Balance disorders in adults: an overview

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Dizziness is a common and potentially disabling complaint. A multitude of medical and otological conditions may manifest as disequilibrium. Symptomatic improvement in peripheral vestibular lesion is the result of central compensation and not restoration of normal labyrinthine function.

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Dizziness is a non-specific symptom, which does not instantly point to any specific organ systems. Disequilibrium may result from disturbance in a number of structures, including the visual, proprioceptive, vestibular, cardiovascular and central nervous system. Each year, 5 out of every 1000 patients consult their GP for vertigo, and a further 10 in 1000 are seen for dizziness or giddiness (Royal College of General Practitioners and Office of Population Census and Survey, 1986). The term dizziness is commonly used interchangeably to describe four categories of balance dysfunction including vertigo, disequilibrium or unsteadiness, near-syncope and non-specific light-headedness.

Figure 1. Classification of vertigo.

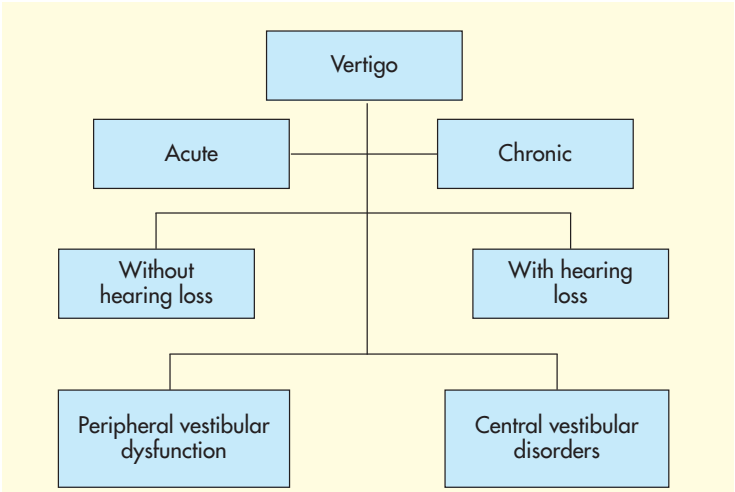

TABLE 1.
Historical features that would help distinguish peripheral from central vertigo

Features	Peripheral	Central
Imbalance	Mild–moderate	Severe
Nausea and vomiting	Severe	Variable (may be minimal)
Auditory symptoms	Common	Rare
Neurological symptoms	Rare	Common
Compensation	Rapid	Slow

Adapted from Baloh and Honrubia (1990)

WHAT IS VERTIGO?

Vertigo is defined as an illusion or hallucination of movement and is typically thought to arise from an abnormality involving the peripheral or central vestibular pathways (*Figure 1*). A clear-cut distinction should therefore be made between vertigo and non-specific light-headedness, dizziness or faintness, which may be caused by a plethora of general medical conditions. A carefully obtained thorough history, combined with targeted physical examination, would pave the way for successful diagnosis in the majority of instances. Dizziness is a common but often untreated symptom, which is associated with extensive handicap and psychological morbidity (Yardley et al, 1998). It is important to realize that not all dizziness is vertigo even though patients may describe vertigo as dizziness. It is therefore useful to ask the patient to describe the exact sensation and any other associated symptoms, such as nausea, vomiting or headache.

CLASSIFICATION OF VERTIGO

The history usually provides the vital information necessary to distinguish peripheral from central causes of vertigo (Baloh, 1998) (*Table 1*). It should include:

1. The mode of onset and description of the first episode
2. Frequency and duration of individual attack
3. Whether it is spontaneous or provoked by certain factors, such as head movement
4. Associated auditory symptoms, such as hearing loss or tinnitus
5. Head trauma
6. Concomitant ear disease or previous aural surgery.

When the symptoms are global and the patient describes the sensation as 'wooziness', being

'about to blackout' or 'disoriented', a presyncopal cause secondary to insufficient blood flow to the central nervous system is assumed. More than one factor may contribute to balance disorder in the elderly, e.g. visual and proprioceptive disturbance. In some patients, an accurate diagnosis may emerge only after a period of review.

PERIPHERAL *VS* CENTRAL VESTIBULAR DYSFUNCTION

Vertigo may present as:

1. A spontaneous attack with prolonged recovery lasting days
2. Recurrent attacks lasting minutes to hours
3. Brief attacks induced by position change usually lasting less than a minute.

A peripheral or a central disorder may cause each of these syndromes. Gait unsteadiness and fear of falling are more common manifestations of neurological disorders. Patients with a history of falls require thorough evaluation of risk factors, such as drug intake, neurological condition, cognitive function, environmental factors and general medical conditions (*Figure 2*). Disorders affecting the peripheral vestibular system often cause associated hearing impairment. However, conditions such as benign paroxysmal positional vertigo (BPPV), vestibular neuronitis and familial vestibulopathy selectively affect the peripheral vestibular system without causing hearing loss.

Bath et al (2000) report peripheral vestibulopathy as the cause of dizziness in 64.7% of adults, with central, psychiatric and unknown causes respectively accounting for 8.1%, 9% and 13.3% of cases (*Table 2*). Duration of the individual attack will usually give an idea about the possible underlying pathology. Sudden rotatory vertigo of less than a minute is commonly caused by BPPV, whereas vertigo of several hours is a manifestation of Ménière's disease or migraine.

In between attacks, there may be periods of long remission. A single acute episode of vertigo with gradual improvement in symptoms is most commonly the result of an acute peripheral vestibular event, such as viral or ischaemic labyrinthitis. Patients with poorly compensated peripheral vestibular disorder may complain of constant disequilibrium, which in the absence of an acute attack should prompt a search for central vestibular pathology.

Figure 2. Causes of dizziness in adults.

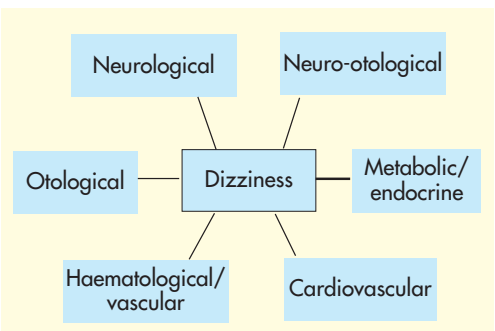

TABLE 2.
Aetiology of dizziness in adults

Systems	Conditions
Otological	Benign paroxysmal positional vertigo
	Labyrinthitis
	Ménière's disease
	Ototoxic drugs
	Vestibular neuronitis
	Otosyphillis
	Cholesteatoma
	Perilymph fistula
Neuro-otological	Cerebello-pontine angle tumours
	Vestibular schwannoma
	Meningioma
	Metastatic tumours
	Arachnoid cysts
Neurological	Multiple sclerosis
	Parkinson's disease
	Cerebellar or brainstem infarction
	Syncope
Cardiovascular	Aortic stenosis
	Carotid sinus hypersensitivity
	Cardiac dysrhythmias
Haematological	Anaemia
	Hyperviscosity syndrome
	Leukaemias
	Sickle cell disease
Vascular	Autoimmune vasculitis
	Carotid artery stenosis
	Vertebrobasilar ischaemia
	Subclavian steal syndrome
Metabolic/endocrine	Hyper-/hypoglycaemia
	Hyperventilation
	Thyroid disease
Others	Psychological disorders
	Cervical vertigo
	Visual vertigo
	Head injury
	Drug induced, other than ototoxic medications
	Multisensory dizziness syndrome
	Chronic fatigue syndrome

VERTIGO ASSOCIATED WITH HEARING LOSS

Vertigo associated with hearing loss may be a manifestation of Ménière's disease, temporal bone fracture, otitic barotrauma, perilymph fistula, vestibular schwannoma, labyrinthitis, otosyphilis, ototoxic drugs and autoimmune inner ear disease.

BENIGN PAROXYSMAL POSITIONAL VERTIGO

BPPV is one of the most common causes (25%) of dizziness (Epley, 1996) and is thought to arise from the presence of abnormal dense particles, most likely otoconial debris, in the long arm of the posterior semi-circular canal. BPPV is characterized by severe paroxysms of rotational vertigo provoked by positional changes of the head. Dix and Hallpike's positional test, which provokes dizziness and a typical geotropic nystagmus, is diagnostic of BPPV (Epley, 1995). It is eminently treatable by a canalith repositioning procedure.

TABLE 3.
Aetiology of poor compensation in peripheral vestibular dysfunction

Drug-related factors	Prolonged use of vestibular sedatives, tranquillisers and other psychotropic drugs
	Very strong hypotensives
	Anticonvulsant use may need to be reviewed and balanced
Psychological factors	Avoidance behaviour
	Anxiety, depression and panic attacks
Visual problems	New glasses: bifocals, change of prescription
	Development of cataract
	Retinopathy: diabetes mellitus, hypertension
	Macular degeneration
Musculoskeletal disorders	Stiff or painful neck or back
	Arthritis of major weight-bearing joints
Neurological disorders	Neuropathy: diabetes mellitus, alcohol
	Recent posterior circulation infarction
Cardiovascular	Poorly controlled or untreated hypertension
	Arrhythmias
	Valvular heart disease
	Inadequate cardiac output as a result of other medical causes
Haematological problems	Anaemia
	Polycythaemia rubra vera
Vestibular factors	Progressive peripheral vestibular dysfunction: Ménière's disease
	Bilateral vestibular failure
	Concomitant untreated benign paroxysmal positional vertigo
	New central vestibular disorder: multiple sclerosis, cerebellar atrophy
Exercise-related factors	Inappropriate exercise regimens
	Poor motivation and compliance

VESTIBULAR NEURONITIS

Vestibular neuronitis is reportedly the third most common cause of peripheral vestibular vertigo following BPPV and Ménière's disease (Strupp and Brandt, 1999). Sudden onset of severe vertigo with nausea, vomiting and absence of auditory symptoms typify vestibular neuronitis.

CERVICAL VERTIGO

Lack of definite diagnostic tests makes cervical vertigo a controversial clinical entity. Vertigo associated with neck movements could result from disorders of vestibular, visual, vascular, neurovascular or cervico-proprioceptive mechanisms. The proponents of this condition claim that dizziness of cervical origin could result from cervical tone imbalance, secondary to defective neck proprioceptors. However, such a tone imbalance has not been demonstrated in whiplash injuries or cervical pain syndromes. Nevertheless, if true cervical vertigo exists, the treatment is similar to that of the underlying neck condition (Brandt and Bronstein, 2001).

BILATERAL VESTIBULAR FAILURE

Bilateral vestibular failure (BVF) may present with recurrent episodes of vertigo associated with unsteadiness in the dark and ossilopsia on head movement. Ototoxic drugs, e.g. gentamycin, commonly cause BVF, which is often associated with some degree of high frequency sensorineural hearing loss. Approximately 20% of patients with BVF have no identifiable cause (Rinne et al, 1998). Management of patients with bilateral vestibular loss is difficult; the emphasis is on maximizing and optimizing visual and proprioceptive inputs. Patients should be counselled regarding safety considerations, e.g. night-lights at home or assistive devices, and should be warned of the risk of drowning while diving. Familial vestibulopathy without hearing loss may be inherited as an autosomal dominant condition (Baloh et al, 1994).

COMPENSATION

Compensation is a central process, therefore peripheral lesions tend to compensate more readily than brainstem or cerebellar causes. About 80% of patients respond favourably to vestibular rehabilitation exercises (Luxon, 1998).

INADEQUATE/POOR COMPENSATION

Vestibular sedatives are useful adjuncts in the management of acute vertigo, but their long-term use may delay compensation. Other causes of poorly compensated vestibular dysfunction include inappropriate exercise strategy, poor vision and psychological factors (*Table 3*).

CENTRAL VERTIGO

Common central causes of recurrent spontaneous vertigo are migraine and vertebrobasilar transient ischaemia. A family history is helpful in supporting a diagnosis of vestibular migraine.

Common central causes of a single attack of vertigo lasting more than 24 hours include posterior circulation infarction, cerebellar or brainstem haemorrhage and multiple sclerosis (Solomon, 2000). Brainstem and cerebellar lesions can cause a persistent positional vertigo with down beat, up beat or torsional nystagmus.

Imbalance in the absence of typical vertigo may be seen in patients with bilateral vestibular loss, significant peripheral neuropathy (diabetes) or spinal cord dorsal column lesions (compressive, vitamin B_{12} deficiency, syphilis). Other causes include cerebellar atrophy, white matter disease, normal pressure hydrocephalus and extrapyramidal disorders (Parkinson's disease, progressive supranuclear palsy). Patients with recurrent or persistent vertigo of more than 6 weeks' duration should be investigated neuro-otologically (Luxon, 1997).

INVESTIGATION OF DIZZINESS

Investigation of the dizzy patient may include some or all of the following tests based on history and clinical findings (*Table 4*).

A magnetic resonance imaging brain scan may be required to rule out cerebello-pontine angle tumours, e.g. vestibular schwannoma. Transcranial Doppler ultrasound is becoming increasingly popular in the evaluation of vascular dizziness.

CONCLUSIONS

Dizziness is a vague symptom, which may be a manifestation of wide-ranging pathological processes. Chronic episodic vertigo resulting from inadequately compensated peripheral vestibular disorder is an important cause of morbidity and time taken out of work. Rehabilitation of patients with peripheral vestibular dysfunction is aimed at improving central compensation by means of physiotherapeutic measures with or without medical and or psychotherapeutic intervention. HM

Conflict of interest: none.

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TABLE 4.
Investigations for dizziness

Audio-vestibular	Pure tone audiogram
	Stapedial reflexes
	Auditory brainstem responses
	Electronystagmography
	Posturography (Equitest, Neurocom International, Clackamas, Oregon, USA)
	Caloric test
Haematological/ biochemical	Full blood count
	Urea and electrolytes
	Fasting blood sugar
	Serum lipids
	Thyroid function tests
	Serology for syphilis
	Autoimmune profile
Radiological	Magnetic resonance imaging
	X-ray cervical spine
	Magnetic resonance angiography
	Transcranial Doppler ultrasound

KEY POINTS

- The aetiology of dizziness is diverse.
- A thorough history and careful clinical assessment will lead to appropriate diagnosis in the majority of instances.
- Peripheral vestibular dysfunction is the commonest cause of dizziness. A structured exercise programme will usually lead to symptomatic recovery.
- Prolonged use of vestibular sedatives may interfere with central compensation, resulting in delayed recovery.
- Rehabilitation of bilateral vestibular failure consists of measures to maximize and optimize visual and proprioceptive sensory inputs.
- Chronic central vertigo is a management challenge because the mechanisms are poorly understood. Persistent vertigo of more than 6 weeks warrants detailed neuro-otological assessment.
- In some patients, an accurate diagnosis may emerge only after a period of review.