⁰ but two others approached the standards of a systematic review.^{41,42} Five studies were drawn from a general practice population.^{2,9,18,21,32,33} All the papers were read and criticised by one author (KH). No papers were rejected, since the evidence base by which vertigo can be assessed in primary care is very limited; however, the more pertinent articles are summarised in Table 1.

Distinguishing vertigo from other causes of dizziness

Drachman and Hart³⁶ first described a 'complaint-orientated' approach to the patient's symptoms, by categorising dizziness into: pre-syncope, disequilibrium, lightheadedness or vertigo. One hundred and twenty-five patients attending a

K Hanley, MRCP, GP principal, Rathmullen, Co. Donegal, Ireland.
T O' Dowd, FRCP, professor of general practice, Trinity College, Dublin, Ireland. N Considine, FRCSI consultant ENT surgeon, Sligo General Hospital, Ireland.

Address for correspondence

Dr Karena Hanley, The Mall, Ramelton, Co. Donegal, Republic of Ireland. E-mail: nunan@gofree.indigo.ie

Submitted: 23 May 2000; Editor's response: 3 October 2000; final acceptance: 6 March 2001.

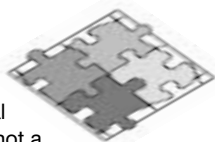
©British Journal of General Practice, 2001, 51, 666-671.

HOW THIS FITS IN*What do we know?*

Vertigo is a relatively common general practice complaint for which there is not a large evidence base to guide the management.

What does this paper add?

This systematic review documents the causes that doctors should consider in a community presentation of this symptom. Treatment options are given for the more common conditions.



dizziness clinic were used to test this approach. It has since been used to classify symptoms in research.^{15-19,21,24,25,32}

Pre-syncope is the sensation of impending loss of consciousness. It is usually caused by a decrease in global cerebral blood flow. Cardiovascular disorders, peripheral neuropathy, hyperventilation, postural hypotension, and vasovagal reactions are common causes. Carotid sinus hypersensitivity with either vasodepressor or cardioinhibitory effects is emerging as a more important cause of dizziness in the elderly, especially if associated with falls or dizziness.⁴³

Disequilibrium, or postural unsteadiness, is a sense of imbalance not strictly associated with motion. It usually occurs while standing and is often made worse by walking. It arises when the brain is processing less information about the body's position in space. Conditions which can produce disequilibrium include decreased lower limb strength (e.g. pseudoparkinsonism) peripheral neuropathy, visual loss,

and poorly compensated peripheral vestibular disorders.²⁹

Lightheadedness, also termed 'giddiness' or 'wooziness' has no clear definition and no clear associated diagnosis.²⁵

A question which has been validated for detecting whether vertigo is present or not is as follows¹⁴: 'When you have dizzy spells, do you just feel lightheaded or do you see the world spin around you as if you had just got off a playground roundabout?'

Confirmation of vertigo as a rotatory illusion significantly predicts a peripheral vestibular disorder ($P < 0.001$)¹⁶ especially if nausea and/or vomiting coexist.²⁵ A number of community-based studies of dizziness indicate the distribution of dizziness into the above four categories. While different proportions were found of the other types of dizziness, the proportion with vertigo was more uniform. Specifically, it formed 28%,¹⁵ 29%,²¹ and 32%^{17,24,32} of presentations in five studies. One study had more presentations of dizziness defined as vertigo at 56.4%,²² but this was in an older population.

Descriptions of the common causes of vertigo

In 1952, Dix and Hallpike declared the probable major causes of vertigo as being owing to Ménière's disease, benign positional vertigo, and vestibular neuronitis. The statement was based on their clinical experience and this case series gave robust descriptions of the clinical and pathological features of these three conditions.³⁵

No research has sought to establish the causes of true vertigo on first presentation. Clues to what may be underlying

Table 1. Primary care management of vertigo. The following studies have been chosen as the best available evidence on which to base primary care diagnosis and management of vertigo.

Trial	Area	Method and setting	Number of subjects	Comments
Yardley (1992) ²⁰	Clinical features of vertigo	Prospective survey in a tertiary centre	171	Validates a vertigo symptom scale to quantify severity. Useful for future research.
Kentala (1996) ²³	Clinical features of vertigo	Prospective survey in a university centre	564	Good description of clinical features of six diagnoses. Useful study, but selected study population.
Lempert (1997) ³⁹	Aetiology of BPV	Open trial plus single blind crossover trial in a tertiary centre	30	Physiological experiment confirming canalithiasis theory.
Froehling <i>et al</i> (1991) ²⁸	Clinical features of BPV	Retrospective survey in primary care	53	Five year follow-up showed no increased risk of stroke or death. Excludes 42% owing to insufficient documentation.
Baloh <i>et al</i> (1987) ³¹	Clinical features of BPV	Retrospective survey in tertiary centre	240	Method poorly described. Establishes a possible cause for BPV in half. Selection bias a problem.
Brill (1982) ³³	Clinical features of AVN	Case series from general practice	50	Substantiates link with previous infection. Weakened by poor definition of AVN.
Grad <i>et al</i> (1989) ³⁰	Clinical features of vertigo of vascular origin	Retrospective survey	84	Method poorly described. Vertigo of vascular origin lasts minutes (TIA) or hours/days (CVA).
Blakely (1994) ³⁸	Treatment of BPV	Prospective RCT in university centre	38	Good methodology but small numbers. One manoeuvre no different from no treatment.
Yardley <i>et al</i> (1998) ⁹	Treatment of dizziness and vertigo	Prospective RCT in general practice	143	Symptomatic improvement demonstrated. Subjects were those with unspecified dizziness making the study population quite general.
Grimley Evans (1990) ⁴⁴	Prognosis of vertigo in the elderly community	Cohort study in a community sample	2025	Suggests vertigo is a risk factor for stroke in the over-65-years age group. Good method but concentrates on detecting TIA symptoms.

BPV = benign positional vertigo; AVN = acute vestibular neuronitis; RCT = randomised controlled trial.

ing come from studies from secondary and tertiary centres that allude to diagnoses found. The proportions of conditions quoted are as follows: vestibular neuronitis (ranging from 10–44% of diagnoses), Ménière's disease (ranging from 17–43%), benign positional vertigo (ranging from 10–27%).^{19, 23, 27, 29, 45}

A clear picture of presenting patterns is also hampered by lack of consensus on diagnostic terminology. For example, in Dix and Hallpike's seminal paper on the condition, the term vestibular neuronitis was chosen owing to the physiological experiments of galvanic testing on the integrity of Scarpa's ganglion and of caloric stimulation on over 100 patients. Their findings, although uncontrolled, led to the conclusion that an organic lesion of the vestibular nervous pathways central to the labyrinth and up to and including the vestibular nuclei³⁵ is causative. However, the terms 'acute/viral labyrinthitis', 'epidemic vertigo' and 'vestibular neuritis'^{36,46} are used interchangeably although pleas continue for uniform nomenclature.⁴¹ Similarly, BPV is sometimes termed benign positional paroxysmal vertigo or nystagmus (BPPV/BPPN) and Ménière's disease is sometimes termed Ménière's syndrome.

A further problem is that for two of the likely three most common diagnoses there is no 'gold standard' of diagnosis. Benign positional vertigo and vestibular neuronitis are clinical diagnoses. They have characteristic findings on history and clinical examination and they do follow a predictable clinical course, but there is no confirmatory investigation of sufficient sensitivity or specificity for either condition. For most other causes of vertigo, including Ménière's disease, neurological or vascular causes, there are definitive investigations.

Descriptions follow of these clinical syndromes and three other categories to consider when vertigo presents; infective, vascular and neurological causes of vertigo.

Vestibular neuronitis

A recent review describes this common condition as occurring in a previously well, young, or middle-aged adult.⁴¹ An association between vestibular neuronitis and preceding or concurrent infectious illness was first postulated by Dix and Hallpike, who found evidence or a history of infection in 57% of cases³⁵ which has since been confirmed in general practice.³³ Sinusitis, influenza, and upper respiratory tract viral illnesses are the most likely precipitants.⁴¹ The causative lesion is thought to be isolated degeneration of the vestibular nerve or its connections. This would explain why there is neither hearing loss nor wider brainstem involvement.

Onset of vertigo commonly occurs on first awakening; however, a minority of patients have a more gradual onset. Nausea is marked and is almost universal, vomiting occurs in half of cases, and unsteadiness can be pronounced.³³

Examination may show a fine horizontal or rotatory nystagmus on gaze and an unsteady gait. In half of patients, the underlying nerve damage recovers within two months,⁴⁷ but as vestibular compensation occurs, the patient's vertigo symptoms usually resolve slowly over a few days.⁴² However, the sensation of disequilibrium may persist for longer. In some patients, attacks of vertigo recur over days or weeks reflecting interference with compensation. If the attacks are not sequentially shorter than another diagnosis

must be considered.⁴¹ The diagnosis of vestibular neuronitis can be correctly made on the basis of sufficient clinical history.²³ Reassurance, explanation, and advice are essential, together with symptomatic treatment only in the first few days,⁴² as vestibular suppressant drugs delay compensation. Prochlorperazine has been stated as the best agent, but on what basis this is recommended is unclear.⁴¹ Prognosis is usually excellent, but development to benign positional vertigo after an attack of vestibular neuronitis is well described; this occurred in 15% in one series.³¹

Benign positional vertigo

This condition causes recurrent bouts of vertigo brought on by changes in head position. A probable cause can be identified in about half of cases, such as viral neuronitis, surgery, infection, vasculitis, trauma, where onset is within three days of head injury,^{31,37,48} vertebro-basilar migraine or drug-induced ototoxicity.³¹ Two theories of pathology exist. It is postulated in the canalithiasis theory^{10,13} that otoliths may have migrated from the utricle, through the long arm of the posterior semicircular canal until they reach the cupula. The corrective manoeuvre of Epley¹¹ aims to empty the semicircular canal of this debris. The cupulolithiasis theory suggests that otoliths have migrated through the other opening of the semicircular canal, through the short arm, adhering to the utricular side of the cupula.¹³ The Semont manoeuvre was developed to treat this. Work in a frog model has supported both theories¹⁰ and physiological experiments lend weight to the canalithiasis theory.⁴⁹

Onset of symptoms is most commonly in the sixth decade. The most quoted estimate^{10,39} of the incidence of BPV at 0.64 per 1000 population comes from a retrospective record review with admitted poor documentation²⁸ and is probably not a reliable estimation. Females outnumber males 2:1.³¹ Episodes of vertigo are typically induced by turning over in bed, bending over and straightening up, or extending the neck to look up.³¹ Individual episodes usually last seconds, never more than five minutes, but may be severe enough to require the patient to stop whatever they are doing.²³ Nausea may be present, but vomiting is rare. Related loss of hearing, tinnitus or a feeling of 'fullness' in the ears, if present, suggest another diagnosis, usually Ménière's disease. Typically, bouts of vertigo occur with variable periods of remission.³¹ Usually the only abnormal sign is a positive Hallpike's manoeuvre (Box 1), found in about half of patients at presentation.²⁸

Although vestibular sedatives are commonly prescribed for all types of vertigo, including BPV, it is now recommended that they should be avoided where the vertigo becomes chronic as they suppress vestibular feedback crucial for the development of compensation and symptomatic recovery.^{7,42,50} More use should be made of a series of exercises aimed at encouraging eye, head, and body movements to facilitate recalibration of the vestibulo-spinal and vestibulo-ocular reflexes that have been developed, termed vestibular rehabilitation.⁹ These are based on therapy developed by Cawthorne and Cooksey in 1945⁸ and are designed to treat poorly compensated vestibular dysfunction of whatever cause.⁷ They have been tested in general practice⁹ where they have been shown to be effective for other types of dizzi-

- Warn before starting that vertigo may recur. Arrange patient such that when recumbent on couch the head will hang over the top of the couch.
- With head rotated 45° to one side, lie patient back rapidly until head is dependent.
- Nystagmus may take between two and 20 seconds to appear, but usually it is two seconds. Will last 20–40 seconds.
- Wait 30 seconds for negative test.
- Repeat on opposite side. The side of nystagmus is the side of the lesion, which may be bilateral.

Box 1. Synopsis of Hallpike manoeuvre.

ness as well as persisting vertigo. The trials on the Epley and Semont repositioning manoeuvres yield mixed results.¹⁰⁻¹² The only placebo-controlled trial of Epley's manoeuvre showed no significant difference in outcome but had small numbers.³⁸ Nor is there yet consensus on the precise positioning, timing, and post-manoeuve patient advice for the above repositioning exercises.^{10,12,13,38} Brandt and Daroff exercises (Box 2) are simpler repositioning exercises that have been suggested for less severe BPV⁵¹ and in the original series achieved complete relief within 3 to 14 days.⁵²

Ménière's disease

In 1861, Prosper Ménière first described the triad of fluctuating hearing loss, tinnitus, and episodic vertigo.⁴⁰ To this is often added a sensation of fullness or pressure in the ear.^{3,48,53}

The main pathology is an increase in the volume of the endolymph leading to pressure within the semicircular canals, according to postmortem studies in 1938.⁵⁴ Why this occurs is not clear but several factors, including immunological, viral, vascular and genetic, have been postulated.⁴⁰ The episodes of hearing loss and vertigo seem to be caused when the pressure mounts, either worsening the distortion or rupturing the membrane that separates the endolymph from the perilymph.³ Recovery occurs as endolymphatic pressure falls. The prevalence has been estimated at 1 per 1000 of the population⁴⁰ with onset of symptoms usually occurring between 20 and 50 years. Men are affected slightly more than women.⁴⁸ There is a familial predisposition with a variety of Ménière's disease that is autosomal dominant, accounting for about 7% of cases. Symptoms are unilateral in 80% of cases but, with longer follow-up, an increase in those with bilateral disease is observed.⁴⁰

The attacks, lasting for hours, are intense, often necessitating bed rest; however, nausea and tinnitus are moderate. Sudden slips or falls are common, as is headache, with hearing loss worsened during an attack.²³ Three stages in the course of the disease are recognised clinically. In stage 1 the predominant symptom is vertigo with associated deafness, but hearing is normal between attacks. Hearing loss becomes established at stage 2 but continues to fluctuate. The attacks of vertigo reach their most severe, and then diminish. The periods of remission between attacks can be very variable. Finally, the hearing loss stops fluctuating and becomes progressively worse. The episodes of vertigo fade⁴⁰ leaving a residual sensori-neural hearing loss.⁴⁸

During an attack the patient may show rotatory nystag-

- Sit the patient on the side of the couch with eyes closed.
- Tilt the whole upper body laterally to the side of the lesion until the lateral aspect of the occiput rests on the bed. This position is maintained until the evoked vertigo subsides.
- Sit the patient upright again for 30 seconds.
- Tilt the body to the opposite and maintain the head on the bed for 30 seconds.
- Repeat every three hours during the day.

Box 2. Brandt-Daroff exercises for benign positional vertigo.

mus, while between attacks examination may be normal in the early course of the illness, with unilateral deafness as the disease progresses. The American Academy of Otolaryngology diagnostic guidelines stipulate at least two spontaneous episodes of rotational vertigo lasting at least 20 minutes, audiometric confirmation of a sensorineural hearing loss, plus tinnitus and/or perception of aural fullness.^{40,55} Audiometric tests show a recruiting sensori-neural hearing loss.⁴⁰ If the diagnosis is in doubt an oral dose of glycerol leads to an improvement in the audiometric response. Referral to an ENT specialist has been recommended for every case where vertigo is associated with hearing loss to exclude the possibility of an acoustic neuroma.²³

Treatment is symptomatic as no agent has yet been shown to alter the course of the illness. Prochlorperazine, promethazine, and diazepam can be used to treat the acute attacks. Data from controlled trials are conflicting on the use of dietary salt restriction or diuretics. Significant improvement in the short term has been shown for the use of betahistine and this, with or without a diuretic, is the favoured treatment currently.⁴⁰ A Cochrane systematic review is currently assessing the effects of betahistine.⁵⁵ Cinnarizine, propranolol (especially where there coexists a history of migraine), and corticosteroids are sometimes used for refractory cases. There are a number of surgical options for the minority of patients with continued symptoms.

Local infective causes of vertigo

Otitis media, chronic otomastoiditis or cholesteatoma can be a cause of vertigo¹⁸ through extension into acute labyrinthitis. Other infective causes are also rare and include mumps, syphilis, Ramsay-Hunt syndrome, and tuberculosis.³ It has been recommended that the term acute labyrinthitis be reserved for specific viral and bacterial infections.⁴¹ Clinically, hearing loss and/or tinnitus accompany vertigo.

Vascular causes of vertigo

The blood supply to the inner ear, brainstem, and cerebellum originates from the vertebrobasilar system.³ Ischaemia within the distribution of the cerebellar arteries can cause vertigo. In a review of 84 cases of vertigo of vascular origin,³⁰ isolated vertigo was the first symptom in 24% of cases, with associated brainstem neurological features usually following within a couple of months. It is recommended to seek associated evidence, such as visual disturbance, dysarthria, and drop attacks in making the diagnosis of vertebro-basilar

insufficiency.^{8,30} Where vertigo has been associated with diplopia this is likely to be a vascular event, at least a transient ischaemic attack, which predisposes the patient to an increased risk of stroke.¹⁴ Central vascular disease, however, is likely to be the commonest cause of dizziness that has not been categorised as vertigo.¹⁷

Vertigo can rarely be an isolated manifestation of migraine, but patients will probably have migraine headaches at other times.³ Basilar migraine, which is most common in adolescent girls, also causes vertigo, along with diplopia, ataxia, and dysarthria.

Neurological causes of vertigo

Lesions in the brainstem, cerebellum, thalamus or cortex can cause vertigo, but all are rare and are likely to have other associated neurological features. Central causes have less nausea and vomiting, but more pronounced imbalance than peripheral causes.³ Nystagmus was part of the classic triad of Charcot in multiple sclerosis⁴⁸ where vertigo is usually one of several neurological symptoms. Vertigo can be associated with epilepsy, usually in the aura phase of a fit. Unilateral progressive nerve deafness is the presenting symptom in 95% of patients with acoustic neuroma, tinnitus is present in most, and vertigo in about half.²³ Toxins that affect the vestibular system include alcohol, and drugs such as aminoglycosides, salicylates, and frusemide but, as damage is usually gradual and bilaterally symmetrical, symptoms of vertigo are usually minimal.⁴⁸ The latter two drugs are more likely to produce deafness.⁴²

Finally, anxiety can be a cause of dizziness.²⁵ Anxiety may also be a product of vertigo of any cause²⁰ and worsen associated autonomic symptomatology, but there is no agreement for anxiety as an actual cause of vertigo.^{20,34}

Future research

There is little research in primary care on this relatively common condition. It is not known what types of vertigo commonly present in general practice or how they are managed. We do not know what proportion suffering vertigo are referred on to a specialist, but less than 10% of patients with dizziness are referred.¹⁸ An attempt has been made to develop guidelines for referral;³² however, these are from a specialist viewpoint only with no input from GPs. More specific managements, such as rehabilitation exercises or repositioning treatments for chronic vertigo, may be applicable in primary care but require more evaluation.

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