Vertigo

Ajay Mehra

Vertigo is a common symptom encountered in clinical practice by all practitioners. Vertigo is an illusion of movement either of the person or of object about the person. It requires systematic approach to diagnose and manage these patients. The causes range from benign diseases to fatal life threatening conditions. The patients history is the best means to establish a diagnosis, as the physical examination and diagnostic test results are often normal.

INTRODUCTION

Most clinicians find it difficult to diagnose dizzy patients, as the potential causes span subspecialties including internal medicine, neurology, otology, ophthalmology and psychiatry.

The term Vertigo is often loosely used by patients, and some times also by physicians. It is defined as an illusion of movement either of the person or of objects about the person, and is quite distinct from mere dizziness, unsteadiness or giddiness.

True vertigo is an unpleasant sensation of imbalance, spinning and disorientation. Nausea and vomiting are common and, during an acute attack, many patients are understandably terrified and frightened that they are going to die.

Vertigo is by no means uncommon, occurring in about 5% of patients consulting a general practitioner and in about 10% of patients examined by the otorhinolaryngologist.

Vertigo is an illusion or hallucination of motion either of oneself or the environment. The feeling may involve the whole body or be limited to head it is always accompanied by imbalance / disequilibrium.

Dizziness

Dizziness is a lay term, frequently misused by both patients and their physicians. It may be used to describe a variety of cephalic sensations including the more specific symptom of vertigo. In general, however, the term dizziness covers sensations like:

- Light headedness
- Faintness
- Unsteadiness of gait without any actual feeling in the head or cephalic sensations.
- The brief period of loss of awareness or contact with the environment that accompanies complex partial seizures.

Dysequilibrium

Dysequilibrium is defined as a loss of balance. It may or may not be associated with a cephalic sensation or vertigo. Dysequilibrium may however occur unaccompanied by vertigo. It is usually present only in the standing position or while walking. In order to understand dysequilibrium it is important to define equilibrium.

Equilibrium is defined as the ability to maintain body posture and a sense of awareness of the body parts

Address for correspondence

D.M. (NEUROLOGY), D.N.B. (NEUROLOGY)
Consultant Neurologist,
Chandrapur.

and their position in relation to self and the external environment. It depends upon the continuous provision of visual, labyrinthine (vestibular), proprioceptive information and its integration in the brain stem and the cerebellum.

Dysequilibrium thus results from diseases that affect:

Peripheral or central vestibular pathway.

Cerebellum

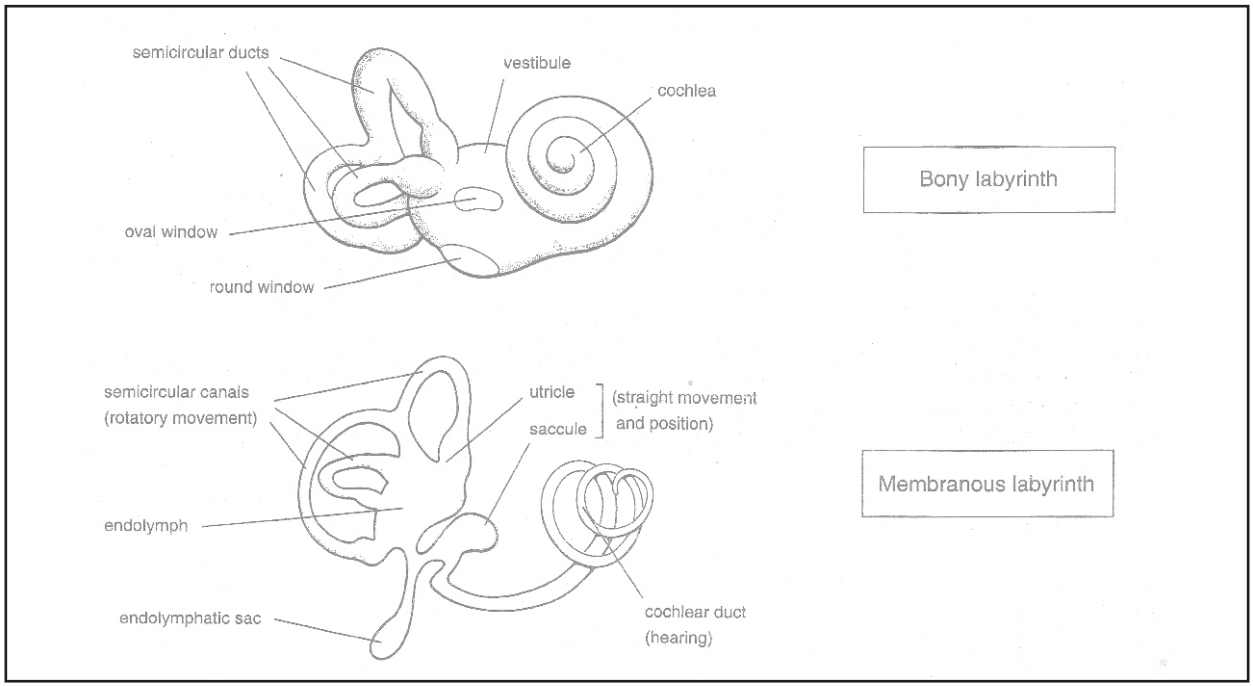
Sensory pathways involved in proprioception.

Symptoms as a clue to diagnosis

Feature	Suspected diagnosis
Presentation type of dizziness	
Rotational vertigo	Acute vestibular disorder (central or peripheral)
Positional vertigo	Benign paroxysmal positional vertigo (BPPV), migraine, central positional vertigo
Unsteadiness	Bilateral vestibular dysfunction, neurological disorder (e.g. polyneuropathy, myelopathy, normal pressure hydrocephalus, cerebral small-vessel disease, cerebellar disorder)
Non-specific dizziness	Orthostatic hypotension, drug toxicity, psychogenic
Duration of attacks	
Seconds	Vestibular paroxysms, cardiac arrhythmia, BPPV
Few minutes	TIAs, panic attacks, migraine
20 minutes to several hours	Meniere attacks, migraine
Days to weeks	Vestibular neuritis, brainstem/cerebellar stroke or demyelination, migraine
Days to weeks	Vestibular neuritis, brainstem/cerebellar stroke or demyelination, migraine
Persistent	Fixed neurological deficit, bilateral vestibular failure, chronic intoxication, psychogenic
Triggers	
Changes of head position	BPPV, other positional vertigo
Menstruation, sleep deprivation	Migraine
Moving visual patterns	Visual vertigo
Elevators and other close spaces, crowds, heights, leaving the house	Panic attacks
Loud noises, Valsalva manoeuvres Standing up	Fistula syndromes Orthostatic hypotension

Associated symptoms	
Photophobia, headache or visual auras	Migraine
Hearing loss, tinnitus, Fullness in the ear	Meniere's disease, autoimmune inner ear Disease, acoustic neuroma
Blackening out of vision, syncope	Vasovagal reaction, orthostatic hypotension, Cardiac arrhythmia
Red eyes, skin rashes, renal disease, arthritis	Autoimmune inner ear disease
Palpitations, choking trembling catastrophic thoughts panic	Anxiety disorder
Diplopia, dysarthria, numbness, paresis clouded consciousness	Posterior fossa lesion (Including ischemia) basilar migraine.

ANATOMY OF THE INNER EAR



The membranous labrynth of the inner ear is filled with endolymph and surrounded by the perilymph and comprises of the:

Cochlea - concerned with hearing.

Vestibular apparatus - concerned with maintenance of body equilibrium.

The vestibular apparatus includes :

Semicircular canals

Anterior - lies in the vertical plane.

Posterior - lies in the vertical plane.

Lateral - lies in the horizontal plane.

The three semicircular canals have a terminal swelling at one end, the ampulla, and are oriented 90 degree to each other and sense rotational acceleration in all three planes. Receptor hair cells embedded in the gelatinous Cupula, record movements of the cochlea endolymph produced by head movement.

Otolith Organs

Utricle - horizontally oriented

Saccule - Vertically oriented

The receptor hair cells in the utricle and saccule sense linear acceleration.

EXAMINATION OF DIZZY PATIENT

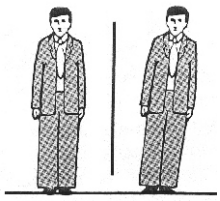
Examination	Interpretation
Spontaneous nystagmus	Peripheral or central vestibular disorder Peripheral: horizontal-torsional, increases without fixation Central: any direction (up, down, torsional, horizontal)
Clinical VOR assessment (head-thrust test)	Detects major peripheral vestibular loss (i.e. >60%)
Eye movements (pursuit, saccades, VOR suppression)	Abnormalities indicate central lesion
Positional manoeuvre	Identifies benign paroxysmal positional vertigo (and only rarely posterior fossa lesion)
Romberg test: normal unidirectional variable swaying with eyes open swaying after eye closure	In most dizzy patients Acute vestibular lesion Acute fall cerebellar/brainstem lesion Dorsal-column spinal disorder / large-fibre neuropathy
Gait abnormalities	Cerebellar, parkinsonian, spastic, apraxic, neuropathic disorders
Gait with eyes closed or Unterberger test	Ipsilesional deviation in peripheral lesions
Postural responses to trunk pushes	Impaired in parkinsonian syndromes

VOR = vestibulo-ocular reflex

Traditional diagnostic tests for vertigo.

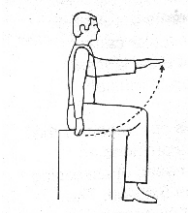
Romberg test

The patient stands with his feet together and eyes closed. A normal subject is stable, but a patient with vertigo tends to lean away from the vertical in an attempt to compensate. For the sensation of movement which he feels. He leans towards the side on which the damaged labyrinth lies.

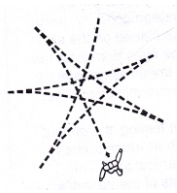


Barany's pointing test

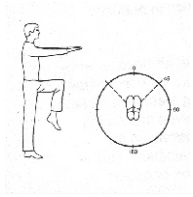
An object is held in front of the seated patient who is then asked to close his eyes and point repeatedly to the object. If the patient has a labyrinth which is not functioning properly he will experience an illusion of the movement of the object and will point past it.



Babinski-Weill's test
The patient, with his eyes closed, repeatedly takes five steps forward and five steps back for 30 seconds. When there is a unilateral vestibular disorder, the patient will start walking in a star shape.



Unterberger's test
The patient closes his eyes and stretches his arms horizontally in front of him. He then walks on the spot, raising the knees as high as possible for one minute. Where there is a vestibular disorder, the patient turns about his axis.

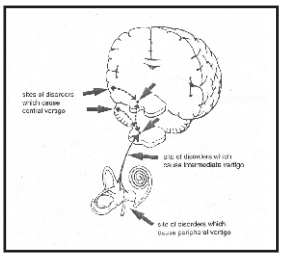


Laboratory Investigations for Dizzy Patients

Test	Interpretation
Pure-tone audiometry	Essential for Meniere's disease (often low frequency loss) Red flag for acoustic neuroma Normal in most other vestibular disorders
Brainstem auditory evoked potentials (BAER)	Useful screening for acoustic neuroma in patients with unilateral auditory symptoms
Caloric and rotational tests	Caloric canal paresis: lack of response from one ear, often observed in peripheral vestibular disorders Directional preponderance (on caloric or rotational tests): indicates vestibular asymmetry—non-specific
Eye-movement recording (electro-/ video-oculography)	Do not replace clinical eye-movement examination May help to detect central Dysfunction
MRI consider when: central symptoms/signs atypical positional nystagmus progressive unilateral sensorineural hearing loss	Identification of posterior fossa lesion (MRI superior to CT)

MRI = magnetic resonance imaging; CT = computed tomography

SITES OF LESIONS CAUSING VERTIGO



Vertigo can be caused by a lesion in any part of the vestibular system,

and some clinicians like to classify vertigo according to the site of the problem.

1. Peripheral (vestibular) vertigo
A disordered generation of nerve impulses by the vestibular apparatus.

2 Intermediate vertigo
Faulty transmission of impulses along the VIII th cranial nerve.

3. Central vertigo
Misinterpretation of information by the vestibular nuclei in the brain.

Causes of Peripheral Vestibular Disorders

Acute and recurrent vestibulopathy.
Benign positional vertigo.
Post-traumatic vertigo.
Meniere's syndrome.
Drug-induced vertigo.

Miscellaneous rare causes : Acoustic neuroma, ischemia, Bacterial otitic infection, Syphlitic labrynthitis, Basal meningitis, Ramsay Hunt syndrome, Endolymphatic fistulae

Causes of Disorders of Central Vestibular Pathways

Vertebrobasilar occlusive disease (TIA & Stroke).
Cerebellar and/or brainstem hemorrhage.
Basilar artery migraine.
Demyelinating disease.
Vertiginous epilepsy.
Drug induced.
Posterior fossa neoplasms.
Posterior fossa malformations.

Miscellaneous rare intramedullary brainstem lesions:
AV malformation, vasculitis, metastatic disease, granulomas, brainstem encephalopathies.

PATTERNS OF VERTIGO

Vertigo is always a symptom and never a disease. At least eighty disorders are reported to have vertigo as a possible symptom, so it is perhaps unsurprising that in about 40% of cases a clear and definite diagnosis is not made.

A distinctions may be made between three patterns of vertigo. Firstly there are those attacks which come on suddenly, last for a few hours or days, and then disappear only to recur at a later date (i.e. acute, recurrent vertigo) Secondly there are those attacks which also come on suddenly, but which are more persistent, lasting perhaps weeks or months until the underlying disorder improves. A third group consist of vertigo of gradual onset, where the symptom is persistent and chronic.

1. Acute, recurrent (episodic) vertigo.

Acute recurrent vertigo may be due to various causes. A classic cause, and one which is often associated with considerable therapeutic difficulties, is Meniere's disease.

The vertigo of Meniere's disease can be so violent that the patient has to go straight to bed and lie still in the dark, prostrated with nausea and vomiting and a sensation of fullness in the ear. Attacks usually last less than 24 hours, but may continue for several days. Nystagmus (rapid eye movement) is seen in all patients during the acute episode.

In the early stages, vertigo may be the only symptom. However, by definition, it later progresses into a triad of symptoms - vertigo, fluctuating hearing loss and tinnitus. As the disease progresses, deafness may worsen until it becomes complete, unless treatment is started early. Sometimes there is distortion of hearing rather than just hearing loss, with hyperacusis and diplacusis.

Tinnitus can precede an attack and thus represent a pre-Meniere's warning (an 'aura'). Rarely, tinnitus may be persistent, and can remain even after destruction of the inner ear by surgery, causing intolerable stress to the patient.

Not all cases of acute recurrent vertigo without deafness will progress to Meniere's disease. For example, some may be due to migraine, hypoglycaemia or other causes; often, however, the underlying cause of recurrent vertigo remains undiagnosed. In recent years, there has been a trend towards referring to vertigo of uncertain origin as Meniere's disease, even if there is no evidence at the time for believing that auditory symptoms will develop. Note that clinical studies have shown that Meniere's disease is often reversible if diagnosed very early and appropriately treated

Causes of Recurrent Vertigo

Peripheral causes

Benign positional vertigo.
Meniere's syndrome.
Otosclerosis.
Perilymph fistula.

Central causes

Vertebrobasilar ischemia.
Migraine.
Vertiginous seizures.

Recurrent vertigo: diagnoses with key features

Disorder	Key features
Migrainous vertigo	Attacks of spontaneous or positional vertigo lasting seconds to days; history of migraine; migrainous symptoms during vertigo; migraine-specific precipitants provoking vertigo
Benign recurrent vertigo (probable migrainous vertigo)	Same dinical features as migrainous vertigo, but relationship to migraine is less obvious, e.g. no personal history of migraine, lack of migraine symptoms during the attack
Meniere's disease	Vertigo attacks lasting 20 minutes to several hours with concurrent hearing loss, tinnitus and aural fullness Progressive hearing loss over years
Vertebrobasilar transient ischaemic attacks	Attacks of vertigo lasting minutes, often accompanied by ataxia, dysarthria, diplopia or visual-field defects Elderly patients with vascular risk factors
Paroxysmal recurrent vertigo	Brief attacks of vertigo (seconds) several times per day with or without cochlear symptoms; often good response to carbamazepine Supposedly caused by vascular compression of the eighth nerve
Perilymph fistula	Vertigo after head trauma, barotrauma, stapedectomy, often provoked by coughing, sneezing, straining or loud sounds Symptom duration variable
Other rare causes	Autoimmune inner-ear disease, syphilis of the inner ear, schwannoma of the eighth nerve, vestibular epilepsy, insufficient compensation of unilateral vestibular loss, otosclerosis, Paget's disease, episodic ataxia type 2, familial hemiplegic migraine

Meniere's disease: key features

History	Attacks of spinning vertigo lasting 20 minutes to several hours with unilateral tinnitus, hearing loss and aural fullness. Fluctuating hearing loss with recovery in the early stages and progression later on
Clinical findings	Initially normal between attacks; later hearing loss on the affected side
Pathophysiology	Increased pressure within the endolymphatic space (hydrops) leading to mechanical and chemical irritation of the labyrinthine sensory organs
Investigations	Repeated audiometry to document fluctuating low-lone hearing loss in the early stages and then progressive hearing loss Electro-cochleography in doubtful cases; caloric testing may show unilateral

	canal paresis
Treatment	Vestibular suppressants for acute attacks; trial of betahistine or low-salt diet+/- diuretics for prophylaxis; intralympanic gentamycin, labyrinthectomy or vestibular neurectomy for refractory patients with frequent and severe attacks

Vertiginous transient ischaemic attack: key features

History	Spontaneous attacks of vertigo with abrupt-onset lasting minutes, often with other posterior circulation symptoms, e.g. facial numbness, dipiopia Elderly patients with vascular risk factors
Clinical findings	Examination may reveal signs of atherosclerosis and of previous cerebral or inner-ear ischaemia, e.g. a carotid bruit, hemiparesis or hearing loss
Pathophysiologv	Transient hypoperfusion of the labyrinth, less commonly of the vestibular nuclei or cerebellum
Investigations	Audiometry and calorics to search for labyrinthine damage; vascular studies: neck vessel ultrasound, MR-angiography; cerebral angiography in selected patients only
Treatment	Control of risk factors; antiplatelet drugs; rarely anticoagulation

2. Vertigo of sudden onset, which gradually diminishes over time

Certain conditions may give rise to vertigo which starts as an unexpected acute attack but then persists, improving gradually over weeks or months as the underlying disorder resolves itself.

Viral labyrinthitis is an example of this, in which the vertigo is present more or less constantly for several days or weeks, but subsides as the viral infection improves.

Bacterial labyrinthitis is now relatively uncommon in Westernised communities, though is still an important cause of vertigo in other parts of the world, often arising through inadequate treatment of otitis media.

Acute, recurrent positional (benign) vertigo is a common diagnosis, though it is often difficult to define the underlying cause. In this condition, vertigo is induced by rapid positional change, usually when the patient is in a supine position and the head is tilted to the right or left. The disorder is self-limiting, but can persist for several months, Drug therapy can be useful in the early stages, though it is important to choose a drug which does not decrease the normal adaptation responses.

Vestibular neuronitis is often a misnomer in the sense that neuronal inflammation may be just one of several possible causes. It involves an acute unilateral loss of vestibular function, characterised by sudden intensive rotatory vertigo. Drug therapy can be useful during the first 3-4 days, though again the medication should not decrease the normal adaptation processes.

Single episode of prolonged vertigo: diagnoses with key features

Disorder	Key features
Vestibular neuritis	Acute onset of vertigo, nausea and imbalance Spontaneous nystagmus towards healthy ear, unilateral VOR failure, falls towards affected side improvement over days to weeks.
Acute brainstem or cerebella lesion (e.g. stroke, demyelination)	Vertigo with brainstem or cerebellar signs Variable time course MRI usually shows lesion affecting central vestibular pathways

First attack of migrainous vertigo	Acute vertigo may last for days. Mostly central types of vestibular nystagmus and ataxia History of migraine and often migrainous features during attack
Fist attack of Meniere's disease	Vertigo lasting hours may be isolated symptom in early Meniere's disease Otherwise, associated hearing loss, tinnitus and aural fullness
Other causes	Labyrinthine infarction, perilymph fistula, bacterial labyrinthitis. drug/alcohol toxicity

VOR = vestibulo-ocular reflex; MRI- magnetic resonance imaging

Vestibuiar neuritis: key features

History	Acute onset of vertigo, nausea, vomiting and veering to one side
Clinical findings	Spontaneous recovery over days or weeks Spontaneous nystagmus towards healthy ear, partly suppressed by visual fixation Unilateral VOR failure with rapid head rotation towards involved side, directional postural imbalance towards affected side
Pathophysiology	Probably viral infection of the vestibular nerve leading to sudden asymmetry of neural activity in the vestibular nuclei.
Investigations	Caloric testing; audiometry not required in clear cases; MRI only when there are neurological abnormalities or in patients with vascular risk factors
Treatment	Exercise therapy promotes restoration of balance-Some evidence in favour of oral steroids for three weeks

Clinical criteria for requesting magnetic resonance imaging in *a* patient with acute vertigo

Clinical criterion	Suspected diagnosis
Abrupt onset of vertigo against a background of old age or vascular risk factors	Ischaemia of the labyrinth or root entry zone of the eighth nerve?
Abrupt onset of profound hearing loss	Labyrinthine infraction due to AICA occlusion Concomitant brains tern/ cerebellar infarction?
Previous episodes of unexplained neurological symptoms for days or weeks in a young patient	Undiagnosed MS, acute lesion in the root entry zone of the eighth nerve?
Central oculomotor findings : saccadic pursuit, pure torsional nystagmus, upbeat/downbeat nystagmus, gaze-evoked nystagmus, central positional nystagmus, skew deviation	Involvement of central vestibular structures in the brainstem or cerebellum?
Cranial nerve abnormalities	Brainstem lesion or tumour /inflammation close to the brainstem ?

Long tract signs: hemi-/ quadriparesis, extensor plantar responses, hemisensory loss, Homer's syndrome	Brainstem lesion?
Cerebellar signs: truncal/ limb ataxia, dysarthria	Cerebellar lesion?

"Skew deviation designates a vertical misalignment of the eyes of central origin. In contrast to squints resulting from paresis of extraocular muscles, skew deviations show a stable squint in all directions of gaze.
AICA = anterior inferior cerebellar artery, MS = multiple sclerosis

3. Vertigo of gradual onset, which persists.

Chronic vertigo which develops slowly can be due to underlying diseases which progressively worsen in severity over the years. For example, tumours pressing on the VIIIth cranial nerve or brainstem can produce vertigo, as can cases of meningitis in which the VIIIth cranial nerve is affected.

A number of systemic conditions can also produce chronic vertigo, such as arteritis, diabetes, hypertension and generalised atherosclerosis.

Trauma to the brain or vestibular apparatus resulting from whiplash injuries or skull fractures can also result in a chronic vertigo.

Causes Of Non Vertiginous Dizziness

- Cerebral hypoperfusion - cardiac arrhythmias, cardiac valvular disease, postural hypotension.
- Hyperventilation syndrome or psychosomatic disturbance.
- Multiple sensory deficits.
- Drug-induced.
- Miscellaneous neurologic disorders affecting brainstem, cerebellum, or spinal cord posterior columns.
- Endocrinopathies.

Recurrent dizziness: diagnoses with key features

Disorder	Key features
Orthostatic hypotension	Brief episodes of dizziness lasting seconds (to minutes) after standing up; relieved by sitting/lying down; drop of systolic blood pressure of >20mmHg after standing up
Cardiac arrhythmia	Dizziness lasting seconds; may be accompanied by palpitations; can be caused by bradycardia <40/s or tachycardia >170/s
Psychogenic dizziness	Variable duration from minutes to permanent; usually related to anxiety or depression Often provoked by specific situations such as leaving the house, riding on buses or driving, height, crowds, lilts Accompanied by choking, palpitations, tremor, body warmth, anxiety
Drug-induced dizziness	Variable clinical presentation according to pharmacological mechanism: sedation, vestibular suppression, ototoxicity, cerebellar toxicity, orthostatic hypotension, hypoglycaemia
Other rare causes	Hypotension, metabolic disorders, height vertigo

Orthostatic hypotension: key features

History	Brief episodes of dizziness lasting seconds two minutes after assuming an upright posture; relieved by sitting/ lying down; may be followed by syncope Risk factors: old age, dehydration, heat, carbohydrate meals, prolonged bed rest, various drugs
Clinical findings	Drop of systolic blood pressure of >20mmHg after standing up
Pathophysiology	Often multifactorial: autonomic failure, volume depletion, vasodilatation, associated anaemia Reflex mechanism in neurally mediated syncope
Investigation	Orthostatic blood pressure; heart rate variability; sometimes other autonomic function tests including tilt table
Treatment	Elimination of offending drugs; increased salt and fluid intake; frequent small meals; head and trunk elevation at night; fludrocortisone, midodrine, erythropoetin Patient education (raise slowly); orthostatic training for neurally mediated syncope

Factors causing or aggravating orthostatic hypotension

Salt/volume depletion
Prolonged bed rest
Fever
Heat
Hyperventilation
Drugs (diuretics, vasodilators, antihypertensives, dopaminergics, anticholinergics)
Anaemia
Bilateral carotid stenosis

Typical triggers for neurally mediated syncope

Prolonged standing
Warm environment
Emotions of fear/helplessness
Sight of blood or injection needle
Venipuncture or any other invasive medical procedure
Sudden pain
Micturition

Cardiac arrhythmia: Key features

History	Episodes with lightheadedness lasting seconds (to minutes), sometimes with palpitations; may evolve to syncope Often history of heart disease
Clinical findings	Either normal in the interval or clinical signs of heart disease.
Pathophysiology	Both bradycardia (<40/s) and tachycardia (> 170/s) may interfere with adequate perfusion of the brain
Investigations	Electrocardiogram; Holler monitoring; rarely invasive electro-physiological testing or implantable loop recorders
Treatment	Correction of metabolic or electrolyte disturbances; antiarrhythmic drugs; cardiac pacemaker; implantable cardioverter defibrillator; radio-frequency ablation

Arrhythmia syndromes that may induce dizziness

Tachycardias
Sinus tachycardia
Atrial tachycardia
Atrial flutter/fibrillation
Atrioventricular tachycardia
Pre-excitation syndromes
Ventricular tachycardia

Bradycardias
Sick sinus syndrome
Atrioventricular block
Atrial fibrillation with bradyarrhythmia

Psychogenic dizziness: key features

History	Episodic (occasionally permanent) dizziness, often accompanied by autonomic symptoms and catastrophic fears; may coexist with vestibular disorder (psychiatric overlay)
Clinical findings	Normal with pure psychogenic dizziness, but abnormalities from subclinical vestibular dysfunction or vestibular disease not uncommon
Pathophysiology	Often related to anxiety disorders with misinterpretation of physiological signals and conditioning responses; sometimes triggered by experience of vestibular dysfunction
	Additional hyperventilation causes dizziness from cerebral hypoperfusion
Investigations	Vestibular testing, audiometry, psychiatric opinion
Treatment	Behavioral therapy, anxiolytics, vestibular rehabilitation

Drugs causing dizziness and imbalance

Mechanism	Class of drugs	Sample drug
Sedation	Tranquillisers	Diazepam, alprazolam
	Barbiturates	Phenobarbitone
	Aliphatic phenothiazines	Chlorpromazine
Vestibular suppression	Antihistamines	Dimenhydrinate, promethazine
	Benzodiazepines	Diazepam, lorazepam]
	Anticholinergics	Scopolamine
Ototoxicity	Aminoglycosides	Gentamyein, streptomycin
	Glycopeptide antibiotics	Vancomycin
	Alkylating agents	Cisplatin
	Loop diuretics (reversible)	Furosemide, ethacrynic acid
	NSAIDS (reversible)	Aspirin, ibuprofen
	Antimalarial drugs (reversible)	Quinine, quinidine
Cerebellar toxicity	Antiepileptics	Carbamazeine, phenytoin, phenobarbitone
	Benzodiazepines	Diazepam, clonazepam
	Inorganic salt	Lithium salt
Orthostatic hypotension	Diuretics	Thiazide diuretics, furosemide
	Vasodilators	Nitroglycerine, isosorbide
	Beta-blockers	Propranolol, metoprolol
	Alpha-blockers	Phenoxybenzamine, prazosin

	Calcium-channel blockers	Nifedipine
	ACE inhibitors	Captopril, enalapril
	Tricyclic antidepressants	Amitryptyline
	Aliphatic phenothiazines	Clorpromazine
	Dopaminergics	L-Dopa, pergolide
	MAO inhibitors	Tranylcypromine
Hypoglycaemia	Antidiabetics	Insulin, Sulfonylurea
	Beta-blockers	Propranolol
	MAO inhibitors	Tranylcypromine
Unknown	Antimalarial	Mefloquin
	Quinolone antibiotics	Ofloxazin, trovafloxacin
	And many more...	

ACE = angiotensin-converting enzyme; MAO = monoamine oxidase

Symptoms, signs and mechanisms of drug-induced dizziness

Symptoms/signs	Mechanism	Sample drug
Imbalance, drowsiness, poor concentration	Sedation	Diazepam, alprazolam Phenobartaitone
Imbalance, drowsiness poor concentration	Vestibular suppression	Dimenhydrinate, promethazine Diazepam
Imbalance (worse in the dark), oscillopsia during head movement, bilateral VOR loss	Ototoxicity	Gentamycin, streptomycin Cisplatin
Drowsiness, gait and limb ataxia, dysarthria	Cerebellar toxicity	Carbamazepine, phenytoin lithium
Orthostatic dizziness, blackouts, syncope	Orthostatic hypotension	Nitroglycerine, furosemide, propranolol, captopril amitryptiline, l-dopa

VOR = vestibulo ocular reflex

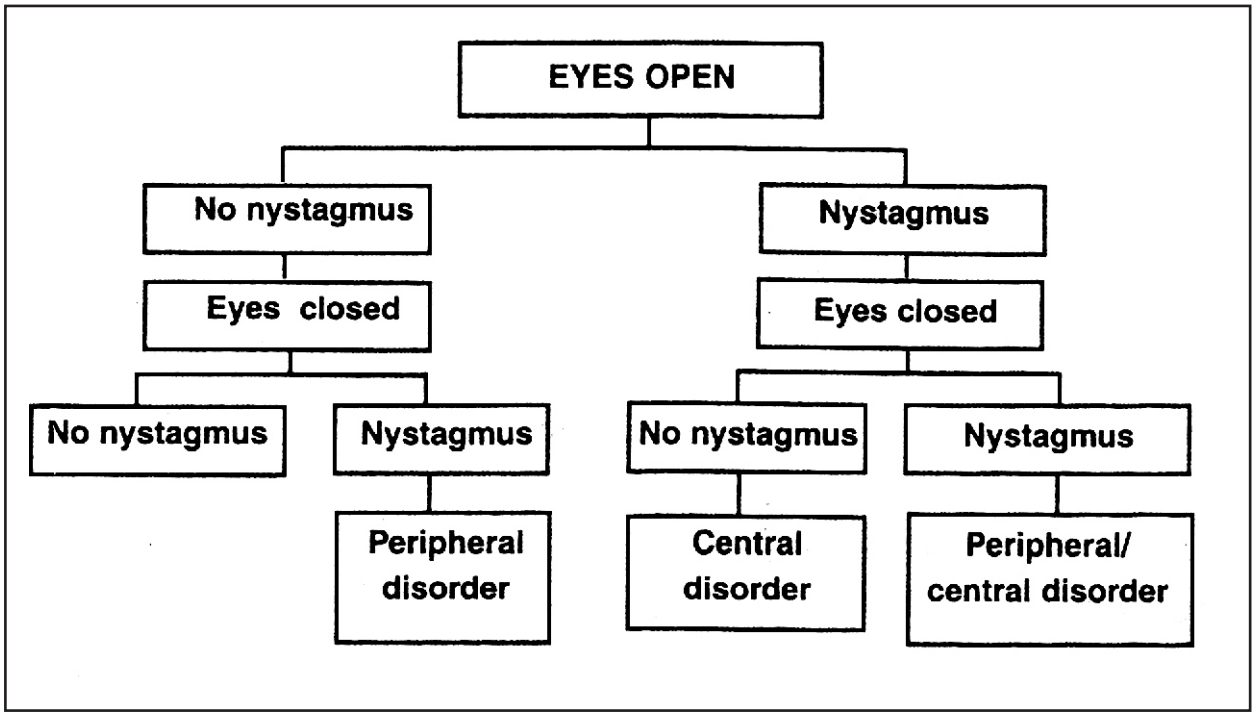
Guide For Peripheral Versus Central Vestibular Disorders

	Peripheral	Central
CNS exam/symptoms	-	+/+++
Auditory exam/symptoms	Frequently +	Usually -
Unsteadiness:		
Acute	+++	+++
Chronic	+/-	+++
Eye-movements	Normal	Usually abnormal
Vertigo:		
Acute	+++	++
Chronic	+/-	+/-

Guide for peripheral versus central nystagmus

	Peripheral	Central
Plane[a]	Horizontal, or horizontal > torsional	Any
Amplitude:		
Acute	++	++/+++
Chronic	+/-	++/+++
Fixation removal*	Appears/enhances	Variable
Waveform[c]	Rectilinear	Exponential (jerky), pendular

[a]This table is for spontaneous nystagmus since peripheral positional nystagmus as in BPPV is predominantly torsional.
[b]By oculography, Frenzel's glasses, infrared viewer or funduscopy in the dark.
[c]By oculography only (specialist procedure).



Interpretation of spontaneous nystagmus
Treatment Of The Dizzy Patient

■There are four components to the treatment of a patient with dizziness or vertigo:

- treatment of the specific condition, such as benign paroxysmal positional vertigo (BPPV) or migraine, to name two of the more common ones
- counselling and reassurance
- rehabilitation

- pharmacological treatment of vertigo and associated nausea

These four aspects should he considered for each individual patient and they are all equally important . However, not all patients will require action in all four domains. For instance, patients with BPPV will usually require repositioning treatment and nothing else. Patients with chronic dizziness usually require counselling and rehabilitation but no drugs. In contrast, patients with migrainous vertigo, if they do not have interictal symptoms, may just require antimigraine drugs but no rehabilitation. As always in medicine, the treatment has to be tailored to the individual patient.

Key elements in the treatment of dizzy patients

Specific treatment of the underlying condition	Examples: Positional maneuvers for BPPV Migraine prophylaxis for migrainous vertigo Gentamycin for Meniere's disease Antiplatelet agents for TIAs Cognitive- behavioural therapy for anxiety disorders
Reassurance, information, counselling	Relieves unnecessary fears; provides the basis for therapeutic cooperation and realistic goals
Vestibular rehabilitation	For treatment of chronic dizziness and imbalance. Includes exercises for; Eye-head coordination Retraining of balance strategies Gail training Visual desensitisation Ball games
Non-specific drug treatment of acute vertigo, nausea and vomiting	For example: dimenhydrinate, prochlorperazine, promethazine
Other treatments	Surgery (e.g. refractory BPPV, Meniere's) Stress management Self-help groups

BPPV =- benign paroxysmal positional vertigo; TIA = transient ischaemic attack

Patient reassurance information and counsellin

Vertigo is a terrifying experience, particularly the very first attack. Most patients associate the sensation with life-threatening or incapacitating conditions such as a heart attack, stroke or even imminent death. Patients with conditions such as BPPV or vestibular neuritis need to be reassured that, despite the unpleasant symptoms they experience, the underlying cause is benign.

The patient with acute vertigo also needs a brief explanation on the process of vestibular compensation. At this point reassurance on the power of the brain to make up for the loss of peripheral function is needed. This is also a good opportunity to explain that rest, in particular bed rest, is not indicated - quite the contrary. They should understand that for (he process of vestibular compensation to be successful they should try to remain active and limit activity moderately only during the hyperacute phase. However, patients 'too keen' should also be told not to overdo it, because of the possibility that nausea or vomiting could lead to exhaustion and future negative associations between physical activity and nausea

Patients with long-term dizziness need quite a lot of reassurance. These patients have often wandered from clinic to clinic, from specialist to specialist, sometimes for months or years. On the basis of normal brain scans some doctor is likely to have said, at some point, 'there's nothing wrong with you', or 'there's nothing that medical science can do for you' or 'it's all in your mind'. We believe that this is not only the wrong approach but also usually not true. Patients may have suffered a genuine vestibular insult in the past, which may not show in conventional vestibular testing, but failures in the vestibular compensation process or secondary added psychological problems complicate the situation. It is a fact that rehabilitation works even in patients with many years of

chronic dizzy symptoms, so you want your patient to cooperate in this process. For this reason you need to explain' the principles of vestibular compensation and rehabilitation. You rnay also need to mention that symptoms of anxiety and depression are very common in patients with dizziness but that this doesn't imply that the symptoms are imaginary or, worse, the result of malingering.

In departments seeing large numbers of patients with dizziness and vertigo it is customary to have leaflets with patient-oriented information.

Rehabilitation

A key aspect in rehabilitation of balance disorders is the understanding that control of balance emerges from an interaction between many sensorimotor systems. The purpose of rehabilitation is to facilitate the ability of the central nervous system (CNS) to compensate for lesions in the vestibular system. The neural basis for vestibular compensation is distributed throughout the nervous system such that lesions in the cerebellum, cortex, spinal cord, brainstem or sensory systems can prevent or reduce the capacity for compensation. Vestibular compensation is a plastic process that allows the CNS to redress functional symmetry after a unilateral peripheral vestibular lesion. Patients with both unilateral and bilateral vestibular loss also compensate by a process of sensory substitution. In both cases, but particularly the latter, patients learn to rely more on non-vestibular information for balance, namely visual and proprioceptive inputs.

Essentially these consist of eye, head and postural exercises of progressive complexity The exercises the patient is asked to perform are intended to include the eye, head or body positions and movements that provoke vertigo. Since the aim is to stimulate the vestibular system, patients should come off medication as early as possible. However, medication can be used to suppress symptoms during the early stages of therapy.

The exercises should be performed for 10-15 minutes twice a day. Pacing of the exercises is crucial. Unless they are performed slowly at first they will induce an unacceptable degree of vertigo and nausea. However, the patient should be instructed to gradually increase the pace and difficulty of the exercises as the provoked vertigo progressively abates. It is important to foster positive but realistic expectations. Patients must be told that their symptoms will at first worsen, and that improvement may be uneven. Clinical trials of the efficacy of exercise programmes typically report improvement in symptoms in 70—80% of those participating.

Movements typically included in vestibular rehabilitation programmes

Head exercises (performed with eyes open and closed)

Bend head backwards and forwards

Turn head from side to side

Tilt head from one shoulder to the Other

Fixation exercises

Move eyes up and down, side-to-side

Perform head exercises while fixating stationary target

Perform head exercises while fixating moving target

Positioning exercises (performed with eyes open and closed)

While seated, bend down to touch the floor

Bend down with head twisted first to one side and then the other

Lying down, roll from one side to the other

Sit up from lying on the back and on each side

Repeat with head turned to each side

Postural exercises (performed with eyes open; eyes closed under supervision)

Practice static stance with feet as close together as possible

Practice standing on one leg, and heel-to-toe

Repeat head-fixation exercises while standing and then walking

Practise walking in circles, pivot turns, up slopes, stairs, around obstacles

Standing and walking in environments with altered surface and/or visual conditions with and without head and fixation exercises

Aerobic exercises including alternative touching the fingers to the toes, trunk bends and twists, etc.

Drug treatment of vertigo, nausea and vomiting

Pharmacological treatment of vertigo is mostly symptomatic since no drug has been shown to influence a vestibular disorder at the causal level. Two classes of drugs are used for acute vertigo: vestibular suppressants and antiemetics.

Table II – Drugs used in Vertigo

No.	Drug	Dosage	Route
1	Dimenhydrinate (Dramamine)	50-100mg (4-6 hrly)	PO, IM, IV, PR
2	Diphenhy dramine (Benadryl)	25-50 mg (TDS or QID)	PO, IM, IV
3	Promethazine (Phenargan)	25 mg (BD or QID)	PO, IM, IV
4	Meclizine (Antivert)	12.5 to 25 mg (BD or QID)	PO, IM, IV, PR
5	Cyclizine (Marezine)	50 mg (4-6 hrly)	PO
6	Hydroxyzine (Vistaril)	25-100 mg (TDS or QID)	PO, IM
7	Prochlorperazine	5 mg (TDS)	PO, IM
8	Clonazepam (Rivotril, Clonotril Petril)	0.5 mg (TDS)	PO
9	Diazepam	5 mg (TDS)	PO, IM, IV
10	Betahistine Hydrochloride (Vertin)	8 mg (TDS)	PO
11	Cinnirazine (Stugeron / cinzan)	25-75 mg (TDS)	PO

How to use vestibular suppressants and antiemetics

The choice of a vestibular suppressant depends on the clinical situation and expected side-effects of the drug. In particular, potency and sedating effects of vestibular suppressants are closely related. Thus, for patients with acute vertigo, nausea and vomiting, the combination of strong vestibular suppression and sedation can be desirable. Typical disorders would be intense motion sickness, acute vestibular neuritis or severe attacks of Meniere's disease or migrainous vertigo. On the other hand, a patient with mild or moderate attacks may get along with a less potent and less sedating drug that allows him or her to continue daily activities.

Occasionally, one may use vestibular suppressants for prophylaxis of recurrent vertigo of unknown cause, which may ameliorate rather than completely eliminate future attacks. When this approach is taken, a less sedating substance should be chosen.

Often, vestibular suppressants are erroneously prescribed for disorders they cannot control, such as benign paroxysmal positional vertigo, bilateral vestibular loss, poorly compensated unilateral vestibular loss, or chronic dizziness unrelated to vestibular dysfunction.

Patients need careful instruction about the potential side-effects of vestibular suppressants, particularly sedation and how this may interfere with driving and operating machines. They should also know that treatment is symptomatic and intended for short-term application only. Some patients develop drug dependency, particularly with benzodiazepines but also with antihistamines such as dimenhydrinate.

Specific situations

Recovery from acute vestibular neuritis occurs spontaneously over days to weeks by central compensation. During the first 24—48 hours, when vertigo, nausea and vomiting are severe, patients need a potent vestibular suppressant and antiemetic (e.g. promethazine or dimenhydrinate). Thereafter, central compensation can be accelerated by vestibular rehabilitation.

Motion sickness can be very distressing to susceptible individuals and is a good indication for vestibular suppressants. Meclizine, which is only mildly sedating, can be tried first. When symptoms are severe, dimenhydrinate is preferable. Transdermal scopolamine is also very effective but works only when applied well in advance, so the patch has to be put in place a few hours before embarking on a journey.

Recurrent vertigo of unknown cause (benign recurrent vertigo) is a common diagnosis, even in specialised neuro-otologic clinics after full investigation. When the term benign recurrent vertigo is used a link to migraine is commonly implicated. Whether patients are actually suffering from migrainous vertigo, Meniere's disease or vestibular paroxysmia may become evident later in the course of the disease. For symptomatic treatment one should prescribe vestibular suppressants. When attacks last longer than seconds migraine prophylaxis can be tried, such as with a beta-blocker or amitriptylie. Alternatively, one may suspect early Meniere's disease, even in the absence of aural symptoms, and try a low-salt diet, provided the patient is ready to take the associated burden or betahistine. A trial of acetazolamide is also worthwhile as it can be effective in migrainous vertigo and in several paroxysmal disorders caused by ion-channel dysfunction such as episodic ataxia type 2. Finally, when all this has failed it can be justified to try a non-sedating dose of a vestibular suppressant for long-term prophylaxis.

References:

F. Vaz. Garcia. Vertigo nine clinical cases 2000

Geeta A Khwaja. Neuroscience Today 1999, 3: 129-143

J. Claes, PM Van De Heyning, Medical treatment of Menieres disease : a review of literature Acta Otolaryngol 1997 ; supplement 526:37-42

M Deiterich, Thomas Brandt, Episodic vertigo related to migraine : vestibular migraine J Neurology 1999 : 246 : 883-892

Adolfo Bronstein and Thomas Lempert . Dizziness a practical approach to Diagnoses and management Cambridge University Press 2007.