

Vertigo: A practical approach to diagnosis and treatment

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Background. Vertigo is a symptom that has diverse causes. The diagnosis may remain elusive even after exhaustive clinical enquiry and there is often also a general perception that little can be done in many cases apart from prescribing vestibular suppressant drug therapy. Many patients are therefore treated symptomatically without a specific diagnosis being made.

Objective. To discuss the diagnosis and management of the more common vestibular syndromes.

Discussion. Many conditions such as benign positional vertigo and migraine can be successfully diagnosed and treated without the need for referral. Exercise therapy rather than chronic drug use should be the mainstay of treatment in patients with 'uncompensated' vestibular lesions. This article provides a practical approach to vertigo that can lead to a successful clinical outcome in many cases.

The assessment of patients with vertigo can be one of the most challenging clinical problems. Vertigo is defined as an illusion of movement. Sensations of spinning are often described, which result from involvement of the semicircular canals. Linear sensations of falling, tilting and sudden dropping are also valid descriptors of vertigo and probably reflect involvement of the otolith organs (utricle and saccule) which sense linear motion.

A sudden unilateral loss of vestibular function causes acute, severe vertigo that persists for hours to days. Even when the underlying vestibular deficit is permanent, resolution of the severe debilitating symptoms occurs due to equilibration of the tonus in the brainstem vestibular nuclei via the CNS process known as compensation. During this recovery period, patients frequently complain of short-lived episodes of motion-induced vertigo. Persistent motion-induced symptoms often respond to exercise therapy or specific vestibular rehabilitation programs, which are designed to promote compensation. In contrast, recurrent *spontaneous* episodes of vertigo reflect an unstable vestibular lesion that demands specific medical or surgical management.

Clinical assessment

During the clinical assessment of patients presenting with dizziness, the answers to the following questions should be sought routinely:

- Does the patient have vertigo? The differential diagnosis includes many conditions including postural hypotension, hyperventilation and pre-syncope.
- If the patient has vertigo, is it the result of peripheral (labyrinthine) or central (CNS) pathology?

The answer to the first question is usually self evident, although many patients do not give a clearcut history which is diagnostic of vertigo. Dizziness which is aggravated by head movements rather than postural changes alone, usually reflects an underlying vestibular problem. The vast majority of cases of vertigo are peripheral in nature, but it is important to exclude the potentially more serious central causes (Table 1).

Table 1. Causes of vertigo

Peripheral	Central
Vestibular neuronitis	Tumours
Labyrinthitis	Vascular disease
Ménière's disease	Multiple sclerosis
Benign positional vertigo	Migraine *
Trauma	Trauma

* End organ or peripheral involvement can be seen uncommonly as well.

Length of attacks

The duration of attacks of vertigo is also of great diagnostic importance. Attacks lasting for seconds only, particularly if they are associated with head movements or postural changes, are highly suggestive of either, an uncompensated peripheral vestibular lesion, or benign positional vertigo. Vertigo that is triggered by lying down or rolling in bed is so distinctive that it should be assumed to be due to benign positional vertigo until proven otherwise. Episodes lasting minutes can be due to migraine or vertebro-basilar ischaemia, the latter being an uncommon condition. The often quoted mechanism of vertebral artery occlusion triggered by neck extension or rotation rarely causes clinical symptoms. Most patients with vertigo triggered by head extension have peripheral labyrinthine pathology such as benign positional vertigo.

Vertigo lasting hours occurs in conditions such as **Ménière's disease**. **Vestibular neuronitis** (isolated vertigo) and **labyrinthitis** (vertigo plus auditory symptoms) typically cause vertigo that develops over several hours and gradually resolves over a period of days to weeks.

Examination

A neurological examination to elicit focal neurological signs is obviously important to exclude CNS causes, but particular emphasis is placed on assessment of standing and walking balance. Careful examination of eye movements is of importance to look for nystagmus and other eye movement abnormalities. Assessment of hearing, tuning fork tests and otoscopy should also be performed routinely.

A Hallpike test should be carried out on every patient presenting with vertigo. With the head tipped back and rotated 45 degrees to one side, the patient is moved briskly to the supine

position so that the head remains at approximately 45 degrees below the horizontal (positioned either over the end of the bed or a pillow). The patient is instructed to report any vertigo and to keep the line of gaze stable with eyes open. The nystagmus, if present, is a mixed upbeating and torsional nystagmus that may not appear for up to 5 seconds or more. Once any dizziness has settled the patient is moved quickly to the sitting position with the head kept in the same plane. The nystagmus may reverse in this position. The manoeuvre is then repeated with the head turned 45 degrees in the opposite direction. It is surprising how often, even without a classic presenting history, this manoeuvre is positive. A positive test will be associated with moderately severe vertigo and should be accompanied by nystagmus that:

- has a mixed torsional (rotatory) and upbeating quality;
- begins after a short latent period;
- fatigues up to 30 seconds later; and
- is often less marked with repeated manoeuvres (habituation).

Nystagmus that does not have all of these characteristics may reflect involvement of other semicircular canals but can also be a rare presenting feature of central lesions in the posterior fossa.

A negative Hallpike test (absence of nystagmus) makes benign positional vertigo an unlikely diagnosis. Occasional patients may have a positive test on subsequent visits, hence it is important to repeat the test if there is a high index of suspicion regarding this diagnosis. Not all positional vertigo is the result of benign positional vertigo. Patients with other labyrinthine disorders may feel dizzy during the Hallpike manoeuvre. The absence of associated nystagmus makes this a non-specific vestibular finding.

Investigations

➤ A CT brain scan should be performed if central pathology is suspected, bearing in mind that some pathology, such as demyelination and ischaemia, may only be seen with magnetic resonance imaging. Generally speaking, temporal bone CT views are not indicated unless there are auditory symptoms suggestive of specific temporal bone pathology.

- Audiometry should be used to document the presence and patterns of hearing loss.
- Brainstem auditory evoked potentials are useful in assessing eighth nerve and brainstem pathology.
- The results of other vestibular investigations, such as caloric and rotational chair

testing, need to be interpreted in the light of the clinical presentation and should usually be ordered only after specialist evaluation.

Common causes

Benign positional vertigo

This is one of the commonest vestibular conditions, accounting for over 25% of patients presenting with peripheral vestibular disorders. As outlined above, the presentation is distinctive. Otoconial deposits or crystals, arising from the utricular matrix, form a heavy mass in the posterior semicircular canal and, in response to a provocative head movement, result in excessive displacement of the sensory organ, or cupula, via a plunger effect.

Benign positional vertigo should be regarded as a syndrome which can occur either without any obvious cause or as a secondary feature of other conditions, particularly head trauma, vestibular neuronitis and Ménière's disease. The condition can occur at all ages, although it is more common with advancing age. The pathology is mechanical and is therefore usually unresponsive to drug therapy. However, in severe cases, the associated nausea and other vegetative symptoms may be alleviated by vestibular suppressant drugs such as prochlorperazine and promethazine. In general, such drugs should only be used for short term treatment of severe, acute vertigo.

In many cases the condition is self limiting, but sometimes the condition may persist for several years without treatment. A home exercise program (Brandt-Daroff exercises, see Patient information sheet later on) will often provide relief after several days but it must be emphasised to patients that the symptoms will need to be provoked during these exercises for subsequent improvement to occur. These exercises probably work by flushing out the otoconial deposits, which are either reabsorbed or displaced to other parts of the labyrinth.

Alternatively, single treatment manoeuvres (Epley and Semont) can be used and have similar success rates. These techniques take some practice to master. The interested reader is referred to a discussion and step by step instruction of these techniques. These manoeuvres and the Brandt-Daroff exercises are curative in more than 90% of cases. Surgery is rarely required.

Other causes of vertigo

Migraine

Migraine is a common cause of vertigo, particularly in younger age groups. It can occur as an aura preceding a migraine headache, or even as an isolated phenomenon (benign recurrent vertigo). Rarely, patients can present with chronic fluctuating spontaneous and positional vertigo, punctuated by intermittent headaches. Prophylactic anti-migraine therapy (eg, pizotifen, propranolol or verapamil) can lead to a dramatic relief of all of these symptoms in some cases. In otherwise unexplained cases of recurrent vertigo, a trial of prophylactic migraine therapy is

often indicated, particularly if there is a history of headache.

Ménière's disease

Ménière's disease or endolymphatic hydrops, classically presents with a history of recurrent vertigo accompanied by fluctuating auditory symptoms (tinnitus, hearing loss and aural fullness). The hearing can recover between attacks but there is often a stepwise or gradual loss of hearing. Vestibular hydrops (recurrent vertigo without auditory accompaniments) has been described but, in the author's opinion, this rarely occurs as an isolated phenomenon for prolonged periods. Many chronic cases of so-called 'vestibular hydrops' have other causes, especially migraine.

Acoustic neuromas

Although arising from the vestibular nerve, acoustic neuromas do not usually present with vestibular symptoms due to the very slow loss of vestibular function that allows central compensation. The most common presentation is with unilateral tinnitus and hearing loss.

Multi-sensory dizziness

This is a term used to describe a syndrome occurring particularly in elderly subjects manifest by disequilibrium and vague, non-specific dizziness when walking. This is an example of a deafferentation syndrome caused by multiple sensory deficits including visual impairment, peripheral neuropathy, vestibular deficits and cervical spondylosis. These patients often have lower limb orthopaedic impairments that will accentuate the disability. This is an important syndrome to recognise as sedative and vestibular suppressant drugs have the potential to exacerbate the problem. Falls and balance rehabilitation programs may provide functional improvement in many cases.

Central causes

Vertebrobasilar ischaemia rarely presents with isolated vertigo. Demyelination occasionally causes an acute vestibulopathy that may superficially resemble vestibular neuronitis. A careful history and examination will usually detect other CNS features.

When to refer

Referral to a neurologist is required when there are any CNS symptoms or signs, or if any of the 'red flags' (Table 2) are present. An ENT assessment should be sought if there are any of the features listed in Table 3. A perilymph fistula is an abnormal communication between the perilymph compartment of the inner ear and middle ear caused by a labyrinthine rupture in the region of the round or oval windows. Urgent surgery is often required for this condition.

Table 2. 'Red flags' indicating possible CNS disease

- Focal neurological signs
- Ataxia out of proportion to vertigo
- Direction changing, or gaze evoked, nystagmus
- Pure vertical (upbeating or downbeating) nystagmus
- Other eye movement abnormalities, eg, gaze palsy, skew deviation (vertical misalignment of the eyes).

Referral for specialist opinion should also be arranged for patients with persistent symptoms where the diagnosis is not apparent. Chronic treatment with vestibular suppressant drugs should be avoided because of the risk of development of drug induced Parkinsonism and tardive dyskinesia.

Table 3. When to refer for ENT assessment

- Presence of auditory associations (hearing loss, tinnitus, pressure or aural fullness, particularly if asymmetrical)
- Signs of suppurative middle disease
- If auditory or vestibular symptoms are triggered by pressure changes (barotrauma or Valsalva manoeuvre), suggesting the possibility of a perilymph fistula.

The risk of psychological complications increases with chronicity of symptoms. In one study, the incidence of complications, such as panic disorder and depression, was up to 50% in patients with chronic vestibular disorders. Such complications make assessment difficult as underlying organic pathology may be overlooked in patients with psychiatric complaints. Psychiatric complications are also important to address and treat appropriately as recovery from the underlying vestibulopathy may otherwise not occur.

Summary of Important Points

- An accurate diagnosis is important to exclude potentially serious central causes of vertigo, and to aid successful treatment.
- Vestibular suppressant drug therapy should only be used for the short term treatment of acute vertigo.
- A Hallpike manoeuvre should be performed on all patients presenting with vertigo.
- Benign positional vertigo is one of the more common causes of vertigo and is readily curable with exercise therapy.
- Chronic motion induced dizziness often reflects incomplete recovery from an acute, unilateral peripheral vestibular lesion and responds best to physical treatment programs.

Management of benign positional vertigo

Benign positional vertigo is caused by abnormal clumps of debris collecting in one of the fluid-filled balance canals of the inner ear. Brandt-Daroff exercises are designed to break up this material and unblock the canal. The exercises should be performed three times daily. In the initial stages it is permissible to take anti-sickness medication if nausea is a problem. The symptoms of dizziness need to be reproduced by the exercises for any benefit to occur. If the exercises are done regularly, the symptoms should resolve in most cases over a period of several days.

Step 1. Sit on edge of bed, turn head slightly to left side (approximately 45 degrees).

Step 2. Lie down quickly on right side (so that the back of the head rather than the front is resting on the bed). Wait for 20-30 seconds or for any dizziness to resolve.

Step 3. Sit up straight, again waiting for 20-30 seconds or for any dizziness to resolve.

Step 4. Turn head slightly to right side and repeat sequence in opposite direction.

Continue as above for 10 minutes (five or more repetitions to each side).