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The bedside assessment of vertigo

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Vertigo, an illusory sensation of self or environmental rotation is a common presentation to the emergency department, affecting approximately 20–30% of the general population.[1] Despite its frequency, most clinicians find acute vertigo challenging. An easy way of approaching it is to have in mind the most common causes and to consider them all during history taking and examination. When acute vertigo presents with other symptoms, the diagnosis is easy, for example with facial numbness in stroke or auditory distortion in Ménière's. This discussion will therefore focus on the clinical approach to patients presenting with acute isolated vertigo.

History taking

What is 'vertigo'?

The vestibular organ detects head motion, so abnormal activity in the vestibular nerves may be interpreted by the brain as self-motion. Lesions in the brainstem may also affect these vestibular signals, thus central lesions such as cerebellar strokes can also cause profound vertigo. Nausea, vomiting and malaise often accompany vertigo since there are connections between the vestibular nuclei and brainstem centres mediating nausea and vomiting. It should be noted that the presence or absence of nausea does not reliably distinguish between a central or peripheral lesion.

Patients often use the term 'dizziness' to describe a variety of subjective sensations. Clarification of the terminology is required to avoid diagnostic mistakes:

- If the patient finds it difficult to explain their sensation, offer words like 'merry-go-round' (vertigo), rocking like a boat (not vertigo).
- A feeling of 'unsteadiness' without true vertigo may be related to lower limb incoordination or weakness.
- Complaints of 'giddiness' or 'light-headedness' may suggest non-vestibular causes such as anaemia, hypoglycaemia or orthostatic hypotension.

Duration and time course of vertigo

Subjective recall of time is inaccurate, particularly when episodes are brief (seconds to minutes). Patients with benign paroxysmal positional vertigo (BPPV) often report that their dizziness lasted 'a few minutes'. It is worthwhile counting out aloud '1...2...3...' and asking the patient to say 'stop' when the recalled duration of intense spinning dizziness has ended. Patients who describe the attacks as lasting 'minutes' will frequently say 'stop' after a few seconds. Patients with BPPV feel unsteady and dizzy (usually of the 'rocking' type) for several minutes or hours after an acute attack.

Physical examination

The core examination in patients in our institution with vertigo and/or balance disorders is shown in Table 1.

Table 1. Core examination of patients with vertigo and/or balance disorders.

Eye movements	• Spontaneous nystagmus • Effect of gaze direction on nystagmus • Head impulse test
Limb coordination	
Hallpike manoeuvre	
Gait assessment	• Romberg test • Tandem walking
Otoscope (exclude Varicella Zoster Virus vesicles)	
Clinical assessment of hearing	

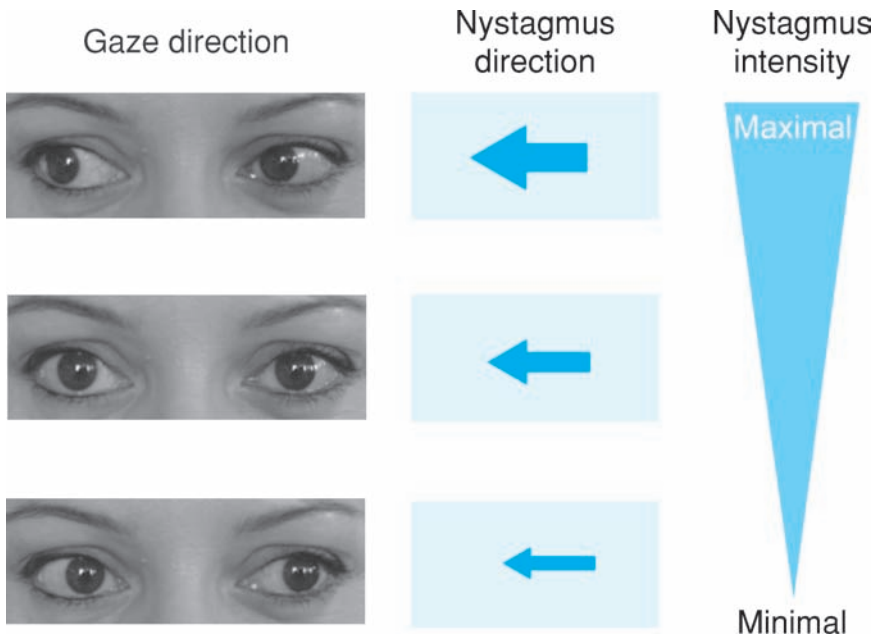

Fig 1. Unidirectional vestibular nystagmus (eg as in a left vestibular neuritis). The figure shows the effect of gaze direction on a unilateral vestibular nystagmus. (For a physiological explanation see Ref 13.)

Nystagmus

Spontaneous unidirectional nystagmus

Patients with vestibular nystagmus are said to have 'undirectional nystagmus' – that is, nystagmus unaffected by gaze direction. In contrast, the intensity of a vestibular nystagmus varies with gaze direction (Fig 1). Whether the nystagmus is of peripheral or central origin can be determined by the head impulse test (see below).

Gaze-evoked nystagmus

Physiological gaze-evoked nystagmus can be observed in normal individuals if the examiner assesses gaze beyond 20° from the straight ahead. Gaze-evoked nystagmus at gaze deviations of 20° or less is usually significant (eg drugs such as carbamazepine, phenytoin or cerebellar pathology). Physiological nystagmus is not associated with other eye movement abnormalities, whereas pathological gaze-evoked nystagmus is associated with eye-movement abnormalities such as impaired smooth pursuit, vestibular-ocular reflex (VOR) suppression and saccadic dysmetria.[2]

Vestibular-ocular reflex

The function of the VOR is to stabilise the eyes during fast head movements (eg when walking or running) and works something like a video recorder's 'steadycam'. When the VOR function is completely lost patients see the world like a bad home movie where the visual world jumps around whenever they move their head. The VOR maintains gaze stability by generating eye movements of the same speed as head movements but in the opposite direction (Fig 2).

With unilateral VOR loss the compensatory eye movements that maintain gaze stability are disrupted for head movement ipsilateral to the vestibular loss. This can be demonstrated by making a rapid movement of the patient's head (face) in the direction of the damaged labyrinth (or vestibular nerve) – a clinical test termed the 'head impulse' or 'head thrust' test.[3] The patient is asked to fixate on a visual target (usually the examiner's nose). In the normal state, a high acceleration, low amplitude movement of the head will activate the VOR and produce an almost instantaneous movement of the eyes in the opposite direction – the patient's eyes therefore do not deviate from the target. The test is positive when the patient makes a catch-up saccade to refixate on the examiner's nose. The side to which the head is moved dictates the side of the lesion. (A video demonstration of the head impulse test is available on www.imperial.ac.uk/medicine/balance/research.)

In summary, when the head impulse test is positive, the lesion causing the vestibular nystagmus is peripheral. This is reassuring if there are no other symptoms or signs. However, when vertigo is accompanied by additional symptoms or signs (eg hearing loss, numbness, ataxia) the doctor must beware because the blood supply to the peripheral audiovestibular apparatus can be occluded in some brainstem strokes (the audiovestibular apparatus is supplied by a branch of the anterior inferior cerebellar artery in 90% of people).

Neurological examination

Focal neurological signs associated with vertigo make localisation straightforward, particularly when there is brainstem involvement. However, isolated cerebellar strokes may present with prominent vertigo and nausea and a paucity of signs, potentially mimicking a peripheral vestibular syndrome.[4]

Differential diagnosis

Acute isolated vertigo is usually benign, but occasionally stroke may present with isolated vertigo. The main causes of an isolated attack of vertigo are:

- BPPV
- acute vestibular neuritis
- cerebellar stroke
- migrainous vertigo
- bilateral vestibular failure (BVF) (less commonly).

Fig 2. Head impulse test: a simple bedside test of the horizontal vestibulo-ocular reflex, performed by holding the patient's head and applying a small-amplitude, high-acceleration head turn, first to one side and then to the other. The patient is asked to fixate on the examiner's nose and the examiner watches for corrective saccades (ie the eyes move with the head rather than staying fixed on the nose). 'Catch-up' saccades occur when the head is turned in the direction of the damaged side. Shown here is a normal response for a right head impulse test in which the right labyrinth is functioning normally.

Benign paroxysmal positional vertigo

The most common cause of acute vertigo among the general population, BPPV, is due to calcium carbonate crystals settling within the endolymphatic fluid of the semicircular canals. The movement of these crystals during changes in head position (eg bending down, looking upwards or turning over in bed) stimulates the vestibular sensory receptors and so precipitates a brief episode of vertigo and nystagmus which lasts only as long as the crystals take to settle again (ca 5 sec). Vertigo precipitated on turning over in bed is almost always due to BPPV. The diagnosis is confirmed by the Hallpike manoeuvre (torsional nystagmus beating towards the lower ear) (Fig 2). Treatment is 60–80% effective[5,6] with a single two-minute procedure: either the Semont or Epley manoeuvre, which have similar efficacy. (A demonstration of these manoeuvres is available at www.imperial.ac.uk/medicine/balance/research.) Chronic drug administration has no role in the management of BPPV (regular vestibular sedatives may be prescribed for no more than 2–3 days).

Vestibular neuritis

Vestibular neuritis has an annual incidence of approximately 3.5 per 100,000.[7] It is characterised by the subacute onset of spinning vertigo that persists for days and weeks and is aggravated by head motion. A viral prodrome is present in approximately 50% of cases.[7] The vertigo reaches a peak intensity within minutes or hours and is associated with autonomic symptoms such as nausea, vomiting, malaise, pallor and sweating. There is also a tendency to veer towards the affected side. Clinical examination reveals spontaneous unidirectional nystagmus, typically horizontal with a torsional component. The head impulse test will be positive when the head is turned towards the side of the vestibular lesion. Vestibular neuritis is a clinical diagnosis, but electro-nystagmography with bithermal caloric testing is often employed to document the unilateral loss of vestibular function and to monitor recovery.

In the acute phase, treatment includes a short period (2–3 days only) of bedrest and vestibular suppressants to control the nausea and vomiting. All patients improve spontaneously as a result of central vestibular compensation and few have residual symptoms. Vestibular exercises can accelerate the recovery of balance following acute vestibular neuritis,[8] but data regarding long-term outcome are lacking.

Cerebellar stroke

Cerebellar stroke is characterised by the abrupt onset of vertigo (within seconds), often accompanied by occipital headache. Other associated signs may include gait or limb ataxia, facial numbness, Horner's syndrome, hearing loss, contralateral hemiparesis and hemisensory loss. The presence of malaise and vomiting can make gait assessment difficult. Importantly, the head impulse test remains normal in cerebellar stroke. Immediate brain imaging is indicated in suspected cerebellar stroke as these patients may require urgent intervention.

Migrainous vertigo

A diagnosis of migrainous vertigo is not widely recognised outside neuro-otological practice. Patients with this form of vertigo commonly report spontaneous or positional vertigo lasting hours to days.[9] The typical patient is a migraineur who has noticed a recent increase in headache frequency and, over the same period, developed vestibular episodes, with headache and vertigo not necessarily occurring together. Other migrainous features such as photophobia, phonophobia and nausea are often present during the vertiginous episode. Migrainous vertigo is a diagnosis of exclusion. Many patients have symptoms and signs (including nystagmus) suggestive of central dysfunction, so acute imaging may be required on first presentation. Treatment is with standard antimigraine prophylactic agents.

Bilateral vestibular failure

The most common cause of in-hospital BVF is aminoglycoside toxicity.[10] A common misconception among clinicians is that gentamicin ototoxicty is synonymous with deafness. In fact, vestibular function is much more sensitive to aminoglycosides. However, 90% of patients with aminoglycoside-associated vestibular loss will *not* develop deafness[11] – a finding that may explain why most cases of aminoglycoside otoxicity go undetected.[10] The typical patient with aminoglycoside vestibular failure[12] will have been in intensive care, often with multi-organ dysfunction. If conscious, 20% experience spontaneous episodic vertigo lasting minutes to hours. This is an unexplained phenomenon: vertigo implies a right-left vestibular imbalance, and simultaneous, bilateral vestibular loss would be expected from a systemic ototoxin. The vertiginous episodes wane after a few days as the vestibular function is ablated.

Key Points

- **Establish from the history that the patient has vertigo**
- **Establish the time course of the vertigo**
- **Examination in acute vertigo must include the Hallpike manoeuvre and head thrust test**
- **In acute vertigo always consider 'ABC': acute vestibular neuritis, benign paroxysmal positional vertigo, cerebellar stroke**

KEY WORDS: acute vertigo, benign paroxysmal positional vertigo, Hallpike manoeuvre, vestibulo-ocular reflex

However, attempts to rehabilitate and mobilise patients with aminoglycoside vestibulotoxicity are severely compromised due to gait imbalance and disabling oscillopsia on head movement. These symptoms may lead to an incorrect diagnosis of a cerebellar stroke, but the diagnosis of BVF must be considered in all patients with a history of aminoglycoside administration who develop head movement-induced oscillopsia and gait imbalance. The diagnosis of BVF is easily made clinically via the head impulse test[3] and confirmed by caloric testing. Aminoglycoside vestibular failure is permanent and can have devastating consequences for a patient's mobility and functional status. Functional recovery occurs over several years, and can be hastened with graded physical activity and vestibular rehabilitation.

When is neuroimaging indicated?

Criteria for neuroimaging in acute vertigo is the presence of vertigo plus one or more of the following:[13]

- new onset headache
- central neurological symptoms or signs
- acute deafness
- intact head impulse test.

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