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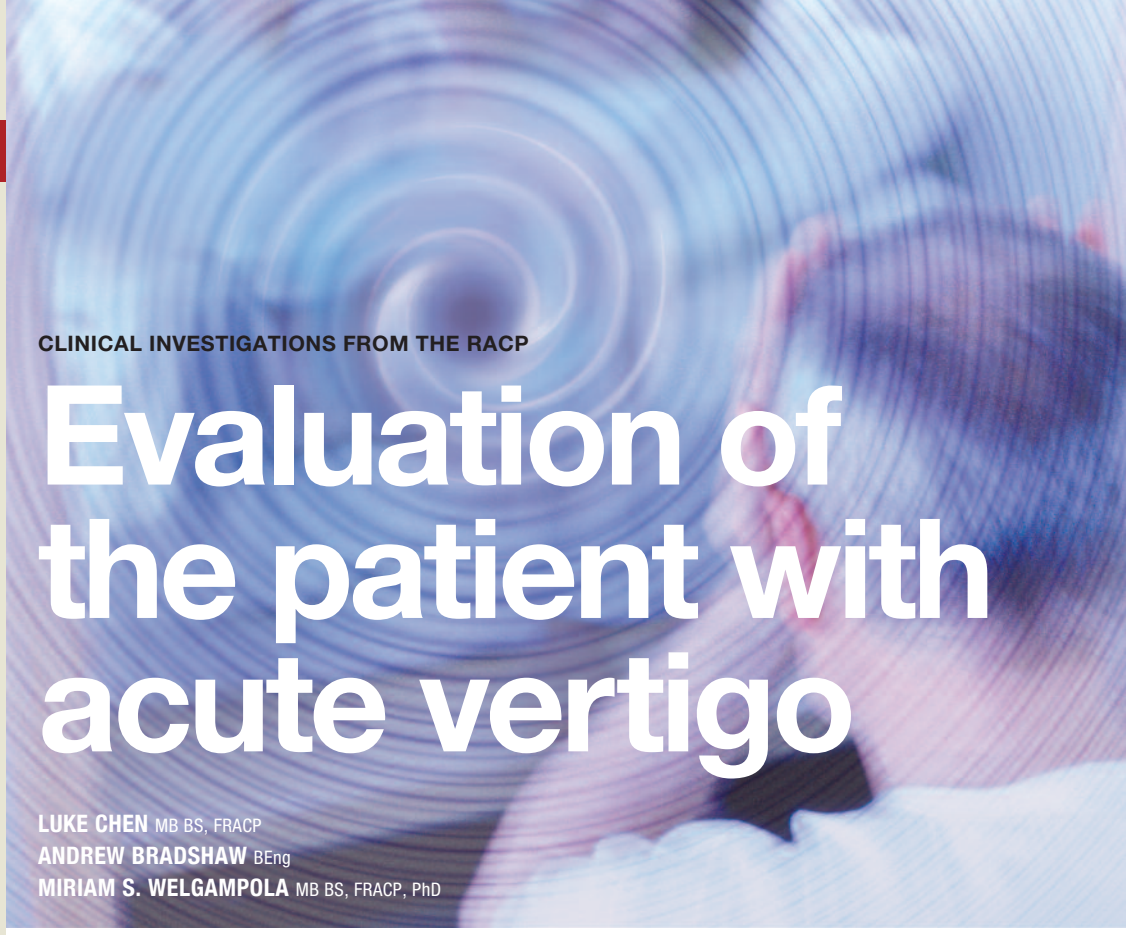

CLINICAL INVESTIGATIONS FROM THE RACP

Evaluation of the patient with acute vertigo

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Key points

- Vertigo is a disabling symptom, but most causes of vertigo are treatable and benign.
- A focused history and careful examination when the patient is symptomatic often leads to a diagnosis.
- When evaluating the dizzy patient, it is important to know whether the vertigo is spontaneous or positional, its duration and its associated symptoms, especially tinnitus, hearing fluctuations and headaches.
- Inspection for (peripheral *v.* central) nystagmus, the head impulse test and the Dix Hallpike test are the three key components of the physical examination.
- Benign positioning vertigo is the most common and correctable form of vertigo.

In this series, we present authoritative advice on the investigation of a common clinical problem, especially commissioned for family doctors and written by members of the Royal Australasian College of Physicians.

Dizziness can arise from vestibular disorders, postural hypotension, anaemia or rhythm disturbances, or as an effect of drug intake, metabolic derangement or even anxiety. A focused history and systematic examination of the patient will help diagnose the underlying cause.[1]

HISTORY

There are some key questions to consider when taking a history from a patient presenting with dizziness.

Is the patient experiencing vertigo?

Vertigo is an illusion of movement (e.g. spinning, rocking or tilting) due to asymmetrical neural activity between the left and right vestibular nuclei. Some patients will give a compelling history of movement of the visual surrounds or 'true vertigo' that suggests a vestibular disorder as opposed to lightheadedness, floating sensations or disorientation that imply nonvestibular causes. Sometimes, pursuing precise symptom quality alone can be unrewarding and misleading.[2]

Is the dizziness spontaneous or positional?

Spontaneous vertigo could be due to vestibular neuritis, Meniere's disease, vestibular migraine or less commonly posterior circulation ischaemia. Positional vertigo is most commonly benign positioning vertigo, typically occurring when rolling over in bed or looking up.

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TABLE. CLINICAL AND LABORATORY FEATURES OF COMMON ACUTE VESTIBULAR PRESENTATIONS

	Vertigo type	Vertigo duration	Associated symptoms	Examination	Investigations
Vestibular neuritis/ Labyrinthitis	First episode Spontaneous	More than 24 hours	Viral prodrome	Spontaneous, unidirectional nystagmus beats towards good ear Positive head impulse test	Abnormal static bias Unilateral caloric weakness Normal audiogram and MRI
Posterior circulation vascular event	First to recurrent Spontaneous	TIA: 20 to 30 minutes Stroke: hours to days	Hearing loss (AICA) Severe imbalance Diplopia, facial or limb sensory or motor loss	Spontaneous, unidirectional or direction-changing nystagmus Skew deviation Negative head impulse test (about 10% could be positive)	Abnormal static bias Normal caloric test usually Normal audiogram or unilateral sensorineural hearing loss Abnormal MRI
BPV	Recurrent Positional	Seconds	None	No spontaneous nystagmus Posterior canal: upbeating, torsional nystagmus on Dix Hallpike testing	None required unless the BPV is secondary to inner ear disease
Vestibular migraine	Recurrent Spontaneous or positional	Minutes to hours Rarely seconds to days	Headache (may not be temporally associated) Visual aura, sensory disturbances, syncope	In between attacks: commonly normal During an attack: normal or horizontal, vertical or torsional nystagmus	Normal vestibular function tests in between attacks Normal MRI
Meniere's disease	Recurrent Spontaneous	Hours	Aural fullness Tinnitus Hearing loss Drop attacks	In between attacks: normal During an attack: horizontal nystagmus	Low-frequency sensorineural hearing loss Unilateral caloric weakness Abnormal electrocochleography Static bias may be abnormal Normal MRI

ABBREVIATIONS: AICA = anterior inferior cerebellar artery; BPV = benign positioning vertigo; TIA = transient ischaemic attack.

Is the dizziness brief or prolonged?

Positional vertigo that lasts seconds is likely to be benign positioning vertigo. Vertigo in Meniere's disease typically lasts hours. Vestibular migraine can cause dizziness lasting from seconds to days. Prolonged severe vertigo lasting more than 24 hours is encountered in both vestibular neuritis and posterior circulation stroke. The vertigo of a posterior circulation transient ischaemic attack (TIA) lasts from 20 to 30 minutes.

Is this the first attack of dizziness?

The first ever attack of prolonged vertigo raises the possibility of a posterior circulation stroke, particularly in older individuals with vascular risk factors. Recurrent isolated vertigo occurring over years is more likely to represent benign positioning vertigo than a posterior circulation vascular event. However, a posterior circulation TIA needs to be considered in recurrent isolated vertigo lasting from 20 to 30 minutes occurring over days to weeks.

What triggers the dizziness?

Vertigo brought on by turning over in bed or arching backwards to look up is very likely to be benign positioning vertigo, whereas positional dizziness on rising rapidly from a supine position suggests orthostatic hypotension. Mild subjective dizziness and imbalance provoked by specific situations in specific places and associated with anxiety symptoms should raise the possibility of psychophysical vertigo.

Are there hearing symptoms (e.g. aural fullness, tinnitus or hearing loss)?

Meniere's disease is characterised by monaural fullness, fluctuating hearing loss and tinnitus that is temporally related to vertigo. Less commonly, binaural tinnitus or fullness may accompany

migraine. Sequential, fluctuating hearing loss may be seen in patients with autoimmune inner ear disease. Hearing loss is uncommon in those with vestibular neuritis. An acute vestibular syndrome with hearing loss raises the possibility of an anterior inferior cerebellar artery stroke.

Are there other accompanying neurological symptoms?

The presence of facial numbness, weakness, speech disturbance, severe gait ataxia or motor loss suggests brainstem or cerebellar involvement (ischaemia or demyelination), and should prompt referral of the patient to the Emergency Department for urgent assessment and imaging. Headache, photophobia or phonophobia can be temporally associated with vertigo in patients with vestibular migraine, but sometimes vertigo occurs in the headache-free interval.

EXAMINATION OF THE ACUTELY DIZZY PATIENT

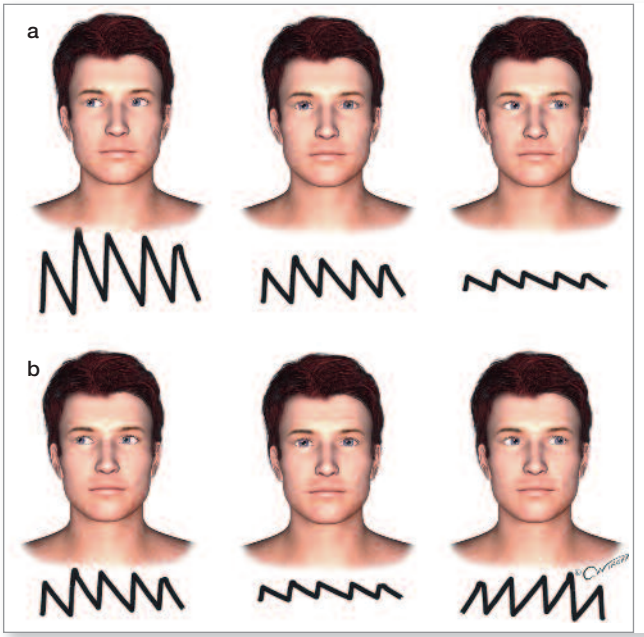
A targeted examination of the acutely dizzy patient is as informative as the vestibular function tests or an MRI scan.[3] The Table summarises the profiles of common vestibulopathies encountered in the Emergency Department and general practice. An examination can be performed in the consulting room, without specialised equipment and will include the following features.

General inspection

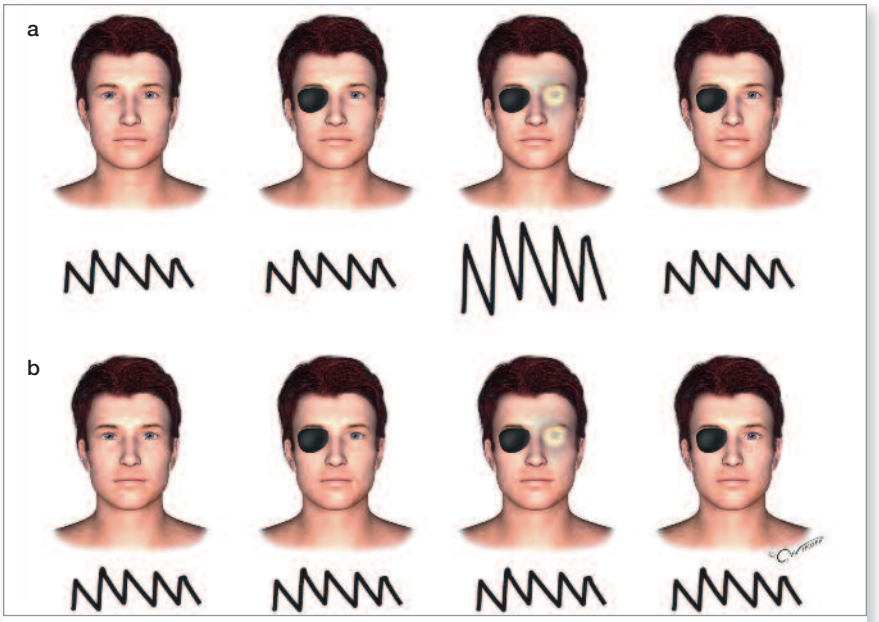
Look for a head tilt (towards the side of a unilateral peripheral vestibulopathy) and evidence of truncal ataxia during a general inspection of a patient with dizziness.

Inspect for nystagmus

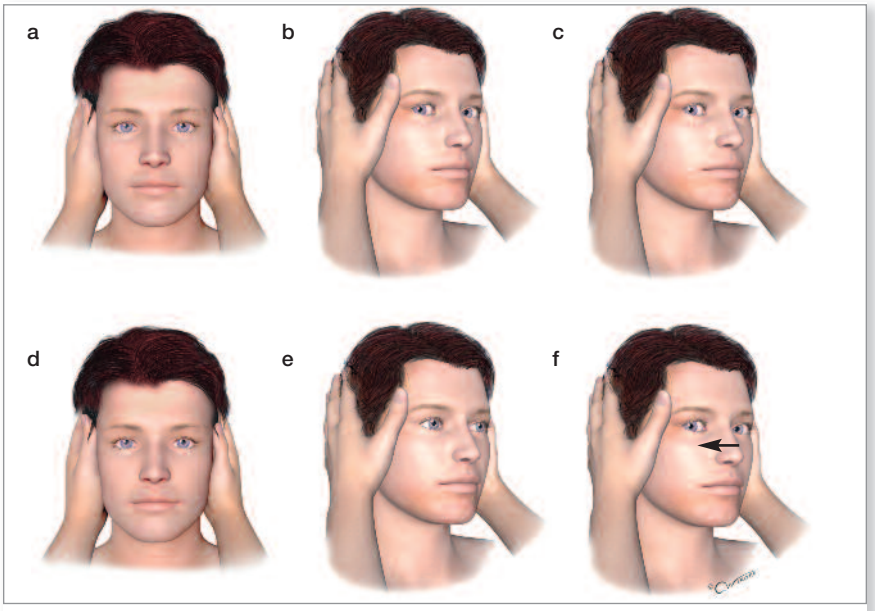
Spontaneous nystagmus usually indicates a left–right difference in resting activity of the vestibular nucleus. Nystagmus consists of a slow phase, which is difficult to see at the bedside, and a fast phase, which is readily seen and will allow the direction of the nystagmus to be identified. In patients with a peripheral vestibular



Figures 1a and b. Nystagmus. a (top row). Nystagmus observed in acute peripheral vestibulopathy. The nystagmus always beats away from the lesion (e.g. right beating with a left ear lesion) regardless of the direction of the gaze. b (bottom row). Central nystagmus changes direction with gaze (i.e. right beating on right gaze and left beating on left gaze).



Figures 2a and b. The penlight test for inspection of the effect of visual fixation on central and peripheral nystagmus. a (top row). First ask the patient to fixate on a distant target. Then occlude the right eye and observe the velocity of the nystagmus. Now shine a penlight torch directly on the left eye intermittently for 10-second periods. When the right eye is occluded and the left eye is illuminated (light 'on'), visual fixation is completely removed and peripheral nystagmus is enhanced. When the illumination is turned 'off', the left eye fixates and peripheral nystagmus suppresses. b (bottom row). With central nystagmus, there is no enhancement with loss of fixation (when the light is 'on') and no suppression when visual fixation returns (when the light is 'off').



Figures 3a to f. The bedside head impulse test. a and d. First ask the patient to fixate on a target such as the examiner's nose. Firmly grasp the patient's head with both hands and ensure the subject is relaxed. Deliver brisk unpredictable head rotations (of about 20º) to each side. b and c. If left vestibular function is normal, leftward head impulse results in compensatory eye movements towards the right and the eyes remain fixed on the target throughout the head movement. If there is a left vestibular loss, on leftward head impulse the eyes will (e) first move with the head then (f) refixate with a catch-up saccade (arrow).

disorder, the fast phase of the nystagmus beats towards one direction regardless of the gaze (unidirectional: Figure 1a and videos 1a and b; all videos can be viewed online at www.medicinetoday.com.au), whereas central nystagmus is more commonly direction changing – that is, left beating in left gaze and right beating in right gaze (Figure 1b). Vertical and pure torsional nystagmus also indicates a central cause. Visual fixation makes peripheral nystagmus less pronounced. The 'penlight cover test' can be used to compare nystagmus with and without visual fixation (Figure 2).[4]

Head impulse testing

The vestibulo-ocular reflex maintains visual stability despite head movement. Thus a small rightward head rotation generates a very rapid, equal and opposite leftward eye rotation that maintains the line of sight on the object of regard. Horizontal head impulse testing allows bedside assessment of the horizontal vestibulo-ocular reflex. The examiner asks the patient to fixate on a distant target, then applies a quick, passive head turn to the right or left and if the vestibulo-ocular reflex is deficient, the patient makes a corrective saccade to maintain fixation (Figure 3 and videos 2 and 3). The head impulse test is positive in patients with acute vestibular neuritis, but not in those with stroke.[5] In patients with an acute vestibular syndrome (severe, spontaneous vertigo lasting more than 24 hours), this test will separate neuritis from a stroke with 90% sensitivity.[6]

Skew deviation

Skew deviation or vertical misalignment of the eyes implies disruption of the pathways from the otolith organs to the eyes. To detect a skew deviation, a cover test can be performed. Although it can occur in both peripheral and central vestibulopathies, skew deviation is more common in patients with central disorders.

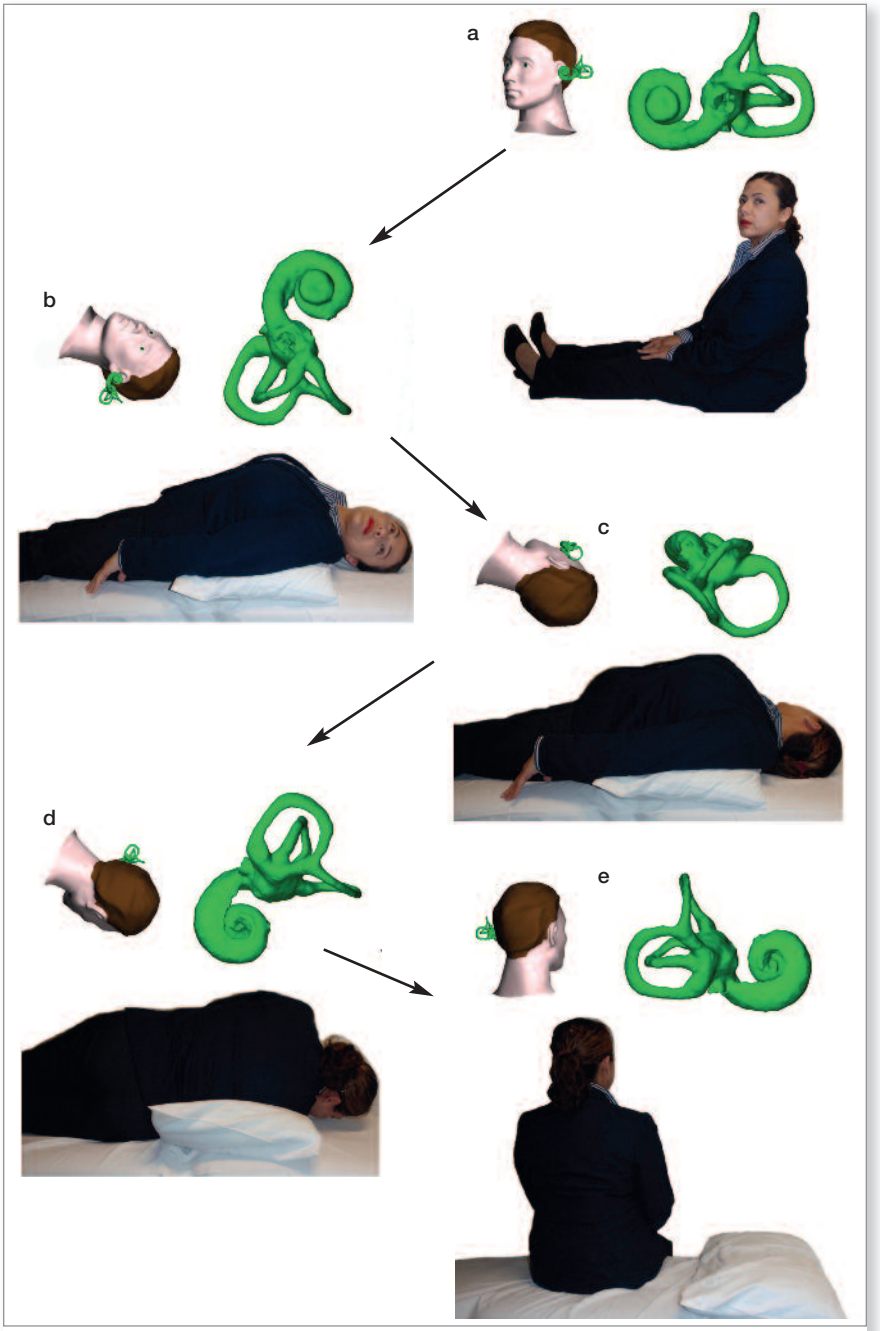
Positional testing

Positional testing is an indispensable part of bedside vestibular assessment. To perform the Dix Hallpike test to detect posterior canal benign positioning vertigo, the patient sits on a couch, with the head rotated 45° to the side that is being tested. The examiner then rapidly lies him down in a head-hanging position (Figure 4). In posterior canal benign positioning vertigo, upbeating torsional nystagmus appears after a latency of five to 10 seconds (video 4); the fast phase of the torsion beats towards the affected ear and upwards. The nystagmus fatigues in 30 seconds or so, and may fatigue after repeated Dix Hallpike testing.

A single bedside Epley manoeuvre will successfully treat posterior canal benign positioning vertigo in about 80% of patients (Figure 4). To perform the Epley manoeuvre for left posterior canal benign positioning vertigo, slowly roll the patient over from the left Dix Hallpike position to the right Dix Hallpike position, then to a nose down position and finally to a sitting-up position.

Gait and vestibulospinal testing

The gait should be tested using the tandem gait and Romberg test. The patient with acute vestibular neuritis will still be able to stand unsupported but they may fall to the affected side on the Romberg test and have difficulty tandem walking. The patient with a cerebellar infarct may have difficulty standing without support even with the eyes open. The Unterberger test can also be performed by the patient marching on the spot with the eyes closed for 30 seconds; if there is excessive vestibular asymmetry, the patient will turn to the weaker side.



Figures 4a to e. The Dix Hallpike test and Epley manoeuvre for left posterior canal benign positioning vertigo with three-dimensional CT reconstructions of the inner ear in each position. a. Ask the patient to sit up in bed with the head rotated 45° to the left. b. Quickly lie the patient down over the pillow; wait for nystagmus and vertigo to abate (at least 30 seconds). c. Slowly roll the patient's head over to the right. d. Roll the patient's entire body to the right; wait 30 seconds. Then rotate the head further to achieve a nose down position; wait for two minutes. e. Slide the patient's feet over the edge of the bed and then sit the patient up.

General examination

A general examination should include measurement of supine and standing blood pressure if orthostatic hypotension is suspected, measurement of blood sugar level by a fingerprick test, a cardiovascular examination for rhythm disturbances and a screening neurological examination for weakness, limb ataxia and sensory dysfunction.

WHEN TO REFER PATIENTS WITH ACUTE VERTIGO

Patients with benign positioning vertigo and characteristic positional nystagmus only require an Epley manoeuvre and follow up within one week to ensure the symptoms have abated. If the benign positioning vertigo is not secondary to an inner ear disease (such as vestibular neuritis or Meniere's disease) and responds to bedside repositioning, no further investigation or referral is needed. Patients with recurrent positioning vertigo and atypical positioning nystagmus that is not consistent with posterior semicircular canal benign positioning vertigo or benign positioning vertigo that is unresponsive to multiple repositioning manoeuvres should be referred to an otolaryngologist or neuro-otologist.

Patients with episodic vertigo of uncertain origin should be referred to a local balance clinic, otolaryngologist or neuro-otologist for further investigation.

An acute vestibular syndrome is characterised by the rapid onset of severe vertigo, nausea, vomiting, nystagmus and unsteady gait lasting one or more days. Most patients with this syndrome have vestibular neuritis, which is self-limiting, yet cerebellar stroke is a close mimicker of neuritis and will only be evident on careful assessment of eye movement. The HINTS ('Head Impulse, Nystagmus, Tests of Skew') battery is useful for clinically differentiating a stroke from vestibular neuritis.[3] A negative head impulse test, direction-changing nystagmus (central nystagmus) or vertical misalignment of the eyes (skew deviation)

is very suggestive of a stroke.

The presence of any one of the above 'central' attributes in a patient presenting with acute vestibular syndrome should raise the possibility of central vertigo and trigger urgent referral of the patient to the Emergency Department for prompt imaging and appropriate intervention. Conversely, the combination of a positive head impulse test, unidirectional nystagmus that enhances when fixation is removed (peripheral nystagmus), the absence of skew deviation and the absence of hearing loss favours a diagnosis of vestibular neuritis.

Based on evidence of less pronounced vestibular deficits in patients treated with corticosteroids,[7] we recommend that vestibular neuritis seen within the first seven days of symptom onset should be treated with use of oral corticosteroids (e.g. prednisolone 1 mg/kg/day for one week followed by a tapered dose over a further week) if there are no major contraindications (such as concurrent infections or uncontrolled diabetes). Patients should be encouraged to ambulate early and be referred to a balance clinic, otolaryngologist or neuro-otologist for vestibular testing, diagnostic confirmation and initiation of vestibular rehabilitation. Prochlorperazine should only be used in the first few days to control nausea and vomiting.

WHAT TESTS ARE DIAGNOSTICALLY HELPFUL?

One or more of the following tests may be helpful when investigating the dizzy patient.

Audiogram

An audiogram is valuable because it will pick up the low frequency hearing loss found in patients with Meniere's disease. New hearing loss with a first attack of prolonged severe vertigo can also suggest an inner ear stroke.

Bithermal caloric test

The bithermal caloric test assesses the function of the individual horizontal semicircular canal of the ear and is the most widely used vestibular function test. Caloric asymmetry is more common in patients with Meniere's disease and vestibular neuritis and is rare in those with vestibular migraine and idiopathic benign positioning vertigo.

Static bias

The static bias or subjective visual horizontal is a perceptual test during which the patient is asked to align an illuminated bar in total darkness to what is perceived to be horizontal. In patients with

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Video 1. Nystagmus observed in acute peripheral vestibulopathy. The nystagmus always beats away from the lesion regardless of the direction of gaze and is more pronounced upon removal of visual fixation (a) and suppresses with visual fixation (b).

Video 2. Normal head impulse test. The patient's eyes remain fixed on the target during delivery of head impulses.

Video 3. Abnormal left head impulse. When the patient's head is thrust to the left, the eye lags behind for a few milliseconds and throws in a 'catch-up saccade' to refixate on the target.

Video 4. Upbeating and leftward torsional nystagmus recorded in the left Dix Hallpike position is typical in left posterior canal benign positioning vertigo.

acute vestibular neuritis, there is a conjugate binocular torsion of the eyes towards the affected ear. This torsional offset results in a shift of the subjective visual horizontal towards the affected side.[8] This test represents otolith ocular pathways and is currently used in balance clinics.

Vestibular evoked myogenic potential

The vestibular evoked myogenic potential is a new test that uses sound or vibration pulses to activate the otolith organs. It complements the information obtained from canal function tests. The cervical vestibular evoked myogenic potential, an inhibitory muscle potential, is recorded from the sternomastoid muscles during active contraction and tests otolith pathways to the neck. The ocular vestibular evoked myogenic potential is an excitatory muscle potential recorded infra-orbitally and tests otolith pathways to extraocular muscles.[9]

MRI scan

An MRI scan with diffusion weighted imaging is indicated if the clinical assessment suggests a posterior circulation stroke. CT scans of the brain image the posterior fossa poorly and are of little relevance when investigating vertigo. As an urgent MRI is not accessible to all patients presenting with acute vertigo, the HINTs battery should be used to triage those more likely to have experienced an acute event.

Video head impulse

The video head impulse is a very recent addition to the vestibular test battery and is likely to be widely used in balance clinics, and neurology and otolaryngology practices in the future. It uses a high-speed video camera mounted on lightweight goggles to record the eye movement during the head impulse test and provides an objective measure of the vestibulo-ocular reflex in the horizontal canal plane (see under 'head impulse').[10]

CONCLUSION

Vertigo is a common presentation in the emergency room and general practice. A focused history and careful examination holds the key to its accurate diagnosis and successful management. MT

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